MEDICAL JURISPRUDENCE AND TOXICOLOGY

Medical Jurisprudence and Toxicology

BY

JOHN GLAISTER, J.P., D.Sc., M.D., F.R.S.E.

Of the Inner Temple, Barrister-at-Law, etc.

Regius Professor of Forensic Medicine, University of Glasgow; formerly Professor of Forensic Medicine, University of Egypt, Cairo; and Medico-Legal Consultant to the Egyptian Government

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PREFACE TO THE NINTH EDITION

THIS edition has been fully revised, brought to date, and has been enlarged to a slight extent. Many changes in the layout of the text have been made and a considerable amount of new material has been added in relation to the Liability of Hospitals and Nursing Homes, the Law and Post-mortem Dissections, the Coroner and His Functions, Finger-prints, Medico-legal Aspects of Artificial Insemination, and Rh Sub-types, in addition to other subjects. Alterations in the Law have necessitated considerable changes, including the addition of legislation touching the National Health Service Acts, the National Insurance (Industrial Injuries) Act, the Law Reform (Personal Injuries) Act, together with relevant changes in the Law relating to the Sale and Control of Poisons.

The opportunity afforded by a new edition of the book has been responsible for the re-writing of a number of the Sections. The subject of War Gases has been omitted, and that of the Effects of Blast has been relegated to the end of the Chapter on Wounds. In place of War Gases, a short Chapter on the Effects of the Atomic Bomb has been substituted. Recent Legal Cases exemplifying various aspects of Medical Jurisprudence have been added and the text has been more profusely illustrated by photographs, the majority of which have been included in the book for the first time.

Some new substances have been added to the Section on Toxicology and include Dichloro-diphenyl-trichlorethane, or D.D.T., Benzedrine Sulphate, Beryllium, and Radio-active Substances. Chemical Tests now find their place in an appendix, where the various substances for test have been arranged in alphabetical order.

I am sincerely appreciative of the far-reaching assistance given by Dr Edgar Rentoul, my University Assistant. Not only has he helped to revise the legal aspects of the entire subject, rendered valuable advice in numerous directions, but, with Dr Robert Richards, Lecturer in Forensic Medicine, University of Aberdeen, and Dr Robert Brittain, presently attached to my Department,

he has greatly lightened numerous tedious tasks, including those of proof correcting and index making.

I am grateful to Dr A. G. Mearns for the revision of his contribution on Food Poisoning, Stings and Bites, and the Identification of Maggots, together with his drawing shown in Figure 44, and to him and my publishers for the use of the photograph shown in Figure 214; also to Dr J. A. Imrie, Principal Medical Officer, City of Glasgow Police, Lecturer in my Department, for revising his contribution on Ethyl Alcohol and for obtaining a few of the photographs which have been used.

It is a pleasure to thank the Chief Constable, City of Glasgow Police, for his valuable assistance and to say that I am indebted to members of his staff, notably Chief Inspector George Maclean, Identification Bureau, Police Headquarters, for his valuable assistance in many directions.

To Archibald R. Jamieson, Esquire, City Analyst, and Magnus Herd, Esquire, Additional City Analyst, I extend my thanks for their energetic co-operation in the revision of the Chemical Tests. My thanks are also due to W. L. Johnstone, Esquire, of that Department for the drawings in this connection.

I desire to acknowledge Messrs Hanovia Limited, Slough, for the block used for Figure 208, and the assistance given by Mr W. J. Elsey, my Senior Technical Assistant.

To the various contributors of information on points arising and of photographs, and to anyone who has rendered service, but whose name has unwittingly been omitted, I tender thanks.

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JOHN GLAISTER.

Department of Forensic Medicine,
University of Glasgow,
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MEDICAL JURISPRUDENCE

CHAPTER I

GENERAL MEDICAL COUNCIL, MALPRAXIS, LEGAL PROCEDURE, AND NATIONAL INSURANCE (INDUSTRIAL INJURIES) ACT, 1946

MEDICAL Jurisprudence, Legal Medicine, or Forensic Medicine, is the science concerned with the application of medical knowledge to certain branches of the law, both civil and criminal. Since members of the medical profession are liable to be called upon to render professional assistance, of the most varied character, in medico-legal cases which may later compel their attendance at court, it is highly important that they should appreciate and understand both the medical and legal aspects of the subject. This involves on the part of the practitioner a knowledge of the medical aspects of the various criminal acts which may come under his consideration, of the medical bearing of various Acts of Parliament, and of the law of evidence.

THE GENERAL MEDICAL COUNCIL

The General Medical Council, instituted by the Medical Act, 1858, is the official body statutorily charged with the supervision of the medical profession in this country. The principal objects of the Medical Act are to supervise medical education and establish an official register of medical practitioners known as the Medical Register.

The General Medical Council consists of thirty-nine representatives, of whom eighteen are appointed by the Universities in the United Kingdom having medical faculties, nine by the Medical Corporations, five by His Majesty in Council, and seven by election by the medical profession. For dental business, three dentists, who are members of the Dental Board, are added. The Council is divided into branch councils for England, Scotland, and Ireland. Eire has had its own medical council and Register since 1927. Each of the branch councils has a separate Register.

The Medical Act enables the public to identify the qualified practitioner, but does not prevent the public from seeking medical assistance from unregistered persons.

The most important limitations which the Act imposes upon the unregistered practitioner are that he is forbidden to use any title which he does not possess, or to pretend that he is a qualified doctor. He cannot recover fees in a court of law, poisons may not

be supplied by him, or dispensed by a pharmacist on his prescription, and he cannot sign valid certificates. He is also precluded from holding medical appointments in the public service, and from practising under the National Health Service Acts, or from holding Crown appointments. He may not, apart from urgent necessity, attend maternity cases unless under the direction and personal supervision of a duly qualified medical practitioner, and he cannot, by virtue of the Venereal Disease Act, 1917, treat venereal diseases. Further, only duly qualified and registered medical practitioners, if they so desire, are exempted from serving on juries.

It is the act of registration which renders the practitioner legally qualified to practise, and not his medical qualifications. Therefore, it is most important that all who become medically qualified should be registered.

The duty of the Registrar is to keep the Register correct, and to erase the names of all registered persons who have died. To enable the Registrar to fulfil this duty, he may write to any registered person, at the address shown on the Register, to inquire whether he has ceased to practise, or has changed his residence, and if no answer is received within six months from sending the letter, the name of that person may be erased from the Register. A name so erased may be restored by direction of the Council.

The Council recommends the curriculum, and range of subjects of medical study, and ensures that adequate standards are maintained.

It is also responsible for the preparation of the British Pharmacopœia, and the regulation of the curricula and examinations for Diplomas in Dentistry and Public Health.

The Council, by enactment strengthened by decision of the courts of law in their interpretation of the Medical Act, has attained the position of a court of justice. A Penal Cases Committee decides in advance which cases shall come before the Council for inquiry. When acting as a court of medical discipline and conduct, the Council conforms in procedure to that of a court of law, except that it has not the power to compel attendance of witnesses, to administer oaths, or to compel production of documents. The verdicts are either guilty or not guilty "of infamous conduct in a professional respect," and when the former verdict is pronounced, the sentence is erasure of the name of the offending person from the Medical Register. In certain cases sentence may be postponed and the offender may be ordered to appear for judgment following a period of probation. Section 29 of the Medical Act, 1858, states that: "If any registered medical practitioner shall be convicted in England or Ireland of any felony or misdemeanour, or in Scotland of any crime or offence, or shall after due inquiry be judged by the General Council to have been guilty of infamous conduct in any professional respect, the General Council may, if they see fit, direct the Registrar to erase the name of such medical practitioner from the Register." A conviction before a court-martial, or the court of another country, or the finding of a Divorce Court against a doctor that he has committed adultery with a patient, or a member of a patient's

family, are not convictions within the meaning of section 29, and the facts must be inquired into. If the practitioner has been convicted within the meaning of this section he may not seek to disprove the facts which the court has found proved. The Council was inclined to apply this rule to the findings of the Divorce Court, but the House of Lords held in the Spackman case [1] that this was wrong and that a practitioner might bring evidence traversing the findings of the Divorce Court. The facts of the case in question were that a registered medical practitioner was found by the Divorce Court to have committed adultery in circumstances associated with professional relationship, and the General Medical Council considered the removal of his name from the Register for " infamous conduct in a professional respect." Before the Council, the practitioner asked permission to call witnesses other than those who had testified at the divorce proceedings, but this request was refused, and the Council proceeded to direct removal of the name. Appeal was then made to the Divisional Court, but this Court upheld the decision of the Council. Subsequently, the Court of Appeal reversed this decision and the case proceeded to the House of Lords. In 1943, the House dismissed the appeal of the Council, and quashed its direction that the name of the practitioner should be struck off the Register. The Lord Chancellor said that the question was whether the Council reached its decision after due inquiry. The decree of the Divorce Court provided a strong prima facie case, but the duty of considering the defence before pronouncing the accused guilty rested with the tribunal. What mattered was that the accused was not to be condemned without being given a fair chance of exculpation. The Council was entitled to attach to the conclusion of the Divorce Court judge all the weight due to the effect of sworn testimony upon a trained judicial specialist, but that did not exonerate the Council from failing to allow the accused to put before it relevant matter in support of denial. This decision, therefore, does not give the Council licence to refuse to hear evidence tendered in mitigation of a serious offence. In March 1944, the Council summoned Dr Spackman to attend a new inquiry, and found the charge of adultery proved but did not see fit to direct the erasure of his name. The president observed that the doctor's conduct during the two years which had elapsed since the divorce proceedings had been irreproachable ; nevertheless, the Council's decision must not be taken as condoning a grave professional offence.

The Hennessy Case [2] is also of outstanding importance. Dr Hennessy was slandered by a former patient, whose complaint to the General Medical Council resulted in the doctor's name being struck off the Register. He won his action for slander, and subsequently the Council restored his name to the Register. After announcing the restoration, the President of the Council said that " this is a case in which a Judge of the High Court, in a judgment delivered by him, intimated that the Council (to quote the learned judge's own words) not having the advantage of the great mass of

evidence that had been put before him, and which was not put before them had ' in the absence of adequate evidence ' : (and they could only deal with the matter in evidence before them) ; come to a conclusion adverse to the practitioner, which had resulted, in the firm and clear view of the learned judge, in a gross miscarriage of justice. The transcript of the additional evidence has been carefully considered by the Council, and in view of this additional evidence the Council have taken the earliest opportunity of restoring the name of the practitioner to the Register.

But I must emphasise that it is imperative in all inquiries held by the Council under Section 29 of the Medical Act, 1858, that an accused practitioner should call before the Council all material and relevant evidence in support of his case. If he neglects to do so, he does so at his own peril. The Council can only deal with any case on the evidence before them. If there is evidence that is relevant and could be adduced on behalf of the practitioner and is not so adduced, the responsibility of any adverse view by the Council must rest with the accused practitioner."

The verdict, guilty of infamous conduct in a professional respect, has been defined in the Court of Appeal in the following terms :— " If it is shown that a medical man, in the pursuit of his profession, has done something with regard to it which would be reasonably regarded as disgraceful or dishonourable by his professional brethren of good repute and competency, then it is open to the Council to say that he has been guilty of ' infamous conduct in a professional respect.' " A doctor, whose name is erased from the Medical Register by the Council, has a right to appeal to the High Court in England, or to the Court of Session in Scotland, only on the ground that the inquiry by the Council was misconducted in some serious manner, or led to a substantial miscarriage of justice.

In the case of dentists, however, the Dentists' Act, 1921, confers upon them the right to appeal against removal of their names from the Dentists' Register, and permits any dentist so aggrieved to appeal to the High Court in England, or to the Court of Session in Scotland, which may rehear the whole inquiry. Under the Dentists' Act, 1921, a dentist may, if he so desires, have his name deleted from the Register. The courts, however, have held that a medical practitioner's name cannot be removed from the Register merely at his own request, without compliance with certain provisions.[3]

From time to time the Council have enlarged the scope of matters embraced within the meaning of the words, " infamous conduct in a professional respect." The following summary of the resolutions and decisions of the Council upon forms of professional misconduct, brought before the Council in the exercise of its disciplinary jurisdiction over the members of the medical and dental professions, has been published by the Council and is referred to popularly as the " Warning Notice."

The summary of instances of professional misconduct does not constitute a complete list of the offences which may be punished

by erasure from the Register, and the Council is in no way precluded from considering and dealing with any form of professional misconduct, as, for example, immorality involving abuse of professional relationship, which may be brought before it, although it may not appear to come within the scope or precise wording of any of the categories set out below.

Certificates, notifications, reports, etc.

Registered medical practitioners are in certain cases bound by law to give, or may be from time to time called upon or requested to give, certificates, notifications, reports, and other documents of a kindred character, signed by them in their professional capacity, for subsequent use either in courts of justice or for administrative purposes.

Such documents include, among others, certificates, notifications, reports, etc., given in connection with :—

The Acts relating to births, deaths, or disposal of the dead ;
The Acts relating to lunacy and mental deficiency and their Rules;
The Vaccination Acts and their Orders ;
The Factory Acts and their Regulations ;
The Education Acts ;
The Public Health Acts and their Orders ;
The National Insurance (Industrial Injuries) Act ;
The Acts and the Orders relating to the notification of infectious diseases ;
The National Health Service Acts and their Regulations ;
The Old Age Pensions Acts and their Regulations ;
The Merchant Shipping Acts ;
Sickness benefit, insurance and friendly societies ;
The issue of Foreign Office passports ;
Excusing attendance in courts of justice, in the public services, in public offices, or in ordinary employments ;
Naval and military matters ;
Matters controlled by the Ministry of Pensions.

Any registered practitioner shown to have signed or given under his name and authority any such certificate, notification, report, or document of a kindred character, which is untrue, misleading, or improper, whether relating to the several matters above specified or otherwise, is liable to have his name erased from the Register.

Beside having his name erased, the medical practitioner may run the risk of criminal prosecution, as instance, cases of medical practitioners having been sentenced to imprisonment for issuing false medical certificates to members of the Armed Forces (see also p. 164).

Unqualified assistants and covering.

The employment by any registered medical practitioner in connection with his professional practice of an assistant who is not duly

qualified or registered, and the permitting of such unqualified person to attend, treat, or perform operations upon patients in respect of matters requiring professional discretion or skill, is in the opinion of the Council in its nature fraudulent and dangerous to the public health ; and any registered medical practitioner who is shown to have so employed an unqualified assistant is liable to have his name erased from the Medical Register.

Any registered medical practitioner who by his presence, countenance, advice, assistance, or co-operation, knowingly enables an unqualified or unregistered person, whether described as an assistant or otherwise, to attend, treat, or perform any operation upon a patient in respect of any matter requiring professional discretion or skill, to issue or procure the issue of any certificate, notification, report, or other document of a kindred character, or otherwise to engage in professional practice as if the said person were duly qualified and registered, is liable on proof of the facts to have his name erased from the Medical Register.

This does not apply to the training of bona fide students, or the legitimate employment of dressers, dispensers, surgery attendants, and skilled mechanical or technical assistants, under immediate personal supervision of a registered medical practitioner.

Sale of poisons.

The employment, for his own profit and under cover of his own qualifications, by any registered medical practitioner who keeps a medical hall, open shop, or other place in which scheduled poisons or preparations containing scheduled poisons are sold to the public, of assistants who are left in charge but are not legally qualified to sell scheduled poisons to the public, is in the opinion of the Council a practice professionally discreditable and fraught with danger to the public, and any registered medical practitioner who is proved to have so offended will be liable to have his name erased from the Medical Register (see p. 500).

Dangerous drugs.

The contravention by a registered medical practitioner of the provisions of the Dangerous Drugs Acts and the regulations made thereunder may be the subject of criminal proceedings, and any contravention of the Acts or the regulations, involving an abuse of the privileges conferred upon registered medical practitioners, whether such contravention has been the subject of criminal proceedings or not, will, if proved to the satisfaction of the Council, render a registered medical practitioner liable to have his name erased from the Medical Register (see p. 510).

Association with unqualified persons.

Any registered medical practitioner who, either by administering anæsthetics or otherwise, assists an unqualified or unregistered

person to attend, treat, or perform an operation upon any other person, in respect of matters requiring professional discretion or skill, will be liable on proof of the facts to have his name erased from the Medical Register.

Advertising and canvassing.

The practices by a registered medical practitioner :—

(*a*) of advertising, whether directly or indirectly, for the purpose of obtaining patients or promoting his own professional advantage ; or, for any such purpose, of procuring or sanctioning or acquiescing in the publication of notices commending or directing attention to the practitioner's professional skill, knowledge, services, or qualifications, or depreciating those of others ; or of being associated with or employed by those who procure or sanction such advertising or publication, and

(*b*) of canvassing or employing any agent or canvasser for the purpose of obtaining patients ; or of sanctioning, or of being associated with or employed by those who sanction, such employment ;

are in the opinion of the Council contrary to the public interest and discreditable to the profession of medicine, and any registered medical practitioner who resorts to any such practice renders himself liable on proof of the facts to have his name erased from the Medical Register.

Association with uncertified women practising as midwives.

Any registered medical practitioner who, by his countenance or assistance, or by issuing certificates, notifications, or other documents of a kindred character, enables uncertified persons to attend women in childbirth otherwise than under the direction and personal supervision of a duly qualified medical practitioner, will be liable to have his name erased from the Medical Register.

By a Resolution adopted by the Council, any registered medical practitioner who knowingly and wilfully assists a person who is not registered as a dentist in performing any operation in dental surgery, either by administering anæsthetics or otherwise, will be liable on proof of the facts to be dealt with by the Council as having been guilty of infamous conduct in a professional respect.

The Council does not of itself initiate proceedings against members of the medical and dental professions. It takes action only in cases of criminal conviction, or of judicial censure, officially brought to its notice, or in cases of formal complaints, supported by evidence, brought before it by responsible persons or bodies. When a member of the medical or dental profession is convicted in a court of law, the fact is reported to the Council. After consideration of the facts

by the Penal Cases Committee, the delinquent may be summoned to appear before the Council or, alternatively, he may be sent a warning that the matter has been noted and that there should not be a repetition of the offence.

A short draft bill has been submitted by the Council to the Government which includes important provisions such as giving practitioners a right of appeal to the courts against penal erasure from the Medical Register, giving the Council statutory power to restore the names of practitioners to the Register after penal erasure, enabling the Council to take evidence on oath and to compel attendances of witnesses by subpœna, and enlarging the direct representation of the profession on the Council.

NATIONAL HEALTH SERVICE ACT, 1946
NATIONAL HEALTH SERVICE (SCOTLAND) ACT, 1947

These Acts contain provisions for the preparation and publication, by Executive Councils, of lists of medical practitioners, dentists, and pharmacists who undertake to provide their respective services under the Acts. Provision is also made for the constitution of Tribunals with the power to order the removal of the names of practitioners, dentists, or pharmacists from these lists. The Tribunals consist of a chairman and two other members. In England, the chairman is appointed by the Lord Chancellor, and must be a barrister or solicitor. In Scotland, he is appointed by the Lord President of the Court of Session, and must be a practising advocate or solicitor. One of the other members is a person appointed, in England, by the Minister of Health, and, in Scotland, by the Secretary of State, after consultation with representatives of the Executive Councils. The other member, referred to as the practitioner member, is a member of the profession concerned, for example, medical, dental, or pharmaceutical, and is appointed, in England, by the Minister of Health, and, in Scotland, by the Secretary of State, after consultation with the appointed representatives of that profession.

The purpose of these tribunals is to inquire into cases where representations are made in the prescribed manner to the Tribunal by an Executive Council, or by any other person, that the continued inclusion of any person in any list prepared under these Acts would be prejudicial to the efficiency of the services in question. The Tribunal, on receiving representations from an Executive Council shall, and in any other case may, inquire into the case and, if they are of the opinion that the continued inclusion of the said person in any list to which the representations relate would be prejudicial to the efficiency of the said services, shall direct that his name be removed from that list, and may also, if they think fit, direct that his name be removed from, or not be included in, any corresponding list kept by any other Executive Council under these parts of these Acts.

An appeal lies, in England, to the Minister of Health and, in Scotland, to the Secretary of State, from any direction of the Tribunal.

VENEREAL DISEASE ACT, 1917

This Act was passed to prevent the treatment of venereal disease otherwise than by duly qualified medical practitioners, and to control the supply of remedies for the purpose of treatment.

CANCER ACT, 1939

Under this Act, it is illegal to take any part in the publication of an advertisement offering to treat any person for cancer, or to prescribe any remedy for it, or to give any advice for its treatment, or to refer to any article in terms calculated to lead to its use in the treatment of cancer. The Act is not intended to apply to those who publish as a reasonable necessity to bring the advertisement to the notice of certain specified persons such as a local authority, those controlling hospitals, medical practitioners, nurses, chemists, students of these professions, or sellers of surgical appliances.

MALPRAXIS

Malpraxis means failure in the exercise of a reasonable degree of skill and care on the part of a medical practitioner in his treatment of a patient. The public who entrust themselves to the care of registered practitioners are entitled to demand and to expect from them the exercise of reasonable skill and care. In order to define the terms " reasonable skill " and " reasonable care " it is necessary to consider the definitions which have been given in the courts of law. The question whether or not malpraxis exists in any given case must depend upon the inherent facts, but, from the consideration of many cases, it seems clear that very considerable carelessness in the conduct of a case is necessary to establish proof of malpraxis in a court of law. Only a judge or jury after hearing the evidence, can say whether the medical practitioner did, or did not, use a proper degree of skill and care. It has been ruled that a doctor is not responsible for an error in judgment, and that he is bound to bring to the discharge of his duty only reasonable care, skill, and knowledge. In the case of Lanphier v. Phipos,[4] the judge, in directing the jury, stated that every person who entered into a learned profession undertook to bring to the exercise of it a reasonable degree of care and skill. He did not undertake to perform a cure or to show the highest possible degree of skill. There might be persons who had higher education and greater advantages than he had, but he undertook to bring a fair, reasonable, and competent degree of skill, and the question was whether the injury must be

referred to the want of a proper degree of skill and care or not. In the case of Rich v. Pierpont,[5] it was stated that a medical man was certainly not answerable merely because some other practitioner might possibly have shown greater skill and knowledge, but he was bound to have that degree of skill and knowledge which could not be defined, but which in the opinion of the jury was a competent degree. It was not enough that medical men of far greater experience or ability might have used a greater degree of skill or that the person charged might have used more care. The question was whether there had been a want of competent care and competent skill to such an extent as to lead to the bad results. It had also to be considered "whether the amount of care and skill bestowed was up to the average of the reasonable skill and diligence ordinarily exercised by the profession as a whole, not that exercised by the thoroughly educated, but the average of the thorough, the well, and the moderate, all, in education, skill, and diligence." To this there must be added an allowance for particular circumstances of position, whether urban or rural, near a centre of population or remote. It is obvious that the term "reasonable" must be applied relatively. For example, a rural practitioner cannot be expected to have the surgical resources of a hospital surgeon. All that the law demands is the exercise of such an amount of skill as should be possessed by everyone who has qualified as a medical practitioner. It has been held that a doctor is not responsible where, in the treatment of a case, a drug has been administered and which, contrary to ordinary experience, has acted disastrously. Errors in diagnosis are not necessarily due to ignorance, and no guarantee is given or implied that a medical practitioner will diagnose correctly the condition from which the patient suffers. In all cases the standard of care and competence varies in accordance with the specific circumstances. A specialist consulted in regard to his specialty might probably be held liable for negligence in respect of treatment which in an ordinary, or junior, member of the profession might be regarded as satisfactory. The forming of fixed standards is impossible, and the degree of skill can only be assessed by having regard to the experience of the one who treats. If it is found that there has been a lack of reasonable skill and care in any given case, the patient is entitled to recover damages from the practitioner in respect of any injury which has resulted from that cause, and it becomes a question for the jury to say whether any injury is attributable to the want of a reasonable and proper degree of skill and care. The case of Whiteford v. Hunter and Gleed [6] has been described as disquieting to the medical profession. Put briefly, the facts of this case are that a seventy-year-old plaintiff sued a surgeon and a general practitioner for damages on the ground of their negligence in wrongly diagnosing his condition. They had told him, he said, that he was suffering from cancer and had only nine months to live. He thereupon gave up his business and went to America to die. An American surgeon had then operated upon him for a bladder complaint which he declared was the sole and obvious cause of the condition. The

plaintiff claimed that he had suffered pecuniary loss and mental agony as a result of defendants' mistake. Mr Justice Birkett held that no negligence could be attributed to the general practitioner. With regard to the surgeon, it was now admitted that the diagnosis was wrong. The surgeon had done nothing afterwards to confirm or verify it. His Lordship said he was clearly of opinion that the surgeon was negligent in not making a microscopical examination ; he was lacking in his duty in taking no step to check or verify his diagnosis, and also in not making a cystoscopic examination. Judgment was entered against the surgeon for £6300. A stay of execution was granted with a view to appeal. The appeal was upheld. Mr Justice Asquith, in giving judgment, said that a doctor was not liable in negligence by reason only that he had made a mistake in diagnosis. Leave has been granted for an appeal to the House of Lords. The basis of these cases is usually founded on badly united fractures, injuries involving nerve complication with resultant loss of power, improperly reduced dislocations or the like, although unusual claims are occasionally intimated. In all cases of likely bone injury, radiological examination should be insisted upon. The case of Baldwin v. Lord and Others is of interest.[7] A man was admitted to hospital and an operation for grafting a piece of bone from his leg into his right wrist was performed successfully. A tourniquet applied to the patient's leg was, however, left in position for a period of eleven hours. The patient died despite a blood transfusion. Both the infirmary and the surgeons were sued. Liability was admitted and £4000 with costs was awarded.

Under circumstances when the negligence shown is so gross in character as to amount to recklessness, criminal prosecution may result. In these cases the nature and degree of the negligence must be of such a degree as to convince the jury that it should be punished. It is clear, therefore, that what might amount to negligence in a civil action would not necessarily warrant a conviction on a criminal charge.

In the case of R. v. Bateman,[8] the Lord Chief Justice said that in a civil action for negligence, the extent of a man's liability depends on how much damage he causes ; in a criminal trial it depends on how negligent he was.

The case of R. v. Wight provides a good example of a criminal prosecution. The accused, a medical practitioner, was charged with having caused the death of a woman while delivering her with forceps, he being at the time under the influence of alcohol. It was shown in evidence, however, that the cause of his condition was not alcohol but chloral hydrate. He was convicted and sentenced to three months' imprisonment. The comments of the judge indicated that no medical man was entitled to practise his profession while under the influence of any drug or other narcotic substance, and that had alcohol been proved the cause of the prisoner's condition, a much more severe sentence would have been passed.

Lord Ellenborough has held that if a person acting in a medical

capacity be guilty of misconduct arising either from gross ignorance or criminal inattention by which the patient dies, he is guilty of manslaughter.

The rarity of criminal proseeutions may be taken as an indication of how serious a degree of recklessness has to be committed before members of the profession are brought within reaeh of the law, and how rarely criminal negligenee is found in medical practiee.

LIABILITY OF HOSPITALS AND NURSING HOMES

It has long been established as a rule of Seots law that a master is liable for the wrongful or negligent acts of his servants.

Hospitals and nursing homes are therefore elearly liable for the negligent aets and omissions of their non-medical staffs.

The medical staffs are, however, in a different position beeause between them and the managers of hospitals there is no true relationship of master and servant. The reason for this is that the managers of hospitals have no right of interference or direction as to the way the work is to be done. The eontraet is not serviee but an agreement to render services, and the obligations of the hospital authorities are fulfilled when they appoint personnel who are fully qualified and competent. Foote v. Greenock Hospital.[9]

The position in England was similar to that in Scotland and it was decided by a number of eases that hospitals were not responsible for the negligent aets of doctors, Hillyer v. St Bartholomew's Hospital,[10] or of nurses, Strangways-Lesmere v. Clayton.[11] Nursing homes were held to be in a different position, and it was decided that the proprietors of a nursing home were responsible for damages for injuries caused by the negligenee of two trained nurses employed by them in the home, Powell v. Streatham Manor Nursing Home.[12]

The law as thus stated was, however, considered in the case of Gold v. Essex C.C.[13] This case decided that a hospital was liable for the negligent acts of a radiographer employed by them. The court in this instanee decided that a hospital does undertake maintenance, treatment, and nursing and that it must fulfil such obligations through its servants, nurses, and radiographers for whose wrongful or negligent acts it is responsible.

This decision raises difficulties. It leaves doubtful the position of house physicians and surgeons and even throws doubt on the position of visiting physicians and surgeons. Under the National Health Service Act, 1946, and the National Health Service (Scotland) Aet, 1947, it is the duty of the Minister to provide hospitals to meet reasonable requirements ; it is also his duty to provide medical, nursing, and other services, and the serviees of specialists. Accepting the reasoning expressed in the case of Gold v. Essex C.C. it might be held that this makes regional hospital boards and boards of governors responsible (under section 13 of the Acts) for the negligent acts and omissions of all those whom they employ to carry

out their duties under the Acts, including visiting physicians and surgeons.

It will thus be recognised that there is lack of finality in the present state of the law with regard to the liability of hospitals and nursing homes.

LEGAL CRIMINAL PROCEDURE

ENGLISH PROCEDURE

In England there is a Director of Public Prosecutions. The duties of this official, under the direction of the Attorney-General, are, among others, to institute, undertake, or carry on criminal proceedings at any stage and in any court, and to give advice and assistance to persons, whether officials or not, concerned in a criminal proceeding. He undertakes all cases punishable by death, and cases of importance or difficulty, or in which, owing to special circumstances, or the refusal or failure to proceed with a prosecution, intervention on the public behalf is necessary for the prosecution of the offender.

English Criminal Courts.

Magistrates' Court of Petty Sessions.

This is a Court of Summary Jurisdiction, or a Court of First Instance.

Two or more Justices of the Peace or one professional magistrate, either sitting alone or with other Justices, preside in the court, which tries petty offences and passes sentence. In all serious charges, before a prisoner can be committed for trial, the witnesses in the case give their evidence on oath before a body of Justices, who elect their chairman, and in the presence of the accused, and in open court. The accused is entitled to give evidence on oath and to call witnesses. When making preliminary inquiry as to whether an accused person shall, or shall not, be committed for trial by a jury, the Justices are termed "examining magistrates" or "examining Justices." The evidence is recorded in writing, and after being read over to the respective witnesses is signed by them and by the presiding Justice. The evidence of each witness so recorded is termed a deposition and these depositions form the basis of the evidence led at any subsequent trial of the accused person, the various witnesses being expected to adhere to their respective depositions. From the evidence given, it remains for the Justices to decide whether or not the accused shall be committed for trial at the Quarter Sessions or Criminal Assize Court according to the nature of the offence. If there is not a prima facie case established against the accused he is discharged, but this is not synonymous with acquittal since he may be arraigned again on the same charge.

Court of Quarter Sessions.

This court is held four times annually in each county, county borough, and city, but "adjourned Sessions" are held at intermediate dates. It has both civil and criminal jurisdiction, and is presided over by an assembly of Justices of the Peace, who elect their chairman, usually a barrister since it is essential that the chairman of such a court should have considerable knowledge of the law. In the cities and the county boroughs this court is presided over by a Recorder, who is a practising barrister elected from the circuit in which the city, or the county borough, is placed. The only whole-time Recorder is the Recorder in London who sits in his special court at the Central Criminal Court ("Old Bailey"). The court may try indictments for any offences within the county with certain exceptions, for example, capital felony, forgery, bigamy, and all offences punishable with imprisonment for life. The usual classes of case which are dealt with comprise the lesser felonies and misdemeanours. As an appellate court, it may confirm, reverse, or vary the decisions of the Courts of Summary Jurisdiction.

Court of Assize.

A Judge of the King's Bench Division of the High Court or a Commissioner specially appointed, presides at the Criminal Assize Court. Each county has three to four Assizes annually, but in London and its suburbs the Assizes are held monthly at the Central Criminal Court ("Old Bailey"). The Judge or Commissioner travels a "circuit" of assize towns and in each he holds a court. The court tries prisoners on indictment before a jury, and an appeal against a conviction lies to the Court of Criminal Appeal.

The procedure at a trial in the Criminal Assize Court may be summed up shortly. The prisoner having tendered a plea of "not guilty," and the jury having been sworn, the prosecuting counsel opens the case against the prisoner by outlining briefly the facts to which the witnesses he is about to call will testify. He next calls and examines his witnesses in turn. This part of the examination of a witness is called the examination-in-chief. Each witness, at the conclusion of this examination, may be cross-examined by counsel for the accused, or by the accused himself if not represented by counsel, after which prosecuting counsel may re-examine each witness on points brought out in cross-examination, but he may not examine the witness on any fresh subject of evidence. If the witness is to be asked further questions, these must be put by the judge. After the witnesses have been called, the case for the Crown is closed. If counsel for the defence calls witnesses, he will conduct their examination-in-chief, and counsel for the prosecution will cross-examine them. Thus the position of counsel with reference to the defence witnesses is reversed. At the conclusion of the evidence, defending counsel addresses the jury on behalf of the prisoner, thereafter prosecuting counsel addresses the jury, and finally, the judge sums up. The jury then retire to consider their verdict.

If, however, defending counsel does not propose to call witnesses, the order of procedure is changed to this extent, namely, that at the end of the evidence for the prosecution, prosecuting counsel proceeds to address the jury, after him defending counsel, and then follows the summing-up of the judge. If the prisoner is not defended by counsel, the right of addressing the jury possessed by prosecuting counsel is denied him.

In any case where a medical witness has given evidence in a court in which an accused person is committed for trial, he, in common with other witnesses, is bound over to give evidence at the Criminal Assize Court.

King's Bench Division of the High Court.

An appeal from the inferior criminal courts lies to this court, by a stated case on a point of law.

Court of Criminal Appeal.

In certain circumstances, a convicted person may appeal to the Court of Criminal Appeal, presided over by the Lord Chief Justice of England, and not less than, but usually, three judges of the King's Bench Division of the High Court. It may decide by a majority. The court has a wide range of jurisdiction and can quash a sentence and substitute the one it thinks ought to have been passed, and it can substitute a verdict for that in fact returned where it is justified by the evidence. Thus it can increase sentences or penalties.

House of Lords.

An appeal lies to the House of Lords from the judgment of the Court of Criminal Appeal, but only where the Director of Public Prosecutions, or the prosecutor, or defendant obtains a certificate of the Attorney-General that the decision involves a point of law of exceptional public importance, or that it is desirable in the public interest that a further appeal should be brought. Such appeals are few. On appeals, only lords who hold, or have held, high judicial office act as judges.

In addition to the foregoing Criminal Courts, there are the County Court, the Court of Assize, the King's Bench Division of the High Court, the Court of Appeal, and the House of Lords, which are concerned with civil actions.

The Coroner and the Coroner's Court.

The office of the Coroner is of great antiquity and there is now no record of its origin.

The Coroner has authority to inquire into the causes and circumstances of deaths, to deal with treasure trove, to execute writs in place of the Sheriff and, in the City of London, to hold inquests on non-fatal fires.

There are three kinds of Coroners, namely, Coroners ex-officio, Franchise Coroners, and County and Burgh Coroners. For present purposes, the County and Burgh Coroners need only be considered, and these are appointed by the county and borough councils, respectively.

By the Coroners (Amendment) Act, 1926, s.1, no person is qualified to be appointed a County Coroner or a Borough Coroner unless, with certain exceptions, he is a barrister, solicitor, or legally qualified medical practitioner, of not less than five years' standing in his profession.

Every Coroner must appoint a deputy Coroner who possesses the necessary qualifications of a Coroner.

The jurisdiction of a Coroner is restricted to the district to which he has been appointed. Where, however, death has taken place in a prison situated in a borough, which has a Borough Coroner, the jurisdiction of the County Coroner prevails to the exclusion of the Borough Coroner.

The main duty of a Coroner is to inquire into the circumstances of certain deaths occurring within his jurisdiction.

There is an obligation on the Coroner to make an inquiry when he is informed that a dead body is lying within his jurisdiction, and there is reasonable cause to suspect that such a person has died either a violent or unnatural death, or has died a sudden death of which the cause is unknown, or that such person has died in prison, or in such place or under such circumstances as to require an inquest. (Coroners Act, 1887, s.3.)

It should be clearly understood that it is contrary to the common law of England for anyone to bury the body of a person dying from a violent death, or to dispose of a body so as to prevent the holding of an inquest, or to obstruct the Coroner and his jury before the Coroner has had an opportunity of holding an inquest upon such body.

The Coroner obtains information by means of his officer. The Coroner's officer is not a statutory official, but his duties are onerous since he is primarily responsible for collecting relevant information on the Coroner's behalf. He is closely connected with the detailed arrangements for inquiries and inquests. Frequently a police officer is permanently detailed for this purpose.

The Coroner's inquiry may take the form of a private inquiry or of an inquest in the Coroner's Court with or without a jury.

Whichever method of inquiry is adopted medical evidence forms an important part of the proceedings, and as a verdict arrived at without medical evidence has little value it is now the practice to call medical evidence at every inquest.

The relationship of medical witnesses to the Coroner is governed by the Coroners Act, 1887, and the Coroners (Amendment) Act, 1926.

Under the Coroners Act, 1887, s.21, the Coroner in the course of his inquiry may summon as a witness any legally qualified medical practitioner who attended the deceased at his death or during his last illness. If a doctor has not attended the deceased, the Coroner

may summon any legally qualified practitioner, who is at the time in actual practice in or near the place where the death occurred, and may obtain evidence from him as to the cause of death.

If a Coroner considers that information to be obtained by a post-mortem examination will make an inquest unnecessary he may instruct a registered medical practitioner to make a post-mortem examination and to report the result to him in writing.

Under the Coroners (Amendment) Act, 1926, s.22, the Coroner, at any time after he has decided to hold an inquest, can request any legally qualified practitioner to make a post-mortem examination of the body, or to make such special examination of the body or parts of the body as he may think necessary to provide evidence concerning the cause of death.

When there is sworn testimony that, in the belief of a witness, the death of a deceased person was caused partly, or entirely, by the improper or negligent treatment of a medical practitioner, or other person, that medical practitioner or other person shall not be allowed to perform or assist at any post-mortem or special examination made for the purposes of an inquest on the deceased. The medical practitioner or other person concerned has, however, the right, if he so desires, to be represented at the post-mortem examination.

The foregoing sections are of great practical importance to practising members of the medical profession, and they have made the evidence of pathologists increasingly important at Coroners' inquests. The 1935 Departmental Committee on Coroners recommended that a list of pathologists should be kept by the Home Office and that all post-mortem examinations ordered by Coroners should, with few exceptions, be undertaken by pathologists whose names were on this list. Such a recommendation shows the trend, a desirable one, to employ pathologists for this class of work.

When the Coroner has reasonable cause to suspect that the deceased has died a violent or unnatural death, or has died in prison or in such place or in such circumstances as to necessitate the holding of an inquest in accordance with the requirements of any Act, other than the Coroners Act, then he must hold an inquest.

The Coroner must summon a jury, which consists of seven and not more than eleven members, in cases where there is reason to suspect that :—

(*a*) the death was caused by murder, manslaughter, or infanticide :
(*b*) the death occurred in prison or in such circumstances as to require an inquest under any Act other than the Coroners Act, 1887 ;
(*c*) the death was caused by an accident, poisoning, or disease, notice of which is required to be given to a government department, or to any inspector or other officer of a government department under any act ;
(*d*) the death was caused by an accident arising from the use of a vehicle in a street or public highway ;

(*e*) the death occurred in circumstances the continuance or possible recurrence of which is prejudicial to the health or safety of the public or any section of the public.

Inquests after judicial execution must be held with a jury by the Coroner within whose jurisdiction the execution has taken place. The inquest must be held within twenty-four hours after death. The jury inquires into the identity of the body and whether sentence of death has been duly carried out.

The Coroner, before holding an inquest, must view the body. The jury do not need to view the body unless the Coroner so directs or a majority of the jury so desires.

Witnesses can be compelled to attend an inquest and give evidence but the summons of a Coroner is limited to his sphere of jurisdiction. If it is necessary to compel the attendance of a witness who is outwith the Coroner's jurisdiction this can be done by obtaining a Crown Office subpœna. Evidence is given on oath and counsel and solicitors who attend inquests in a representative capacity may question witnesses.

In cases of murder, manslaughter, and infanticide, the evidence must be recorded in writing and signed both by witnesses and Coroner. These statements of evidence are termed depositions, and can be used in subsequent legal proceedings (see pp. 15 and 22). It is, however, essential that they should be taken in the presence of the accused person if they are to be used at a criminal trial.

When the evidence is completed it is customary for the Coroner to sum up for the jury, who then give their verdict. The Coroner can accept a majority verdict provided that the minority is not larger than two. The verdict is in simple form and embodies such facts as the identity of the deceased, the cause, time, and place of death, and classifies the death as "natural" or "violent." It further classifies the death as "not criminal" or "criminal." If some part of the inquest is unanswered, an open verdict is returned.

When, as a result of the verdict, some person is charged with murder, manslaughter, or infanticide, or of being an accessory before the fact to a murder, the Coroner must issue a warrant to arrest such person, if a warrant has not already been issued. He must also bind the witnesses by recognisance. The warrant of commitment should include a direction as to the court in which the accused is to appear. This will usually be the next Court of Assize for the county or the next session of the Central Criminal Court, but the Coroner has power to send the accused to the next convenient Assize even though this is not within his own jurisdiction. The Coroner is bound to attend a court where a case is being tried on an inquisition taken before him.

The Coroner while acting in his court is in the position of a judge and is therefore immune from actions at the instance of any individual, e.g., no action could be taken by an individual for slander in the Coroner's summing up. This immunity is effective only while the Coroner is acting within his jurisdiction.

It is the duty of every Coroner to make an annual return to a Secretary of State of all cases on which he has held an inquest.[14]

Relation of medical men to Coroner and Inquest:

There are certain questions which affect the medical profession with respect to their relation to the Coroner. Probably the most important is whether or not it is the duty of a medical practitioner in attendance upon a person who has died from a violent or any unnatural cause to report the fact to the Coroner. Although there does not appear to be any statutory obligation, a medical practitioner would appear to owe a duty as an ordinary citizen to report violent, or unnatural death, a sudden death, the cause of which is unknown, or the kind of death into which the Coroner is obliged by statute to inquire. This would include deaths which result from the administration of an anæsthetic. It is commonly, but erroneously, thought that the Births and Deaths Registration Act, 1926 (see p. 153) restricted the practitioner's duty to report suspicious deaths to the authorities and that he had discharged his duty merely by completing the death certificate and delivering it to the Registrar. This Act has not relieved the practitioner from the duty which he already owed before this Act was passed. In all cases of doubt an informal conversation with the Coroner will provide a clear indication of what he considers is the proper line of conduct having regard to the innate circumstances of the specific instance under review.

In 1914, Mr Justice Avory, in delivering judgment in the case of a woman who died from the effects of an illegal operation, and who had been seen by a number of doctors prior to her death, said, "It may be the moral duty of the medical man, even in cases when the patient is not dying, or not unlikely to recover, to communicate with the authorities when he sees good reason to believe that a criminal offence has been committed. However that may be, I cannot doubt that in such a case as the present, where the woman was, in the opinion of the medical men, likely to die, and, therefore, her evidence was likely to be lost, it was his duty, and that some one of these gentlemen ought to have done it in this case."

Medical practitioners when summoned to appear as witnesses must obey the summons or become liable to penalty.

SCOTTISH PROCEDURE

The prominent characteristic of criminal procedure in Scotland is the existence of the office of Public Prosecutor.

The Lord Advocate, the Solicitor-General, and four Advocates-Depute are the public prosecutors, on behalf of the Crown, before the High Court of Justiciary. In each county there is a Procurator-Fiscal for whose appointment the Lord Advocate is responsible. He initiates the prosecution of crimes and offences in the Sheriff Court, and conducts such prosecution either personally or by depute. The Lord Advocate and the Advocates-Depute, who

are responsible for the direction of criminal proceedings, preside at the Crown Office, Edinburgh. When a Procurator-Fiscal has collected all the evidence in any case, it is sent to the Crown Office. There, the evidence is fully reviewed and decision is taken with regard to the nature of further proceedings and as to the court in which the case will be tried. It may be that no further proceedings will be authorised through insufficiency of evidence or other good reason. The most serious cases are tried before the High Court of Justiciary, but in less serious cases they will probably be disposed of by the Sheriff.

In Scotland, the legal advisers at the Crown Office assess the adequacy of evidence in any given case and instruct proceedings. In this way the evidence to be led at a trial is not available to the public and to potential members of a jury who may be called upon subsequently to pass a verdict.

In contrast, English procedure necessitates the disclosure of the evidence at the Magistrates' Court prior to the actual trial at Assizes, and thus the evidence becomes known to the public prematurely.

The duty of bringing all accused persons to a bar of justice falls primarily upon the Procurator-Fiscal as local Public Prosecutor. In addition, he is charged with the duty of inquiring into the cause of sudden or suspicious deaths, which inquiry takes the form of precognition of witnesses, both lay and medical. This is ordinarily a private inquiry. After he has performed this duty, it is his further responsibility to report and certify the cause of death to the Registrar of the district in which the death took place.

Some explanation of the term "precognition" is necessary. A precognition is a statement from a person who will probably be called by the Crown as a witness in a case. This statement embodies the facts to which the potential witness will testify at a trial, and is recorded by a Procurator-Fiscal or one of his deputes. The statement, which is not given on oath or signed by the witness, is usually made at the office of the Procurator-Fiscal and, as will be noted, is obtained with much less formality than the English deposition, already described. A witness is not bound by the terms of his precognition, the object of which is to form a basis for the preparation of the prosecution, and to facilitate the leading of evidence at the trial. A precognition taken in this way cannot be used to contradict what he says when giving evidence. Solicitors acting for an accused person may also take precognitions and thus prepare the case for the defence. It should be clearly understood that in criminal prosecutions every person competent and compellable as a witness for the prosecution may be compelled in the preliminary investigations to disclose what he knows concerning the crime under investigation. This does not apply to the opinion of experts. If a person is unwilling to submit to precognition by the Procurator-Fiscal in the ordinary way, he may be compelled to attend before a magistrate, and may be imprisoned if he refuses to answer pertinent inquiries.

By the Summary Jurisdiction (Scotland) Act, 1908, any witness

who, after being duly cited fails, without reasonable excuse after receiving at least twenty-four hours' notice, to attend for precognition by a Procurator-Fiscal or Burgh Prosecutor at the time and place mentioned in the citation served on him, or refuses when so cited to give information within his knowledge in regard to any matter relative to the commission of the offence in regard to which such precognition is taken, shall be deemed guilty of contempt of court and be liable to be summarily punished forthwith for the same by fine or imprisonment.

In civil actions, the court will not order persons against their will to submit to precognition. There is no duty or obligation on any person to give information before the trial regarding any matter which may form the subject of litigation or to allow himself to be precognosced with a view to his being called as a witness. The statements which a witness voluntarily makes on precognition are in the same position with regard to privilege as statements made on oath in the witness box (see pp. 60 and 64).

If a medical witness assumes the attitude that his function is to aid the ends of justice, his duty becomes clear. Should, therefore, the prosecution or the defence desire to precognosce a medical witness who has been called by the defence or the prosecution, he should not have the slightest hesitation in submitting both his findings in, and views on, the case at issue, just as he has done for the side who first requested his professional services. The same facilities should be at the command of either the prosecution or the defence.

The Procurator-Fiscal is also charged statutorily with the duty of making public inquiry into the causes of fatal accidents, and, in special circumstances, of sudden deaths. The former is held under the Fatal Accidents Inquiry (Scotland) Act, 1895, as amended by the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act, 1906, and the Criminal Procedure (Scotland) Act, 1938, and the latter under the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act, 1906. The Procurator-Fiscal presents a petition to the Sheriff craving him to hold a public inquiry in regard to the cause of the death and the circumstances of the accident, and the Sheriff in granting the petition pronounces an order, directing that a public inquiry be held, and grants warrant to cite witnesses at the instance of the Procurator-Fiscal. In the last mentioned Act it is enacted that, in any case of sudden or suspicious death in Scotland, the Lord Advocate may, whenever it appears to him to be expedient in the public interest, direct that a public inquiry into such death and the circumstances thereof shall be held.

Fatal Accidents Inquiry.

The Inquiry is held by a Sheriff and jury, the jury consisting of seven jurors, of which neither an employer of the person regarding whose death the Inquiry is held, nor any person engaged under the same employer as the deceased, shall be a member. After hearing the evidence, of which part is frequently medical, and following

the summing-up by the Sheriff, the jury returns a verdict which sets forth when and where the accident and the death took place, together with the cause or causes of the accident and death, and additional facts as to fault or negligence of anyone, or defects in mode of working in the case of accidents. The verdict is usually unanimous, but it may be returned by a majority. It is the duty of the Fiscal at the close of the inquiry to transmit all the documents, including reports and productions, with a copy of the verdict, together with the usual schedule for the Registrar of deaths of the district, to the Crown Agent.

The duties of the Procurator-Fiscal, therefore, include the sphere of action of the Coroner in England. In carrying out his duties in connection with the investigation of crime, when serious cases are reported to him, he, or one of his deputes, will proceed to the scene and there make an examination. In such cases as rape, child-murder, or concealment of pregnancy, where invaluable evidence of the commission of the crime may be lost by delay, he arranges for early medical examination of the assaulted person or of the accused. When death results from violence, he ensures that a post-mortem dissection of the body is made by experienced medical men who, as soon as possible, furnish him with a report of the autopsy together with their opinion as to the cause of death. In the event of a serious assault to the danger of life, and where death after an interval takes place, he will obtain evidence respecting the treatment of the injured person during that interval. In cases of assaults likely to prove mortal, he must proceed with a Sheriff so that the deposition of the dying person may be taken (see p. 57). The inquiry into the cause of sudden deaths by the Procurator-Fiscal is to eliminate possible crime. In deaths from accident, and in all cases of sudden deaths reported to him, he precognosces witnesses, among whom is a medical witness who reports on the cause of death, without a post-mortem examination when the cause of death can be so ascertained, otherwise after a post-mortem dissection.

As a medical witness may require to appear in any of the law courts, the following indicates the names of these courts, and the class of cases therein :—

Police Court: for certain common law and statutory offences of a minor kind, or as the court of first instance in remits to a higher court.

Sheriff Court: in which the Sheriff of the county, or his substitute, may act alone, or with a jury. The jurisdiction of this Court is of a very extensive character since it is concerned with both civil and criminal cases. Certain common law and statutory offences of less serious character are tried before the Sheriff summarily, but more serious cases are heard before a jury, and the conduct of the prosecution is in the hands of the local Procurator-Fiscal. All except very few crimes may be tried, and as much as two years' imprison-

ment may be given when the trial is on indictment, namely, when the trial is heard before a jury.

The High Court of Justiciary: which ordinarily sits in Edinburgh also sits quarterly or oftener in certain large towns, when it is popularly called the Circuit Court, or the High Court on Circuit. This Court deals with major crimes, and after conviction, in a charge of murder, a sentence of death may be pronounced. The judges of the court are the Lord Justice-General, the Lord Justice-Clerk, and twelve other judges. A single judge, sitting with a jury composed of fifteen members, presides. Appeals from this court lie to the Criminal Appeal Court.

Court of Session: is the Supreme Civil Court in Scotland and, in certain cases of importance, an appeal to the House of Lords from this Court may be made. The Court of Session is composed of fourteen judges, who are the same as those composing the High Court of Justiciary, but as civil judges they are termed Lords of Session. The Lord Justice-General has the title of Lord President of the Court of Session. The Court has a wide and exclusive jurisdiction and is primarily concerned with actions connected with considerable sums of money, divorce and nullity, and civil trials by jury. The Inner House, when sitting as an appellate court hears appeals from the Outer House of the Court, together with certain appeals from the Sheriff Court. The Lord President presides over the First Division, and the Lord Justice-Clerk over the Second Division of the Inner House.

Criminal Appeal Court: this Court was instituted by the Criminal Appeal (Scotland) Act, 1926, and to it an appeal from the High Court of Justiciary is permitted on special grounds. The right of appeal is given only to the accused, and is usually based on questions of law.

House of Lords: is the ultimate Court of Appeal in civil causes.

Procedure in investigation of a serious crime.

The police lay information of the crime, for example, murder, before the Procurator-Fiscal, and provide him with a list of the witnesses in the case. The prisoner on apprehension is brought before a Police Magistrate on the given charge, by whom he is remitted to the Sheriff. By this time, the report on the post-mortem examination of the body of the victim, together with all other relevant reports in the case are in the hands of the Procurator-Fiscal, who has previously made the requisite arrangements for those necessary investigations. The Fiscal then proceeds to precognosce the witnesses. The Sheriff, or his substitute, gives the prisoner an opportunity, in the presence of his law agent, if he is legally represented, of emitting a declaration concerning the charge preferred against him, the prisoner having been duly warned that he need not say anything, but that if he chooses to do so his statement will

be written down, and used at his trial. When the accused is brought before the Sheriff for examination on any charge, and he or his agent intimates that he does not desire to emit a declaration in regard to such a charge, no declaration is taken.

At this stage the Fiscal has concluded his inquiry. The precognitions of witnesses, together with the medical reports, are now forwarded to the Crown Office, where they are placed before one of the Advocates-Depute. After reviewing all the documents, the Lord Advocate or one of his Deputes makes an order on the following lines : that no further inquiry is necessary ; to continue the inquiry ; to prosecute in the manner suitable to the circumstances of the crime. Depending upon the nature and gravity of the crime, the order may be "Sheriff summarily," or "Sheriff and jury," or "Indict," which necessitates trial before the High Court of Justiciary, or the High Court on Circuit.

Indictment.

When the Lord Advocate orders trial, either before a Sheriff and jury or in the High Court, an indictment or charge is carefully drawn up, setting out the nature of the crime, the place and time of its perpetration, and the productions or exhibits in the case. Productions may comprise garments, weapons, or documents, etc., which are to be produced at the trial as evidence against the accused. An indictment may be either written or printed, or partly written and partly printed, and consists of the following sections :—

1. (*a*) Name and designation of accused ; (*b*) name of Lord Advocate ; (*c*) the charge, which condescends upon (1) time, (2) place, (3) and manner ; (*d*) aggravations.
2. Numbered list of productions.
3. Numbered list of witnesses.

The indictment is signed by the Lord Advocate or by one of his Deputes or, where the trial takes place before a Sheriff, by the Procurator-Fiscal, who prefixes to his signature the words "By authority of His Majesty's Advocate."

At the trial the productions bear a number and have individual labels attached to them. On these labels will be found the signatures of the principal witnesses who will speak to their identity. These have been signed by the witnesses on a previous occasion following identification. Therefore, all that any of these witnesses has to do, in order again to identify an article when giving evidence, is to examine the label for the presence of his or her signature.

The indictment is served upon the accused prior to trial, together with a notice calling upon him to appear and answer the charge.

The precognitions and reports in the case form the brief of the prosecutor at the trial of the accused, and from them he leads the evidence of the witnesses.

Another step in the procedure prior to trial is what is termed the pleading diet. This consists of the accused or his solicitor intimating a plea of "guilty," "not guilty," or "not guilty" with the

intimation of a special defence, in relation to the charge preferred, in the presence of the Sheriff, or his Substitute, in court.

Procedure at trial in Scotland.

In Scotland, the procedure at the trial of an accused person is very similar to that in England. There are, however, a few differences.

In Scotland, when the court has assembled, the diet is called against the prisoner, who is asked by the judge to plead to the indictment against him. Should he plead guilty the judge passes sentence. If he plead not guilty, then a jury of fifteen is impanelled and put on oath by the clerk of court. By custom, the first person selected by ballot usually becomes the foreman. The prosecutor then calls the first witness who, after being put on oath by the judge, is examined by the prosecutor. At the conclusion of this examination-in-chief the witness is next cross-examined by counsel for the prisoner at the bar, or the "panel," as the prisoner is commonly called. The witness may then be re-examined by the prosecutor on points brought out in cross-examination. Thereafter, the judge may, as at any time during the course of the preceding examinations, ask questions of the witness. Any juror, through the medium of the judge, may also ask questions. In this manner witnesses for the Crown give their evidence, and, when the last has been heard, the case for the prosecution is closed. Next, witnesses for the defence, including the prisoner himself, may be called. If they are called, the position of respective counsel is reversed with respect to the leading of evidence. At the conclusion of all the evidence, the prosecutor addresses the jury, then counsel for the prisoner, the evidence is summed up by the judge, and the jury finally delivers the verdict. Counsel for the prisoner is entitled to have the last word with the jury before the summing-up of the judge. Notes of the evidence must be taken by the judge during the trial and a complete record is made by official shorthand writers previously sworn. The jury may return a verdict without retiring from the box, or after retiral, and the verdict, which is announced by the foreman, may either be unanimous or by a majority.

When the case is heard by a sheriff and jury, the local Procurator-Fiscal prosecutes, and the accused may be defended either by counsel or by a solicitor.

Differences in English and Scottish court procedure.

It will be apparent that procedure in Scotland differs from that in England. There are additional differences with regard to juries in the Court of Assize and in the High Court of Justiciary. An English jury is composed of twelve persons, a Scottish jury of fifteen. The former may fail to return a verdict, or, if a verdict is returned, it must be one of two only, namely, "guilty" or "not guilty." The latter must return one of three verdicts, "guilty," "not guilty," or "not proven." An English jury can convict or acquit only when unanimous; the Scottish jury may do so by a majority.

Workmen's Compensation Acts, 1925-45.

These Acts remain in force in respect of injuries received before July 5, 1948.

The National Insurance (Industrial Injuries) Act, 1946.

This Act substitutes for the Workmen's Compensation Acts, 1925 to 1945, a system of insurance against personal injury caused by accident arising out of and in the course of a person's employment and against prescribed diseases and injuries due to the nature of a person's employment.

Industrial injuries contributions are collected from employers and workers. These contributions together with sums provided by taxation form the Industrial Injuries Fund.

Three types of benefit are provided by this fund.

Where an insured person dies as a result of an industrial accident or prescribed disease "death benefit" is paid to his dependants in the form of a pension or gratuity.

During the period an insured person is incapacitated for work as a result of an accident or prescribed disease, injury benefit is paid at a flat rate with allowances for dependents, up to a maximum period of twenty-six weeks from the date of the accident or development of the disease.

If after the cessation of injury benefit the injured person still suffers from a substantial or lasting "loss of faculty," he is entitled to disablement benefit.

"Loss of faculty" means the partial or total loss of the normal use of organs or parts of the body, or the destruction or impairment of bodily or mental functions. It includes disfigurement.

The degree of disablement is assessed as a percentage. Where disablement is 20 per cent. or more, benefit takes the form of a pension. Where the assessment is under 20 per cent., benefit is provided as a lump sum.

Claims are made to the Ministry and routine questions are decided by an insurance officer. Where, however, there are special difficulties, the insurance officer may refer the case to a local appeal tribunal which consists of a chairman, a lawyer appointed by the Minister, and two members representing employers and workers. In all cases there is an appeal from decisions of an insurance officer to the local appeal tribunal.

The Crown appoints a barrister with high legal qualifications as Industrial Injuries Commissioner. Appeals can be made to him from decisions of the local appeal tribunal.

Questions of disablement are decided by medical boards. These boards normally consist of two doctors. An appeal lies to a medical appeal tribunal which consists of a legal chairman and two doctors.

Questions as to whether employment is insurable employment or not are decided by the Minister. An appeal on points of law lies to the superior courts.

The prescribed diseases and injuries are given in the First Schedule, Part I to the Act which is as follows :—

FIRST SCHEDULE

Part I

Description of disease or injury	Nature of occupation
Poisoning by :	Any occupation involving :
1. Lead	The use or handling of, or exposure to the fumes, dust or vapour of, lead or a compound of lead, or a substance containing lead.
2. Manganese	The use or handling of, or exposure to the fumes, dust or vapour of, manganese or a compound of manganese, or a substance containing manganese.
3. Phosphorus	The use or handling of, or exposure to the fumes, dust or vapour of, phosphorus or a compound of phosphorus, or a substance containing phosphorus.
4. Arsenic	The use or handling of, or exposure to the fumes, dust or vapour of, arsenic or a compound of arsenic, or a substance containing arsenic.
5. Mercury	The use or handling of, or exposure to the fumes, dust or vapour of, mercury or a compound of mercury, or a substance containing mercury.
6. Carbon bisulphide	The use or handling of, or exposure to the fumes or vapour of, carbon bisulphide or a compound of carbon bisulphide, or a substance containing carbon bisulphide.
7. Benzene or a homologue	The use or handling of, or exposure to the fumes of, or vapour containing, benzene or any of its homologues.
8. A nitro- or amido-derivative of benzene or of a homologue of benzene.	The use or handling of, or exposure to the fumes of, or vapour containing, a nitro- or amido-derivative of benzene or of a homologue of benzene.
9. Dinitrophenol or a homologue	The use or handling of, or exposure to the fumes of, or vapour containing, dinitrophenol or any of its homologues.
10. Tetrachlorethane	The use or handling of, or exposure to the fumes of, or vapour containing, tetrachlorethane.
11. Tri-cresyl phosphate	The use or handling of, or exposure to the fumes of, or vapour containing, tri-cresyl phosphate.
12. Tri-phenyl phosphate	The use or handling of, or exposure to the fumes of, or vapour containing, tri-phenyl phosphate.
13. Diethylene dioxide (dioxan)	The use or handling of, or exposure to the fumes of, or vapour containing, diethylene dioxide (dioxan).

Description of disease or injury	Nature of occupation
Poisoning by :	Any occupation involving :
14. Methyl bromide	The use or handling of, or exposure to the fumes of, or vapour containing, methyl bromide.
15. Chlorinated naphthalene (excluding the condition known as chlor-acne).	The use or handling of, or exposure to the fumes of, or dust or vapour containing, chlorinated naphthalene.
16. Nickel carbonyl	Exposure to nickel carbonyl gas.
17. Nitrous fumes	The use or handling of nitric acid or exposure to nitrous fumes.
18. Gonioma Kamassi (African boxwood).	The manipulation of Gonioma Kamassi or any process in or incidental to the manufacture of articles therefrom.
19. Anthrax	The handling of wool, hair, bristles, hides or skins or other animal products or residues, or contact with animals infected with anthrax.
20. Glanders	Contact with equine animals or their carcases.
21. Infection by leptospira icterohæmorrhagiæ.	Work in rat-infested places.
22. Ankylostomiasis	Work in or about a mine.
23. (*a*) Ulceration of the corneal surface of the eye.	The use or handling of, or exposure to, tar, pitch, bitumen, mineral oil (including paraffin), soot or any compound, product, or residue of any of these substances.
(*b*) Localised new growth of the skin, papillomatous or keratotic.	
(*c*) Epitheliomatous cancer or ulceration of the skin, due in any case to tar, pitch, bitumen, mineral oil (including paraffin), soot or any compound, product, or residue of any of these substances.	
24. (*a*) Chrome ulceration	The use or handling of chromic acid, chromate or bichromate of ammonium, potassium, sodium, or zinc, or any preparation or solution containing any of these substances.
(*b*) Inflammation or ulceration of the skin or of the mucous membrane of the upper respiratory passages or mouth produced by dust, liquid or vapour (including the condition known as chlor-acne but excluding chrome ulceration).	Exposure to dust, liquid, or vapour.
25. Inflammation, ulceration or malignant disease of the skin or subcutaneous tissues or of the bones, or leukæmia, or anæmia of the aplastic type, due to *X*-rays, ionising particles, radium or other radio-active substance ; or inflammation of the skin due to other forms of radiant energy.	Exposure to *X*-rays, ionising particles, radium or other radio-active substance or other forms of radiant energy.

Description of disease or injury	Nature of occupation
26. Cataract produced by exposure to the glare of, or rays from, molten glass or molten or red-hot metal.	Any occupation involving: Frequent or prolonged exposure to the glare of, or rays from, molten glass or molten or red-hot metal.
27. Compressed air illness . .	Subjection to compressed air.
28. Telegraphist's cramp . . .	The use of Morse-key telegraphic instruments for prolonged periods.
29. Writer's cramp	Handwriting for prolonged periods.
30. Twister's cramp	The twisting of cotton or woollen (including worsted) yarn.
31. Subcutaneous cellulitis of the hand (Beat hand).	Manual labour causing severe or prolonged friction or pressure on the hand.
32. Subcutaneous cellulitis or acute bursitis arising at or about the knee (Beat knee).	Manual labour causing severe or prolonged friction or pressure at or about the knee.
33. Subcutaneous cellulitis or acute bursitis arising at or about the elbow (Beat elbow).	Manual labour causing severe or prolonged friction or pressure at or about the elbow.
34. Inflammation of the synovial lining of the wrist joint and tendon sheaths.	Manual labour, or frequent or repeated movements of the hand or wrist.
35. Miner's nystagmus . . .	Work in or about a mine.
36. Poisoning by beryllium . .	Any occupation involving the use or handling of, or exposure to the fumes, dust, or vapour of, beryllium, or a compound of beryllium, or a substance containing beryllium.
(Also see Addendum at end of book.)	

Where an insured person has developed a disease prescribed in the above schedule, that disease shall, unless the contrary be proved, be presumed to be due to the nature of his insurable employment if that employment was in any occupation set against that disease in the second column of the schedule and he was so employed on, or at any time within one month immediately preceding, the date on which he is treated as having developed the disease. This does not apply to the disease numbered 24(*b*) in the schedule.

Pneumoconiosis and byssinosis are dealt with separately. The occupations recognised as causing pneumoconiosis are given in the First Schedule, Part II, which is as follows :—

PART II

1. Any occupation involving—

(*a*) the mining, quarrying, or working of silica rock or the working of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials (including any occupation in which any of the aforesaid operations are carried out incidentally to the mining or quarrying of other minerals or to the manufacture of articles containing crushed or ground silica rock) ;

(*b*) the handling of any of the materials specified in the foregoing subparagraph in or incidental to any of the operations mentioned therein, or substantial exposure to the dust arising from such operations.

2. Any occupation involving the breaking, crushing, or grinding of flint or the working or handling of broken, crushed, or ground flint or materials containing such flint, or substantial exposure to the dust arising from any of such operations.

3. Any occupation involving sand blasting by means of compressed air with the use of quartzose sand or crushed silica rock or flint, or substantial exposure to the dust arising from such sand blasting.

4. Any occupation involving—

(*a*) the freeing of steel castings from adherent siliceous substance ;

(*b*) the blasting of metal castings to free them from adherent siliceous substance by means of any abrasive, by a blast of compressed air, by steam, or by a wheel ;

(*c*) the moulding of iron castings with the use of siliceous materials as a facing powder or parting powder ;

(*d*) substantial exposure to the dust arising from any of the foregoing operations.

5. Any occupation in or incidental to the manufacture of china or earthenware (including sanitary earthenware, electrical earthenware, and earthenware tiles), and any occupation involving substantial exposure to the dust arising therefrom.

6. Any occupation involving the grinding of mineral graphite, or substantial exposure to the dust arising from such grinding.

7. Any occupation involving the dressing of granite or any igneous rock by masons or the crushing of such materials, or substantial exposure to the dust arising from such operations.

8. Any occupation involving the use, or preparation for use, of a grindstone, or substantial exposure to the dust arising therefrom.

9. Any occupation involving—

(*a*) the working or handling of asbestos or any admixture of asbestos ;

(*b*) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos ;

(*c*) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures, and appliances for the collection of asbestos dust ;

(*d*) substantial exposure to the dust arising from any of the foregoing operations.

10. Any occupation involving—

(*a*) work underground in any coal, tin, slate, or hæmatite iron ore mine ;

(*b*) the working or handling above ground at any coal or tin mine of any minerals extracted therefrom, or any operation incidental thereto ;

(*c*) the trimming of coal in any ship, barge, or lighter, or in any dock or harbour or at any wharf or quay ;

(*d*) the sawing, splitting, or dressing of slate, or any operation incidental thereto.

Where an insured person in relation to whom pneumoconiosis is prescribed in Part II of the First Schedule has developed pneumoconiosis, the disease shall, unless the contrary is proved, be presumed to be due to the nature of his insurable employment if he has been employed in one or other of the occupations set out in Part II for a period or periods amounting in the aggregate to not less than two years in employment which either was insurable employment or would have been insurable employment if it had taken place on or after July 5, 1948.

Where an insured person, in insurable employment at any time on or after July 5, 1948, in cotton rooms, blowing rooms, or card rooms in factories where the spinning of raw cotton is carried on, for a period or periods (whether before or after July 5, 1948) amounting in the aggregate to not less than twenty years, develops byssinosis, the disease shall, unless the contrary is proved, be presumed to be due to the nature of his insurable employment.

Law Reform (Personal Injuries) Act, 1947.

This Act repeals the Employers Liability Act, 1880, and abolishes the defence of common employment. It also amends the law relating to the liability in damages for breach of statutory duty and to the measure of damages for personal injury or death. By section 3 of the new act, the measure of damages for personal injuries is connected with the National Insurance (Industrial Injuries) Act, 1946. The terms of this section are as follows :—

> In an action for damages for personal injuries (including any such action arising out of a contract), there shall in assessing these damages be taken into account, against any loss of earnings or profits which has accrued or probably will accrue to the injured person from the injuries, one half of the value of any rights which have accrued or probably will accrue to him therefrom in respect of industrial injury benefit, industrial disablement benefit, or sickness benefit for the five years beginning with the time when the cause of action accrued.

The Factories Act, 1937.

By this Act every medical practitioner attending on or called in to visit a patient whom he believes to be suffering from lead, phosphorus, arsenical, mercurial, aniline, manganese, chronic benzene and carbon bisulphide poisoning, or toxic jaundice, chrome ulceration, epitheliomatous ulceration, compressed-air illness, anthrax

and toxic anæmia contracted in any factory, shall forthwith send addressed to "The Chief Inspector of Factories, Ministry of Labour and National Service, London," a notice stating the name and full postal address of the patient and the disease from which, in the opinion of the practitioner, the patient is suffering, and the name and address of the factory in which he is or was last employed, and shall be entitled in respect of every notice sent in pursuance of this section to a fee of 2s. 6d. Failure to notify carries a liability to a fine not exceeding 40s. The foregoing will not apply if a notice, so required, has been previously sent.

By the Lead Paint (Protection against Poisoning) Act, 1926, a practitioner must similarly notify any case of lead poisoning contracted in painting any building unless such notification has already been made.

References

1. R. v. G.M.C., ex parte Spackman (1942), 58 T.L.R., 167, 234, 321, and [1943], A.C., 627.
2. Hennessy case, 1946 Lancet, Vol. I, 616, 622, 862.
3. R. v. G.M.C., ex parte Kynaston [1930], 1 K.B., 562.
4. Lanphier v. Phipos (1838), 8 C. and P., 475.
5. Rich v. Pierpont (1862), 3 F. and F., 35.
6. Whiteford v. Hunter and Gleed, 1948 Lancet, Vol. II, 232.
7. Baldwin v. Lord and Others, 1947 Med. Leg Jour., Vol. XV, Part II, 92.
8. R. v. Bateman (1925), L.T. (K.B.), 792.
9. Foote v. Greenock Hospital (1912), S.C. 69.
10. Hillyer v. St Bartholomew's Hospital [1909], 2 K.B., 820.
11. Strangways-Lesmere v. Clayton [1936], 2 K.B., 11.
12. Powel v. Streatham Manor Nursing Home [1935], A.C., 243
13. Gold v. Essex C.C. [1942], 2 All E.R., 237, and (1942), 58 T.L.R., 357.
14. "Jervis on the Offices and Duties of Coroners," W. B. Purchase. Sweet and Maxwell, and Stevens and Sons, Ltd., London, 1946.

CHAPTER II

MEDICAL EVIDENCE

MEDICAL evidence resolves itself into two forms, namely :—

Documentary, and
Oral.

As documentary evidence, however, must eventually be spoken to on oath in the witness-box, the above distinction is more theoretical than practical.

DOCUMENTARY EVIDENCE

This may take the following forms :—

The medical certificate.
The medical report.
Notes or memoranda.
Dying declarations and depositions.

The medical certificate.

This is the simplest form of documentary evidence, and may, for example, consist in the certification, by a duly qualified and registered practitioner, of the inability of attendance in court of a witness or juror, by reason of illness. In England no particular form of words is required. A simple statement of facts, embodying the name, address, designation, and nature of the illness, of the witness or juror, signed by a practitioner, is all that is necessary. In Scotland something more is required to render the certificate legal. The statement of facts must be attested "on soul and conscience." Without this attestation no certificate or report in a Scottish Court is legal. Certificates of death, however, do not require such form of attestation, as they are in accord with the form scheduled in the Registration Acts.

The medical report.

This form of evidence is given at the instance of the Procurator-Fiscal by a medical witness in circumstances such as the following :—

In a case where injuries have been inflicted upon an assaulted person.

As the result of a post-mortem examination of the body of a person who has died suddenly, or under suspicious circumstances, or from culpable violence, or from any other form of violent death.

For purposes of the Fatal Accidents Inquiry (Scotland) Act.
For purposes of an inquiry regarding the death of a person while in prison, under the Prisons (Scotland) Act.
As the result of the examination of a body after exhumation.
Following an examination of stains on clothing.
Following toxicological examination in cases of poisoning.

In Scotland such reports must be signed "on soul and conscience."

Reports upon the examination of assaulted persons should include an accurate description of the character, number, situation, measurements, and direction of the injuries found, together with an opinion of the class of weapon which has been used in their production.

When a post-mortem examination of a body is required, procedure differs in England and Scotland. In England, the request of the Coroner to undertake the examination is the authority for performing it (see p. 19). In Scotland, the procedure is so different that some detailed consideration is necessary.

The Procurator-Fiscal presents a petition to the Sheriff in the following terms :—

"GLASGOW, , 19 .

"Unto the Honourable the
SHERIFF OF THE COUNTY OF LANARK.

"The Petition of Procurator-Fiscal of Court for the Public Interest,

"*Humbly Sheweth,*

"That from information received by the Petitioner it appears that died at or in on the day of .

"That the circumstances call for investigation and a dissection of the body is necessary.

"May it therefore please your Lordship to grant warrant to cite witnesses for precognition in the premises, and to grant warrant to , to dissect said body and to report.

"According to Justice, etc.

"(Signed)

"Procurator-Fiscal."

" 19 . Grants warrant as craved.

"(Signed)

"Sheriff."

The above warrant is transmitted to the doctor or doctors named in it, together with the information as to where the body is lying. At the mortuary, witnesses who have been summoned for the identification of the body will be in attendance along with a representative of the police. The warrant entitles those named in it to have possession of the body until the examination has been completed. When death is due to culpable violence by an assailant, two medical men are named in the warrant, and both are responsible for the performance of the examination. If they are agreed upon the

findings both will sign the report, but if they should fail to agree, which is very unusual, each will write a report.

Identification of body.

Before commencing the examination of the body, it is the duty of the medical examiners to establish its identity. For this purpose, identification may be either personal or legal. Personal identification is made by two persons who are usually near relatives or close friends of the deceased. Their names, designations, and addresses must be recorded by the examiners for incorporation in their report. Thus the body is identified as a given person who was well known to the witnesses during life. Under certain circumstances, however, it is not possible to secure personal identification, and in this event the body has to be identified in the legal sense, namely, that the body in question is the body which was found or seen by those called upon to identify it, and not as the body of a given person known to them during life. Full particulars of these witnesses must be embodied in the medical report, and special care must be taken to record in the report any possible identifying features present on the body, in order to facilitate subsequent identification of the body as that of a particular person.

No one, except the practitioners instructed to conduct the examination and their assistants, is allowed to be present at a post-mortem examination except with consent of the Crown. The presence of a doctor who has attended the case or who represents the interests of an accused person may be sanctioned by the Crown authorities, but only on condition that he is present as an onlooker, and will not interfere in any way with the examination. No formal intimation is given that the autopsy is to be held, but in all cases where any person is in custody on suspicion of causing death, this examination is made as a matter of course, so that an accused person or his advisers cannot be in doubt as to the necessity of making immediate application for permission to send a medical representative to witness the examination if they so desire. Such application should be made to the Procurator-Fiscal. He will communicate with the Crown agent for the instructions of Crown counsel. If an independent post-mortem examination is desired, for the purposes of the defence, it can be made after the Crown examination.

It is expedient for one examiner to perform the manual work and the other to observe the findings and take notes of the facts observed during the currency of the examination.

External examination.

A careful external examination of the body must be made. The appearances indicative of the time of death and the position in which the body has lain after death should be noted, together with any marks of violence, or other marks, which might point directly or indirectly to the cause of death.

If identity of the body has not been established as that of a

known individual, all identifying features, such as height, approximate age, sex, colour of the eyes and hair, condition and number of teeth, bodily deformities or abnormalities, scars and tattoo marks, together with other characteristic features, should be carefully noted, so that subsequent identification may be established. In the ordinary course of events, the examiners need not extend their observations to the clothing, but it would fall within their duty to note carefully, for example, the wrappings in which the body of a newly born child was found, or the clothing or wrappings of a body which has been discovered in an advanced state of decomposition. Clothing should be examined separately for stains, cuts, or perforations, in cases of injury. It is advisable that this examination should be made in the laboratory, where adequate facilities are available, and should form the subject of a separate report. When garments are considered to have an important bearing on the investigation of a case, by instructions of the Procurator-Fiscal, they are brought by the police to the laboratory where detailed examination can be made.

The points to be ascertained with regard to the time of death include the rectal temperature, the presence and extent, or absence, of rigor mortis, the incidence, extent, colour, and degree of fixation of post-mortem lividity, and the presence, character, and extent of putrefaction. The points relating to the likely cause of death must include any lesion which is indicative of violence. All wounds must be measured accurately, their characters described in detail, and their position fully recorded. Such data will prove invaluable in the determination of the class of weapon which has been responsible for the wounding, and will throw light on the important question as to whether the wounds owe their origin to accident, suicide, or homicide. When injuries are situated in the line of the usual incisions necessary for post-mortem examination, such incisions must be modified to enable the lesions to be examined in their entirety. When bruises or similar marks are present, all of them, after measurement, should be incised, to verify the presence of extravasated blood, since questions may arise in court regarding their possible confusion with post-mortem lividity (see p. 130). Full notes should be made of ligature marks, and evidence of bruising and abrasions on the neck. The hands should be inspected for defensive wounds, blood-staining, weapons, or other objects. Common sites, and other parts of the body surface, should be carefully examined for puncture marks which might have been caused by a hypodermic needle, and the possibility of the presence of wounds which have been produced in the process of embalming should not be overlooked. When evidence of burning or corrosion is present, full particulars of the incidence, character, extent, and degree should be recorded. The condition of the natural orifices of the body should be ascertained. The possibility of fractures being present should be verified or eliminated.

The foregoing is merely illustrative of some of the points which must receive attention, but the circumstances of each case should

suggest the important lines of investigation, and these will receive detailed description, at a later stage, in the consideration of the various medico-legal forms of death.

Internal examination.

When the external examination has been completed, the examiners proceed to the internal examination which must include an examination of all the organs and parts of the body, despite the fact that the apparent cause of death has been previously found in one of them, since evidence contributory to the cause of death may be found in one or more of the others. The importance of this cannot be too strongly insisted upon, as inadvertent omission of a complete examination may readily invalidate a report.

A doctor was asked to examine the body of a man for whose death two men were in custody on a charge of murder. He stated that he found certain marks about the head and upper part of the body, but he attributed death to "failure of the heart's action, due to shock." Owing to the unsatisfactory character of the evidence, a re-examination of the body was made, and the doctor had to admit that in the first examination he had overlooked a dislocation of the first and second cervical vertebræ with fracture of the odontoid process and rupture of the lateral ligaments. This case indicates very emphatically that every post-mortem examination for medico-legal purposes must be completely and carefully performed.

Notes on post-mortem technique.

Instruments.

The following instruments will be found useful :—

Two post-mortem knives.
Cartilage knife.
Several scalpels.
Straight probe-pointed bistoury.
Curved probe-pointed bistoury.
Brain knife.
Two pairs of strong forceps.
Pair of sharp-pointed scissors.
Pair of long-bladed, blunt-ended scissors.
Pair of fine, sharp-pointed scissors, for exploration of the coronary arteries.
Pair of bone forceps.
Pair of rib shears.
Periosteal elevator.
Bone saw.
Coronet, to hold head during severance of calvarium.
Needles, curved and straight.
Ladle.
Virchow chisel, with transverse handle, for separation of the severed calvarium.
Blunt probe.

Accessories.

Twine, for stitching.
Sponges.
Measuring jug.
Metal ruler, marked in inches and centimetres.
A thermometer (Fahrenheit).
Two pairs of rubber gloves.
Talc powder.
Full length waterproof apron.
Full length overall.
Small glass-stoppered bottles containing preserving fluid for portions of tissue, and similar empty containers for samples of urine or blood.
Notebook and pencil.
Bottle of Dettol or similar disinfectant.
Small bottle of tincture of iodine.
Small bottle of 70 per cent. alcohol.

The last three solutions may be used in the event of accidental hand injury.

In cases of suspected poisoning, and in some other cases, a series of glass jars should be at hand for the retention of viscera, also labels for attachment to the jars, and a suitable medium for sealing the containers.

General practical procedure.

Having extended the head, a median incision, reaching from a point about one inch below the symphysis menti to the symphysis pubis and avoiding the umbilicus, is made. The abdominal portion of the wound should first be opened and clearance of the tissue from the ribs effected from below upwards. The lower ends of the recti muscles should be severed to allow greater retraction of the abdominal flaps. To open the thorax, the costal cartilages are severed obliquely and the sterno-clavicular joints disarticulated or sawn through. The sternum is removed by separating the underlying tissue from below upwards. The skin and under-tissue on each side of the neck is reflected outwards. A block is placed under the neck, and the knife is passed round the inner margin of the lower jaw. The attachments of the tongue are separated, and the tongue pulled down through the opening. Using gentle downward traction of the tongue and by dissection, the pharynx, epiglottis, larynx, trachea, and upper part of the œsophagus are freed. The great vessels of the neck are next severed at the root of the neck. The trachea and œsophagus are now pulled downwards and the mass of thoracic viscera is raised out of the chest cavity. The aorta is severed together with the œsophagus before these structures pass through the diaphragm. The œsophagus should be severed between a double ligature to prevent escape of stomach contents. The pericardium is next severed at its diaphragmatic attachment and the inferior vena cava divided. The mass of thoracic viscera

may now be removed entire for detailed examination. As an alternative method the heart and lungs may be removed separately.

With regard to the abdominal cavity, this should be inspected carefully before the contents are disturbed. When this is completed, the small intestine should be removed from its mesentery from the cæcum upwards to the duodenum, where a double ligature should be applied before severance of the intestine. The large bowel is next removed, and a double ligature applied before it is divided at the junction of the sigmoid and rectum. The abdominal organs are then removed individually.

In abortion cases, particularly, the removal of the female pelvic viscera is of importance, and the following method is recommended. Retract the bladder from the pubic arch, and separate the peritoneum and areolar tissue. Carry an incision round the level of the pelvic brim and continue downwards to the pelvic outlet. The legs of the subject are then abducted widely and an incision is commenced under the symphysis pubis and continued downwards and backwards on each side of the labia minora to the posterior border of the vagina. The dissection commenced within the pelvis is continued until a communication with the external incisions is established. The entire genital tract, together with the bladder and rectum, is then removed.

To remove the brain, the first step is to sever the scalp transversely, from ear to ear, and reflect the anterior part of the scalp forwards to the superciliary ridges, and the posterior part backwards to a point just below the occiput. A coronet is then applied, to steady and control the head, and the skullcap is removed by a circular line of severance, using a saw. The line of severance follows a point just above the superciliary ridges in front and through the occiput behind. The skullcap should be completely severed prior to removal and leverage should not be resorted to since such force might fracture the skull or extend an existing fracture. The skullcap removed, the dura mater is cut along the line of skull severance and also along the falx cerebri. When the surface of the brain has been exposed, the fingers of the left hand are inserted between the dura and the frontal poles of the cerebrum and the latter are gently raised. Vessels, nerves, tentorium cerebelli, and spinal cord are severed. The brain, which has been pulled backwards gradually, is now resting on the left hand, and with the use of both hands is transferred to a platter for examination and dissection.

In conducting a post-mortem examination it is advisable to have a basin containing warm water at hand in order to keep gloves and instruments clean.

The foregoing simple points may prove to be of some help, but, for those who are inexperienced in practice, further theoretical guidance from works dealing with post-mortem technique should be obtained.[1] Those who undertake post-mortem examinations should familiarise themselves with the appearances of healthy and diseased organs.

The average weight of adult organs and those of the newborn

child, together with some useful post-mortem room data, in connection with weight, measurement, and temperature conversions will be found on pp. 700 and 701.

Examination of stomach.

In all cases attention should be directed to the stomach contents. The odour, especially if alcoholic, should be noted. In accident cases, in deaths from culpable violence, and in any other case in which an assessment of the degree of possible intoxication may have a bearing, a generous sample of blood should be obtained for the purpose of determining the alcoholic content by analysis (see p. 631). A sample of urine should also be taken for the same purpose. The analyst prefers, when possible, to get at least 2 ounces of blood or urine. The stomach contents will give indication of the extent of digestion which has taken place and, in an approximate manner, the interval of time since the ingestion of the last meal prior to death. An ordinary meal leaves the stomach in about four hours. Maile and Scott,[2] following radiological surveys, have shown that a large meal may be retained for five hours and that a meal, not necessarily large, but containing a considerable quantity of butter or cream, is retained in the stomach for a longer period. Concentrated carbohydrates, such as sugar, leave the stomach much more quickly than natural carbohydrates, such as banana or potato. Fat in abnormal proportions causes the stomach emptying time to be prolonged in marked degree.

Some important points.

The membranes should always be stripped from the base of the skull, and the bone carefully examined for evidence of fracture.

The vertebral column should be examined for fracture or dislocation.

Incisions should be made between the ribs when examination for fracture is undertaken, since this procedure enables the examiner to test the mobility of each rib in turn.

Parts of the body, necessary for further detailed dissection, should be removed and placed in containers to which a 2 per cent. solution of formalin in saline has been added.

In every case of fatal wounding the dimensions of the wound or wounds should be recorded, together with the depth and direction of the track. These particulars have a direct bearing, not only in relation to the weapon which caused the wound, but to the relative positions of the assailant and assaulted person when the injury was inflicted.

When blood or other fluid is found within a cavity, the quantity should be measured and recorded.

In cases of firearm injury, a bullet retained in the body must be located (see p. 272).

The eyes should be examined for evidence indicative of impaired vision, for example, an artificial eye or corneal opacity. This is

of great importance sometimes, especially in cases of vehicular accident.

The finger-nails should be inspected for evidence of blood-staining and for small portions of epidermis, indicative of struggle.

In cases of suspected strangulation, manual or otherwise, the structures of the mouth and neck should be removed for detailed dissection and histological examination.

The coronary arteries should always be examined carefully.

The entire length of the aorta should be opened and examined for evidence of degenerative changes.

A B

FIG. 1

Comparison photomicrograph showing matching layers in paint flakes. *A*, Paint from damaged vehicle. *B*, Contact traces removed from car in collision.

(By courtesy of the City of Glasgow Police.)

In cases of head injury following vehicular accident, specimens of hair, close to the edges of the wounds, should be removed. Comparative examination of hairs found on the suspect vehicle may be necessary at a later stage in the investigation (see pp. 110 and 116). In such cases, the wounds and scalp generally should be examined for glass or paint fragments (see Fig. 1). When found, these should be retained.

In cases of suspected carbon monoxide poisoning and in burning

cases, separate specimens of blood should be taken from the left and right sides of the heart for laboratory examination.

In cases of culpable violence, a specimen of blood should be retained for blood-grouping purposes (see p. 346).

When there is any doubt as to whether an injury was inflicted before or after death, tissue should be removed for histological examination. This is of special importance in connection with bruises (see p. 248 and Fig. 99).

In fatal cases associated with rape, a specimen of pubic hair and vaginal swabs should be taken (see pp. 119 and 432).

In cases of burning, a specimen of mucus from the air passages should be examined microscopically for the presence of carbon particles.

In cases of drowning, when the stomach contents indicate the swallowing of unusual fluids, a specimen should be retained for examination.

In cases of firearm wounding, a surface scraping should be removed from any area of blackening surrounding the wound of entry, together with a specimen of hair, adjacent to the wound, especially in the instance of head injury. Microscopical examination of the particles and of the hairs may prove important with regard to the assessment of the range of fire.

The notes made during the currency of a post-mortem examination should be scrutinised carefully before the body is closed.

Effects of keeping bodies in refrigerating chamber.

Under these circumstances, if the body has been exposed to a low temperature soon after death, the onset of rigor mortis will be retarded. If exposed at a later stage, when rigor is passing off, putrefaction will be retarded. Bodies which have been kept in a cold chamber most frequently show reddish, patchy colorations on the body surface, especially in the hypostatic regions and sometimes in the organs, the blood is a brighter reddish colour, and surface injuries, such as bruises and abrasions, have an intensified appearance. We have frequently noted these manifestations, and consider that a necessary allowance should be made in their interpretation. Prior to autopsy, bodies which have been chilled should be removed from the cold chamber and placed on the table for about two hours. This will allow the tissues to become softer and easier to manipulate.

Effects of embalming.

Embalming of a body is effected by the injection of a solution such as 40 per cent. formaldehyde and 10 per cent. methyl alcohol into one of the large arteries either by gravity or by positive pressure. It is necessary to drain a considerable quantity of the blood from a vein so that there will be a replacement by the embalming fluid. Openings into both the chest and abdominal

cavities are also made and a quantity of the solution is injected into them. The object of embalming is to preserve the body by the destruction of micro-organisms. It is important to note that both post-mortem dissections and the removal of specimens from bodies for investigation should be completed prior to embalming, since the tissues of the body will be hardened and the detection of certain poisons made difficult if not impossible by the process.

Procedure in suspected poisoning.

In cases where poisoning is the suspected cause of death, special precautions are adopted. Careful attention should be paid to the condition of the mouth, the œsophagus, and the gastro-intestinal tract. The lower end of the œsophagus, about three inches above the cardiac orifice of the stomach, should be tied with a double ligature, and severed between the ligatures. A double ligature should be placed round the upper part of the duodenum, and the stomach, with contents, removed. Next, the whole of the large and the small intestines are removed by division between double ligatures. The various parts should be placed in separate glass jars, labelled, signed, and sealed. In further jars are placed the liver, kidneys, spleen, a specimen of blood, and urine in addition to other organs and tissues as the circumstances of the specific case dictate. When these containers are formally handed over to the analyst, a receipt should be obtained. Should the stomach and intestines be opened and examined in the post-mortem room, care must be taken to keep the various parts separate and free from contamination not only from each other but from the other organs. It is only by such procedure that an opinion, as to the possible interval of time which has elapsed between the ingestion of the poison and death, can be expressed after the results of chemical analysis have been considered. This is of particular importance in cases of metallic irritant poisoning, such as arsenical poisoning. The necessity for taking all the important parts of a body in a case of poisoning will be obvious when it is explained that after analysis by the Crown experts, the remainder may be requested by the accused for examination by experts nominated by him for his defence. In cases of suspected arsenical poisoning, specimens of hair, skin, bone, and nails should also be removed for analysis (see p. 551).

Exhumations.

It is sometimes necessary in cases of suspected crime, in cases of civil actions for damages, in certain instances of life insurance, where the cause of death is disputed, and in other contingencies, to disinter bodies for the purpose of determining the actual cause of death. In any case of comparatively recent interment it is an unsavoury operation, and one not unattended by danger.

In England, at common law, a Coroner may order a body to be disinterred for the purposes of inquisition. If exhumation is required for other purposes, the licence of the Home Secretary

must be obtained under Section 25, Burial Act, 1857. In Scotland, a Sheriff, after petition, may grant a warrant. In a civil case, a petition is presented to the Sheriff, or to the Court of Session, usually to the former, by the party desiring the exhumation. Thereafter, the relatives are informed that such a petition has been made, and a date is arranged for hearing any objections. After the hearing, the petition may or may not be granted, but if it is granted, the name of the practitioner, or practitioners, is put on the warrant for exhumation, and authority given to the graveyard authorities to have the body exhumed.

The necessary preparations having been made with the authorities of the graveyard, the body is disinterred, and the examination made. The coffin should be identified by the undertaker, and the grave-digger should identify the grave.

Certain precautions should be adopted in exhumations. For example, the examination should be made during the lightest period of the day and, when possible, the dissection should be made in the open. It may be necessary to have a temporary screen erected. When the examination is made inside a building, the accommodation used should permit of free ventilation. When the coffin has been raised to the surface, adequate time should be given for the drainage of fluid from the interior of the coffin, and after the body has been removed from the coffin, it should be exposed to the air for a short period to permit the dissipation of foul gases. Care should be taken to ensure that the gloves worn are in perfect condition.

The dissection is made in the same manner as that of the routine post-mortem examination.

In cases of suspected arsenical poisoning it is important to preserve a portion of the earth from above the coffin, against a possible defence that this poison, which may be found in the body, was imparted to it by the soil. A piece of the shroud enveloping the body, together with a portion of the wood from the coffin, should also be retained in such cases for the purpose of eliminating the presence of arsenic (see p. 550).

Important and decisive information, respecting the cause of death, may be obtained from an examination of a body following exhumation.

Report on a post-mortem examination.

From the result of a post-mortem examination the report of the examiners is framed.

The opinion expressed by the examiners in their report must be founded solely on the facts comprehended within the report, and not upon facts which are not specified in detail in the description of the organs as found.

The examiners should send to the Procurator-Fiscal, within two days after the examination, their report of the examination, embodying their opinion of the cause of death, and the reasons for

that opinion, which should be stated clearly. Along with the report the warrant, still in their possession, should be returned to the Procurator-Fiscal. If further investigation is necessary, the report, by arrangement, may be delayed until the investigation is completed.

The examiners should frame the terms of the report in simple but clear terms, remembering that it may in all probability be read in court for the information of the court, counsel, and jury. The report should be as brief as possible, compatible with clearness. It is not necessary to describe each organ in detail, and it is sufficient to say, where the organs of a cavity have been found healthy, that "all the organs of this cavity were examined, and found healthy." Should one or more organs only be found diseased or injured, after description of the facts found with respect to these, it is more advisable to say that "the other organs of this cavity were examined and found healthy," than to describe their normal appearances. While in certain circumstances it may be impossible to avoid the use of technical terms, the examiners should try to give them popular interpretation, within brackets. Conciseness, brevity, and clear language are of high value in the expression of the opinion, since without these attributes the issues may readily become confused, and the report occasion dubiety, and unnecessarily prolonged cross-examination.

Examiners should avoid the use of such terms as "about," or "nearly," when measurement is in question, or of comparative or superlative adjectives with respect, for example, to amounts of fluid or blood, when such can be determined. The report should be exact in its terms. The original notes made at the examination should be retained, together with a copy of the report sent to the Procurator-Fiscal, for future reference in the preparation of evidence.

Framing a report is often a matter of some difficulty for the less experienced, and for this reason the following skeleton basis for a report is given :—

FORM OF MEDICAL REPORT

Preamble.

Date and place from which report is written.

Warrant for the examination, by whom granted, together with place of examination.

Names, ages, designations, and addresses of witnesses who have, in the presence of the examiners, identified the body.

External examination.

External appearances of body indicative of time of death.

Presence or absence of external marks of violence. Their site, character, dimensions, and relation to each other, and of any other markings bearing upon the cause of death.

Internal examination.

Description of—

Brain, its membranes, condition of calvarium, and of base of skull.

Spinal column, and, if necessary, condition of spinal cord and membranes.

Description of organs and contents of thoracic cavity—

General disposition of organs.

Presence of fluid in pleural cavities.

Condition of pericardium, heart, and large blood-vessels.

Condition of larynx, trachea, œsophagus, lungs, ribs, etc.

Description of organs and contents of abdominal cavity—

General disposition of organs.

Presence of abnormal fluid in abdominal cavity.

Condition of stomach, intestines, liver, spleen, kidneys, urinary bladder, pancreas, aorta, etc.

Description of organs and contents of female pelvis—

Vaginal tract.

Uterus and appendages.

The reasoned opinion of the cause of death based upon the facts found.

In Scotland, attestation of the report "on soul and conscience."

Signatures and medical qualifications of examiners.

NOTE.—In reports, all numbers, except the date of report, should be expressed in words, and not in figures.

All interpolated words, or deleted words, should be initialled by the signators. At the foot of each page, the number of words deleted should be indicated and initialled by the signators.

When the report occupies more than one page, the signatures of the examiners should be appended to each page.

The following report embodies greater detail and applies many of the points suggested :—

Place.
Date.

By virtue of a warrant of the Sheriff of , and at the instance of , Esquire, Procurator-Fiscal of the said County, we, the undersigned, on this date and within the mortuary of (*here name institution or police station*) made a post-mortem examination of the body of (*insert full name, if known*), which was identified in our presence by the following persons :—

1. A. B., 22 years, son of the deceased, residing at 24 Blank Street, Glasgow, and
2. B. C., 58 years, confectioner, cousin of the deceased, residing at 261 Blank Street, Glasgow.

External examination.

The body was that of a well-nourished and well-developed person. Death-stiffening, or rigor mortis, was present generally. The only mark of violence visible on the body surface was an incised, or clean-cut, wound situated one and a half inches below the level of the left collar-bone, and two and a quarter inches from the middle line of the breast-bone. There was faint bruising at the edges of the wound. It measured three-quarters of an inch in length and a quarter of an inch in breadth. It was ovate in shape.

Internal examination.

Head.

The brain and its coverings were found normal. The skull was intact.

Chest.

On opening the chest cavity, a considerable quantity of clotted blood was found lying in the tissues below the breast-bone, and extending into the left side of the chest. Within the pericardium, or heart-bag, fluid and clotted blood, amounting to nine ounces, was found. There were, in addition, six ounces of blood in the left side of the chest. Before the organs of the chest were removed, careful inspection of the direction of the wound on the front of the chest was made. This inspection showed that the wound had a direction downwards, from left to right, and that it had penetrated the chest wall, between the first and second ribs. Dissection showed that the wound continued through the heart-bag, and into the main artery of the body, the aorta, which leads from the left chamber of the heart. The wound penetrated this vessel at a point one and a quarter inches above the point where the vessel leaves the heart. The wound in the wall of the heart-bag measured three-eighths of an inch in length, and in the blood-vessel, a quarter of an inch. These two wounds lay, with relation to one another, in a slightly slanting line, the latter just below the level of the former. The structure of the heart was normal. The lungs were healthy.

Abdominal cavity.

The organs of this cavity were examined individually, and, with the exception of the liver, which was somewhat fatty, were found healthy.

Opinion.

From the foregoing examination we are of opinion : (1) that the cause of death was wounding of the aorta, or principal artery of the body ; (2) that the wound, which penetrated the chest and injured the pericardium and aorta, was produced by a sharp-pointed,

sharp-edged instrument of at least some inches in length; and (3) that the injury must have been inflicted with some measure of force.

These are attested on soul and conscience.

(Signed)
(Qualifications)

The law and post-mortem dissections.

Post-mortem dissections are made for three principal purposes—

To obtain information about the cause of death for the purposes of the Crown authorities.

To provide instruction for medical students and doctors in the study of anatomy.

To acquire knowledge of the causes and nature of the diseases which affect the body.

The legal position with regard to the first category is clear: the doctor's authority for conducting the post-mortem examination is in England the request from the Coroner and in Scotland the Sheriff's warrant (see pp. 19 and 36).

Post-mortem dissections in the second category are regulated by the provision of the Anatomy Acts 1832 and 1871.

The relevant provisions of these Acts are that—

doctors and medical students are licensed to practise anatomy;

anyone who has the lawful possession of a body, except for the purposes of interment, may permit it to undergo anatomical examination unless to his knowledge the deceased in his lifetime expressed a wish to the contrary;

the objection of a near relative is always sufficient to stop the examination;

the person carrying out the examination must be duly licensed and he must carry out the examination at a place notified to the Minister of Health.

forty-eight hours must elapse after death before the body is removed for dissection;

notice of the intended removal must be sent to the Inspector of Anatomy for the district, and the body can be removed for the purposes of dissection only after twenty-four hours have elapsed from the giving of this notice;

the person making the dissection must send to the Inspector for the district, within twenty-four hours of removing the body for this purpose—

(*a*) the certificate of the cause of death,
(*b*) the name of the person from whom the body was received,
(*c*) the day and time when the body was received,
(*d*) the date and place of death,
(*e*) the sex, name, age, and last place of residence of the deceased;

the body must be removed in a proper coffin and the person removing it must arrange for its suitable disposal, either by burial or cremation, in a manner consistent with the religious convictions of the deceased ;

a certificate of interment of the body must be sent to the inspector within two years of the day on which the body was removed for examination ;

a medical practitioner who has been duly licensed and who makes an anatomical examination according to the provisions of the Act is not liable to any prosecution, penalty, forfeiture, or punishment for so doing (Section 14).

Lawful possession of the body usually rests in the nearest relative, but if a patient dies in a hospital then the person controlling the hospital is the lawful possessor of the body and, in the absence of objection from a near relative, he can give permission for removal of the body for anatomical dissection or post-mortem examination.[3]

The relatives have a right to forbid an anatomical dissection or a post-mortem examination, but if a patient has died in a hospital there is no obligation on the Controller of the hospital to ask for the consent of relatives and he does not even require to notify them of his intention to authorise a dissection. It is, however, customary for the person in possession of the body to obtain the consent of the relatives before authorising a dissection.

Provisions for the retention of organs of the body or skeletons are not included in the Anatomy Act.

The legal position with regard to post-mortem dissections in the third category, namely, the routine post-mortem dissections carried out in pathology departments, has now to be considered.

The present practice is that post-mortem dissections are conducted in pathology departments on the basis that these dissections are legal provided consent has been obtained from the person having possession of the body and the nearest relative.

This has been the practice for a great many years and so far as we know, it has never been questioned. There does not, however, appear to be any legal authority for this position.

In order to determine the legal position it is necessary to consider further the Anatomy Act of 1832. This Act was passed at a time when there was great public concern about the crimes which were being committed in order to procure bodies for the purpose of anatomical dissection. The case of Burke and Hare had made a very deep impression.

The Act is described as "An Act for regulating Schools of Anatomy." The preamble reads—

"Whereas a Knowledge of the Causes and Nature of sundry Diseases which affect the Body, and of the best Methods of treating and curing such Diseases, and of healing and repairing divers Wounds and Injuries to which the Human Frame is liable, cannot be acquired without the Aid of Anatomical Examination : And whereas the legal supply

> of Human Bodies for such Anatomical Examination is insufficient fully to provide the means of such Knowledge : And whereas, in order further to supply Human Bodies for such Purposes, divers great and grievous Crimes have been committed, and lately Murder, for the Single Object of selling for such Purposes the Bodies of the Persons so murdered : And whereas therefore it is highly expedient to give Protection, under certain Regulations, to the Study and Practice of Anatomy, and to prevent, as far as may be, such great and grievous Crimes and Murder as aforesaid ; be it therefore enacted by the King's Most Excellent Majesty, . . . "

Then follows the provisions which have already been given.

It will be recognised that the terms of this preamble are both wide and indefinite having regard to present-day conditions. It would appear that the legislators intended the provisions of the Act to apply not only to dissections for anatomical instruction, but also to dissections made for the investigation of disease.

In addition to the terms of the preamble, support is also given to this view by Section 15, which reads—

> " And be it enacted, that nothing in this Act contained shall be construed to extend to or to prohibit any post-mortem Examination of any Human Body required or directed to be made by any competent legal Authority."

Speller in his " Law relating to Hospitals " [4] is of the opinion that the provisions of the Anatomy Acts are also applicable to present-day routine pathological post-mortem dissections and states that, " Anatomical examination of the body of a deceased person otherwise than at the request of, or on the instructions of, the coroner is lawful only if in accordance with the Anatomy Acts."

Since 1832, there have been great developments in the study of pathology, and post-mortem dissections for pathological purposes are no longer conducted in schools of anatomy. The procedure of pathology departments has now the authority of a considerable period of uncontested usage and it would seem that post-mortem dissections conducted in this way do not require the protection of Section 14 of the Anatomy Act of 1832. The interpretation of the Anatomy Acts is however a matter for the law courts and until an interpretation relevant to this matter has been given it is impossible to state a definite opinion on the issue.

Cremation of bodies used for dissection in schools of anatomy.

Where a body which has been given for anatomical dissection is to be subsequently cremated, the clerical procedure in connection with the cremation (see p. 159) must be carried out at the time of death and the relative documents retained until the anatomical dissection has been completed.

Procedure for bequeathing a body for anatomical examination.

A person wishing to leave his body to a medical school, attached to a particular hospital, writes either to H.M. Inspector of Anatomy, Ministry of Health, Whitehall, S.W., or to the Licensed Teacher of Anatomy at the medical school which he wishes to benefit, stating his desire ; upon which the necessary forms will be sent to him.

He should then leave clear instructions in writing and inform his executors of his wishes. If no particular institution is named, the Inspector of Anatomy will send the body to the next Metropolitan School due to receive one.

When death occurs, the executor completes the form already received and posts it along with the medical certificate of the cause of death to the Inspector, who will then issue a warrant to an undertaker to remove the body and place it in the charge of some particular Licensed Teacher of Anatomy.

During the examination of the body care is taken that every particle removed from it is placed in the coffin in which it will ultimately be buried.

The burial is conducted by a clergyman of the faith which the deceased professed during life. The undertaker must present the burial certificate to the Inspector of Anatomy.

It must be understood that no individual has a right of property in his body after his death and if any near relative objects to the body being used for anatomical dissection, H.M. Inspector of Anatomy will refuse to issue a warrant for its removal to a medical school.

Reports on nature of stains.

Reports regarding the nature of stains upon clothing and upon many other articles may be required. No warrant is necessary for this examination. In Scotland, the examination is made at the request of the Procurator-Fiscal, who sends a police officer, with the articles to be examined, to the laboratory of the examiner. This officer receives from the examiner a receipt for the articles, and, in turn, a receipt is required from him when the productions are returned on completion of the examination. This is for the purpose of maintaining their identity and the sequence of events so that the chain of evidence given subsequently in court may be complete.

Work of this character should be undertaken only by experts. The report on the examination embodies such particulars as the nature of the instructions received and from whom, the date the articles were received and from whom, together with a detailed description of the articles examined and the wording on the labels attached. Further details in relation to the presence or absence of stains, their number and situation, relative to their position on the article and to one another, their shape, colour, and other physical characteristics must be included. With regard to the technique employed in the examination of the stained material, it is not necessary to enter into detail in the report. It is sufficient to

state that after an examination by chemical analysis or microscopic or spectroscopic examination, certain substances were or were not found. Then follow the opinions, the attestation "on soul and conscience," if in Scotland, and signature. The evidence of what has been found should be preserved by the examiner for subsequent production in court, should this be requested.

The following is a more or less general example of such a report :—

Place.
Date.

Acting upon instructions received from A. B., Esquire, Procurator-Fiscal, County Buildings, Glasgow, I, the undersigned, on 24th June 1949, received from C. D., Detective-Inspector, Southern Division, City of Glasgow Police, the undernoted article for examination and report thereon : A blue cloth jacket, labelled "Found in the possession of E. F., 216 Blank Street, Glasgow, and referred to in the case against E. F."

Examination.

The following reddish-coloured stains were present upon the garment :—

1. On left lapel of jacket (outer surface)—
   - (*a*) A circular, red-coloured stain, close to the junction of the neck seam and lapel, which measured a quarter of an inch in diameter.
   - (*b*) A similar stain, one and a half inches below (*a*), which measured half an inch in diameter.
   - (*c*) A triangular-shaped, smeared stain, situated close to (*b*), which measured one inch and a half by half an inch ; and
   - (*d*) Adjacent to (*c*), a group of seven small and indefinite stains.
2. On left side of jacket (inner surface)—
   - (*e*) A stain, half an inch in length, shaped like an "exclamation mark," situated three inches above the level of the top button-hole, and at the same distance from the free edge of jacket.
   - (*f*) A stain, which measured a quarter of an inch in length, of similar shape to (*e*), situated one inch above the level of the top button-hole, and two and a quarter inches from the free edge of jacket.
   - (*g*) A circular stain, situated one inch from (*f*), which measured half an inch in diameter ; and
   - (*h*) An irregularly-shaped stain, situated five inches below (*f*) and in direct line, which measured one inch at its point of greatest breadth.

Portions from the stains lettered (*a*), (*c*), (*f*), and (*h*) were excised, and after suitable preparation, were examined chemically, microscopically, and microspectroscopically. They were also examined serologically.

Opinion.

As the result of the foregoing examination I am of the opinion that—

1. Stains lettered (*a*), (*c*), (*f*), and (*h*) are composed of human blood ;
2. The "exclamation mark" stains resulted from forcible projection of the blood against the garment.

These are attested on soul and conscience.

(Signed)
(Qualifications)

Photographs are automatic records of appearance and may be used in evidence to assist oral description of the objects photographed. Medical reports used for legal purposes in Scotland must be certified "on soul and conscience" and must be sworn to in court as true reports by the maker of them. They must exclude facts elicited by the signator on hearsay.

Notes or memoranda.

Notes or memoranda may be used in a court of law by a medical witness but, if used, they may be scrutinised by cross-examining counsel for the purpose of cross-examination, since they are then treated as productions, or exhibits, in the case. It is necessary, therefore, to know under what circumstances reference to them is permissible. Such notes may be used by a witness for the purpose of refreshing his memory if :—

The notes were made at the time of, or shortly after, the occurrence of the event to which they refer, and were made by the witness, or by another and reviewed at the time by the witness.

On the other hand, they may not be used if :—

They were made only after a lapse of time following the event to which they refer, they were made by another and not supervised by the witness at the time, or they are used for the purpose of bringing forward facts which the witness has forgotten.

It must be clearly understood that the use of notes or memoranda is restricted solely to the purpose of refreshing the memory of a witness with regard to some point which may arise in the course of his evidence. Having refreshed his memory, on the point in question by reference to the notes, he must then be able to testify from his own recollection.

Dying declarations and dying depositions.

Since it may become the duty of a medical practitioner to record statements made by dying persons, which might be of great importance in the attainment of justice, it is necessary for him to know his duty in this connection.

England.

The principle on which evidence of this description is admitted is "that such declarations are made in extremity when the party is at the point of death, and when every hope of this world is gone; when every motive of falsehood is silenced, and the mind is induced by the most powerful considerations to speak the truth; a situation so solemn and so awful is considered by the law as creating an obligation equal to that which is imposed by a positive oath administered in a court of justice." [5]

In the ordinary course of events these statements are taken in the form of a dying deposition. The examination is conducted by a Justice of the Peace and the deposition is taken on oath in answer to his questions. The statement is reduced to writing, read over to the deponent, and signed by him, if he is able, and by the examiner. If the deponent cannot write, that fact and its cause should be recorded. The writing itself must be produced at the trial, and be sworn to by two witnesses, of whom one should be the examiner, as correct and as made voluntarily when the deponent was in his sound mind.

If a witness is dying and there is no time to arrange for administration of the oath and a formal deposition being taken, his evidence may be written down as a declaration by any creditable person.

The conditions necessary for the acceptance of dying declarations and dying depositions as valid evidence are :—

1. The case must be a criminal one.
2. The case must be one of homicide.
3. The death of the deceased must be the subject of the charge, and the circumstances of the death the subject of the declaration or deposition.
4. The words used must be those of the deceased and if questions have been put these must be given together with the answers.
5. The person making the statement must be actually dying, must believe that he is dying, and must express no hope of recovery. It is not necessary that he should have stated his expectation of immediate death, if this can be inferred from other circumstances.
6. The person who made the statement must be dead.

The use of depositions as evidence is also controlled by statute. The Criminal Law Amendment Act, 1867, makes provision, in the case of indictable offences, for the taking of depositions out of court where information is likely to be lost owing to the probable death or continuing illness of a witness. If the deponent dies, or is unlikely ever to be able to give evidence in court, then the deposition is acceptable as evidence.

Scotland.

The law of Scotland gives a wider validity to dying declarations and depositions than the law of England.

It holds that a dying deposition or a dying declaration of a person who subsequently dies is admissible as evidence whether that person were the party injured or not, if in life he would have been a competent witness.

The circumstances necessary for this type of evidence to be valid in Scotland therefore are :—

1. The declarant or deponent must be a competent witness in a criminal trial.
2. He must be a material witness.
3. His life must be in danger.
4. His mind must be sufficiently clear to give reliable evidence.

It should be noted that there does not appear to be any legal obligation on a medical attendant to record the declaration of a dying man, but as he is often the only person present who is capable of undertaking the duty, he should in the interests of justice do so in an emergency. Where there is time, it is his duty to ensure that notice is given to the legal authorities so that in Scotland a Sheriff, or in England, a Justice of the Peace, may attend and take the statement in the form of a dying deposition.

ORAL EVIDENCE

Oath and affirmation.

Whatever form medical evidence may assume, it must ultimately take the form of oral evidence, which can only be valid in court when given upon oath or affirmation. In Scotland, the oath is administered to the witness by the judge, who, standing in his place and speaking the words of the oath, requests the witness to repeat the words after him. This is done clause by clause. The words of the oath in Scotland are as follows :—" I swear by Almighty God, as I shall answer to God at the great Day of Judgment, that I will tell the truth, the whole truth, and nothing but the truth."

Certain judges omit the words " as I shall answer to God at the great Day of Judgment." The words are repeated by the witness while holding up his right hand.

In England, the witness is sworn by an officer of the court, by repeating the following words :—" I swear by Almighty God that the evidence I shall give to the court touching the matters in question shall be the truth, the whole truth, and nothing but the truth."

In the Coroner's Court the oath taken is :—" I swear by Almighty God, that the evidence I shall give to this inquest on behalf of our Sovereign Lord the King, touching the death of . . . shall be the truth, the whole truth, and nothing but the truth."

In regard to affirmation, by virtue of the Oaths Act, 1888, every person upon objecting to being sworn, and stating as the reason either that he has no religious belief, or that the taking of an oath is contrary to his religious belief, shall be permitted to make his solemn affirmation instead of taking an oath in all places, and for all

purposes where an oath is or shall be required by law. This affirmation has the same legal force and effect as the oath. The form of an affirmation is as follows :—" I, A. B., do solemnly, sincerely, and truly declare and affirm," and then follow the words of the oath prescribed by law, omitting any word of imprecation.

Medical evidence.

In giving evidence there are certain principles which should never be forgotten by the medical witness. The language used in the witness-box should be clear, concise, and as untechnical as possible. Such terms as " syncope," " comatose," " highly vascular," " œdematous," and others should not be used. It is impossible to expect a jury to know what is meant by the terms " pericardium." " meninges," and " calvarium," but the substitution of " heart-bag," " brain-coverings," and " skullcap," or " vault of skull," will make matters clear.

The language should be concise. Adjectives of degree, especially superlatives, should be used sparingly, and only when absolutely necessary, since their use may be regarded as biased opinion, and this discounts the value of the evidence. The voluble witness is often a godsend to opposing counsel with a weak case, since the witness saying more than is required is apt to say more than he means, and in doing so increases his vulnerability while under cross-examination. Categorical answers, when possible, are the best, and when not possible, answers should be concise and clear.

The replies of a witness should invariably be courteous. This is not difficult during examination-in-chief, since both witness and examiner are in accord, but it frequently becomes less easy during cross-examination, when the object of the cross-examiner is to weaken or, if possible, negative the evidence given in the previous examination. However trying the situation may be, the witness should keep the fact clearly before him that he is giving expression to honest opinion, and that he has but consistently to hold by what he has formerly said, and to give fully the reasons for his belief, to convince the court of his sincerity. It is usually with reference to opinions or inferences that differences between counsel and witness arise.

It may be of assistance to the witness under cross-examination to bear in mind that it is the business of the cross-examiner to make the best case he can for his client. Calm but persistent restatement of former evidence will sooner or later break down even the most pressing cross-examiner, and a witness may rely upon the judge interfering when he considers that counsel is overstepping the bounds of legitimate cross-examination. In short, if the witness can preserve himself free of the assumption that cross-examining counsel is his natural enemy, and if he does not, therefore, assume the mental attitude appropriate to that view, he will leave the witness-box, if otherwise he has been well prepared, with credit. There are occasions, however, upon which it is absolutely necessary for a medical witness to maintain a very firm

attitude, and to decline strongly to have words attributed to him which have not been stated. Occasionally cross-examining counsel may ask a question, which is based upon a statement which he desires the witness to understand he has already made in reply to a previous question, the answer to which tends to put an entirely different complexion upon the tenor of his evidence. If the witness is collected he will detect the misstatement and at once challenge it. Should counsel persist in stating that the witness made the statement, the witness should appeal to the judge who, by having reference made to the notes of the shorthand writer, in court, will quickly settle the issue.

Evidence should always be given distinctly, deliberately, and audibly. It has often been said by judges that no witnesses are so difficult to be heard and to be understood as medical witnesses: difficult to be heard from want of clearness in articulation, and difficult to be understood by reason of the nature of the evidence.

The general rule is that ordinary lay witnesses must speak only to matters of fact and not of opinion. A medical witness, however, usually speaks both to matters of fact and of opinion, and therefore is regarded by the court as an expert witness. Under certain circumstances he may only be called upon to give evidence on matters of opinion and not of fact. A skilled witness should not be asked to give an expression of opinion which is a direct answer to the issue under trial since this is a matter to be decided by the judge or jury as the case may be. As a general rule, subject to certain exceptions, hearsay evidence is not valid evidence, and a witness, therefore, must speak only to facts which come within his own personal knowledge. Any opinion which he expresses from a given series of facts must be his own opinion. The published opinions of writers on the subject may be adopted by skilled witnesses and made a part of their evidence. A skilled witness may be taken as concurring in his evidence with the opinion expressed in full by a preceding skilled witness. An expert may speak to the recognised authority of a particular writer, and may adopt as his own opinions, specified parts of a published work. The passage is then regarded as being supported by the weight of the author as well as of the witness. The published work need not be produced in court, and it does not matter whether the author is living or dead. This is not an unimportant point since, quite frequently, cross-examining counsel will quote certain passages from standard books, with the intention of rebutting evidence given by a witness in his examination-in-chief, and ask the witness whether he agrees or disagrees with opinions of the author. In such a case a witness is well advised never to offer any reply until he has been permitted to read for himself the quotation as given in the book together with the context.

Any statement which a witness has made on precognition, but which he has modified in the witness-box, cannot be challenged on the ground that he made a different statement on a former occasion, although this does not apply to a deposition in England (see pp. 15, 20, and 22).

Members of the medical profession should understand clearly that evidence given by them is absolutely privileged and that no witness can be compelled to answer any question which might have the effect of incriminating him, or which would tend to degrade his character socially or professionally. To this general rule there is an exception. By the Criminal Evidence Act, 1898, a person charged, and tendering evidence on his own behalf, may be asked any question in cross-examination notwithstanding that the answer might incriminate him in relation to the offence charged.

In a trial for rape a medical witness was asked, in cross-examination, if he had been in the way of having connection with the woman said to have been ravished. He hesitated for some time whether to answer or not, when it occurred to him to ask if he must answer the question, and on being assured that he need not do so unless he chose, he said, "Then I refuse to answer."

Certain principles should always be remembered by medical witnesses. These are:

Study the case, and be conversant with the facts, and the literature on the subject.
Always have adequate reasons for opinions.
Be fair and unbiased, concede points which should be conceded.
Answer "I do not know" when you do not know.
Never express an opinion on the merits of the case. That is the function of the judge or jury as the case may be.
Do not "sit on the fence." A doctor who will not commit himself to his opinion is not worth calling as a witness.

If a medical witness has made a thorough examination, is conversant with his subject, has made an accurate report on his findings, is truthful, unbiased, remains composed, and is fair in all his opinions, his integrity and professional reputation will remain untarnished, even under the most exacting cross-examination.

PROFESSIONAL SECRECY AND PRIVILEGE

From the days of Hippocrates until the present, members of the medical profession have bound themselves not to divulge professional secrets.

The declaration taken by medical graduates of the University of Glasgow is:—"I do solemnly and sincerely declare that, as a Graduate in Medicine of the University of Glasgow, I will exercise the several parts of my profession, to the best of my knowledge and abilities, for the good, safety, and welfare of all persons committing themselves, or committed to my care and directions; and that I will not knowingly or intentionally do anything or administer anything to them to their hurt or prejudice for any consideration or from any motive whatever. And I further declare that I will keep silence as to anything I have seen or heard while visiting the sick which it would be improper to divulge. And I make this solemn declaration in virtue of the Provisions of the Promissory

Oaths Act, 1868, substituting a Declaration for Oaths in certain cases."

Lord Riddell [6] sums up the legal position of doctors, in relation to professional secrecy, both tersely and informatively in this way :—

"A doctor being in a fiduciary capacity must preserve his patient's confidences unless relieved from the obligation by some lawful excuse, for example, legal compulsion, the patient's consent, the performance of a moral or social duty, or protection of the doctor's interests. A doctor shares with other citizens the duty to assist in the detection and arrest of a person who has committed a serious crime. Everyone recognises the necessity and importance of medical confidences. Everyone recognises that they are sacred and precious. But we must recognise also that the rules regarding them exist for the welfare of the community, and not for the aggrandisement or convenience of a particular class. We must recognise also that they must be modified to meet the inevitable changes that occur in the necessities of various generations."

Legal decisions have shown that secrecy is an essential condition of the contract between a medical man and his employers, and breach of secrecy affords a relevant ground for an action of damages. This, therefore, may be taken as a general expression of the law.

In Britain, however, it is the law that secrets of patients obtained by doctors in the course of their profession cannot be withheld from a court of law if their divulgence is necessary for the purposes of the law.

The question of professional secrecy, therefore, presents two different aspects, namely, the ethical aspect and the legal aspect. It seems clear that a medical witness, in court, cannot claim any privilege and that he must disclose, when a judge so rules, any secret information which he may have obtained in the course of his professional relationship to a party involved in any case. Medical practitioners must therefore reckon upon their liability to be called upon to make such disclosures when necessary. At the same time, a medical witness should always be reluctant, in a court of law, to disclose such secrets, and he should ask for a ruling of the court before he makes such a disclosure. Under certain circumstances, permission may be given the witness to write his answers to certain questions, thus obviating divulgence in public. Such action will, at least, convince the public that the medical profession guards that which has been committed to its members as confidential, and that it is most reluctant to divulge unless compelled by law to do so.

The observations of Mr Justice Horridge, in the Divorce Court in 1921, regarding the reluctance of a medical witness to give evidence with regard to venereal disease on the ground that he, with other medical men, had undertaken these duties at a clinic on the distinct understanding that professional secrecy would be observed, reaffirmed the claim of courts of law to compel the disclosure of professional confidences. In the same year, also in the Divorce Court, a similar point was raised before Lord Mersey, who said that there was no statement in the Acts relating to venereal diseases

to the effect that the information was confidential, and that he did not consider the production of a card would be against public policy.

In a recent case [7] a wife developed venereal disease after a short period of marriage. She and her husband went to a clinic, where the former was diagnosed, but the latter was found free of disease. The husband instituted divorce proceedings and his wife requested the doctor to furnish her solicitor with particulars of her illness and the considered date of its commencement. The doctor stated that the disease was secondary syphilis but withheld information required to defend divorce proceedings. Subsequently, both parties and their solicitors agreed to send questions to the doctor with a request for answers. The importance of this step lay in the fact that if they had been answered in a given direction the husband would have been unable to prove his case. The doctor refused the information required, but intimated his willingness to give the information as evidence in court. This he did later. His Lordship ruled that it was of the greatest importance that proper secrecy should be observed in connection with venereal disease clinics and that nothing should be done to diminish the confidential relationship existing between doctor and patient. These considerations, however, did not justify a doctor in refusing to divulge confidential information to a patient, or to any named person, or persons, when asked by the patient to do so. The information should have been given, and in such circumstances the doctor is not guilty of any breach of confidence.

The case of St Clair Gray v. The Northern Accident Insurance Company in the Scottish law courts shows another aspect of the question. The pursuer alleged that he had accidentally cut his finger while operating, and that later, while attending another patient, was infected specifically, in consequence of which he was unable to work for some time. He held a policy against accident in the defendant's company, and made a claim in the Sheriff Court, which the defendants resisted. After hearing evidence, in which he declined to reveal the name of the patient by whom he was infected, the Sheriff-Substitute awarded him damages. This judgment was appealed to the Sheriff-Principal, who reversed the decision on the ground that the pursuer had failed to prove that the disease was communicated as stated by him, or was the result of an accidental injury, within the meaning of the policy. The pursuer appealed against this decision to the Court of Session, which affirmed the decision of the Sheriff-Principal. The Lord Justice-Clerk, who gave leading judgment, said that there was no reason to doubt the bona fides of the pursuer, who had acted in an honourable manner in not disclosing the name of the patient whom he was attending. While that was so, however, his lordship was bound to conclude that the pursuer had not proved his case.

The general question of professional secrecy in relation to medical witnesses was fully commented on by the judge in the case of Kitson v. Playfair. He stated that the medical profession had no right to

legislate on the matter. They might make their own rules, for their guidance as professional men, but they could not impose upon the public their self-made laws. Although the judge was the person who had to rule whether or not a witness was to answer a question, he would exercise discretion in ordering a witness to answer or not. There might be some matters which the judge thought most unreasonable to be divulged by a professional man, and he might refuse to permit it, and allow the witness to say, " I refuse to answer." Each case had been considered by its own particular circumstances and by the ruling of the judge who happened to preside on the occasion. The judge would decide according to law. There was always a rule to set a judge right if he went wrong.

When it was said that there was a general rule existing in the medical profession, that when they saw, in the course of their medical attendance, that a crime had been committed, or was about to be committed, they were in all cases to go off to the Public Prosecutor, he was bound to say that it was not a rule which met with his approbation, and he hoped it would not meet with the approbation of anybody else (see p. 387). There might be cases when it was the obvious duty of a medical man to speak out, for instance, in cases of murder. A man might come with a wound which it might be supposed had been inflicted in the course of a deadly scuffle. It would be a monstrous thing if the medical man screened him, and tried to hide the wound which might be the means of connecting the man with a serious crime. That was a different thing altogether. Communications between a doctor and his wife, or children, were said to be privileged when it was necessary to reveal them in order that the wife or children might be protected. He thought that that required a great deal of limitation, because cases might be imagined where the wife might be living under circumstances in which she did not want any such protection at all, and giving to her a secret belonging to a patient would be only a wanton violation of the rule. This was a very delicate question.

The opinion expressed by Lord Justice-Clerk Inglis, regarding the conduct of Dr Paterson, one of the medical witnesses in the Pritchard poisoning case, is important to practitioners. Dr Paterson, a witness for the Crown, stated in the witness-box that when called to see Mrs Pritchard for the first time, he had formed the opinion that she was under the influence of antimony. His lordship said that Dr Paterson was under the decided impression, when he saw Mrs Pritchard on these occasions, that somebody was practising upon her with poison. He thought it consistent with his professional duty, and his duty as a citizen of this country, to keep that opinion to himself. His lordship could not say that Dr Paterson had done right, and he would be sorry to lead the jury to think so. He cared not for professional etiquette or professional rule. There was a rule of life and a consideration that was far higher than these, the duty of every citizen of this country which was owed by every right-minded man to his neighbour, to prevent the destruction of human

life in this world. In that duty he could only say that Dr Paterson had failed.

It should be remembered that a medical practitioner is not under any legal obligation to divulge any information to the police, when asked for a statement, if such conflicts with the principles of professional secrecy.

Privileged statements.

Since this subject is important to members of the medical profession, and is intimately associated with that of professional secrecy, it should next receive consideration. A privileged communication can be defined, in a general manner, as a communication made bona fide upon any subject-matter in which the party communicating has an interest, or in reference to which he has a duty, if made to a person having a corresponding interest or duty, although it contain incriminatory matter which, without the privilege, would be slanderous or actionable.

While the question as to whether or not a statement is privileged must be answered on the merits of each particular case, the summing-up of the judge in that of Kitson v. Playfair deals comprehensively with many of the points which determine privilege. His lordship in his summing-up defined a statement of privilege in the following terms :—"The rule was, that if a man made a charge derogatory to the character of another, if he could show that it was made upon an occasion upon which he was privileged in speaking out, or writing the words if they were contained in a letter, and that he made it honestly and bona fide, and merely in the discharge of a duty which he felt was incumbent upon him, without any sinister motive and without any ill-feeling, it might happen that he would be entitled to say words, even though they did great injury, under the plea of privilege.

"The plea of privilege could not prevail if a man did not really make the communication under a sense of duty and obligation cast upon him to make it. If he did it for the mere purpose of doing a bad turn to a man, or getting gain for himself, or putting pressure on a man he had no right to put, or in a spirit of irritation or anger, or for anything of that sort, it was not privileged." The questions which the judge put to the jury for answer were :—(1) Were the words uttered as alleged ? (2) Did the defendant believe the words to be true ? (3) Were they uttered in good faith and without malice ? (4) Were they uttered not from a mere sense of duty, but from some indirect motive, or to gratify feelings of ill-will, anger, or irritation against the plaintiff or her husband ?

When privilege is claimed, the facts alone must determine the issue.

It would appear that a doctor must not disclose to an insurance company information as to the cause of death of a deceased person without the consent of the nearest surviving relative. A request for such information may be made by a company which has accepted

an insurance without examination. It is open to the company to refer the matter to the Industrial Insurance Commissioner, and then the family practitioner will be requested to make an affidavit for the consideration of the court over which the Commissioner presides. This avoids attendance at this court and the statement, so made, is protected by privilege since the proceedings are quasi-judicial.

Another important case, involving the subject of privilege, was that against the late Sir Patrick Heron Watson for damages for alleged slander.

The question at issue was : Does the privilege which protects a witness from an action of slander in respect of his pertinent evidence in the witness-box also protect him against the consequences of statements made to a solicitor and counsel in preparing the proof for trial ?

This case decided that the preliminary examination of a witness by a solicitor was within the same privilege as that which he would have if he had said the same thing in his sworn testimony in court.

From what has been said with regard to professional seerecy and privileged statements, the importance of medical practitioners being members of a Medical Defence Society, which will represent them when legal difficulties arise in the course of their professional relationships, becomes clearly established.

Presence of medical witnesses in court.

In England.

It is usual for medical witnesses to be present in court, and they may hear the evidence of one another.

In Scotland.

Except by arrangement, medical witnesses are not allowed to be present in court when medieal evidence is being given by others, unless the witnesses have already given their evidence when, thereafter, they may remain in court. Expert witnesses who are to speak to matters of opinion may by agreement of counsel remain in court while witnesses are deponing to matters of fact, but they are not permitted to remain while expert witnesses are giving evidence on matters of opinion.

Unless special permission is granted, medical witnesses for the defence are not allowed to remain in court when medical witnesses for the Crown are giving evidence.

Attitude of medical examiner towards examination of assaulted, accused, and other persons.

In the first place, permission for the examination must be obtained from the person who is to be examined. With regard to persons under the age of sixteen, permission must be obtained from a parent or guardian, since those under that age are not sufficiently

adult to give permission. In exceptional instances, however, for example, a young person under sixteen who has no parent, relative, or guardian, the permission of such young person would be regarded as acceptable. In all cases the permission for examination must be freely given, since there is no power which can compel a person to submit his or her body for examination against consent. An assaulted person, by laying a charge, is presumed to be willing to afford all evidence, even to examination of the body: nevertheless, consent must be obtained. It is advisable, when the examination is to be uncorroborated, to have a suitable person in attendance when consent to the examination is given, and while the examination is proceeding. The object of the examination should first be explained, and the examinee informed that the findings will be embodied in a medical report.

When accused persons have to be examined, the question of obtaining necessary consent is a matter of extreme importance, since the possible consequences might be the discovery of facts which would be used against the accused person at the trial. It is not sufficient that the person to be examined offers no resistance or objection, for that may result solely from ignorance of his or her rights. The proper procedure is to inform the accused, in the presence of a third party, that the examiner has been asked by the authorities to make an examination, but that he can only do so after obtaining consent, which can be withheld, and that he will report the results of the examination whatever they may be. Should consent be refused, the duty of the examiner is to report this fact.

Should consent be given, the examination of either an assaulted or accused person will be directed along the lines of the specific investigation necessary in the circumstances of the individual case. Few, if any, questions should be asked, and thoughts should not be expressed aloud. All relevant findings should be carefully noted and subsequently embodied in the report. Judgment should not be biased by any statements made, and opinions should be based solely upon the results of the examination. Statements made by an accused person should not be embodied in the report.

The legal consequences of want of knowledge of, or inattention to, the necessity of obtaining consent may be serious. In a case tried at Manchester, where a woman was accused of abandoning her infant a few hours after its birth, the judge said in his summing-up of the case "that no medical man may suppose himself armed with authority to proceed contrary to the express will of the person he is instructed to examine." In another case, a doctor, acting on the verbal request of the Coroner and an inspector of police, proceeded to examine a female to ascertain whether or not she had recently been delivered of a child. The woman refused to be examined and offered to send for a medical man whom she knew to make the examination, but the doctor proceeded to examine her. The jury returned a verdict for the plaintiff, and assessed damages at £200.

The attitude of a doctor in relation to the conduct of certain other medical examinations forms another angle of this subject.

Medical practitioners are sometimes asked by ladies to examine their domestic servants whom they suspect of being pregnant. From a desire to oblige, doctors have unthinkingly made the required examination, have informed the mistress of the result of the examination as confirmatory of her suspicion, and the servant, in consequence, has been dismissed. Such an action is contrary to law since, consent not having been expressly given, a technical assault has been committed and professional secrecy violated. A doctor who has acted in this manner may very probably be involved in an action for damages. The proper course to pursue, under such circumstances, is to advise the employer to tell the servant of her suspicion, and to ask her to consult a medical man of her own choosing, or to offer the girl the services of the family doctor. A medical man should never proceed to examine a girl surreptitiously for such a purpose by making pretence to examine her for some non-existent condition, when his sole intention is to discover whether or not she is pregnant. When a medical practitioner is asked, by mistress or employer, to examine a servant or employee, he must proceed with caution and, having explained the object of his examination, must receive full consent to the examination.

References

1. "The Post-Mortem Examination," Sidney Farber. Charles C. Thomas, Baltimore, 1937. "Post-Mortem Manual," Box. J. & A. Churchill, London, 1919, and other works.
2. Lancet, Vol. I, 21, 1935, Maile and Scott.
3. R. v. Feist (1858), Dears and Bell 590.
4. "Law Relating to Hospitals and Kindred Institutions," S. R. Speller. H. K. Lewis & Co. Ltd., London, 1947.
5. R. v. Woodcock (1789), 1 Leach, 504.
6. "Medico-Legal Problems," Lord Riddell. H. K. Lewis & Co. Ltd., London, 1929.
7. Med. Annual, 155, 1947, and C. v. C. (1946), 1 All E. R., 562. Also referred to, "Law of Evidence in Scotland." W. J. Lewis. Wm. Hodge, Edinburgh and Glasgow, 1925.

CHAPTER III

IDENTIFICATION

THE question of personal identity frequently arises in the law courts not only in the identification of criminals but also in the identification of other persons and dead bodies.

The detection of criminals by identification falls essentially within the province of the police, but in matters pertaining to identification of the dead, and in problems touching upon identification generally, the services of the medico-legist are likely to prove important.

Identification of criminals.

The establishment of the identity of criminals will first be considered.

Anthropometry or Bertillon's system.

This system of identification is employed on the Continent and chiefly in France.

In addition to descriptive data of prisoners, the Bertillon system depends on exact measurements of the body. These measurements are recorded on cards which are retained in specially arranged cabinets in the Bureau of Identification. The following are the essential measurements :—

Height when standing.
Span of outstretched arms.
Height when sitting.
Length of head.
Breadth of head.
Length of right ear.
Breadth of face between outer surfaces of the zygomatic arches.
Length of left foot.
Length of left middle finger.
Length of left little finger.
Length of left forearm and hand to tip of the middle finger.

The five measurements which constitute the basis of primary classification are :—

Length of head.
Breadth of head.
Length of left middle finger.
Length of left foot.
Length of left forearm and hand to tip of middle finger.

These are selected because they are the most constant in each adult person, are the most varied in different persons, are the least

correlated to one another, and are the most simple for accurate measurement.

Ancillary to his main system, Bertillon developed a method for the scientific indexing and filing of the descriptions of certain facial characteristics of criminals. This was called " portrait parlé " and laid the foundation for the modern descriptive indices of convicted criminals in use throughout the world to-day.

For methods employed in the classification of the measurements the reader may consult the work referred to.[1] The chief disadvantage of the system lies in possible inaccuracy when taking the measurements.

Dactylography or finger-print identification.

The method of identifying criminals by means of their digital or palmar prints is used universally and can be divided into two main branches :—

1. The filing of criminals' finger-prints for criminal records ;
2. The searching and identification of chance impressions from scenes of crime.

The papillary ridges which cover the inner surfaces of the hands sometimes run in " parallel " curves, and in certain areas, such as the bulbs of the fingers and parts of the palms, they form various patterns. It is because of the occurrence of these patterns that the essential features of the system are impressions which are taken from the bulbs of the fingers and thumbs. It is a simple matter to record the impressions on paper by means of printer's ink, thus permitting of easy examination by means of a hand lens, in order that the pattern may be classified for search and filing purposes.

On the official finger-print form, the imprints of the fingers and thumbs are taken in two different ways, and from the method used are called " rolled " and " plain " impressions. First, the rolled impressions are taken by rolling each digit from one side to the other. The resulting print ensures, as far as possible, the recording of all the detail necessary in classification. Plain impressions are taken of all ten fingers for the purpose of checking that the rolled impressions have been taken in the proper order. These plain impressions are taken by simply placing the inked fingers and thumbs directly on to the form, without any rolling movement.

Galton was the first to devise a system of classification of the finger-print patterns, and along with Henry [2] classified all these patterns into four main types, Arches, Loops, Whorls, and Composites (see Fig. 2). In the Arch, the ridges pass from one side of the finger to the other without making any backward turn, and have no delta. In the Loop, one or more of the ridges make a backward turn, but without twist, and, since the downward slope may tend towards the ulnar or radial side of the finger, loops are designated ulnar or radial. There is one delta. In Whorls, some of the ridges make a turn through at least one complete circuit. In Composites, are included patterns in which combinations of the

Arch, Loop, and Whorl are found in the same print. Whorls and Composites have two deltas. Of the four main types, that of the Loop predominates.

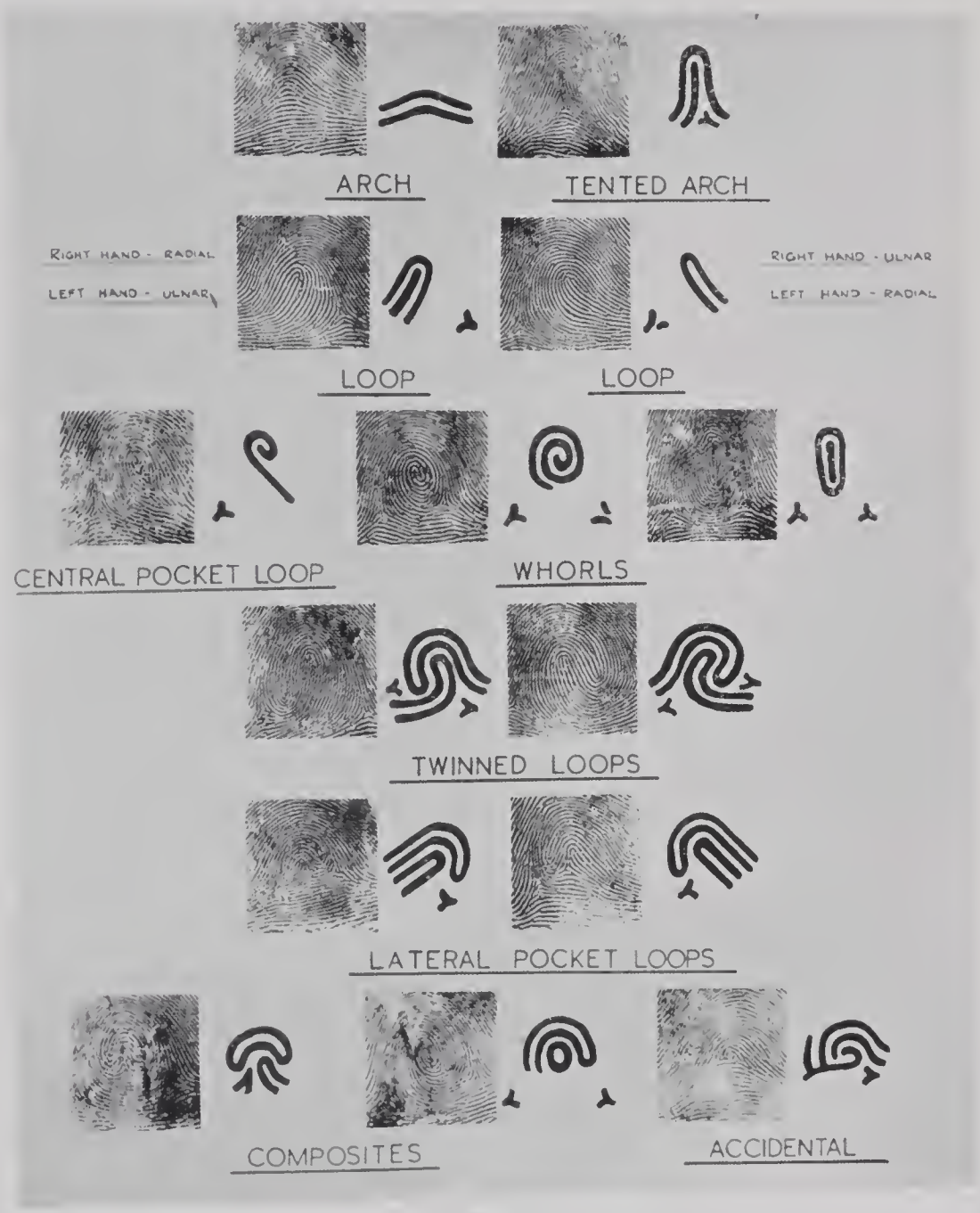


FIG. 2

Principal patterns, formed by papillary ridges, used in finger-print classification. Cores and deltas are shown in diagrammatic form.

(By courtesy of the City of Glasgow Police.)

Various systems for the classification and filing of finger-prints exist, many of these systems being based on the Henry method. It is felt, however, that the classification of finger-prints is a matter for finger-print experts, and details in this connection are omitted. It is interesting to note, however, that even in large collections,

like that at New Scotland Yard, where over a million sets are filed, the system is so perfect that when the finger-prints of a person have been recorded and classified, their location is readily obtained for comparison with the same individual's finger-prints, should these be taken by the police on some future occasion, irrespective of what name the person may take. The system has also been adapted to permit of the transmission of finger-prints for identification purposes by means of wireless or of code telegraph. Battley [3] has devised a practical method for classifying single finger-prints in order that they may be produced readily for comparison with chance impressions found at the scene of a crime (see Fig. 4).

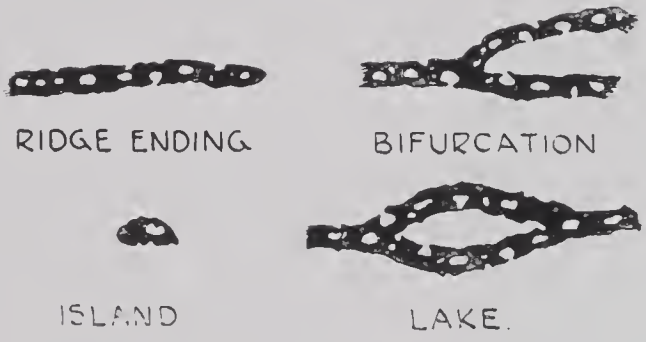


Fig. 3

Ridge characteristics (Galton details).

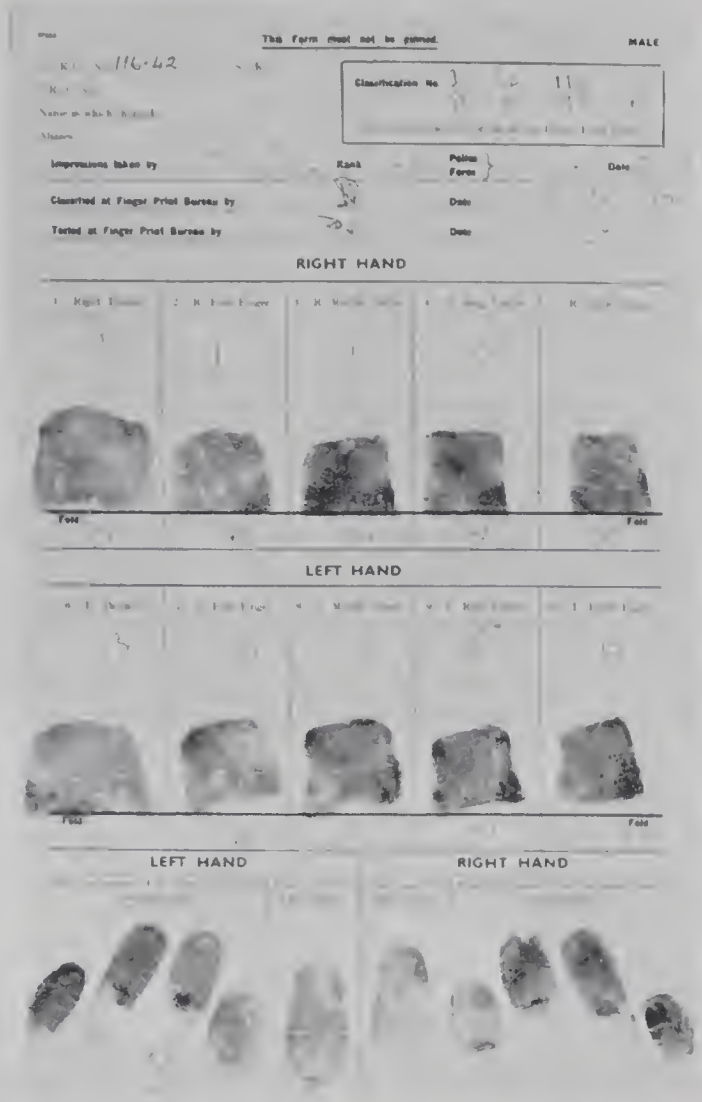
The results of this system over a considerable period have been most satisfactory.

Prints from the soles of the feet are as reliably identifiable as finger or palm prints, and many Eastern countries utilise this method of identification in relation to chance foot-prints found at the scene of crime.

The final identification of any finger or palm print is not made by comparison of patterns, but by comparison of the numerous details or characteristics which occur throughout the ridge areas, and of the sequence in which these characteristics occur. The characteristics of a print, known originally as the "Galton details," may take the form of ridge endings, bifurcations, lake formations, or island formations (see Fig. 3).

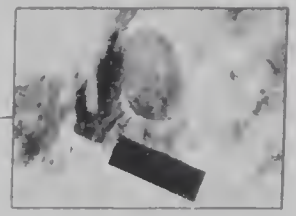
The accuracy of the finger-print and palm-print system of identification is based on the facts—

1. that the patterns and characteristics are present and constant from before birth until decomposition after death, remaining the same except for accidental damage as long as the body survives ;
2. that no two separate finger or palm prints have exactly the same arrangement and formation of papillary ridges ;
3. that in practical experience throughout the world, no two finger or palm prints have been found which were identical in the sequence of their ridge details ; and
4. that mathematical calculations indicate the extreme improbability of chance duplication.

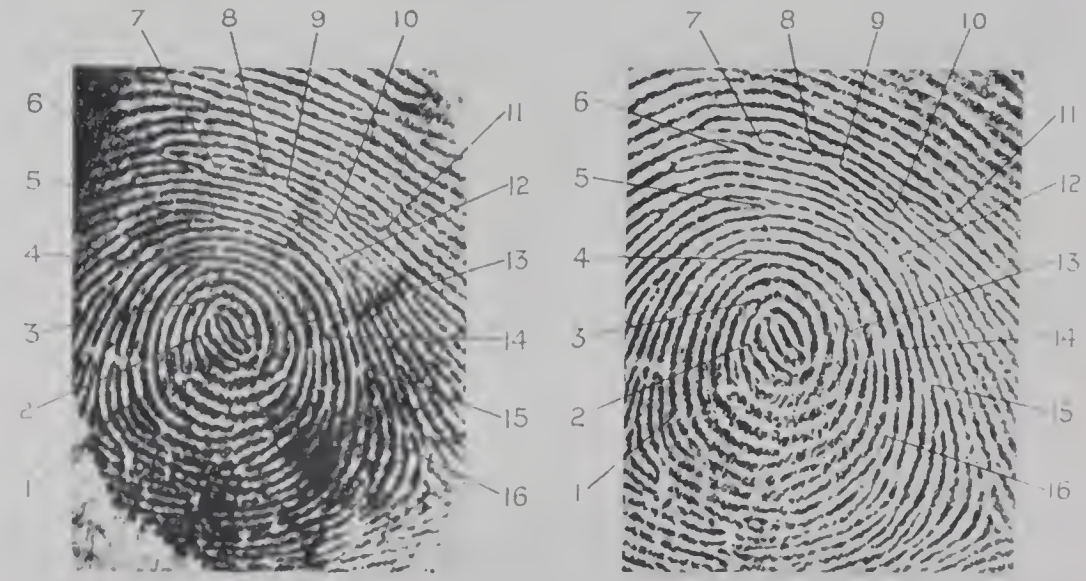


1

2



3



4

FIG. 4

(By courtesy of the City of Glasgow Police.)

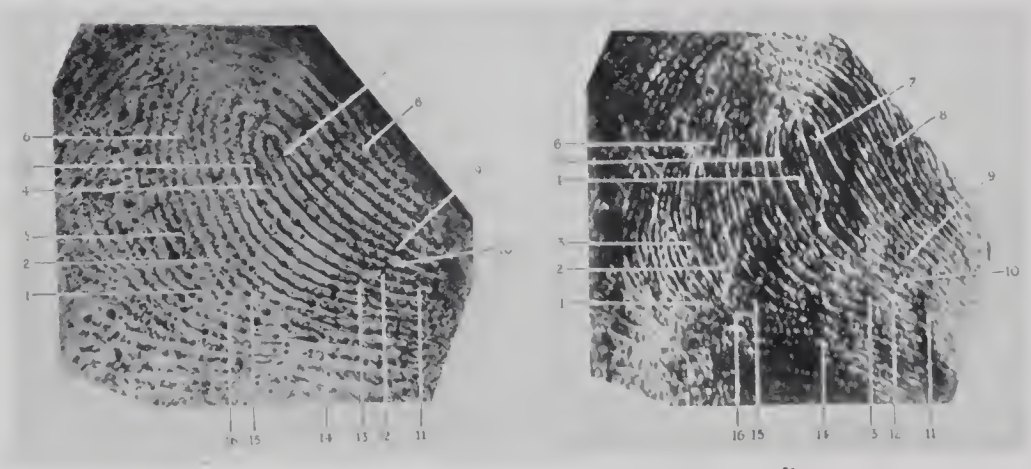
For legal purposes, it is generally accepted that the chances of two finger-prints disclosing sixteen ridge characteristics which are identical and occur in the same sequence are less than one in ten thousand million million. To prove the identity of a single finger-print in court it is necessary to have sixteen ridge characteristics identical and occurring in the same sequence as sixteen ridge characteristics in the comparison print.

Prior to the Criminal Justice Act, the procedure in England with regard to apprehended persons was that, if the person did not object to his finger-prints being taken, they could be taken by the officer arresting or by any other police officer after the person had been charged. When the person refused to have his finger-prints taken, he was remanded in custody and sent to prison where, under the Penal Servitude Act, 1891, his finger-prints could be taken on a warrant granted by a magistrate. The Criminal Justice Act, 1948, enacts that where any person not less than fourteen years of age who has been taken into custody is charged with an offence before a Court of Summary Jurisdiction, the court may, on the application of an officer of police not below the rank of inspector, order that the finger-prints of that person shall be taken by a constable. Finger-prints taken in pursuance of such an order are taken either at the court or, if the person is remanded in custody, at any place to which he is committed. A constable may use such reasonable force as may be necessary for that purpose. Where finger-prints of any person have been so taken, if the person is acquitted or discharged under Section 25 of the Indictable Offences Act, 1848, or if the information against him is dismissed, the finger-prints and all copies and records of them must be destroyed.

In Scotland, at common law, the police may competently take finger-prints of arrested persons who are in police custody without the consent of such persons and without a warrant. Without consent, finger-prints of a person who has not been arrested may not be taken by the police, and they may not be taken without consent in the case of a person who is not in police custody, for example, a person who has been arrested but has subsequently been released on bail.

On 2nd June 1947, a dwelling house in the city was entered by means of forcing the vestibule door with bodily pressure and a quantity of linen and jewellery was stolen. Search was made for finger-prints, and an impression of a right thumb of the whorl type was found inside the lid of a writing bureau in the sitting-room (2). This was photographed (3) and compared with the fingerprints of all persons having legitimate access to the bureau, with a negative result. The impression was then classified and searched in the Single Finger Print Collection. It was identified as the right thumb-print of ——, GCRO. 116/42. This man was arrested, charged with the housebreaking, and finger-printed (1). He intimated a plea of "Not Guilty," and productions were prepared for court. Enlarged photographs of the digital mark on the writing bureau and the right thumb-print on the finger-print form were prepared. On each of these, sixteen identical ridge characteristics occurring in the same sequence were marked (4). Accused was convicted and sentenced to two months' imprisonment.

Since the doctor and the police investigator have to work frequently in close co-operation in the elucidation of certain cases, it is highly important that the former should realise the necessity for the adoption of strict precautions to prevent any unwitting interference with finger-prints left by a criminal at the scene of crime. He should therefore refrain from handling or touching any object until the finger-print expert has completed his examination. The prints left at the scene of crime may be visible or almost invisible. The latter may be developed artificially by dusting the area, by means of a camel's-hair brush, with light or dark coloured powders, depending on whether the surface on which the latent print is situated is dark or light, the object being to provide a contrast. Following development the prints may be photographed. Latent prints may also be examined by oblique lighting.



A *B*

FIG. 5

Comparison of finger-print on bottle in cellar, No. 2 Dalton Square (*A*), with photograph of dermis of right thumb of Body No. 1 (*B*). Sixteen points of agreement are marked (see Ruxton Case, p. 99).

Of importance to those who are attempting the identification of a dead body is the fact that even after the epidermal covering layer of the finger-tips has been shed, as the result of putrefaction, the characteristics of the exposed surfaces of the dermis are identical with those of the voided epidermis, since the ridges of the papillary layer of the dermis or true skin are the primary cause of the ridge pattern of the epidermis. Dermal impressions may lack clear definition, and it may be necessary to employ enlargements of direct photographs of the dermis. Many difficulties may arise when an attempt is made to obtain direct impressions. When the print bearing surfaces are either sodden or hardened, modification of the ordinary ink method of recording prints must be used to meet individual circumstances. Hardened skin may be rendered pliant by the use of oleaginous preparations such as oil or vaseline which should be massaged into the tissues. Excess must be removed. Injection of the print-bearing portions of the fingers with special paraffin-wax may be resorted to in an attempt to restore contour,

In one case of drowning, the terminal phalanx of a thumb was removed and boiled in water, with marked success, in that the process, although reducing the size of the specimen, rendered the skin almost normal in character. When the shed epidermis is available, it should be preserved carefully between two sheets of glass for subsequent examination by a finger-print expert. Successful photographs of the print may be obtained by photographing from the back of the glass by means of transmitted light. The identification of dead bodies by means of finger-prints is, of course, only possible when there are prints available for comparison purposes.[4]

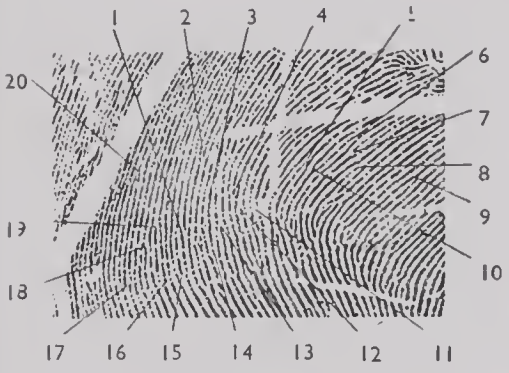


FIG. 6

Palmar impression left at scene of crime.

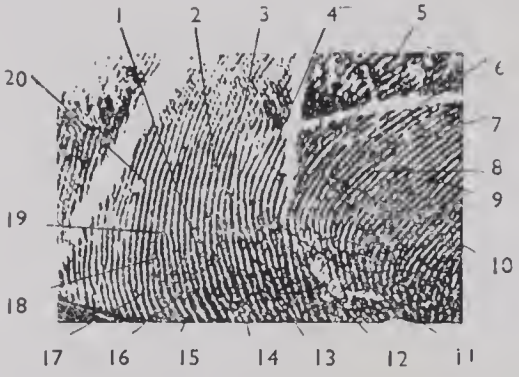


FIG. 7

Palmar impression of suspect of crime.

Identification by teeth marks.

This form of identification has proved of value. Two men were tried at Cumberland Assizes on a charge of theft. On examination of the premises, a piece of cheese, with teeth marks upon it, was discovered. A cast of the teeth of one of the suspects was made and found to fit exactly the impressions on the cheese. Expert evidence was given by a dentist, who stated that no two sets of teeth are exactly alike. The prisoner requested that his mouth should be examined again to ascertain if his teeth would fit the impression on the cheese. On complying with the request, it was found that, since the original impression had been taken, he had removed a stump.[5]

Identification by wounds on the body.

On rare occasions the presence of wounds on the body may assist in associating a suspected criminal with a given crime.

In Glasgow, a thief broke the plate-glass window of a jeweller's shop, stole some jewellery and escaped. On examination of the window, a small, almost circular, piece of skin was found adhering to the edge of the broken piece of window. The police rounded up all the thieves whose speciality was shopbreaking, and their hands were examined. On the finger-tip of one of the men an unhealed

wound was found. It was almost circular in shape, and when the piece of preserved skin was applied it was found to fit almost exactly.

A young girl of thirteen was brought to the police by her mother who alleged that the girl had been criminally assaulted. Examination of her genitals showed both swelling and bruising. The hymen was ruptured, and the underclothing soaked with blood. Examination of the scene of the crime showed a considerable amount of blood on the floor of the stair landing. A man was apprehended, and the girl identified him as her assailant. On examination of the prisoner, a recent rupture of the frænum of the penis was found. Although his trousers had been recently washed, the inner surface showed the presence of blood in the seams, and the soles and nails of his boots also gave unmistakable evidence of blood. The accused strenuously denied the accusation, but at his trial he pleaded guilty.

Dust and debris.

Dust and debris removed from the surfaces of clothing, pockets, and the turn-ups of trouser legs may, when submitted to careful microscopical examination, give some indication of the occupation of a person. Deposits from the ears, nostrils, and from under the nails should also be examined, since particulate matter found in the atmosphere of certain factories and workshops may be found in these regions. Debris from pockets may disclose certain habits. The turn-ups of trouser legs may contain some forms of vegetation which may provide important information in some cases, so may debris associated with bodies retrieved from water, or with footwear or motor cars (see Fig. 1). Not infrequently the services of a botanist, zoologist, entomologist, or chemist may prove essential in the investigation. Spectrographic analysis of traces, often insufficient in quantity for the application of ordinary chemical tests, may yield highly important results. Among many of our cases in this connection, two may be cited. In one of the cases, in connection with a contravention of the Criminal Law Amendment Act, a young girl was the subject of a sexual offence which took place in an out-door lavatory in a quiet country district. Certain stains upon the overcoat of the accused were found on analysis to be of the same chemical composition as the material covering the walls in the lavatory. The second case was one of murder. A young woman was strangled. Her coat and one stocking were stained with a yellowish-green pigment which in all respects was the same as the pigment found upon the sleeve of the accused, and on the door of an out-door lavatory in which her body had been placed (see Fig. 165). Reconstruction experiments demonstrated the fact that there was only one position in which her body could have been carried into the lavatory to deposit it in the position found, and that in doing so the position of the pigment staining on the garments belonging to the two participants in the experiments was closely similar to that on the garments submitted for examination in the case. With regard to photomicrography of particulate matter, photographs

should be taken both by transmitted and reflected light. For detail, infra-red photography by reflected light is recommended.

Tattoo marks.

The practice of tattooing is common among certain classes of persons, and is very prevalent among sailors and soldiers. The process consists of the injection of bright pigments into the true skin to form various patterns or designs. The extent of such marks varies among individuals. It may extend over the major portion of the body or be confined to small areas. The devices assume the most varied character.

FIG. 8

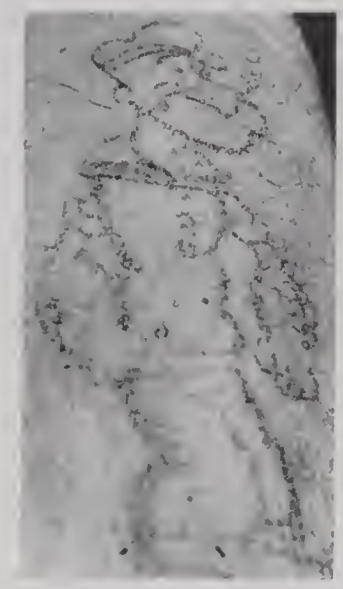


FIG. 9

Appearance of tattoo marks following shedding of epidermis after thirty-seven days' immersion in water. The arms showed green putrefactive change. Although the skull was completely denuded of tissue, these marks established identity.

(By courtesy of Dr Robert Richards, Aberdeen University.)

In many cases the presence of such marks on the body may lead to the identification of an individual not only during life but after death. The nature of the devices may prove of assistance in establishing identity since, in a number of cases, in addition to the initial letters of the name of the person tattooed, further identifying details may be available from the nature of the emblems.

Tattoo marks produced by unstable pigments tend to fade and disappear after variably long intervals, but stable pigments produce permanent marks. Carbon, indian ink, vermilion, and prussian blue are the most permanent pigments. Cinnabar, cochineal, aniline dyes, and ordinary ink, are much less permanent. Tattoo marks made professionally are composed of stable pigments which are injected by several methods and are permanent. Various means have been devised for the elimination of these marks, but the only

perfect method is surgical removal, when the resultant scar is minimal. A tattoo design may be altered, or a second may be superimposed.

Tattoo marks on unidentified putrefied bodies may be photographed with sharp definition if the loose epidermis is first removed and the design on the dermis is recorded. This method is of special

Fig. 10

Fig. 11

Tattoo marks.

value in the case of bodies recovered from water. The use of the ultra-violet lamp or of infra-red photography may prove helpful in revealing latent tattoo marks (see p. 432).

Teeth.

An examination of the teeth may prove of high value in regard to identification, more particularly if dental work has been carried out. The state of the teeth, if the professional records of a dentist are available, is of the greatest importance, and the following, among other, particulars should be noted :—

The number and position of teeth present in, and missing from, each jaw.
Whether extraction of the teeth has been recent or remote.
The presence and position of cavities, fillings, and crowns.
Whether a denture has been worn, and a denture known to have been made for the particular person fits the mouth.
The quality of any dental work found present.
The existence of any recognisable peculiarity of the jaws or teeth.

The following case [6] has been reported. A woman was found unconscious in the black-out, having been hit on the head by a passing motor van. She was wearing dentures. The upper one remained in her mouth, but the lower one was missing. The police traced the van. The driver denied hitting anyone, although marks

were present on the van. In the back of the vehicle was the missing denture. The police took the dentures to a dentist who was asked if he could identify them as made for a patient some years previously. The upper denture was immediately recognised and the lower, which was broken, was found when held together to fit into normal occlusion. The finding of the lower denture in the van, and proving that it belonged to the upper one, provided conclusive evidence that the patient had been knocked down by the van.

Identification of living and dead persons.

Since many of the features used in the identification of dead bodies are equally applicable to the establishment of identity of living persons, to obviate repetition, these aspects of the subject will be discussed together.

The solution of identity, reviewed broadly, depends upon a consideration of the following features :—

Age.
Sex.
Stature.
Deformities, peculiarities, and individual characteristics.

Age.

Evidence of age for medico-legal purposes may be required in several types of cases, for example, as an aid to identification, in rape and sexual offences, in relation to capacity for procreation, in relation to a fœtus, and in connection with age of marriage.

In the instance of young children, a fairly accurate opinion as to age may be arrived at from the state of eruption of the teeth, the general development, height, and weight of the body, and the condition of ossification of the bones. The assessment of age based solely on the state of eruption of the teeth may occasionally prove misleading, but further allusion will be made to this point when consideration is given to the ages at which the first and second dentitions appear. One may also be led astray by the condition of development, height, and weight, more especially in the case of children born and reared in cities, by reason of rickets, or syphilis, which disturb these factors prejudicially, and it will be found that the difficulty does not become lessened as the scale of years is ascended. The time of onset of puberty in girls and of virility in boys is very variable, and although from a large mass of cases we may be able to formulate some average age at which it appears, the calculation does not apply to the individual. It is also necessary to remember the possibility of precocious sexual development which is a condition of childhood characterised by genital development which approximates adult proportions during the first decade, whereas precocious puberty is sexual precocity with further maturation of the child as manifested by spermatogenesis and menses.[7] We are aware of a case of a male child who, from a clearly visible moustache, well-developed sexual organs, growth of suprapubic hair, and other signs, might

readily have been mistaken for a young man. The epiphyses of the long bones on X-ray examination were found to be united to the diaphyses by osseous union. His age, as attested by the mother, the medical man who attended the birth, and the certificate of birth, was only ten years and seven months. He was charged with indecent conduct towards a girl.

Kerr [8] records a case of pubertas præcox in a girl aged six years and ten months, who at the age of three and a half years began to menstruate. The menstrual periods occurred four times annually and each lasted from one to two days. Her appearance was that of a well-developed girl of twelve to fourteen years. The breasts were enlarged, and there was prominence of the mons veneris on which there was some dark, fine hair. The uterus on bimanual examination was of almost adult size. She probably suffered from pineal syndrome due to neoplasm or hyperplasia of the pineal gland. Such precocious development is usually attributable either to pineal or suprarenal disease, commonly tumour.

Approaching middle life, the greatest difficulty in the assessment of age from external appearances is experienced, the result of the cumulative effect of inherited conditions and the vicissitudes of life upon the individual. Prematurely old men and comparatively active old men are quite frequently seen. It is clear, therefore, that in the assessment of age, little reliance can be placed on external manifestations, and that dependence must rest upon general developmental and growth changes in the skeleton.

The importance of developmental changes is clearly illustrated in the assessment of fœtal age. This is sometimes important in connection with charges of criminal abortion, infanticide and concealment of pregnancy or concealment of birth, since it is important for the authorities to know whether certain structures found are the product of conception, and, if so, what stage in development has been reached. As will be appreciated later, when the subject of infanticide is reviewed, the estimation of fœtal age in medico-legal work is of frequent importance (see p. 80). It is necessary, therefore, to recognise the stage of development of the products of conception. The following data will prove of assistance in arriving at an opinion.

Development of fœtus.

End of first calendar month.

The entire ovum measures about 2 centimetres in diameter. The chorion is entirely covered by villi. The umbilical vesicle is fully developed. The embryo measures about 1 centimetre in length. The caudal extremity is well marked. The eye-spots appear as dark dots, and the limbs as buds or dot-like processes.

End of second month.

Between the fifth and sixth weeks, the embryo measures about $1\frac{1}{2}$ centimetres in length. The bud-like limb processes begin to show

evidence of three distinct segments. By the end of the eighth week, the fœtus measures about 2 centimetres in length, and the caudal extremity shows signs of disappearance. The rudimentary eyelids begin to appear, and also the conchae of the ears. The whole chorionic sac measures about 4½ centimetres in diameter.

End of third month.

The fœtus measures from 5½ to 7½ centimetres. Weight is about 4 ounces. Sex is still indefinite. Rudimentary nails, like thin membranes, appear on fingers and toes. The placenta is formed and differentiated.

End of fourth month.

Length = 5 to 7 inches (12½ to 17½ centimetres), weight is from 3 to 9 ounces, and the skin is fairly dense. The pupillary membrane is distinct. The nails on fingers and toes begin to appear. The genital organs are sufficiently developed to enable sex to be recognised. Downy hair appears on the body and scalp. A small quantity of meconium may be found in the intestine.

End of fifth month.

Length = 6 to 10 inches (15 to 25 centimetres), and weight about 6 to 12 ounces. The nails are now distinct. Growth of hair commences, and skin may show sebaceous secretion. Dental germs appear in the maxillae. Ossific centre is found in the calcaneus.

End of sixth month.

Length = 9 to 12 inches (22 to 30 centimetres), and weight about 1½ lbs. Skin, now divisible into cutis vera and epidermis, is wrinkled. Eyelids still closed by pupillary membrane. Umbilical cord is situated a little above pubis. Hair of eyebrows and eyelashes begins to form. Testes are found lying close to kidneys.

End of seventh month.

Length = 13 to 15 inches (32 to 37 centimetres), and weight about 3 to 4 lbs. Nails do not yet reach extremities of fingers. Pupillary membrane difficult to detect. Testes found in process of descent towards scrotum, and in the vaginal process of peritoneum. Fœtus capable of life if born (viable). Centre of ossification is present in the talus.

End of eighth month.

Length = 14 to 17 inches (35 to 42 centimetres), and weight about 4 to 5 lbs. Sebaceous secretion begins to be formed on skin. The nails now practically reach the extremities of the fingers. Pupillary membrane has now entirely disappeared. One testis, usually the left, may be found in scrotum, and the other well advanced in downward descent. Centre of ossification appears at lower end of femur.

End of ninth month.

Length = 18 to 24 inches (45 to 60 centimetres), and weight from $6\frac{1}{2}$ lbs. upwards, the average weight being about 7 lbs. Umbilical cord inserted about three-quarters of an inch below centre of body. Hair is found on scalp and on portions of body. Sebaceous secretion is likely to be found in flexures of joints, and more or less over the whole body. Both testes are in scrotum. The finger nails are fully formed and developed. The child is mature. The centre of ossification at lower end of femur is about the size of a pea. Small centre in cuboid bone, and possibly an ossific centre in upper epiphysis of tibia.

To make a rapid approximation of the age of a fœtus, Hesse's rule may be applied. It is that the length of the fœtus in centimetres is equal to the square of the number of months of gestation. After the fifth month of gestation has been reached, the length of the fœtus, in centimetres, divided by 5, gives the age of the fœtus in months. From the fourth month onwards, the length of the fœtus, in inches, is approximately equal to the number of months multiplied by 2.

Teeth.

An examination of the state of eruption of the temporary or deciduous teeth in infants will offer a reliable indication of age in most cases, since the respective periods of eruption of the different teeth are fairly constant. It must be remembered, however, that these teeth may appear either abnormally early, indeed, in rare cases, some of them may be present at birth, or their appearance may be abnormally delayed. We have examined a boy of eleven who did not then possess, and never had possessed, any teeth. Precocious dentition has been frequently recorded. The teeth which usually signalise this precocious dentition are the central incisors.

The following dental charts indicate approximately the period at which the different temporary and permanent teeth appear :—

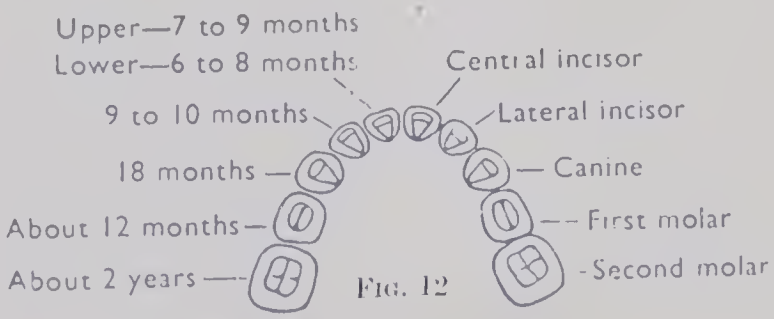


Fig. 12

Temporary or deciduous teeth.

The temporary teeth are twenty in number, and the permanent teeth thirty-two. The former begin to be shed from the fifth to the seventh year.

It will be evident that the first permanent molars appear about the sixth year, the second molars about the twelfth year, and the

third molars about the eighteenth year, earlier or later. There is greater uncertainty respecting the time of eruption of the third molars than of the other teeth. In odd cases they do not appear till adult age is well advanced.

Spokes [9] states that of 638 boys between thirteen and fourteen

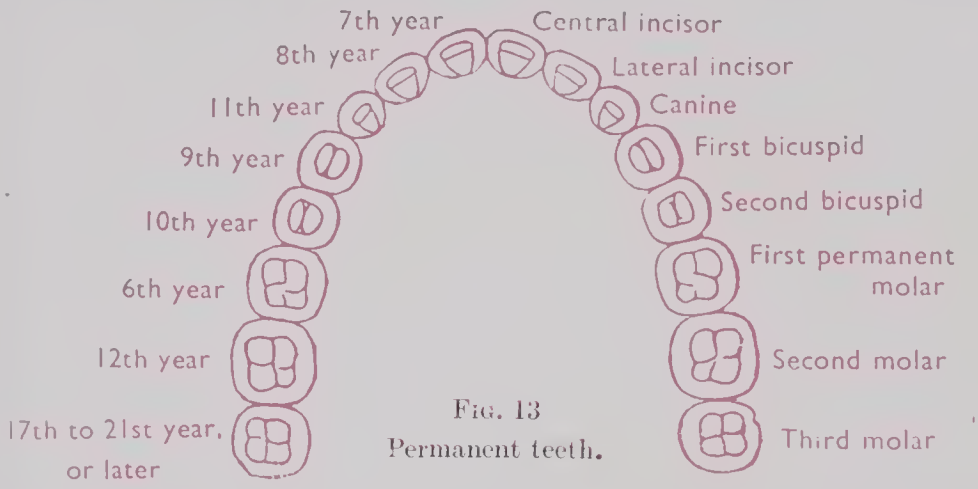


Fig. 13
Permanent teeth.

years of age, the majority had all four second molars erupted, 4 per cent. had all absent, 5 per cent. had three absent, 7 per cent. had two absent, and 7 per cent. had one absent. Of 492 boys between fourteen and fifteen years, 82 per cent. had all four second molars and 92 per cent. had three erupted or visibly erupting.

In the younger subject, age from permanent teeth can be assessed relatively, but not with absolute accuracy, as the formation and eruption of teeth vary in individual subjects.

Times of appearance of centres of ossification in the epiphyses and their fusion with diaphyses.

An approximately accurate estimate of age is given by the centres of ossification, and the progress of that ossification in the unification of the bones.

Flecker [10] holds the view that a perusal of the various anatomical authorities shows a considerable discrepancy regarding the ages at which the various centres of ossification in the epiphyses appear, and at which the epiphyses fuse with their respective diaphyses. The following is a résumé of some of his findings based upon radiological examination of a large series of living subjects of both sexes and of different ages :—

CHRONOLOGICAL ORDER OF APPEARANCE OF CENTRES OF OSSIFICATION IN, AND FUSION OF, SOME OF THE EPIPHYSES. (FLECKER)

Before birth—

Both sexes .	Appearance.	Head of humerus, distal femur, proximal tibia, calcaneus, talus.
Female .	,,	Cuboid.

During first year—

Both sexes .	Appearance.	Head of femur, third cuneiform.
Female .	,,	Capitulum of humerus, distal radius, distal tibia, distal fibula.
Male . .	,,	Cuboid.

During second year—

Both sexes .	Appearance.	Proximal phalanges of the four fingers.
Female .	,,	First and second cuneiforms.
Male . .	,,	Capitulum of humerus, distal radius, distal fibula.

At age two—

Both sexes .	Appearance.	Inner four metacarpals.
Female .	,,	Middle row of phalanges of fingers.
Male . .	,,	First cuneiform.

At age three—

Female .	Appearance.	Patella, proximal fibula.
Male . .	,,	Tarsal navicular, second cuneiform.

At age four—

Female .	Appearance.	Head of radius.
	Fusion.	Greater tubercle to head of humerus.
Male . .	Appearance.	Lunate.

At age five—

Both sexes .	Appearance.	Carpal navicular, greater trochanter.
Female .	,,	Medial epicondyle of humerus, distal ulna.
Male . .	,,	Head of radius, patella, proximal fibula.
	Fusion.	Greater tubercle to head of humerus.

At age six—

Male . .	Appearance.	Medial epicondyle of humerus, distal ulna.

At age seven—

Female .	Appearance.	Distal phalanx of little finger.
	Fusion.	Rami of ischium and pubis.

At age eight—

Female .	Appearance.	Olecranon.

At age nine—

Female .	Appearance.	Trochlea, pisiform.
Male . .	Fusion.	Rami of ischium and pubis.

At age ten—

Male . .	Appearance.	Trochlea, olecranon.

At age eleven—

Female .	Appearance.	Lateral epicondyle of humerus.
Male . .	,,	Pisiform.

At age twelve—

Male . .	Appearance.	Lateral epicondyle of humerus.

At age thirteen—

Female .	Fusion.	Lower epiphysis humerus, bodies ilium, ischium, and pubis.
Male . .	,,	Capitulum of humerus to trochlea and lateral epicondyle of humerus.

At age fourteen—

Female .	Appearance.	Acromion, iliac crest, lesser trochanter.
	Fusion.	Olecranon, upper radius, head of femur, greater trochanter, distal tibia and fibula.
Male . .	Appearance.	Proximal sesamoid of thumb.

At age fifteen—

Female .	Fusion.	Medial epicondyle of humerus.
Male . .	Appearance.	Acromion.
	Fusion.	Ilium, ischium, and pubis.

At age sixteen—

Female .	Appearance.	Tuber ischii.
Male . .	Fusion.	Lower epiphysis humerus, medial epicondyle of humerus, olecranon, head of radius.

At age seventeen—

Both sexes .	Fusion.	Acromion.
Female .	,,	Upper epiphysis humerus, distal ulna, distal femur, proximal fibula.
Male . .	,,	Head of femur, greater trochanter, distal tibia and fibula.

At age eighteen—

Female .	Fusion.	Distal radius.
Male . .	,,	Proximal tibia.

At age nineteen—

Male . .	Appearance.	Tuber ischii.
	Fusion.	Upper epiphysis humerus, distal radius and ulna, distal femur, proximal fibula.

At age twenty—

Both sexes .	Fusion.	Iliac crest.
Male . .	,,	Tuber ischii.

At age twenty-one—

Both sexes .	Appearance.	Clavicle (medial end).
Female .	Fusion.	Tuber ischii.

At age twenty-two—

Both sexes .	Fusion.	Clavicle.

According to Brash,[11] growth changes in the skeleton, although providing a very reliable basis for the estimation of age, do not permit of an exact determination, but only within a range, since there is variation in relation to growth and age. In referring to this point, he states that, " before puberty, when the skeleton begins to consolidate, so many active growth changes are going on—including the development of the growing (epiphyseal) ends of the limb bones, and the progressive calcification and the eruption of the teeth—that it is relatively easy to determine probable age within a year or so. From puberty, until the consolidation of the skeleton (at twenty-two or twenty-three, or at the most twenty-five years), a fairly close estimate—within a range of two to three years—may still be made, mainly on the progress of the union of the epiphyses. Thereafter, the range must lengthen ; and after thirty years, when the mature skeleton already begins to show signs of 'ageing'—including the beginning of the progressive closure of the cranial sutures—it will be hardly safe to estimate more closely than in decades."

The development and consolidation of the bones of the skeleton which ossify in cartilage occur, as a rule, about two years earlier in the female than in the male, but the obliteration of the sutures of the vault of the skull sets in a little later and proceeds more slowly in the female than in the male.,

When all the epiphyses of the limb bones are completely united it indicates a minimum age of twenty-two years, or, at the most, twenty-five years, but after apparent adult age has been attained, and particularly toward later adult life, some indication of age may still be ascertained. For example :—

> The absence of any sign of closure of any of the sutures of the skull points to a strong probability that the age does not exceed thirty years. The closure of the sagittal, coronal, and lambdoid sutures has usually begun by the age of thirty. The parieto-mastoid and squamous sutures commence at a later stage, between thirty-five and forty years, but do not show great advancement until between fifty and sixty years. The spheno-parietal suture commences to close about thirty years, but is not usually completely closed until about seventy years.

Evidence of commencing union of the sutures is first seen on the inner surface of the bones. Franchini [12] states that, following research on 629 skulls, 369 male and 260 female, and grouping them into decades in relation to age, he has found exceptional cases in the skulls of both sexes in which at the ages of 45, 48, 55, and 74 years, there was not a trace of obliteration of sutures. He says that obliteration may be of subsidiary help in association with other appearances, but the variations are such that it is impossible to rely on it alone for determining the age of the subject.

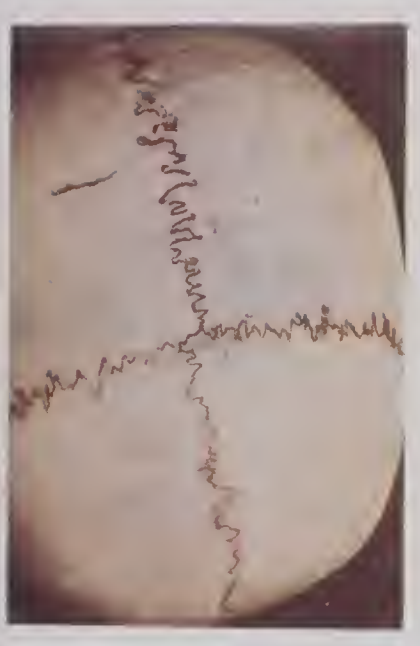


Fig. 14

Absence of closure of sutures of skull.

Fig. 15

Closure of sutures of skull.

Beyond middle age, ossification of the laryngeal cartilages and in the hyoid bone may usually be found, although commencing ossification of the larynx may be present at an earlier age.

Ossification of the costal cartilages is usually associated with advancing years, but may be present in younger subjects.

The xiphoid process usually fuses with the body of the sternum about the age of forty, and in advanced life, the manubrium is occasionally joined to the body by bone, although only the superficial parts of the intervening cartilage is converted into bone.

After middle age, the angle of the lower jaw tends to become more obtuse, its alveolar margin shows some evidence of absorption of bone, and any teeth, which may still be present, are frequently found in a loosened condition.

With respect to the age of a body of which only mutilated remains are available for examination, a fairly accurate estimate of age can frequently be arrived at. The ability to do this will depend

very largely upon the parts available for examination, with respect to their amount, nature, and variety. The Ruxton case, which will be considered later, illustrates several of the points already touched upon in addition to further aspects which find a place in the subject of identification of the dead. This case involved a very wide and varied field of investigation, and the results are regarded as highly instructive in the practical application of available methods for the identification of mutilated and dismembered remains.

Sex.

Difficulty about this question may arise in the examination of bodies when decomposition is so far advanced that the external and internal organs of generation have disappeared, either from exposure to air or water, after burial in the ground, as the result of mutilation and dismemberment, or when only portions of a body are available for examination.

Brash states that, in the examination of bones, occasionally the skull and limb bones present general features which are in some respects equivocal for the diagnosis of sex. This necessitates a careful assessment of the balance of the anatomical features before anatomical proof becomes available for the correct opinion as to sex.

The more outstanding general characters, apart from the genital organs and breasts, which differentiate the sexes may be summed up as follows :—The male is generally of larger build and greater muscular development than the female, although the effeminate male and the masculine female must be remembered. The adult male is broader at the shoulders than at the hips, the adult female broader at the hips than at the shoulders. The waist is marked in woman and ill-defined in man, and the gluteal regions in the former are full and rounded whereas in the latter they are square and contractile. The legs of the female are more rounded and the wrists, ankles, and nails more delicate. Hair only covers the mons veneris in the female, but in the male it covers the pubis and may extend upwards on to the abdomen, and more or less over the anterior surface of the chest. The hair of the male head is shorter, thicker, and coarser than that of the female. The larynx of the male is more prominently developed than that of the female.

In the case of mutilated remains, identity is simplified when those parts of the body bearing sexual characters are available. In this connection it is well to remember that the unimpregnated uterus is one of the organs of the body to resist rapid putrefaction, and consequently when abdominal remains are available, this organ and its appendages should be looked for carefully. When the uterus and its appendages are absent, it becomes necessary to rely upon the general appearances of the parts, the disposition of the hair, the presence or absence of the mammæ, the presence or absence of lineæ albicantes, as indicative of previous pregnancy, and any part of the external genitals which may remain. The prostate gland is resistant to putrefaction, and it should always be looked for since its presence indicates male sex.

When sexual characteristics of the soft parts are unavailable, it becomes necessary to base the diagnosis of sex on the secondary sex characters displayed by bones.

By consideration of the character of the long bones with reference to muscle-attachments, and especially of the character of the pelvis, skull, and sternum taken as a whole, an opinion may usually be arrived at with considerable accuracy. Generally the bones of the female skeleton are lighter, are not so large, and muscle-attachment marks are not so pronounced as in the male. Apart from certain measurements of the long bones, the pelvis, skull, sternum, and sacrum must be reckoned as the chief means of differentiating sex in a skeleton. The following are the principal points of differentiation :—

THE PELVIS

Male.	Female.
Bony framework massive.	Bony framework less massive.
Deep.	Shallow and more capacious.
Walls not splayed.	Walls splayed.
Pubic arch narrow, not more than about 70°.	Pubic arch wide, practically right-angled.
Great sciatic notch narrow.	Great sciatic notch almost a right angle or even greater.
Obturator foramen ovoid.	Obturator foramen more triangular.
Preauricular sulcus not marked.	Preauricular sulcus pronounced.
Iliopectineal line well marked and rough.	Iliopectineal line rounded and smooth.
Body of pubis approximately triangular.	Body of pubis approximately square.
Ramus of pubis is like a continuation of body of pubis.	Ramus of pubis has a pinched or narrowed appearance and is squat in character.
Acetabulum is large, averaging 52 mm. in diameter, and faces laterally.	Acetabulum is small, averaging 46 mm. in diameter, and faces forward as well as laterally.
Sacrum is relatively narrow and long, and its curve is more or less equal over the entire length.	Sacrum is relatively wide and short, and its curve is practically confined to a point commencing below centre of third vertebra.
Promontory fairly prominent.	Promontory less prominent.
... ...	Transverse, oblique, and antero-posterior diameters greater than in male.
... ...	Pelvic outlet is roomy and admits the passage of the clenched fist.

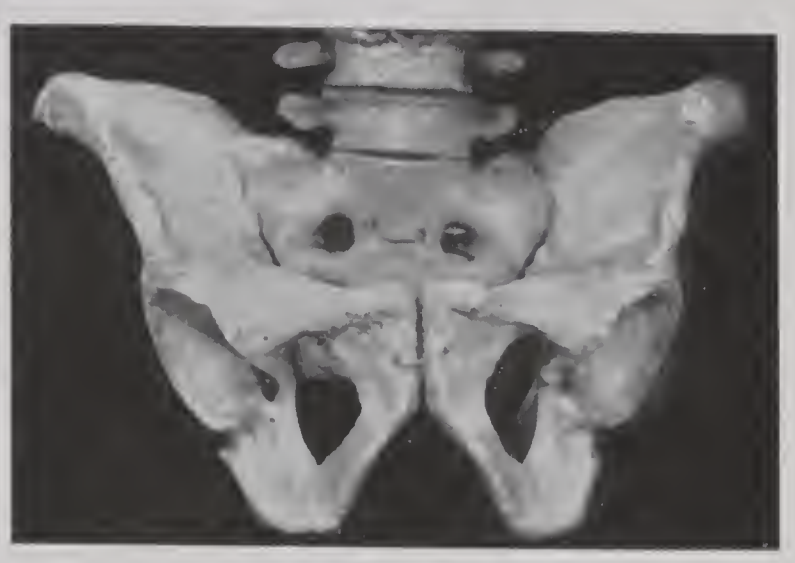


FIG. 16

Male pelvis. Note narrow pubic arch.

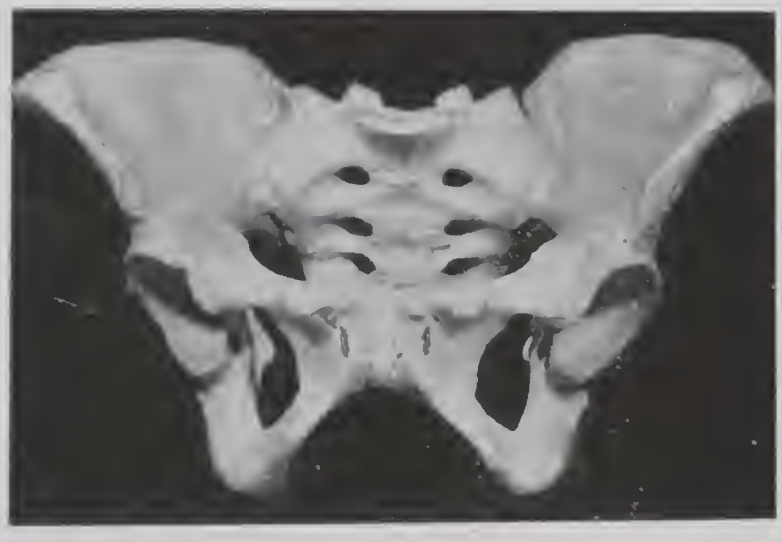


FIG. 17

Female pelvis. Note wide pubic arch.

The sexual characters of the pelvis are of the highest importance in the sexing of skeletal remains. Since the female pelvis is constructed for the function of child-bearing, most usually it presents very distinctive features which contrast with the pelvis of the male. Despite this fact, there are instances in which a pelvis shows variable secondary characters, mixed male and female characters, and in such cases an opinion regarding sex must be limited to one of greater or lesser probability.

Sacrum.

Fawcett advocates a method for sexing the sacrum. It consists of comparing the transverse diameter of the first sacral vertebra

with the transverse diameter of the base of the sacrum. The formula used is :—

$$\frac{CW \times 100}{BW} = \text{Index of body.}$$

CW = corpus width.
BW = basal width.

Readers are referred to the original article.[13]

	White Races. (Adults.)	
	Male.	Female.
Number . . .	134	79
Age range . . .	25–49	22–54
	Corporo-basal Index.	
Mean	45·041	40·486

THE SKULL

Male.	Female
... ...	Usually smaller.
Mastoid processes massive.	Less pronounced.
Superciliary ridges marked.	Less marked.
Fronto-nasal junction prominent.	Less prominent.
Sites of muscular insertions at base well marked.	Less pronounced.

The sex of a subject can frequently be determined from the general characters of the skull, but here again a variability in the sexual characters may be encountered.

In arriving at a conclusion regarding the sex of a pelvis or of a skull, it is necessary that the balance of all the characters should be considered with care.

Sternum.

The proportion between the manubrium and the body of the sternum, according to several authorities, is definitely influenced by sex. In the female, the manubrium is larger in proportion to the body. The following is a method for calculating the percentage index :—

$$\frac{\text{Length of manubrium} \times 100}{\text{Length of body}}.$$

The average female index is 54·3, while the average male index is 46·2 (Krause).

Female. Male.

FIG. 18

Skulls—Front view.

Female. Male.

FIG. 19

Skulls—Side view.

Sexing of the limb bones.

According to Brash, the measurements of the limb bones are reliable and conclusive if within a certain range, and the two most important bones for this purpose are the humerus and femur. There is, however, a considerable variation in the lengths of the bones in each sex which occasions a considerable overlap in the range of the length of each bone in the two sexes. He is of the opinion, however, that an indication of probability of sex, more or less great, may be obtained from such measurements. The table below shows the mean measurements of the maximum lengths of the humerus and femur, from a particular series.[14]

Maximum Length (Mean).	Humerus.	Femur.
Male	326·18 mm.	459·3 mm.
Female . . .	298·66 ,,	426·27 ,,

Other figures for the femur are[15] :—

Male, 447·24 mm. Female, 409·19 mm.

The sizes of the heads of the humerus and of the femur offer even more significant sex characters than the lengths of these bones. The average measurements of the humeral head (with cartilage in position), according to Dwight,[16] are :—

	Head of Humerus.	
	Vertical.	Transverse.
Male (average) . .	48·7 mm.	44·6 mm.
Female (average) .	42·6 ,,	38·9 ,,

According to his measurements, the boundary between male and female lies between 45 and 46 millimetres for the vertical diameter and between 41 and 42 millimetres for the transverse diameter.

Parsons' measurements of the vertical diameter of the head of the femur (including cartilage) have shown that when the measurement exceeds 48 millimetres it indicates a male bone and when less than 44 millimetres, a female bone.

Karl Pearson has formulated a method for "sexing" femora on a basis of values which may be assigned as follows :—

RULES FOR "MATHEMATICAL" SEXING OF FEMUR (PEARSON)

Measurement.	Female.	Female ?	?	Male ?	Male.
Vertical diameter of head . . .	41·5	41·5–43·5	43·5–44·5	44·5–45·5	> 45
Popliteal length .	<106	106–114·5	114·5–132	132–145	>>145
Bicondylar width .	< 72	72–74	74–76	76–78	> 78
Trochanteric oblique length . .	<390	390–405	405–430	430–450	>450
Mark assigned to each . . .	+2	+1	0	−1	−2

The probability of a correct result is greatly increased by the summation of these marks. The measurements are in millimetres, and it should be noted that Parsons' figures and those of Pearson

for the vertical diameter of the head correspond closely when 3 millimetres (representing cartilage) are deducted from the former, since the latter measurements are of dried bone devoid of cartilage (Brash).

Of the above measurements, the maximum bicondylar width is considered highly important especially for sex differentiation. The popliteal length, that is, the measurement from the apex of the popliteal surface to the centre of the intercondylar line, is much less reliable since this measurement cannot always be appreciated with accuracy. The maximum bicondylar width is taken parallel to the infra-condylar plane, that is, with the bar of the calipers touching the lowest points of both condyles in the infra-condylar plane, and the arms of the calipers touching, respectively, the external condyle, externally, and the internal condyle, internally, at the maximum distance. The trochanteric oblique length is the vertical distance from the top of the great trochanter to the infra-condylar plane.

Stature.

The calculation of stature comes to be a matter of necessity when an examiner is called upon to deal with a part or parts of a body or skeleton. The following summary provides some information which may be found helpful in arriving at approximate and preliminary estimates of stature.

Some research data on the relative length of the bones of the human skeleton (M. de S. Luca).[17]

The length of the head equals one eighth of the total body height.

The head is divided into two equal parts by a line drawn immediately below the eyes; each of these parts equals one sixteenth of the total body height.

The nostrils subdivide the lower half of the face equally; each of the resultant parts equals one thirty-secondth of the total body height.

The pubis is a central point between the two extremes of the body; therefore, head to pubis, or pubis to the soles of the feet, equals one half of the total body height.

The height of a man equals the distance which separates the extremities of the two hands when the arms are extended in a horizontal line from the body.

The upper extremities can be divided into five parts, the hand representing one part, the forearm, two parts, and the upper arm, two parts. The elbow is the boundary between the lower and the upper arm.

Irrespective of the length of the hand, five times its length equals the length of the whole of the upper extremity.

Half the length of the hand equals the length of the area occupied by the carpal and metacarpal bones.

When certain of the long bones are available, more particularly the humerus or femur, reliable estimations of stature may be made by the employment of certain formulæ. In the case of a dismembered body, estimation of stature becomes possible by direct measurement, provided the parts of the body, including at least one of the lower limbs, are available and an accurate reconstruction of the body is possible. It will be obvious that this is a preferable method of estimation to the use of formulæ when the circumstances of the case permit. In assembling parts of a body with a view to direct measurement, it is necessary to arrange the parts in their correct and natural positions as securely as possible so that the measurement of the reconstructed body may be determined accurately. After measurement, a small deduction of 1·25 centimetres from the male and 2 centimetres from the female length, the amount by which, on the average, the body lengthens after death, should be made in order to arrive at the probable living stature. It must also be remembered that information regarding the height of missing persons supplied to the police by relatives and friends, especially in the case of women, may prove to be rather misleading, since the estimate given may in the first place be inaccurate, either because the actual measurement has not been taken during life or on account of added height by reason of the hair and footwear. (Brash.)

If bones only are available for examination, the formulæ now employed, because of their comprehensiveness and accuracy, are those of Karl Pearson [18] based on a mathematical study of measurements of limb bones and statures. The formulæ for the reconstruction of female stature differ from those of male stature, and also differ in accordance with whether the bones are in the humid state or have been cleaned and dried. The bones which should be used for measurement are the femur, humerus, tibia, and radius.

Pearson's formulæ for reconstruction of living stature from dried long bones from which all tissue has disappeared.

Male.	Female.
Stature = 81·306 + 1·880 femur.	Stature = 72·844 + 1·945 femur.
Stature = 70·641 + 2·894 humerus.	Stature = 71·475 + 2·754 humerus.
Stature = 78·664 + 2·376 tibia.	Stature = 74·774 + 2·352 tibia.
Stature = 89·925 + 3·271 radius.	Stature = 81·224 + 3·343 radius.

(The results are in centimetres.)

The femur is measured from the head to the apex of the inner condyle. The tibia is measured from the upper articular surface to the tip of the malleolus, excluding the spine. The humerus and radius are measured over their greatest length.

In addition to the above, there are also formulæ for the calculation of stature of both sexes by the employment of a combination

of measurements of more than one of these bones at the same time.

In the case of a male subject, when only dried long bones are available, stature = 67·049 + 0·913 F + 0·600 T + 1·225 H − 0·187 R, or when the bones are with cartilage and are in a humid state, stature = 66·918 + 0·913 F + 0·600 T + 1·225 H − 0·187 R. In the female subject, a similar computation of stature can also be made, for example, when dried long bones are available, stature = 67·469 + 0·782 F + 1·120 T + 1·059 H − 0·711 R, or when the bones are measured with cartilage and are in humid state, stature = 67·810 + 0·782 + 1·120 T + 1·059 H − 0·711 R. Stature can also be estimated by using formulæ in conjunction with the measurements of femur and tibia, humerus and radius, and in other combinations.

Pearson's formulæ for reconstruction of stature as corpse.

Male.	Female.
Stature = 81·231 + 1·880 femur.	Stature = 73·163 + 1·945 femur.
Stature = 70·714 + 2·894 humerus.	Stature = 72·046 + 2·754 humerus.
Stature = 78·807 + 2·376 tibia.	Stature = 75·369 + 2·352 tibia.
Stature = 86·465 + 3·271 radius.	Stature = 82·189 + 3·343 radius.

(The results are in centimetres.)

The maximum lengths of the femur, humerus, tibia (without spine), and radius are measured with the cartilage on and in a humid state. The points of measurement have already been given. In addition to the above formulæ, there are also formulæ for the calculation of corpse stature of both sexes by the employment of a combination of measurements of more than one of these bones.

Body deformities and peculiarities.

The presence of congenital deformities, such as hare-lip, cleft palate, polydactylism, syndactylism, defective development of the ribs and acquired deformities, including mallet-fingers and hammer-toes, together with evidence of joint diseases and fractures, are important aids in establishing the identity of persons. Moles, nævi, scars, and tattoo marks, when present, should be noted carefully since their presence may be of considerable value in this respect. Amputations, also, have frequently played a part in identification. Little requires to be said about these conditions with the exception perhaps of scars.

Scars.

A surface scar always follows an injury of the true skin or dermis, and scar tissue is usually devoid of hair and sweat glands. Scar tissue is formed in about four weeks following moderate injury, and in the course of time the newly formed connective tissue undergoes

cicatricial contraction by the diminution in size of the blood-vessels and cells, together with the consolidation of the young avascular fibrous tissue. When sepsis has supervened or when there has been considerable loss of tissue due to trauma, the resultant scar-tissue formation may be considerable in amount. The healing of an uncomplicated incised wound occasions relatively little scar formation, except in instances where the wound, although gaping, has not been stitched. Scars increase in size if the part on which they are situated increases as the result of growth. A well-developed scar never disappears spontaneously, but scars resulting from wounds in which there has been no appreciable loss of tissue may become very difficult to detect. Scars can be removed artificially, but a new scar is created as the result. The age of a scar can only be arrived at in a very approximate manner, due to the variable periods which different wounds take to heal. A recent scar is of reddish colour, and with increasing age becomes progressively whiter and glistening. The duration of each stage, however, is liable to wide variation. The other factors attendant upon scar formation include the original extent of the wounding, the amount of tissue destruction, the manner of healing, and the health of the subject. The furthest a medical witness should be prepared to go in appraising the age of a scar must depend upon the circumstances of the individual case before him, and he should give a wide margin in his computation of age. Scars may result from various causes and these include, for example, lineæ albicantes of an old pregnancy, healed ulcers, varicose and others, also vaccination. Extensive and disfiguring scar formation frequently results from burning. The well-known keloid condition needs no description. In the case of a living person, when it is important to discover a suspected scar or scars, not readily visible, certain methods may have to be employed. On account of the relative or complete avascularity of scar tissue, depending upon its degree of maturity, any influence which will produce marked increase in the activity of the cutaneous circulation in the surrounding skin will prove of assistance. The application of heat, or the use of surface friction, will produce the desired result. The employment of filtered ultra-violet light may in some cases be beneficial in revealing the presence of faint scarring (see p. 432). A magnifying glass may prove serviceable.

Mutilation and dismemberment.

In approaching the medico-legal aspects of the investigation of cases in which there has been mutilation and dismemberment of a body or bodies, a number of salient questions must receive special consideration. These are :—

Are the remains of human or animal origin ?
If human, do they represent one or more bodies ?
What is the sex ?
What is the age ?
What is the stature ?

Do the mutilation and dismemberment indicate, by their character, the use of anatomical and medical knowledge? For example, disarticulation at the joints as opposed to the use of a saw.

Has there been purposive removal of identifying features?

Do any features still remain which are likely to facilitate identification?

What is the state of the parts in relation to the process of putrefaction?

FIG. 20

Severed head showing site of decapitation by a saw.

Are all the parts in a similar state so far as preservation or putrefaction is concerned?

Are there any marks indicative of the application of ante-mortem violence?

Do the remains show evidence suggestive of an attempt to destroy them?

Are there any articles associated with the remains which might lead to identification?

What was the cause of death?

In certain cases other subsidiary questions will probably arise, but the answers to the foregoing questions usually assume a high degree of importance. Much could be written under each of these headings, but the subject-matter will be dealt with in more practical

form by giving an illustration, in some detail, of the application of medico-legal methods employed in the Ruxton case.

The legal importance of the establishment of the identity of remains, however, should first be mentioned. In law, it is not necessary that there should be a dead body in order that a charge of murder may be presented against some person or persons; but where no body or part of a body, which is proved to be that of the person alleged to have been killed, has been found, the accused person should not be convicted either of murder or manslaughter, unless there is evidence either of the killing or of the death of the person alleged to have been killed. In the absence of such evidence there is no onus upon the prisoner to account for the disappearance or non-production of the person alleged to be killed. (Halsbury's "Laws of England.")

THE RUXTON CASE

The case[19] is notable primarily on account of the extent and character of the mutilation of the two victims. This provided a problem of reconstruction demanding for its solution anatomical work, in detail, not previously required in such cases. On account of the purposive removal of identifying features a novel comparison of skull and portraits was used which, with other circumstantial evidence, helped to place identification beyond doubt (see Fig. 21).

Facts of the case.

On September 29, 1935, human remains were discovered in the bed of Gardenholme Linn, below the bridge on the Moffat-Edinburgh road. Some of the remains were tied up in bundles around which were pieces of cotton sheeting, and associated with some of the parts were a blouse, a pair of child's rompers, pieces of newspaper, straw, and pieces of cotton-wool. Over a succession of days, further portions of human remains were recovered in and around the linn. On October 28, a left foot was found on the roadside some nine miles south of Moffat, and on November 4, a right forearm with hand was also discovered by the roadside about half a mile south of the linn. Seventy portions of human remains had been recovered by this time. All the remains were decomposing and maggot-infested.

The dismembered remains.

Among the remains were the following parts :—

Two heads, each with a portion of neck (including cervical vertebræ) attached ;

Two portions of trunk (thorax and pelvis), which fitted together to form a trunk ; and

Fifteen limb segments divided at the main joints, among them no single region represented in more than duplicate.

Thus there were :—

Four upper arms (separated at shoulder and elbow), two right and two left, which could be matched in pairs ;

Three forearms and hands, one right and two left, including a pair ;

Four thighs (separated at hip and knee), two right and two left, which could be matched in pairs ; and

Four legs, two with feet, a pair, and two without feet, also a pair.

Attached to each of the thighs forming one of the pairs was a patella, and two other patellæ, forming a pair, were found with the detached soft parts. The left foot and the right forearm with hand, subsequently found (see p. 104), completed two sets of limbs with the exception of one foot. In addition to the limb segments, the soft parts included three female breasts which had been mutilated, a uterus with appendages, and two irregularly skin-covered pieces of tissue which were portions of female external genitals.

Fig. 21

Cast of face of Head No. 1, showing mutilations. (Ruxton case.)

Upper trunk portion.

This included the two scapulæ, the two clavicles, the sternum, the costal cartilages, and ribs. The portion of the spine included in this portion of the trunk comprised two cervical vertebræ, all the thoracic vertebræ and two lumbar vertebræ.

Head No. 1.

Attached to this head were only four cervical vertebræ (the first to the fourth) together with fragments of the fifth cervical vertebra which had the appearance of having been cut off obliquely with a knife.

Head No. 2.

Attached to this head there were five cervical vertebræ (the first to the fifth). These were complete except for a small fragment which had been shaved off, apparently by a knife, from the under surface of the fifth vertebra.

The sorting out of the parts indicated that the remains represented at least the bodies of two persons, both apparently well developed and well nourished. As a matter of convenience the parts were designated according to their assignments to one or other of the bodies as determined by the reconstruction.

Nature of the mutilations and dismemberment.

Both heads had been mutilated by the removal of the ears, eyes, nose, lips, and skin of the face ; and teeth had been extracted,

probably after death. The terminal joints of the fingers had been removed from two of the hands. A large amount of tissue had been removed from the surfaces of the bodies, and many of the missing portions might have led to the identification of the bodies, and have borne marks of violence or signs suggestive of the cause of death. The reader is referred to Figs. 22 and 23, which will give some conception both of the extent and character of the mutilations. The bodies had been neatly dismembered into portions convenient for transport, and the dismemberment had been effected by disarticulation with a knife. An obvious attempt had been made to efface any evidence which would lead to ready identification of the bodies, and it seemed evident that the person responsible had some medical as well as anatomical knowledge. This view was strengthened by the facts that there was very little damage to the joints, that the teeth had been extracted after death with some degree of skill, and that the uterus with its appendages had been removed from one of the bodies.

Summary of reconstruction of Body No. 2.

The thorax and pelvis were found to fit in the lumbar region, and it was proved by detailed evidence that they formed part of the same body.

Head No. 2 was found to fit the reconstructed trunk, and it was proved by detailed evidence that the cervical vertebræ, as a whole, formed a complete series which must have come from the same body. It was independently proved that Head No. 1 could not have belonged to the reconstructed trunk.

The nineteen portions of limbs (including the separate patellæ, the left foot, and right forearm with hand found separately) were assembled into two sets of limbs (*a*) a " longer set " and (*b*) a " shorter set." The " longer set " was proved to belong to Body No. 2.

As the result of the foregoing reconstruction, Body No. 2 was complete, so far as the skeleton was concerned, with the exception of the right foot and portions of fingers which had been removed from both hands (see Fig. 23).

Summary of reconstruction of Body No. 1.

When reassembled, this body was represented, so far as the skeleton was concerned, by :—

A head with four cervical vertebræ and a fragment from the fifth.
Right and left upper limbs with the exception of the shoulder girdles.
Right and left lower limbs, complete (see Fig. 22).

There was, however, no direct evidence that these parts all belonged to the same body since :—

The trunk was missing, but
They remained after the reconstruction of Body No. 2. ;
The limbs formed a consistent " shorter " set :

The general features of the head were not inconsistent with the features of the limbs ; and

All the features of the skull and the limb bones, which were used for the diagnosis of sex and age and for the estimation of stature, were entirely consistent with the conclusion that all belonged to the same body.

Sex of the bodies—

Body No. 1.

In the absence of the trunk, the determination of sex had to be based upon the examination of the head and limbs. These showed the following evidence of female sex :—

The secondary sex characters were obviously " female."

The mutilated scalp indicated that there had been an abundant covering of long hair which had been cut short.

There was no evidence of male beard hair.

The contours of the limbs were rounded, and the limbs had a typical female appearance.

The larynx was typically that of a woman.

Measurements of the sizes of the heads of the humeri and femora, together with other measurements of the femora in accordance with Pearson, showed figures which indicated female sex (see pp. 93 and 94).

Body No. 2.

When the reconstruction of the remains of this body was completed, no doubt whatever could be entertained about the sex of the body, since it included a pelvis which contained a portion of female genital organs, namely, vagina.

A point of interest lay in the fact that the general features of the skull and limb bones were in some respects equivocal for the diagnosis of sex, since at first sight they appeared to be of male rather than female type. By a careful assessment of the balance of the anatomical features of the skull and by measurements of the limb bones it became possible to give the opinion " probably female " before the anatomical proof was available that they belonged to the female trunk. Of the measurements of the humeri and femora, on the whole they were nearer to female standards, but it could not have been asserted that they were beyond the range of measurements that might be obtained from male bones (see p. 92).

Age of the bodies—

Body No. 1.

Absence of closure of any of the skull sutures indicated the age was not over thirty years.

Complete bony union of the spheno-occipital cartilaginous joint, probably recent, indicated almost certainly, that the age was below twenty-five years, and almost certainly over eighteen years.

Since all of the four wisdom teeth or third molars were unerupted

and fully calcified, as shown by X-ray examination, they were indicative of eighteen to twenty years.

Since all the epiphyses of the limb bones were united but some of them not completely fused, there was indication that the age lay between eighteen, and the extreme upper limit of twenty-five years.

The opinion expressed was that the age certainly lay between eighteen and twenty-five years, but that the most probable age lay between twenty and twenty-one years.

Body No. 2.

X-ray examination showed that all the epiphyses of the limb bones were completely united, therefore minimum age was twenty-two years, but the features of their fusion indicated that the minimum age was twenty-five years.

Examination of the sutures of the skull indicated an age probably between thirty and fifty-five years.

Examination of the spheno-occipital cartilaginous joint which was completely ossified, and of a section through the joint which showed that the ossification was of some years' standing, indicated minimum age was twenty-five years.

Bone changes associated with osteo-arthritis in some degree in all the cervical vertebræ and in the right hip-joint, considerable ossification in the thyroid and cricoid cartilages, the commencement of bony union between the right greater horn and body of the hyoid bone, together with a considerable degree of calcification in the costal cartilages were also present.

The probable age was given as lying between thirty-five and forty-five years.

Stature of the bodies—

Body No. 1.

The probable stature was estimated from the lengths of the limb bones by means of Pearson's formulæ for the calculation of the corpse length of female stature (see p. 96). Nine different formulæ, based upon the measurements of the bones in a wet or humid condition with cartilage, were employed, not because the succeeding eight added anything to the reliability of the first, but to demonstrate their consistency, which corroborated the conclusion that the different parts belonged to the same body. Two centimetres, the amount by which on the average the female body lengthens after death, were deducted, and the living stature was estimated at 149·5 centimetres. Since Pearson holds that, by the use of his formulæ, the stature of an individual is always subject to an error of not less than 2 centimetres, and may occasionally be as much as 8 centimetres, the living stature probably lay between 147·5 centimetres (4 feet 10 inches) and 151·5 centimetres (4 feet 11½ inches).

Body No. 2.

Since this body was capable of reconstruction, direct measurement was 5 feet 4¼ inches. After the deduction of ¾ inch (2

centimetres), the probable living stature was 5 feet $3\frac{1}{2}$ inches. As a test of the accuracy of Pearson's formulæ, in this instance,

FIG. 22

Body No. 1 reassembled.

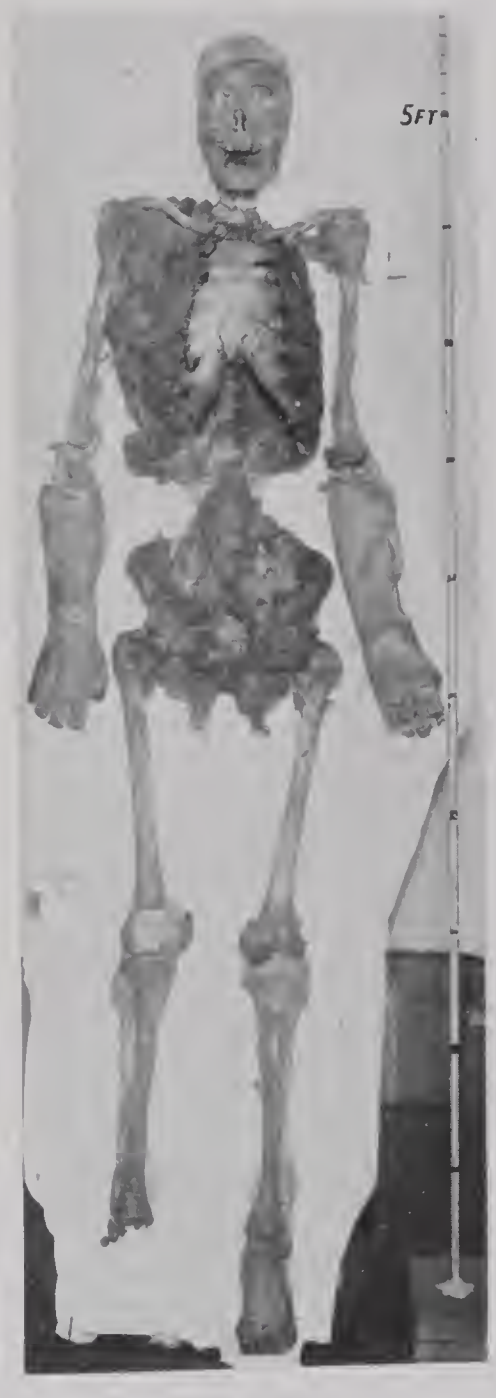


FIG. 23

Body No. 2 reassembled.

Note the scale, the lower end of which was adjusted to the level of the left heel, is marked at 6-inch intervals and in inches at the top.

the calculation of stature from the long bones, before reconstruction of the body was complete, was 155·2 centimetres.

155·2 centimetres − 2 centimetres (margin of error) = living

stature = 153·2 centimetres, which shows a discrepancy, but since Pearson has indicated that the error, on occasion, may amount to

FIG. 24

Positive portrait of Mrs Ruxton and negative of Skull No. 2 photographically superimposed by Professor J. C. Brash. Note the coincidence of the registration marks by means of which the superimposition was effected.

as much as 8 centimetres, if this figure be added to 155·2 centimetres the stature becomes (163·2 centimetres) 5 feet $4\frac{1}{4}$ inches, which is the same length as the reassembled body before correction for living stature. Brash has pointed out that this discrepancy indicates the

desirability of direct measurement in preference to calculation from the lengths of limb bones.

Identification of the bodies.

It is not possible to enter into the details of the case in this book, but it should be mentioned that certain of the articles associated with the remains found in the linn played important parts in the identification of the remains. The medico-legal aspects in connection with the identification are appended in tabular forms on pp. 107 and 108.

FIG. 25

Casts composed of a mixture of gelatin, glycerin, and zinc oxide. Left—Left foot Body No. 1. Right—Left foot Body No. 2. (Ruxton case.)

The methods adopted for the estimation of sex, age, stature, and reconstruction of the bodies, in this case, have been described in some detail, since it is felt that it is without precedent as an example of the difficulties which may be encountered in the investigation of problems connected with mutilation and dismemberment of bodies.

Subsequent to the discovery of the remains, it was ascertained that Mrs Ruxton and her nursemaid, Mary Rogerson, had disappeared from the house of Dr Ruxton in Lancaster on September 15, 1935. They were never seen alive again, and Dr Ruxton was arrested and charged with murder. In his house, human blood-stains were found in the bathroom, on the stair rails and banister, on stair carpets and pads, and on a suit of clothes belonging to him. Particles of human tissue were found in the drains. He was found guilty of murder and hanged.

Age	Twenty years (October 8, 1935).	Certainly between 18 and 25. Probably between 20 and 21.
Stature	About 5 ft.	4 ft. 10 in. to 4 ft. 11½ in. (without shoes).
Hair	Light brown.	Hair from scalp and body light brown.
Eyes	Blue. "Glide" in one.	Removed.
Complexion	Light. Freckles on nose and cheeks.	Ears, nose, lips, and most of skin of face removed; complexion of remainder of skin consistent.
Teeth	Old extraction of six teeth, four of them named.	Old extraction or loss of eight teeth, including the four named.
Neck	Short neck.	Very small larynx very highly situated.
Tonsils	Subject to tonsillitis.	Microscopic evidence consistent with recurrent tonsillitis.
Vaccination marks	Four on left upper arm.	Four on left upper arm.
Finger-nails	Maidservant.	Trimmed but not regularly manicured: scratches indicating some form of manual work.
Scars	1. Abdominal scar—appendix operation.	1. Trunk missing.
	2. Operation for septic thumb which had left a mark.	2. Terminal segment right thumb denuded of tissue; no scar on left thumb.
Identifying peculiarity	Birth-marks (red patches) on right forearm near elbow.	Skin and soft tissues removed from upper third of forearm, and lower two-thirds of front only.
Size and shape of feet	Left shoe as evidence.	Cast of left foot fitted shoe.
Form of head and face	Two photographs in different positions.	Outlines of photographs of skull in same positions fitted.
Finger-prints	Numerous imprints from house at 2 Dalton Square.	Positively identified as the finger-prints of both hands and palmar impressions of left hand.
Breasts	Age 20, unmarried.	Single breast, appearance and structure consistent.

	Isabella Ruxton.	Body No. 2—Female.
Age	34 years 7 months (October 3, 1935).	Certainly between 30 and 55. Probably between 35 and 45.
Stature	5 ft. 5 in. to 5 ft. 6 in.	5 ft. 3½ in. (without shoes).
Hair	Soft texture, mid-brown with patch of grey slightly to right of top of head.	Scalp completely removed; a few adherent hairs light to medium brown. Eyelashes dark brown. Available body hair mid-brown.
Eyes	Deep-set; grey-blue.	Removed.
Complexion	Fair.	Ears, nose, lips, and skin of face removed.
Teeth	Denture replacing three named teeth in gap which would show during life; old extraction of one other named tooth.	Old extraction or loss of fifteen teeth, including the four named.
Fingers and nails	Long fingers. Recognisable nails—bevelled, brittle, growing tight at corners, rounded at ends, regularly manicured.	Terminal segments of all fingers removed.
Legs and ankles	Thick ankles. Legs of same thickness from knees to ankles.	Soft tissues removed from legs.
Left foot	Inflamed bunion of left big toe.	Hallux valgus of left foot; tissues removed over metatarso-phalangeal joint down to bone and joint opened. X-rays showed exostosis of head of metatarsal.
Size and shape of feet	Left shoe as evidence.	Cast of left foot fitted shoe.
Nose	Bridge uneven.	Removed, but bone and cartilage arched.
Form of head and face	High forehead, high cheek-bones, rather long jaw. Two photographs in different positions.	Corresponding features. Outlines of photographs of skull in same positions fitted.
Breasts	Pendulous breasts: three children.	Appearance and structure of pair of breasts consistent.
Uterus	Three children.	Separate uterus. Could not be assigned but structure consistent.

THE DOBKIN CASE

The medical evidence led in the case against Harry Dobkin, in 1942, who was charged with the murder of his wife, tried at the Old Bailey, London, and found guilty, recalled the technique first employed in the Ruxton case. Put briefly, the following were the facts. A demolition worker clearing debris, following bombing damage, found in a cellar of a chapel in Kennington Lane, London, the remains of a woman partly covered by a paving stone. Medical evidence was to the effect that the body, which had been cut up by someone without anatomical knowledge, had been dead for about twelve to fifteen months, that death had been due to strangulation, and that twenty-four points in the proof of identity had been discovered. Following dismemberment of the body the parts had been buried in lime, and these included the skull. Apart from the assessment of sex, age, and stature, a dentist identified his fillings in the teeth of the upper jaw, the uterus showed a large fibroid, and inquiry at two London hospitals showed that Mrs Dobkin had been examined at their out-patient departments, had been diagnosed as suffering from a fibroid tumour of the uterus, and had refused operation. Mrs Dobkin's dentist was satisfied that following extraction, some buried roots of teeth were still present, and radiological examination of the jaw of the victim showed the roots in the expected position. A photograph of the dead woman was superimposed on a photograph of the skull found in the cellar of the chapel and the superimpositions fitted. This is the second occasion on which this process had been employed. The technique originated in the Ruxton case was used in this instance, and acknowledged at the trial.

It is commonly and erroneously believed that quicklime accelerates the destruction of human remains. Experiments have shown the contrary, namely, that lime, whether quick or slaked, has a definite preservative effect.

Occasionally portions of human remains are submitted by the police for examination and for expression of opinion on their significance and bearing in relation to the possible commission of crime. Sometimes an examination will show quite clearly that they are merely anatomical specimens which have been disposed of in an irregular way. The following case is intended to illustrate the fact that the finding of human remains, even in the most unlooked-for places, although not necessarily indicative of the commission of crime, demands the closest investigation.

The specimens consisted of a human hand and a portion of tibia. These were found in a water-barrel at a housing scheme which was in the course of erection. Examination, including a radiological examination, showed that :—

The hand was probably that of a female subject between thirty and forty years of age.

It had been artificially preserved both by fluid and "mass" injection, the process employed in the preservation of anatomical subjects for the purposes of dissection and operative surgery.

It had been disarticulated at the wrist-joint, but the condition of the hand did not conform to the condition which would result from anatomical dissection, although it was consistent with disarticulation performed in a class of operative surgery, with added lesions the objects of which were not apparent.

The fractures present were most probably of post-mortem origin, since X-ray examination did not show any evidence of a healing process, but the likely interval of time which had elapsed since their occurrence could not be estimated.

From the general appearance of the tissues, notably the fat, the specimen might have remained in its present state for many years, since it was similar in appearance to a specimen of a body, preserved for anatomical purposes, after a period of twenty-six years, with which it was closely compared.

The portion of tibia, from its general characters, appeared to have belonged to a second body, probably male, and from radiological examination, to a subject of older years than that of which the hand had formed a part.

From the presence of articular cartilage, fragments of ligamentous-like structure, and irregular portions of periosteum, still adherent, it was probable that these might have been fixed as the result of methods employed for the preservation of anatomical subjects, since the specimen had the appearance of being one of fairly long standing.

Hairs and fibres.

In the examination of blood-stains on weapons, wood, or upon any other surface, hairs or fibres present should be set aside for separate examination, as they may prove of value in elucidating certain points in evidence at the trial of accused persons. Specimens of hair removed from various parts of motor cars are frequently sent by the authorities for examination, and comparison with the hair on the head of deceased persons who have been the victims of road accidents. In sexual offences, especially in rape and bestiality, an examination of hairs frequently becomes important. Fur thefts form another class of case in which an examination of hairs is requested, and there are many other forms of crime or offence in which this investigation is necessary. We have undertaken the examination of hairs, at the request of the authorities, in a large number of different types of cases. In rare instances, hairs may show vivid colours, red, blue, purple, orange, or other colour, due to contact with dye which has emanated from some brilliantly coloured garment worn by a deceased person. We found this condition in a case of murder. The female victim wore some highly coloured clothing which, having become soaked with rain, had liberated the

Fig. 26

Various hairs.

1. Sheep.
2. Cat.
3. Goat.
4. Dog.
5. Horse.
6. Cow.
7. Rat.
8. Rabbit.

dye. The condition of the hair proved important in assisting the police to arrive at an opinion regarding the approximate time of occurrence of the crime, following investigations in connection with weather conditions which obtained on the night in question. With experience, it is not difficult to differentiate between the hairs of man and those of the lower animals. If a hair-root has been forcibly extracted the bulb will be irregular in form, due to rupture of the sheath, and will show an undulating surface, together with excrescences of different shapes and size. A naturally shed bulb has a rounded extremity, a smooth surface, and most probably will show signs of atrophic or fatty degeneration, especially in elderly persons.

In view of the number of cases in which the finding of hairs on various productions has presented itself, and the value of these in relation to the identity of accused persons, a close investigation of the hairs of the entire mammalian class of animals was made. The investigation was approached from three aspects, namely: the study of hairs belonging to the mammalian group, excluding human hairs; the examination of human hairs from different parts of the body; and the examination of cross-sections of all the hairs in the preceding groups. The object of the research in the above scheme was to determine whether the hairs of animals belonging to the same zoological order, sub-order, or family showed characteristics so similar that they could be depended upon for the identification of the animal order, sub-order, or family to which the hair belonged. In the case of human hairs, to find whether there were any specific differences in their appearance with regard to sex, age of the subject, site on the body from which they were taken, and pigmentation. The examination of transverse sections supplemented the information previously obtained from examination in the longitudinal plane.

A collection of hairs was made from many sources, and some 1700 photomicrographs of specimens were taken and classified.[20]

It is hardly necessary to do more than remind the student that the average hair may be differentiated into three layers, an outer layer or cuticle composed of a single layer of cells, a middle or cortical layer of varying thickness composed of longitudinal fibres and a varying amount of pigment, and an inner layer or medulla made up of variously shaped cells usually containing a considerable amount of pigment. The principal conclusions drawn from this investigation were: First, the appearance of the majority of animal hairs is such as to permit the identification of the order, sub-order, or family of the animal to which they belong; second, the characters of the hairs of animals belonging to all the mammalian orders, excepting a few belonging to the Primates or monkey families, are such that their naked-eye appearances are likely to reveal that they do not belong to the human race, and that in the odd instances mentioned, a detailed examination of the structure will show that the pigment in the cortical layer is coarser, and the medulla broader and less regular than in human hair; third, that where doubt as to differentiation between animal

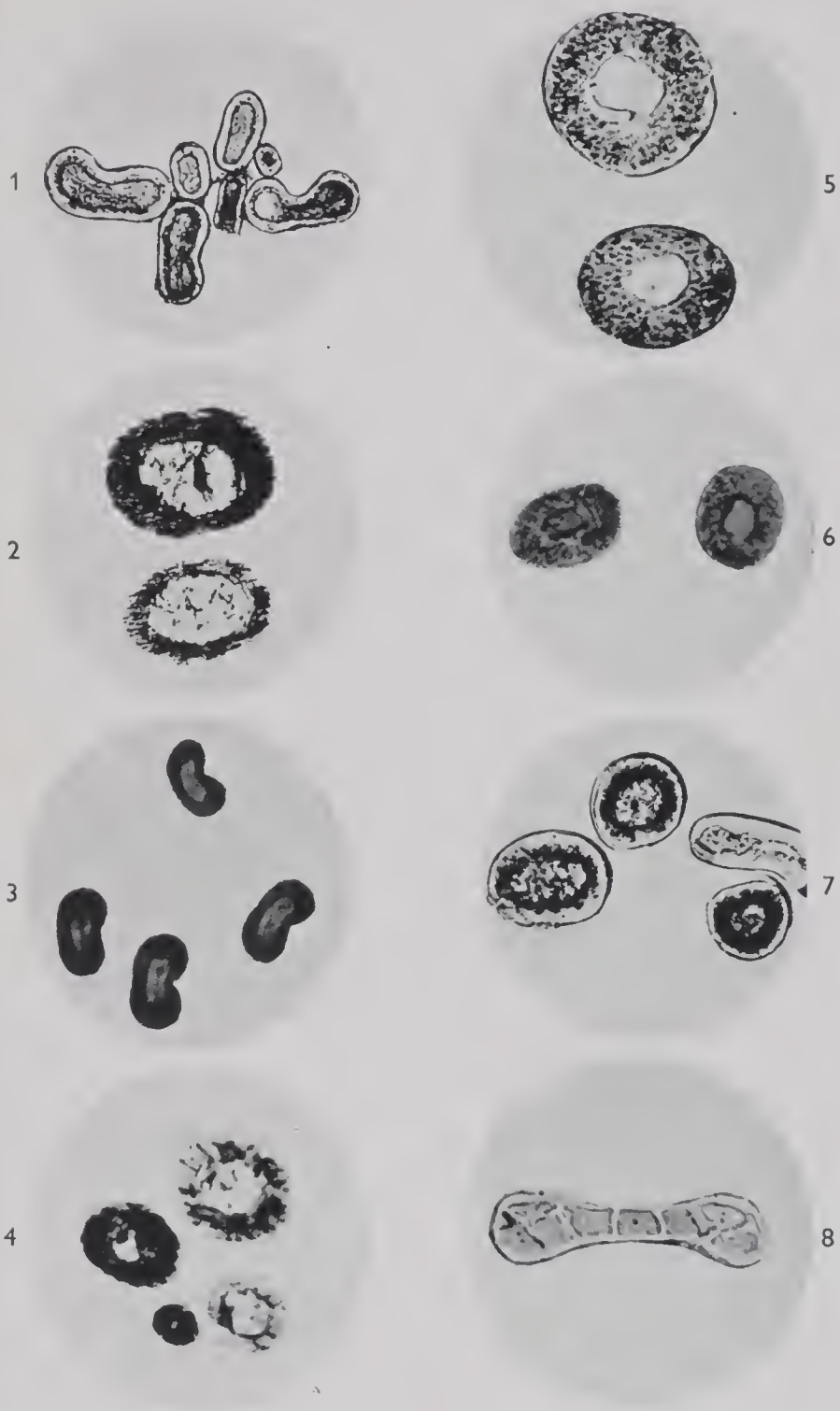


FIG. 27

Cross-sections of hairs.

1. Squirrel.
2. Cat.
3. Goat.
4. Dog.
5. Horse.
6. Cow.
7. Rat.
8. Rabbit.

and human hair should arise from examination in longitudinal plane, the appearance of the transverse or cross-section is likely to settle the question, by reason of the fact that the core or medulla of human hair is invariably smaller than that of any of the other Primates ; fourth, that as a general rule the breadth of the medullary layer is less in the hairs of the higher than in those of the lower-grade mammals.

It may be possible from the examination of human hair to determine the sex of the person in some instances only. On the other hand, it is comparatively easy to determine whether hair is natural or has been dyed. Hairs from different parts of the human body sometimes present differentiating characteristics, as, for example, the hairs of moustache, eyebrow, eyelash, and pubic hair.

From the medico-legal point of view, the examination of hairs should include the following particulars : (1) colour to naked eye and on microscopic examination ; (2) length, ascertained by actual measurement ; (3) texture ; (4) approximate breadth, by micrometer ; (5) hair-tip or hair-end characters, whether intact, cut, or torn ; (6) condition of bulb if present, whether forcibly pulled out, degenerated, or cut across by a sharp instrument or crushed by a blunt instrument ; (7) character of cuticle, extent and character of cortex, presence or absence of medulla and, if present, the character and breadth ; (8) whether hairs are dyed or undyed ; (9) contour of transverse sections in respect of points set down in No. 7.

It is advisable to build up a collection of animal hairs, especially those of domestic animals, so that comparison with given specimens may be readily possible.

The dyeing of hair is a common practice. Hair may be made darker or lighter. The number of different hair dyes and tinting materials available is innumerable, and a large variety of shades can be produced by their use. Alteration in the colour of hair may be effected in a variety of ways by the use of decolorants, vegetable dyes, dyes with a metallic base, or by synthetic organic dyes. Hydrogen peroxide is the most commonly employed decolorant, henna the most frequently used vegetable dye ; and of the dyes with a metallic base, lead and bismuth form the bases in many instances. Of the synthetic organic dyes, phenylenediamine is the principal example, and serious cases of dermatitis together with toxic manifestations have been produced by its use (see p. 611). The detection of hair dyes is important in some cases, and its presence is indicated usually by a lack of uniformity in the colour of the hair, which is often lacking in lustre and brittle in character. The natural colour of the more recently grown hair at the roots strikes a contrast with the shade of the remainder of the hair on the scalp, and this is well demonstrated by the use of filtered ultra-violet light, when dyed hair appears lustreless and like tow. The use of infra-red photomicrography, especially when the hair has been dyed or bleached, is an important adjunct in the examination of hair. Comparison of the colour of the hair on the head with that of the hair

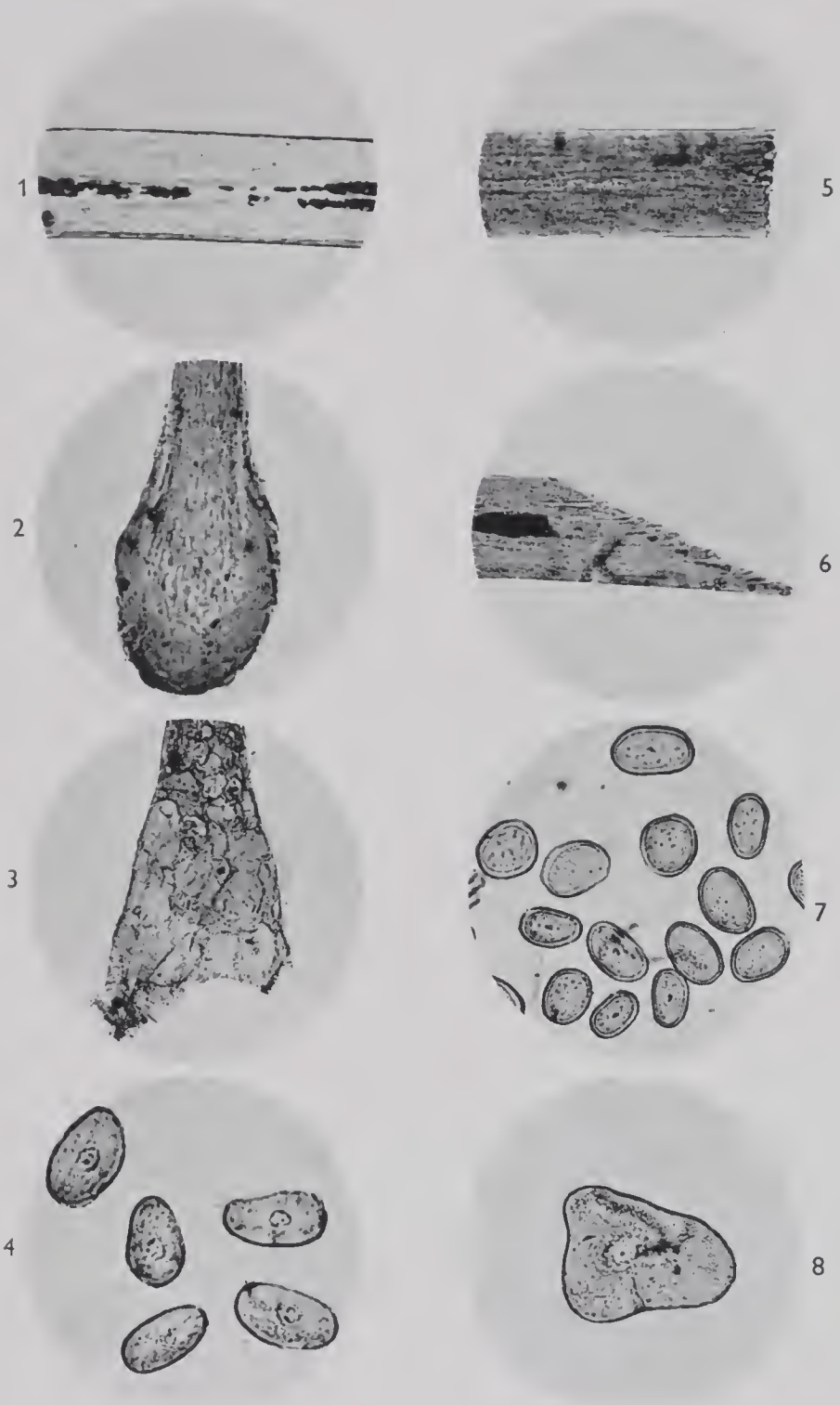


FIG. 28

Certain hair features.

1. Grey hair.
2. Healthy bulb.
3. Degenerate root.
4. Sections of pubic hair.
5. Hair cut by sharp blade.
6. Severed by blunt blade.
7. Sections of head hair.
8. Section of moustache hair.

on other parts of the body will frequently indicate the presence or absence of dye in the former. The scalp may occasionally show staining due to the dye which has been employed. On applying appropriate chemical tests, the nature of many of the dyes commonly used can be detected. A portion of the hair may be steeped in diluted nitric acid, and this will give a solution which can be tested by qualitative chemical methods. On examining dyed hairs microscopically, the intimate structure appears hazy and shows a uniformity in general shade which is not seen in hairs of natural

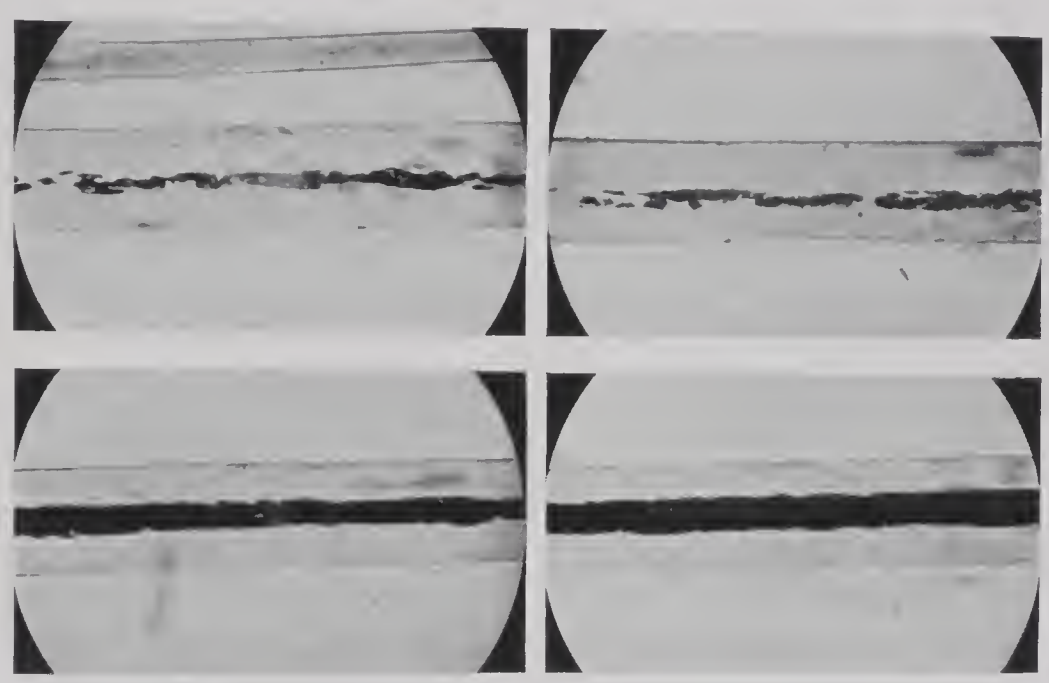


FIG. 29

Comparison of hairs adherent to a motor car (left), with those removed from the head of a person fatally injured (right).

colour. This haziness is more marked when the darker dyes have been employed, but when bleaching substances have been used, such as hydrogen peroxide, the substance of the hair shows a typical bleached appearance which differs from the character of natural white hairs.

The undernoted are but a few examples of the types of cases in which an examination of hairs has been made :—

1. Blood-stains were found upon the direction indicator of a motor car, and hairs were adhering to the offside front mudguard and the frame of the offside front door. When these hairs were compared with the head hair of a deceased man the general and detailed characters were so similar as to be consistent with a common source (see Fig. 29).

2. In a case of theft of silver fox furs, hairs taken from a sack, in which the stolen pelts were alleged to have been placed, were compared with hairs removed from the recovered furs. The examination proved conclusively that the suspect hairs conformed in all respects with the hairs taken from the silver fox pelts for examination (see Figs. 30 and 32).

3. In a case of attempted rape, hairs were found entangled on the middle button of the left cuff of the alleged assailant's jacket.

FIG. 30

Comparison of silver fox hairs. Top—Hairs from silver fox furs alleged to have been stolen. Middle and Bottom—Hairs found in cartons and sack thought to have contained the stolen furs (see Fig. 32).

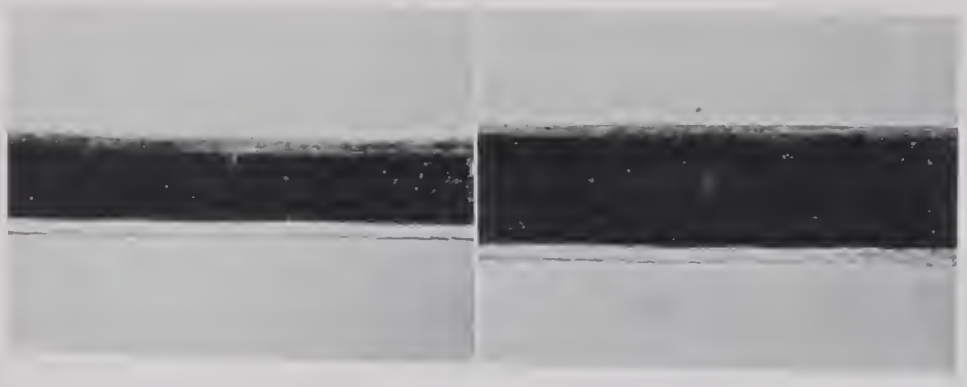


FIG. 31

Hair comparison in bestiality case. Left—Hair from clothing of accused man. Right—Hair from hind quarters of a mare.

These were compared with the head hairs of the assaulted woman, and the characters found were consistent with a common source. A point of interest was that the four hairs around the button showed marked irregularities, consistent with the effects of hair-waving,

and the same characters were present in the head hair due to that cause.

4. A man was charged with bestiality with a female goat. On the clothing of the accused, hairs were found. Hairs were obtained from the region of the sexual parts of the animal. The hairs from these sources were examined, and found to correspond closely both in longitudinal plane and in cross-section.

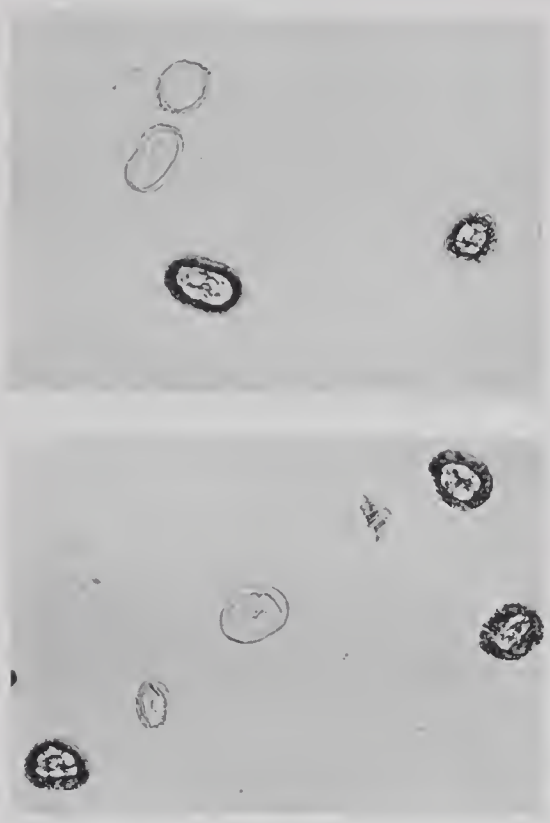


FIG. 32

Comparison of transverse sections of silver fox hairs. Top—Hairs from furs alleged to have been stolen. Bottom—Hairs from cartons thought to have contained the stolen furs (see Fig. 30).

5. Assaulted and robbed in a tenemental entry, during the "black-out," a seaman slashed at his assailants with a knife. The police received information that a man had been assaulted and was lying in an infirmary. They were also informed by the man who had been robbed that he had assaulted a man by slashing him about the head and face with a knife. At the site of the assault the police found two locks of hair and a quantity of blood. They traced the blood-staining to a tramway stopping place where they were informed by a witness that he had assisted a man suffering from facial wounds on to a tramway car. The man was making his way to an infirmary. A lock of hair was obtained from the man detained in the infirmary. Microscopical comparison of the three locks of hair showed general and detailed structural characters which were fully consistent with the three sets of specimens having been derived from a common source.

6. In a murder case, a comparison was made between short beard-hairs found upon the cutting edges of a safety-razor blade, discovered among bracken, and three-day-old hairs shaved from the face and chin of the accused man while in custody. Gross and detailed structure of these specimens were so similar that they were consistent with a common source of origin.

TECHNIQUE FOR THE PREPARATION OF HAIRS FOR MICROSCOPIC EXAMINATION[21]

Preparation of specimens for examination in longitudinal plane.

Prior to mounting the specimens, it is necessary to use some

cleansing reagent to remove adherent debris which may mask structural detail. One of the most efficient consists of equal parts of ether and absolute alcohol. The specimens should be placed in a small test tube with this solution and gently shaken. The hairs are then removed from the tube and dried between sheets of filter-paper. Before mounting, they should be steeped in a good quality benzol or oil of turpentine, which acts as a clearing agent. After drying, the specimens are mounted preferably in "Euparal" or in Canada balsam, long cover-slips applied, and the slides allowed to stand for twenty-four to forty-eight hours, if time will permit, for clearing purposes. By so treating the hairs, they become permanent specimens. In some cases, debris adherent to human hairs may prove valuable by giving a hint as to the site of the body from which the hairs have come. For example, the presence of sweat, vaginal or menstrual secretion, seminal fluid, fæcal or food material, nasal secretion or cerumen, are important indications. It is therefore expedient to make a routine preliminary microscopical examination of the hairs before washing and finally mounting them.

Preparation of cross-sections of hairs.

The following are the stages recommended :—

1. Cleanse hairs in equal parts of ether and absolute alcohol.
2. Place in a solution of 2 per cent. alcohol to which has been added an equal quantity of 5 per cent. liquor ammonii fortis or ammonium hydrate. Steep for five minutes.
3. Soak in 10 per cent. potassium hydrate for one or two minutes. Maintain temperature at 50° C.
4. Wash specimens in a solution composed of equal parts of 5 per cent. sulphuric acid and absolute alcohol for some minutes.
5. Wash in xylol or benzol for a few minutes.
6. Dry hairs between sheets of filter-paper.
7. Steep hairs in liquid paraffin-wax for fifteen minutes.
8. Block specimens in paraffin.
9. Cut sections about 8 to 10 μ in thickness.
10. Float sections on water at temperature of 40° C.
11. Place on albuminised slides and put in incubator at temperature of 37° C. for twenty-four hours.
12. Dissolve paraffin-wax with xylol, dry slides, and add Canada balsam, or preferably, "Euparal."

The objects of stages 1 to 5 are to cleanse, soften, and dehydrate the hairs ; step 7, to ensure adherence of paraffin during blocking stage ; step 10, to uncurl the sections ; and step 11, to ensure adherence of the sections to the slides.

A variety of paraffin-waxes of different melting-points should be available in the ovens, since success lies in the selection of a wax of suitable melting-point, having regard to the thickness and

consistency of the hairs to be sectioned. For general purposes a wax of melting-point at 52° C. should prove satisfactory. During the various stages, the hairs are immersed in the form of a bundle. The bundle is made by binding the strands together, at intervals, with fine silk thread. When only a single hair is available, the various stages may be carried out in a watch-glass, and the specimen embedded by means of fine forceps.

Kneberg [22] has introduced a method for embedding hairs for sectioning purposes. The hairs are mounted on a piece of three-ply drawing cardboard, 4 by 3 centimetres. Slanting cuts are made in the ends of the cards, and an opening, 2 by 1 centimetre, made in the centre of the card. The hairs are slipped through each end, and are then held taut by small rubber-tipped clamps until the glue, which fastens them, is dry. Fish gelatin dissolved in warm water is recommended, since it is insoluble in the alcohols. When the specimens have been put through the various stages, and are finally embedded, the block is cut down leaving only the part containing the hairs. It is also advised that, during the cutting of sections, the microtome blade should be kept moist with 75 per cent. alcohol to obviate curling of the sections.

Examination of cuticular scales.

In 1937, Professor Alan R. Moritz, when working in our laboratories, made investigation in connection with cuticular scale impressions. Experimentation then showed that the most simple and suitable method for making such impressions was to use a plastic film of cellulose acetate in the form of finger-nail varnish diluted with an equal quantity of amyl acetate. A drop of this solution should be spread thinly over the surface of a microscope slide, and the cleansed hair, with some tension exerted upon its ends, should be laid over the wet film. After the film has dried, usually in about ten minutes, the hair is stripped off the slide by a quick movement. The scale impressions are then examined microscopically.

This method is very useful in the instance of hairs which are deeply pigmented and when examination of the cuticular scales is desired.

The colour of the varnish affords clear definition of the structural detail and facilitates photography.

In 1945, Chief Inspector George Maclean and Detective-Inspector Charles McNeill, both of the Identification Bureau, City of Glasgow Police, experimented with all the known formulæ for the media employed in the preparation of cuticular casts, and finally originated the following formula :—

Ethyl lactate . . .	5 millilitres
Amyl acetate . . .	39 ,,
"Celloidin" . . .	6 grammes

The excellence of the results obtained, in addition to the fact that

the solution is equally suitable for hairs of all textures, justifies the recommendation that this plastic medium should invariably be used in making hair casts (see Fig. 33).

In a recent murder case we employed photomicrographs of casts of the cuticular cells, obtained by use of the above described medium, as an additional demonstration of obvious differences in the instance of hair specimens obtained from two sources.

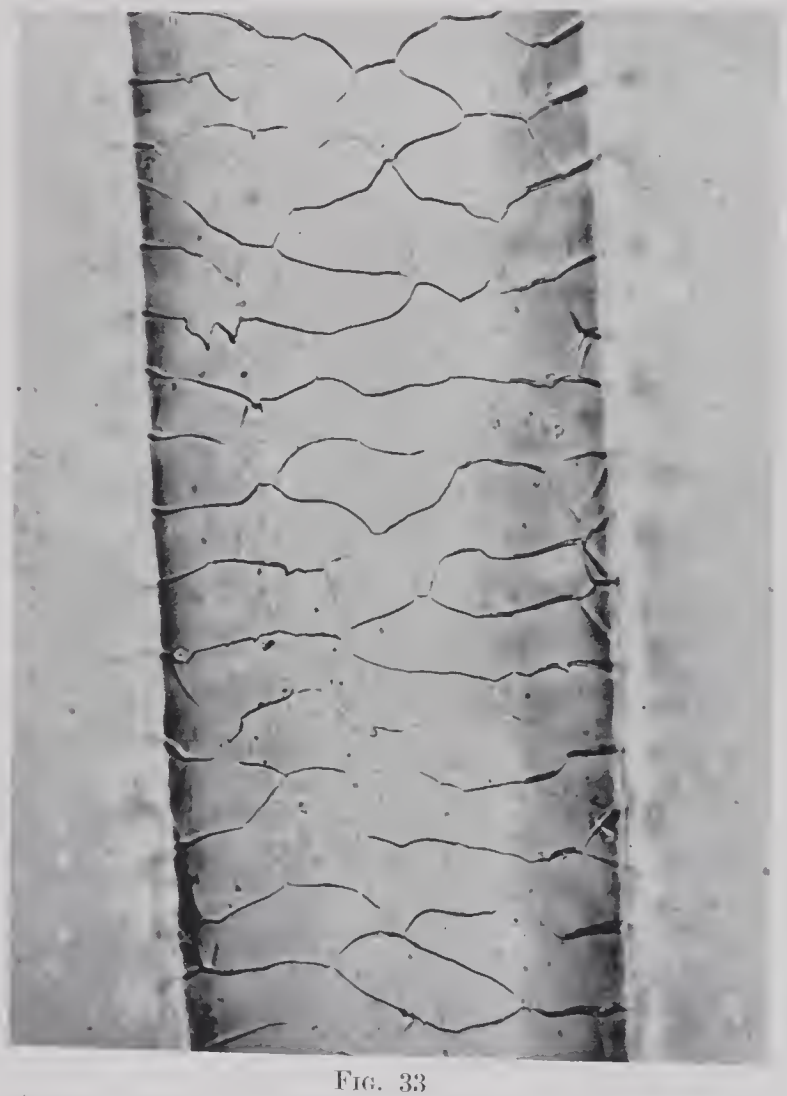


FIG. 33

Case of bestiality. Photomicrograph of cuticular cast of horse hair.

(Method of Maclean and McNeill.)

Davidson and Taylor [23] describe a method for staining the cuticular cells of hair. The root and tip of the hair are removed, the hair is placed in equal parts of absolute alcohol and ether for fifteen minutes, and then washed thoroughly in distilled water. Next, the specimen is placed in distilled water in a vacuum embedding oven at room temperature, and a pressure of 18 to 30 mm. mercury is maintained for two hours. This process ensures the withdrawal of air from the medullary part of the hair. The specimen

is now bleached in a solution composed of 90 volumes hydrogen peroxide, diluted 1 in 3 with distilled water, 50 millilitres, 5 per cent. aqueous ferric chloride, one drop, and excess of liquid ammonia which is added immediately before use. The stage of bleaching occupies a period of from fifteen minutes to twelve hours, depending upon the degree of pigmentation of the hair. The specimen is washed thoroughly in distilled water and stained for at least fifteen minutes in 1 in 100 carbol fuchsin (Ziehl Neelsen) in distilled water in a vacuum embedding oven at room temperature and at a pressure of 18 to 30 mm. mercury. The hair is then washed in distilled water, decolorised in absolute alcohol for five to ten minutes, cleaned in benzene and mounted in Canada balsam. The writers assert that this method permits the study of the cuticular scales and the longitudinal section of the hair in detail, and that, with the use of suitable plates and colour filters, photographs showing maximum detail and contrast can be obtained.

Examination of fibres.

Cotton.

This is the seed hair of the genus Gossypium. When undyed, it has a whitish colour.

Flax.

This is the bark of Linum usitatissimum and has either a grey or cream colour. When the fibres are damped, they curl or turn in a clockwise direction.

Hemp.

This is the bark of Cannabis sativa and is creamy-white or grey. When the fibres are damped, they curl or turn in an anti-clockwise direction.

Jute.

This is the bark of Corchorus capsularis or Corchorus olitorius. It is yellowish-brown or yellowish-white.

Manila hemp.

This is made from the leaf of Musa textilis and is of light brown colour.

Sisal hemp.

This is made from the leaf of the genus Agave and has a light yellow colour.

We have been requested to examine fibres in a large variety of cases, for example, in cases of burglary, when fibres from the garments of accused persons have been found adherent to the surfaces of broken windows, or when incriminating fibres have been discovered

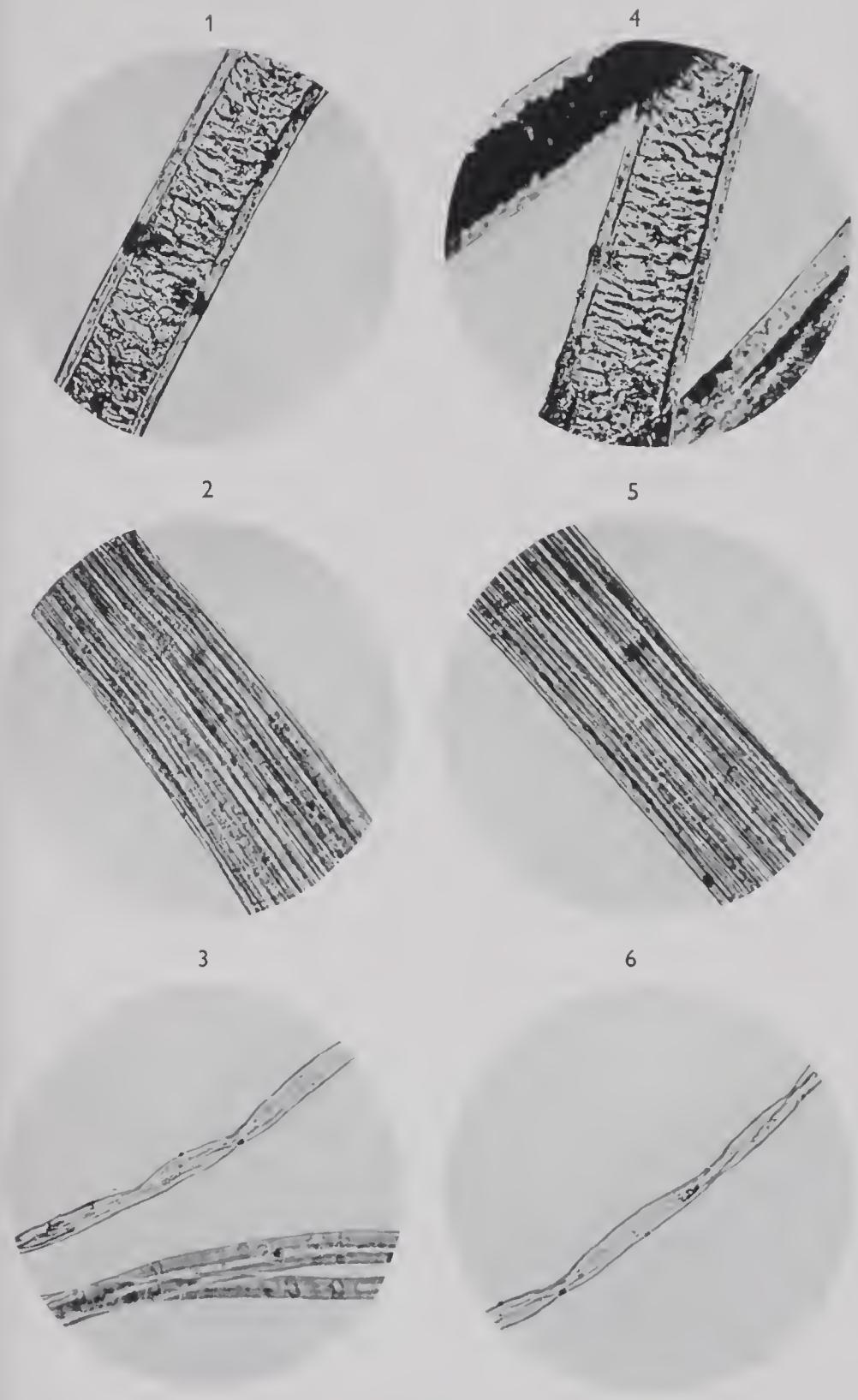


FIG. 34

Comparison of fibres.

1. Fibre from large foot mat.
2. Fibre from large rug.
3. Fibres from large rug.
4. Fibres on trousers worn by accused.
5. Fibre on black sock worn by accused.
6. Fibre from under surface of heel of right boot worn by accused.

CHARACTERS AND IDENTIFICATION OF FIBRES

Cotton.	Flax.	Hemp.	Manila Hemp.	Jute.	Wool.	Silk.	Linen.
Flattened, twisted fibres with thickened edges. Irregularly granulated cuticle. Blunt apex. No transverse markings. Fibres show a spiral twist. Fibres swell in a solution composed of copper sulphate and sodium carbonate dissolved in ammonia. Insoluble in strong sodium hydroxide, but soluble in strong sulphuric acid and partially soluble in hot, strong hydrochloric acid.	Apex tapers to fine point. Transverse sections are polygonal, and show a small cavity. The fibres consist of cellulose and give blue or bluish-red colour when treated with a weak solution of potassium iodide saturated in iodine and sulphuric acid. The fibres, which show transverse lines and are usually seen in grouped formation, dissolve in a solution composed of copper sulphate and sodium carbonate in ammonia.	Fibres show transverse lines and consist of cellulose. Large oval cavities seen in transverse sections. Ends usually blunt, and there is often a tuft of hair at the knots. Stain bluish-red with phloroglucine and yellow with both aniline sulphate and with a weak solution of potassium iodide saturated with iodine and sulphuric acid.	Fibres are smooth without transverse or longitudinal marking. The cavities are large and uniform. The walls are lignified. The tips are fine-pointed.	Fibres are quite smooth without either longitudinal or transverse marking. The fibre has a typical, large cavity, which is not uniform in size, but varies with the degree of contraction of the walls of the fibres which are lignified. The ends are blunt. The fibres are stained red with phloroglucine and yellow with aniline sulphate, also with iodine and sulphuric acid.	These fibres can easily be distinguished from vegetable fibres, since the former show an outer layer of flattened cells and imbricated margins. The interiors are composed of fibrous tissue, but sometimes the medulla is present. They do not dissolve in a solution composed of copper sulphate, sodium carbonate and ammonia. Stain yellow with iodine and sulphuric acid also with picric acid. Do not dissolve in sulphuric acid. Smell of singeing on burning.	Manufactured silk is almost structureless, microscopically. Fibres stain brown with iodine and sulphuric acid, yellow with picric acid. They dissolve in sulphuric acid, but dissolve slowly in a mixture of copper sulphate, sodium carbonate and ammonia.	Fibres are straight and taper to a point. Cortical area shows transverse lines which frequently intersect, simulating a jointed appearance. The medullary region shows a thin, dense line. They do not dissolve in concentrated sulphuric acid. If placed in 1 per cent. alcoholic solution of fuchsin and then in a solution of ammonium hydroxide, they assume a bright red colour.

upon, or close to, safes which have been blown, and also in cases of fatal motor car accidents, in connection with cars thought to have been involved. In one case, a bundle of fibres, similar to those composing a carpet at the scene of a burglary, was found embedded in debris attached to the heel of footwear worn by a man accused of the burglary (see Fig. 34).

When making an examination of fibres, it is advisable to examine them after bleaching, in addition to examining them in their natural state, since detail, masked by colour, may then be seen clearly. For the purpose of bleaching, only a portion of the fibres should be utilised, and hydrogen peroxide or, preferably, a solution composed of equal parts of bleaching powder and sodium carbonate dissolved in water should be used.

The table on p. 124 will be found useful in the identification of various fibres.[24]

References

1. "Signaletic Instructions," American Edition, 1896, translated by McClaughry.
2. "Classification and Uses of Finger-Prints," 1928, Henry.
3. "Single Finger-Prints," H.M. Stationery Office, London, 1930, Battley.
4. "Medico-Legal Aspects of the Ruxton Case," Livingstone, Edinburgh, 1937, Glaister and Brash.
5. Brit. Med. Jour., Vol. I, 343, 1906.
6. Med. Leg. Rev., Vol. XIII, Part II, 99, 1945. Also Brit. Dental J., 78, 76, 1945.
7. J. Pediatrics, 31, 142, 1947, Bergman.
8. Brit. Med. Jour., Vol. II, 620, 1937, Kerr.
9. Ibid., 568, 1905, Spokes.
10. Jour. Anat., London, Vol. LXVII, 118, 1932, Flecker.
11. See Reference No. 4.
12. Boll. del. Soc. Ital. de. Biol. sper., 22, 151, 1946, Franchini.
13. Jour. Anat., Vol. LXXII, 633, 1938, Fawcett.
14. Philos. Trans. Roy. Soc., Vol. CLXXXIX, 135, 1897.
15. "A Study of the Long Bones of the English Skeleton," Part I. "The Femur." Drapers' Company Research Memoirs, Cambridge University Press, 1919, Pearson and Bell.
16. Amer. Jour. Anat., Vol. IV, 19, 1905, Dwight.
17. Cosmos, Revue Encyclopédique Hebdomadaire des Progrès des Sciences, Paris, 386, 22, 1863, Luca, and "Taylor's Principles and Practice of Medical Jurisprudence," 10th edition, Vol. I, 157, 1948, Churchill, London.
18. Philos. Trans. Roy. Soc., A, Vol. CXCII, 169, 1899, Pearson.
19. See Reference No. 4.
20. "Hairs of Mammalia from the Medico-Legal Aspect," published by the Egyptian Government, 1931, Glaister. "Recent Advances in Forensic Medicine," 2nd edition, Churchill, 1938, Smith and Glaister. "Le Poil de l'Homme et des

Animaux," Paris, 1910, Lambert and Balthazard. "Das Haupthaar nnd seine Bildungsstätte bei Rassen des Menchen." Berlin, 1912 (also Supplement, 1915).

21. "Reeent Advanees in Forensic Medicine," Churchill 1938, Smith and Glaister.
22. Amer. Jour. Physieal Anthropology, Vol. XX, 51, 1935, Kneberg.
23. Quekett Microscopical Club, Series IV, Vol. 1, No. 6, 289, 1943, Davidson and Taylor.
24. Additional information can be obtained from "Forensic Chemistry," Arnold, 1946, Lucas; and from "Animal Fibres of Industrial Importance: Their Origin and Identification," Wool Industries Research Association, 1939, Wildman; "Textile Fibre Atlas," American Wool Handbook Company, New York, 1942, Von Bergen and Krauss.

CHAPTER IV

THE MEDICO-LEGAL ASPECTS OF DEATH

DEATH may be defined as complete and persistent cessation of respiration and circulation. Death of the individual is termed somatic death, and death of the tissues, molecular death. These do not happen concurrently, since molecular life remains for a variable period after the occurrence of somatic death.

SIGNS OF DEATH AND CHANGES FOLLOWING DEATH

Every sign of death is of importance when considered relatively to the supposed time of its occurrence, and it is by an assessment of the various changes which occur in the body after death that an approximate opinion regarding the interval which has elapsed since death can be formed. It is necessary, therefore, to consider these changes in some detail.

Cessation of respiration and circulation.

The former may be tested by :—

Auscultation ;
Placing a hand-mirror or downy feather in front of the mouth and nostrils, and noting the absence of bedewing or movement ;
Placing a saucer filled with water on the chest or abdomen, and observing the absence of rippling.

The latter by :—

Auscultation ;
Absence of pulsation in the principal arteries ;
Applying a ligature tightly round a finger, and observing the absence of a bloodless zone at the seat of the ligature and a zone of livid redness on its distal side ;
Applying pressure to the finger-nail and observing that application of pressure, followed by relaxation of the pressure, fails to produce any alteration in colour ;
Holding the webs of the fingers against a strong light and noting an opaque appearance instead of a pink, translucent colour ;
The severance of an artery disclosing the cessation of circulation.

The absence of vital reaction to these tests, or to some of them, five minutes after death is supposed to have taken place, will indicate the occurrence of death.

General muscular flaccidity of the body.

This condition has been termed primary flaccidity to distinguish it from the condition of secondary flaccidity which occurs after the departure of rigor mortis. In the former condition which immediately follows death, there is complete relaxation of the muscles (see p. 134.) During this period, which persists for a few hours, there is loss of lustre of the eyes, which assume a glazed appearance.

For a variable time after somatic death, usually a few hours, while molecular life remains, electrical stimuli will produce some measure of muscular response, and by the application of atropine or physostigmine to the eye, dilatation or contraction of the pupil will occur.

As a result of cessation of circulation, the skin of the body shows pallor. In death from inhalation of carbon monoxide, however, a pinkish colour of the skin on certain parts of the body is shown. Reddish, irregular areas over the exposed parts of the body may be seen in death from cold and exposure. In certain cases of death by drowning, a rosy colour of the lips and of the malar prominences also may be observed. In death from hydrocyanic acid and cyanide poisoning, pink, patchy colorations may be seen upon the body surfaces. Certain colorations, present during life, such as jaundice, will be readily discernible after death.

Cooling of the body.

After death, heat production ceases and the loss of heat is continuous, the rapidity of the loss being dependent upon a number of circumstances. During a period of a few hours following death the temperature falls comparatively quickly, but later progressively, and more slowly the nearer it approaches the temperature of the medium in which the body lies. Generally, the body surface will be cold in from eight to twelve hours, but the temperature of the medium in which the body is lying exerts an important influence on the time factor, and must be taken carefully into consideration. The surface of the trunk cools less rapidly than the extremities, and it should not be overlooked that the surface temperature is not a true indication of the temperature of the body interior. A much longer interval will elapse before there is complete cooling of the interior of the body. In ascertaining the temperature, the axillary and rectal temperature should be taken, and a suitable thermometer, not a clinical thermometer, must be used. The visceral temperature may be recorded at post-mortem examination by placing a thermometer against the under surface of the liver. The average fall in temperature during the first few hours after death is variable and dependent on many circumstances but is approximately in the region of 1·5° F. per hour, or slightly more:

thereafter the rate of heat loss is slower. Under average conditions, at room temperature, a clothed adult body of good nutrition will have attained the temperature of the surrounding medium in about twenty to thirty hours. To make a very approximate estimate of the duration of death from the body temperature, the following formula has been suggested :—

$$\frac{\text{(normal temperature) } 98{\cdot}4^{\circ} - \text{rectal temperature}}{1{\cdot}5} = \text{approximate number of hours.}$$

The important point to observe, however, is that the cooling of the body is progressive. When the temperature of the body has fallen from 15° to 20° F., below normal, it is incompatible with life. Environmental circumstances exert a very important controlling effect. These include water, clothing, bedding, heated atmosphere, outdoor exposure, and seasonal variations.

The rate of cooling of the body, when considered in greater detail, is modified by the following circumstances :—

Condition of the body.

The bodies of obese subjects retain heat longer than the bodies of thin subjects, the bodies of robust persons longer than those of debilitated persons, and clothed bodies longer than naked bodies.

Medium in which the body is placed.

In water, bodies cool more rapidly than in air, and the colder the medium, the more rapid is the cooling. The difference between body temperature and the temperature of the atmospheric surroundings has an important controlling influence.

Mode of death.

In deaths by lightning, and by asphyxia, bodies may retain heat for a longer period. In diseases of bacterial origin, such as cholera, yellow fever, smallpox, septicæmia, and others, the temperature of the body may even rise after death, for a short time, as the result of development of the bacteria in the fluids and tissues. The same fact has been recorded in death from acute rheumatism, injuries to the nervous system, and from certain abdominal conditions.

Contact flattening of the muscles.

During the period of primary flaccidity, the soft, convex portions of the body, for example, the buttocks and calves, which lie in contact with a hard surface become flattened from pressure, and lose their convexity. On this account, alterations in the position of a body after death may be indicated by the presence of areas of contact flattening on parts of the body which no longer lie in contact with a solid surface (see Fig. 35).

Hypostasis, post-mortem staining, or post-mortem lividity.

This condition may be observed upon all bodies, irrespective of the cause of death. The condition is commonly seen even in deaths from hæmorrhage. An exception is the instance of dismembered bodies from which the blood has been fully drained very shortly after death. The extent, colour, and incidence of the condition vary in different cases. It is found upon parts of the body which are the most dependent in relation to the position of the body and is due to the engorgement of the capillaries by downward gravitation of the blood. In a body placed upon its back after death, hypostasis

FIG. 35

Hypostasis, or post-mortem staining, and areas of contact flattening.

will be found on the dorsal aspect of the trunk, the posterior aspect of the head and neck, the extensor surfaces of the upper limbs, and the flexor surfaces of the lower limbs. In bodies placed in positions other than this, the incidence of the lividity will be found accordingly. From its distribution, therefore, it is possible to determine the position in which a body has lain after death, and to form some estimate with regard to the time of death. In medico-legal work, especially in cases where there are marks of violence upon the body, the incidence of hypostasis must be noted carefully. Hypostasis does not appear upon parts of the body which are in actual contact with the surface upon which the body is lying. It is also absent over constricted parts, even although these are dependent, as, for example, under the site of the collar, or at the pressure marking of a waistband, garters, or corsets which have been laced tightly. This is due to the fact that the pressure occludes the underlying capillaries and therefore precludes their engorgement with blood.

Hypostasis is fully pronounced on a body in from eight to twelve hours after death. It commences shortly after death and the staining is usually fixed, as the result of blood coagulation, in from six to eight hours. Hypostatic areas are of bluish-red colour, are irregular in

contour, and affect considerable areas of the body surface. In cases of carbon monoxide poisoning, the areas show a cherry-red colour due to the presence of carboxyhæmoglobin in the blood (see Fig. 226). A similar coloration may be evident on bodies when death has resulted from hydrocyanic acid, or cyanide poisoning. In asphyxial deaths, the colour may be markedly livid. The manifestations of hypostasis are also seen internally, for example, in a body which has been lying on its back during the occurrence of its onset, the bases and posterior parts of the lungs, the posterior wall of the stomach and the lowermost parts of the intestines, together with the kidneys, will be found affected. It is important that the appearances of hypostasis affecting viscera should not be confused with evidence of disease.

Hypostasis must be differentiated decisively from bruising in cases of suspected violence. Differentiation between these two very different conditions is effected by making an incision into the tissues through the centre of the discoloration. If the discoloration is the result of hypostasis, only a few oozing points at the severed capillaries will be observed, but if it is due to bruising, blood will be found extravasated into the cutaneous and subcutaneous tissues. In all cases where the effect of violence is suspected, sections of the tissue should be examined microscopically (see p. 248).

The colour alone is no guide in the differentiation between hypostasis and bruising.

Rigor mortis.

The condition of primary flaccidity, or muscular relaxation of the body, is replaced by a progressive rigidity which affects the muscular system and includes both the voluntary and involuntary muscles of bodies of all ages.

Research by E. C. Smith [1] has shown that the stiffening of muscle in rigor mortis is closely related to gelation of the muscle plasma, and that, apart from the formation of lactic acid, no reaction has been found to occur post-mortem which can be held responsible for the coagulation of the plasma. The stiffening of rigor is very closely associated with the accumulation of lactic acid in the muscle. In his view, it remains to be discovered whether there is not a third change to which both stiffening and lactic acid production are intimately related. He states, "still further possibilities of chemical change exist, and it is well to restrain speculation until more is known of these reactions."

Szent-Györgyi [2] has recently investigated the chemistry of muscular contraction and his work is still in progress. He is of the opinion that one of the principal factors concerned in the maintenance of suppleness and plasticity of muscle is the degree of hydration of the protein. This in turn is dependent on the amount of adenosine triphosphate which is adsorbed in the myosin of the muscle. After death, the adenosine triphosphate is gradually decomposed, dehydration takes place and this results in the condition known as

rigor mortis. The subsequent relaxation is due to the final disorganisation of muscle.

The earliest manifestations of rigor mortis will be found in the muscles of the eyelids and those of the lower jaw. Rigidity of the eyelids usually preeedes that of the jaw muscles, which are affected in about three to four hours after death. It next appears progressively in the muscles of the neck, the face, the thorax, the upper extremities, the trunk of the body, and the muscles of the lower extremities. Rigor mortis brings about slight shortening of the muscle fibres. The muscles in which rigor mortis first appears

Fig. 36

Rigor mortis, or death stiffening. Note flexion of arms and legs.

are those from which it usually first disappears. Rigor also affects the involuntary muscles, and might be mistaken for hypertrophy, for example, in the instance of the heart.

The period of invasion can only be stated in broad terms, since there are so many modifying factors. As a rule, general stiffening will be established after ten to twelve hours, although in some cases the period may be shorter. Under certain circumstances, however, it may appear unusually early, or its appearance may be retarded very considerably. When rigor mortis is established, the jaw, neck, and extremities become fixed in position, and movements at the joints are possible only within a very limited degree unless the rigidity of muscle is overcome by effort. When force has been used in this way, the rigidity of muscle does not subsequently recur. In the majority of cases it will have commenced to pass off in about thirty-six hours but, in cold weather, and for other reasons, a much longer interval may elapse.

The stronger muscularly the person is at the time of death, the later is the time of onset, and the longer the duration. The more feeble or exhausted the muscular condition, the more rapid is the time of onset, and the shorter the duration. Exercise or fatigue reduces the muscle glycogen content, and may ultimately bring about its complete disappearance, with an accumulation of acid substance in the muscle plasma.

If a body, already the subject of newly established rigidity, is exposed to a temperature of 75° C., the rigidity becomes more pronounced, since the albuminates in the muscles become coagulated. If, on the other hand, a body is exposed immediately after death to

a temperature at, or below, freezing-point, the onset of rigor mortis is retarded so long as this temperature continues. When the temperature is gradually elevated to 10° C., or 50° F., rigor comes on rapidly, but disappears more quickly than had the body not been subjected to the process.

In all long-continued, febrile, and chronic diseases, in addition to continuous convulsive disorders, rigor appears early and passes off quickly.

Conditions simulating rigor mortis.

Heat stiffening occurs in bodies exposed to intense heat by burning, or immersion in hot liquids, and is due to coagulation of the albuminates in the muscles. Rigor mortis does not supervene in such cases, and the primary rigidity persists until the onset of putrefaction. When heat stiffening affects a body, the position of the limbs simulates the general attitude of a boxer, and to this condition the term pugilistic attitude has been aptly applied (see p. 219).

Stiffening of the body as the result of exposure to cold may simulate rigor mortis. When the general stiffness of the body is due to freezing, passive flexion of the joints may be accompanied by faint crepitant sounds due to the breaking of the frozen synovial fluids. When the body thaws, this stiffening passes off, and true rigor may become established (see p. 233).

Instantaneous rigor or cadaveric spasm.

This condition is of considerable medico-legal importance, and is unconnected with rigor mortis. It occurs at the moment of death, and the muscles of the hand are most usually affected. We have

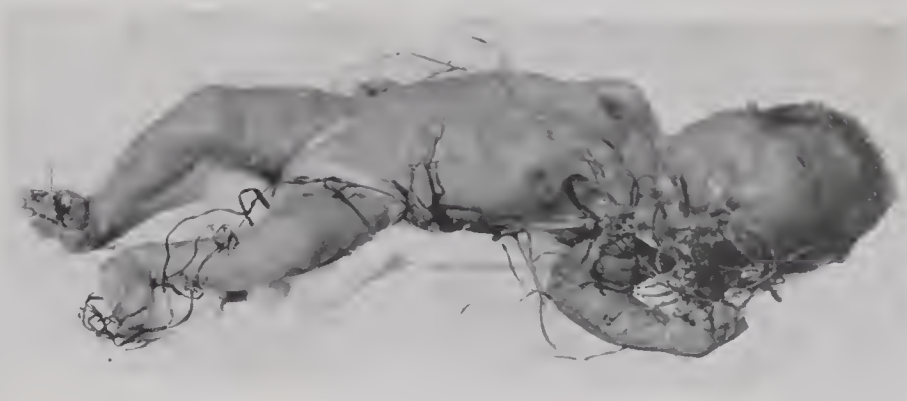


Fig. 37

An unusual case of instantaneous rigor, or cadaveric spasm, in a newly born child who died from the effects of incomplete respiration. Note the grass tightly grasped in the left hand. Birth occurred in the open.

seen it several times in cases of suicidal cut-throat, and suicidal shooting, when the weapon was so firmly grasped in the hand that considerable force was required to disengage it. In cases of drowning, the finding of articles grasped firmly in the hands of the victims is not unusual. This condition is due initially to a voluntary vital act,

probably accompanied by a high degree of emotion, immediately preceding death.

It does not appear that an adequate explanation of the phenomenon has been ascertained. That it is not simply an unusually early local onset of ordinary rigor mortis seems clear, since the local muscles do not share in the general primary flaccidity of the muscular system, else the weapon would fall out of the hand, or the postural attitude of the body would be lost. The condition of the nervous system at the time of death would appear to play an important part. Cadaveric spasm may affect only certain groups of muscles, or all the muscles, of a limb or limbs.

When a weapon, or other article, is found firmly clutched in the hand of a dead person, it is proof that it was in the hand of the person during life, and that the grasp occurred at, or about, the moment of death. Nothing can simulate instantaneous rigor, or cadaveric spasm, and it cannot be produced by any method after death. Personal experiments have been unsuccessful.

Muscular states following death.

The different muscular states which a body undergoes between the period of somatic death and putrefaction may be summed up as follows :—

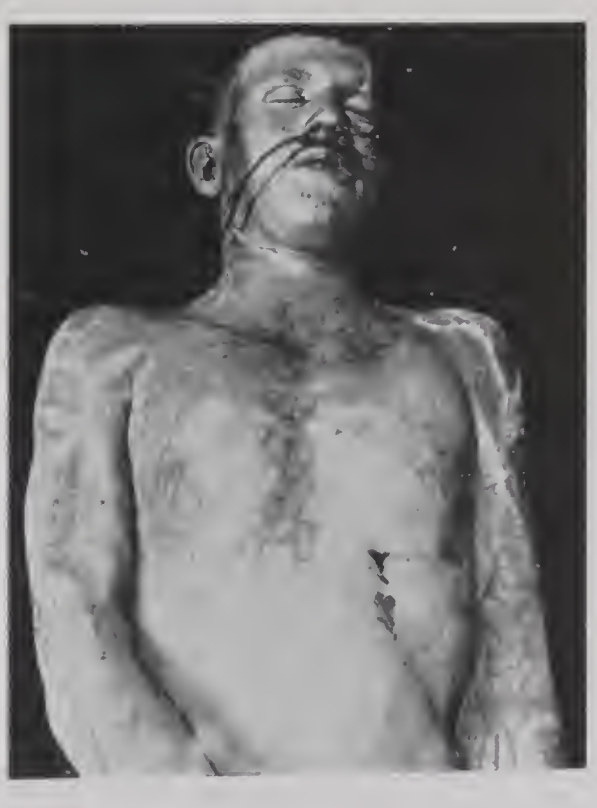


Fig. 38

Discoloration of superficial veins due to putrefactive changes.

1. The condition of primary flaccidity — relaxed muscles — responsive to electrical stimuli ;

2. The condition of rigor mortis — muscular stiffening of the body—irresponsive to electrical stimuli ;

3. The condition of secondary flaccidity which is synchronous with the onset of putrefaction.

PUTREFACTION

Putrefaction is the last stage in the resolution of the body from the organic to the inorganic state.

The factors responsible for the process of putrefaction are micro-organisms, the action of warmth, the presence of air, and the presence of moisture.

Burn [3] has investigated the question of post-mortem bacteriology, and states that B. coli, Staphylococcus, non-hæmolytic Streptococcus, Streptococcus

viridans, B. welchii, diphtheroid and proteus types are the most frequently isolated strains of bacteria, while others isolated are usually associated with disease processes within the body. In his opinion, the most frequently recovered types are known to be normal inhabitants of either the respiratory or the intestinal tract, while those less frequently found are usually present in association with some focus within the body, or a disease process. He noted

Fig. 39

Early putrefactive discoloration involving abdominal surface.

no significant differences in either the frequency, or the kind of bacteria isolated from the organs, post-mortem, between the first and forty-eighth hour after death.

External signs of putrefaction.

The various steps in the process of putrefaction need not be described in detail, and the following summary will indicate the progressive and predominant features :—

Greenish coloration over the right iliac fossa.
Extension of greenish colour over the whole of the abdomen, and the other parts of the body.
Discoloration and swelling of the face.
Swelling and discoloration of the scrotum, or of the vulva.
Distension of the abdomen with gases.
Development of bullæ, of varying size, on the surfaces.
Bursting of bullæ, and denudation of large irregular surfaces due to the shedding of epidermis.
Escape of blood-stained fluid from the mouth and nostrils.
Brownish coloration of the surface veins giving an arborescent pattern on the skin.
Liquefaction of the eyeballs.
Increasing discoloration of the body generally, and greater and progressive abdominal distension.
Presence of maggots.
Shedding of the nails, and loosening of the hair.
Facial features unrecognisable.

Conversion of tissues into a semi-fluid mass.
Bursting open of the abdominal and thoracic cavities.
Progressive dissolution of the body.

The evolution of gases of putrefaction, sulphuretted hydrogen, phosphoretted hydrogen, carburetted hydrogen, ammonia, and carbon dioxide, and the liquefaction of the tissues are the responsible factors in the bursting open of the natural cavities of the body.

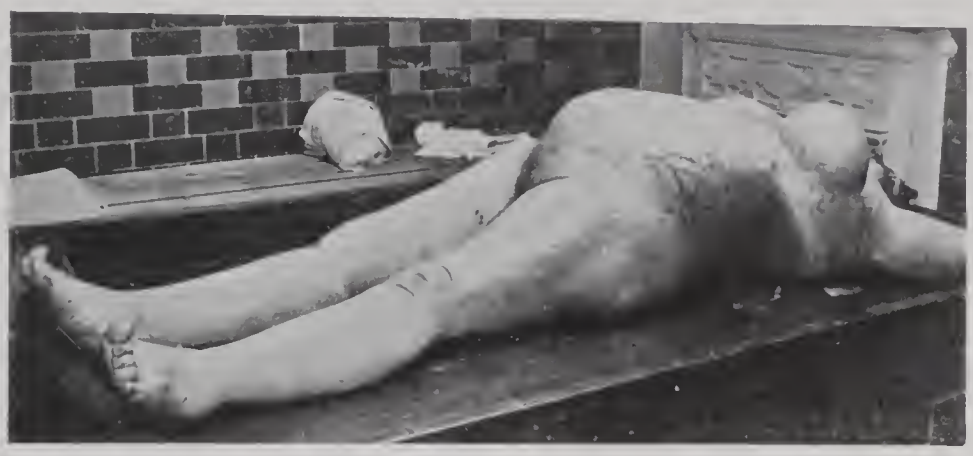


FIG. 40

Distension of abdominal cavity and breasts due to the formation of putrefactive gases.

Different views are held as to the nature of the greenish and dark colours produced over the abdomen, and other surfaces of the body, but it is sufficient to add that these are produced by changes in the blood pigment which result from bacterial action.

Putrefaction of internal organs.

The following is a tabulated statement of the order of putrefaction of the organs, which may be divided into two classes :—

(*A*) Those which putrefy early.
(*B*) Those which putrefy late.

(*A*)	(*B*)
Brain.	Œsophagus.
Lining of trachea and larynx.	Diaphragm.
Stomach and intestines.	Heart.
Spleen.	Lungs.
Liver.	Kidneys.
Uterus (pregnant or in puerperal state).	Urinary bladder.
	Uterus.
	Prostate gland.

It will be seen that the organs composed of muscular tissue resist putrefaction longer than the parenchymatous organs, with the

exception of the stomach and intestines, which, by reason of their contents at the time of death, decompose quickly. The slowness of the process also depends, in some measure, upon the amount of fibrous tissue which an organ contains, as, for example, the lungs. The internal organs of the young putrefy more rapidly than those of middle-aged or elderly persons. Wide variations in the order of putrefaction are frequently met, and the specific factors present in each case must be the subject of special consideration.

FIG. 41

Putrefactive changes affecting the face.

Factors which modify rate of putrefaction.

These may be divided into :—

External factors.
Internal factors.

External.

The principal external factors are :—

Presence or absence of micro-organisms ;
Presence or absence of air ;
Temperature of the environment ;
Presence or absence of moisture ;
Character of medium in which the body lies.

Presence or absence of micro-organisms.

This is very largely determined by the second factor. If air is freely present, bacteria have access to the body, but a certain amount of moisture, and a moderate degree of temperature must also be present to enable them to develop. Occasionally bodies show unusually rapid external manifestations of decomposition characterised by a bloated and swollen state of the skin and underlying tissues of an emphysematous nature. We have quite frequently isolated B. welchii in such cases.

Presence or absence of air.

The presence of air promotes, and its absence retards, decomposition. Under uniform circumstances, a nude body will decompose

more rapidly than one which is clothed. Porous sandy soils accelerate decomposition, while clayey, dense, or water-logged soils retard it.

Temperature of environment.

Putrefaction begins at about 50° F., and is most favoured by temperatures ranging from 70° to 100° F. The process is retarded

FIG. 42

Fungus growth on face of body found in a warm, dry cellar under a house. Body was fully clothed and lay on bare soil, apart from an intervening rainproof coat.

by temperatures between 100° and 212° F. since the fluids are dried up, and there is a probability of mummification of the body tissues. Bacterial development is retarded at extremes of temperature (see p. 144).

Presence or absence of moisture.

Moisture is essential for decomposition of an animal substance, and normally the body contains sufficient for the process. Any additional moisture, however, aids the rapidity of the action. This is one of the reasons why bodies decompose more quickly in water than in the earth. If warmth is added, the rate of decomposition is much accelerated.

Type of medium in which the body lies.

These media are three in number, air, earth, and water. It may be accepted as a general principle that a body decomposes in air twice as quickly as in water, and eight times as rapidly as in earth. Putrefaction develops rapidly in bodies lying in sewage-polluted water.

Bodies retrieved from water after lengthy immersion very frequently show advanced putrefactive changes involving the face, neck and chest. This is possibly due to exposure of these parts to the air after flotation (see Figs. 41 and 53).

Internal.

The following are the principal factors :—

Age ;
Sex ;
Condition of body at death ;
Nature of death.

Age.

Usually the bodies of very old persons do not putrefy rapidly on account of the relative absence of fat.

Sex.

This has but little influence, except in the case of bodies of women dying after child-birth, more especially when death has been caused by septicæmia. In such cases putrefaction is rapid.

Condition of body at death.

The physical state of a body influences the rapidity of putrefaction to some extent. Fat, flabby subjects putrefy more quickly than lean bodies, doubtless due to the larger amount of fluid in the tissues and the presence of an excess of fat.

Nature of death.

Bodies of persons dying suddenly in apparent health decompose less quickly than those of persons dying from acute or chronic diseases, especially infective diseases. Mutilation of tissue favours decomposition on account of the larger area exposed to bacteria. Bodies of chronic alcoholics, and those who have died from poisoning by such substances as carbolic acid, arsenic, antimony, and chloride of zinc, resist putrefaction longer, owing to the preservative action of such substances on the tissues, or to their destructive, or inhibitive, action on the organisms which induce decomposition.

MAGGOTS

Identification.[4]

In certain cases, with a view to checking the accuracy of the estimate of time between death and the examination of the remains, identification of the maggots found on a putrefied body may prove of value, since having identified them it becomes possible to deter-

mine the age reached in the cycle of their development. By this means an estimate of the time which has elapsed since the eggs of the flies were deposited on the remains becomes possible within fairly accurate limits. It is, however, necessary for the examiner to

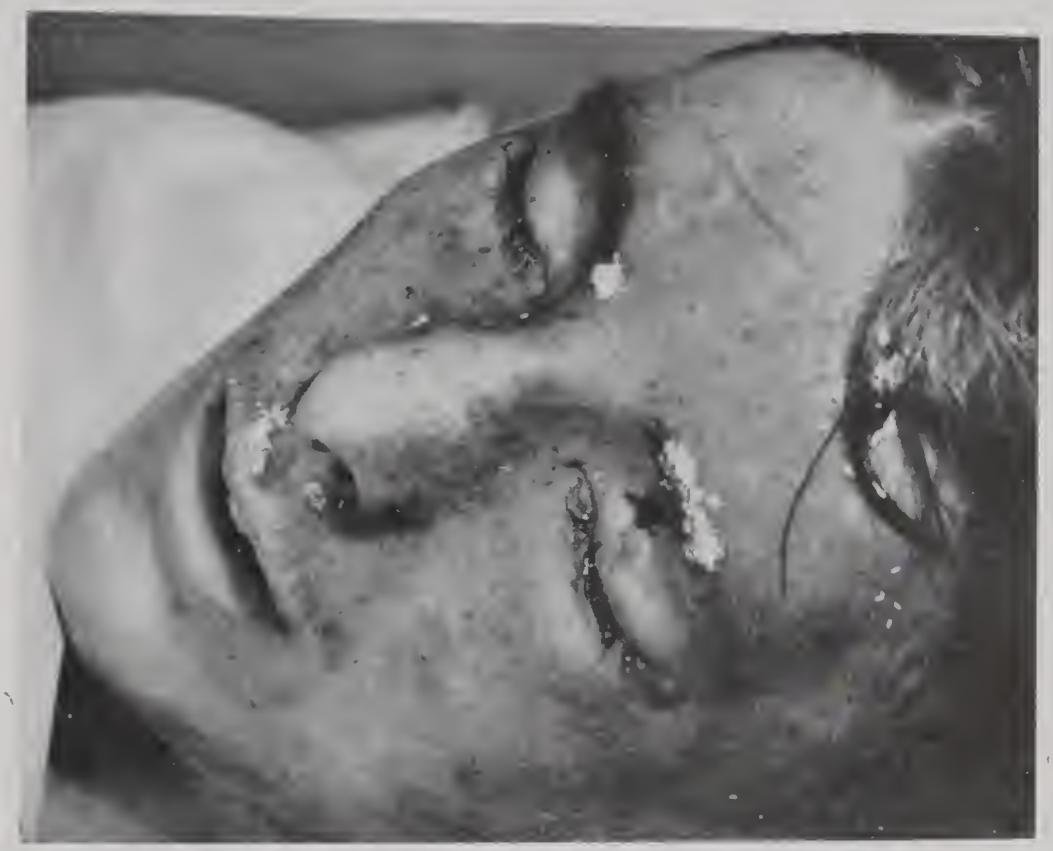


Fig. 43

Body recovered from the sea probably seven to ten days after death. Calliphorine eggs were deposited, as shown, after body had been in mortuary for twenty-four hours. Note presence of fly on left eyebrow. (By courtesy of Dr Robert Richards, Aberdeen University.)

have knowledge of the life histories of the various maggots, and for this reason the examination should be left to an entomologist. It would be out of place to deal with this subject in great detail in a book of this description, but it is felt that the life histories of certain of the muscid larvæ may usefully be included.

Life histories of three types.

The common blue-bottle (Calliphora erythrocephala).

The eggs are laid readily on meat when it is fresh—less commonly when it is decayed. The maximum number of eggs laid by a single adult fly is generally 2,000. They are deposited in groups of about 150, and hatch in from eight to fourteen hours, depending upon the temperature of the environment. Bodies which have lain exposed after immersion in organically polluted waters may retain surface residues which encourage female Calliphorines to deposit eggs (Fig. 43). Cold weather delays hatching. The first larval instar (stage in the life history between two successive castings of the cuticle or outer skin) persists for eight to fourteen hours. The

skin is then shed and the second larval instar, similar to the first although larger, appears. It persists for two to three days. The third instar is the fully-grown maggot, which feeds voraciously for six days. The larva, now creamy white, migrates during the night to some distance from its food and burrows into the soil, where it pupates. Owing to the possession by the larvæ of a liquefactive ferment, meat parasitised by them rapidly becomes putrid and for this reason, should the infestation be heavy, a body is more advanced in putrefaction than it would normally be. The pupal stage lasts approximately twelve days. Calliphorine flies are found from early spring until late autumn.

The green-bottle (Lucilia cæsar) and the **sheep maggot fly** (L. sericata).

These flies, which have a bright metallic lustre, show a life history similar in all respects to that of Calliphora. Their larvæ may also invade wounds (myiasis).

The common house-fly (Musca domestica).

The female lays about 150 eggs at a time, mainly in manurial matter. Oviposition on dead bodies is rare. The eggs hatch in eight to twelve hours, and the first larval stage lasts thirty-six hours. The second instar persists for one to two days, and the final for three to four days, depending on food, moisture, and temperature. The pupal stage usually lasts seven days, or less during warm weather. The full-grown third larva migrates, usually at night, and burrows into the soil to pupate.

Differentiation between the three types.

The larva of Musca is distinguished from those of Calliphora and Lucilia by mounting on a slide the thinnest possible section, removed from the posterior end of an alcohol-hardened specimen by means of a safety-razor blade, and examining the stigmata or breathing pores with the $\frac{2}{3}$-inch lens. The contained spiracles are convoluted in Musca but straight in Calliphora (Fig. 44).

In order to differentiate between the maggots of Calliphora and Lucilia, it is necessary to examine the anterior end of the specially prepared larva microscopically. The mandibular sclerite or jaw-hook (Fig. 44) of Calliphora has an internal horizontal process not present in Lucilia.

In the Ruxton case, the maggots which infested the remains were identified as those of Calliphora, and the estimation of the time since deposit of Calliphorine eggs was as follows :—

Stage.	Time (outside limit).
Egg	8 to 14 hours.
1st larva	8 ,, 14 ,,
2nd ,,	2 ,, 3 days.
3rd ,,	7 ,, 8 ,,
Total	10 ,, 12 ,, (approx.).

The total life of the largest larvæ could not have exceeded twelve days, but was probably less. It was unlikely that the eggs had been laid more than a day or two after the deposit of the remains in the linn. The possibility of a laying of eggs by the progeny of the first generation of blue-bottles could be disregarded. That

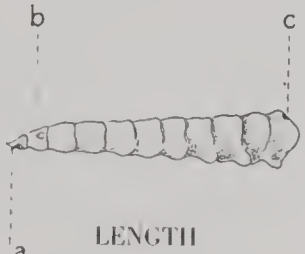
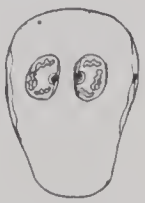
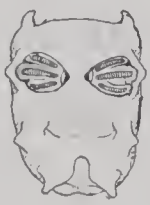
SPECIES	LARVA	TRANSVERSE SECTION POSTERIOR SPIRACLES	PUPA	WING VENATION
MUSCA	 LENGTH $\frac{1''}{4}$	 NOTE FORM OF BREATHING PORES	LENGTH  $\frac{1''}{4}$	
CALLIPHORA	MORPHOLOGY AS IN MUSCA BUT LENGTH $\frac{1''}{2} - \frac{3''}{4}$		MORPHOLOGY AS IN MUSCA BUT LENGTH $\frac{3''}{4}$	MORPHOLOGY IDENTICAL WITH MUSCA

FIG. 44

Musca and Calliphora. Differential taxonomic characters.

(*a*) Jaw hook.
(*b*) Position of anterior spiracle.
(*c*) Position of posterior spiracle.
(*d*) Characteristic angling of median vein.

would have required a month, which was quite irreconcilable with the stage of putrefaction of the remains.

The stage of development of the larvæ was compatible with the remains having been deposited in the linn about twelve to fourteen days before their examination on October 1. The result of this line of investigation not only corroborated the opinion expressed on other grounds but fitted the hypothesis of the prosecution that the parts of the bodies had probably been placed in the ravine during the early hours of the morning of September 16, 1935 (see p. 99).

Specimens of larvæ intended for laboratory examination should be dropped alive into boiling absolute alcohol which kills them instantaneously in the extended state. Several should then be transmitted to the laboratory in a spirit-proof container charged with 50 per cent. alcohol.

SAPONIFICATION AND MUMMIFICATION

Putrefaction of the body may be modified in its progress, and in the character of the products formed.

The two principal modifications are:—

Saponification.
Mummification.

Saponification.

A body upon which moisture has been constantly acting, whether it is immersed in water or in damp soil, may undergo this change.

The cause of saponification is the change of the fatty constituents of the body into new chemical compounds, more or less stable in composition, and the process is brought about by the gradual hydrogenation of pre-existing fats in the body to form higher fatty acids. Adipocere, the term applied to the material resulting from the process of saponification, is a fatty-looking substance which varies in colour from white to yellowish-white, imparts an unctuous feeling to the fingers, melts in flame, burns with a feebly luminant flame, and has a faint mouldy, cheesy odour.

Adipocere in mature form is largely composed of palmitic, stearic, oleic, and hydroxystearic acids. Calcium soaps, proteins, and some other constituents are variable incidental components.

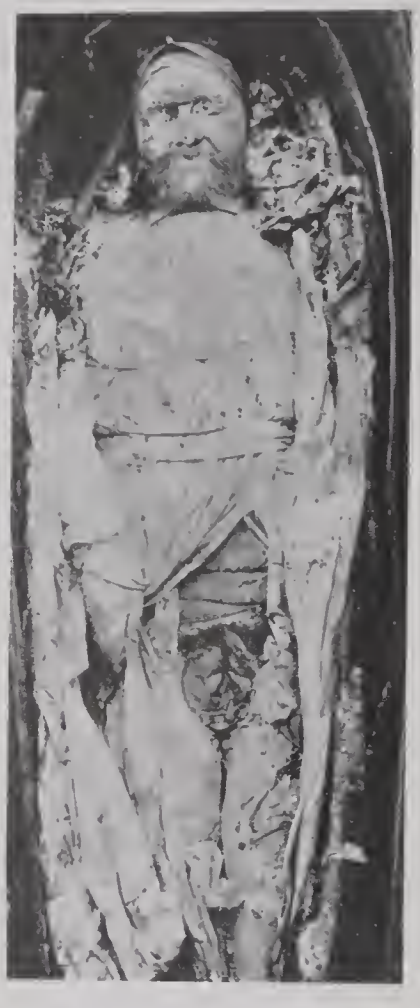


Fig. 45

A body converted, almost completely, into adipocere.

It is not usual for the entire body to be converted into adipocere, although instances have been quite frequently seen (see Fig. 45). More generally, the limbs, chest wall, buttocks, or other parts of the body may become saponified. When the face is the seat of this process, the features are well preserved, and when the entire body is involved, the form and contours are well retained.

The change of tissues into adipocere takes a considerable time in temperate climates, but its development in warm climates is much more rapid. It takes several months to effect the change even in limited parts of the body. The process, however, is more rapid in the bodies of infants and young children than in those of adults. Occasional cases of early production of adipocere have been recorded.[5] It is not possible to prescribe any specified time in which adipocere will be produced, on account of the effects of variable operative factors.

The necessary conditions for the production of adipocere are a superabundance of moisture and a relative diminution of air and micro-organisms. A warm temperature is a further facilitating factor.

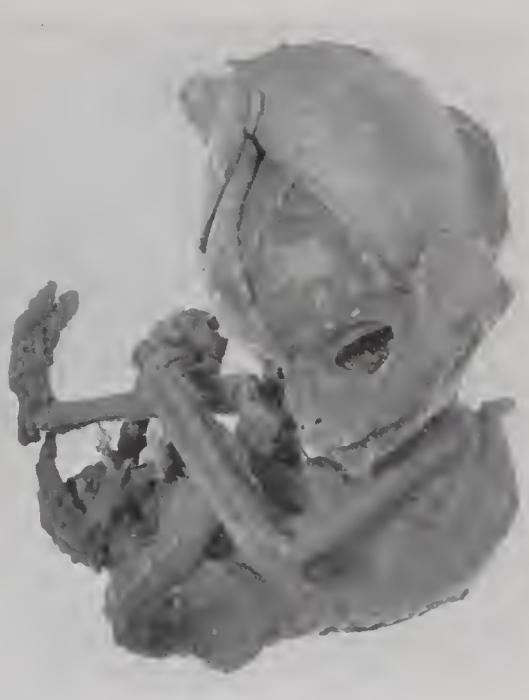


FIG. 46
Mummified fœtus found behind a kitchen fireplace.

Mummification.

This is a common result following embalming in which artificial measures are employed (see p. 44), but in temperate climates it occurs naturally only under certain circumstances. Cases are more numerous in the tropics and sub-tropics. The mummified bodies of infants are occasionally found in unusual places of concealment which favour the production of this process, for example, in dry-stone walls, suitcases, trunks, and at the back of fireplaces. The factors necessary for the production of mummification are the absence of dampness and the continuous action of a current of dry or warmed air. The appearance of a mummified body is quite typical. The whole structure is desiccated, shrivelled, brownish-black in colour, and the anatomical features are well preserved. The skin, which clings closely to the shrunken framework of the body, the hair on the scalp, and the skeletonised features of the face are well preserved. A body in this condition is practically odourless.

Forbes [6] has written on the brown house moth as an agent in the destruction of mummified remains. He reports a case of mummified twins found in a "false roof" under the slates of a house. The bodies had probably been concealed there for a period of from fourteen to seventeen years. The skin had entirely disappeared, and the soft tissues were riddled with roughly circular holes which led into burrows filled with moth eggs and excreta of moths. The type of moth responsible for the larvæ was the brown house or false clothes moth—Borkhausenia pseudospretella. The larvæ of this moth eat furs, skins, and dried specimens of animals and birds. He states that given time, the soft tissues of the bodies would probably have completely disappeared. Larvæ were present in the abdominal, thoracic, and cranial cavities. No traces of fly infestation were found in the bodies by entomological examination.

PROXIMATE CAUSES OF DEATH

All deaths may be resolved into one of three proximate causes, namely, coma, syncope, or asphyxia. Since life is maintained by

the interdependent action of the brain, the heart, and the lungs, the arrest of the function of one of them is quickly succeeded by the arrest of the function of the others, and life ceases. From clinical observation and post-mortem appearances, it is found that there is also a mixed form, due to the combined effects of coma and asphyxia, to which the term comato-asphyxia has been applied.

Coma.

The principal causes of coma are compression of the brain, due to depressed fracture of the skull, cerebral hæmorrhage, and tumours of the brain. The effects of poisons introduced into the body, for example, hypnotics and narcotics, and the effects of substances produced within the body during the currency of certain diseases, such as Bright's disease and diabetes mellitus, may also produce coma. Coma may exist for some time before death, and the characteristic signs consist of more or less profound stupor, imperfect action of reflexes and, not infrequently, relaxation of sphincters.

As the result of interference with respiratory function, the post-mortem signs usually resolve themselves into a combination of both coma and asphyxia. Most frequently seen are a hyperæmic condition of the brain and its covering membranes, together with signs of a varying degree of asphyxia.

Syncope.

This term is applied to a sudden cessation of the action of the heart, which may prove fatal. Syncope may be brought about by a large variety of causes, including shock, emotion, a blow on the epigastrium, or the evacuation of fluids, for example, the over-rapid withdrawal of fluid from an ascitic abdomen, or from a pleural cavity. The injection of fluid into the uterus may also induce syncope. Excessive hæmorrhage, infective and chronic diseases, extensive injury, and disease of the heart, especially of the degenerative type, also coronary thrombosis, are frequent causes. Among many other causes, sudden immersion of the body in cold water, with impingement of the water upon the nasal and post-nasal mucous membrane, and sudden constriction of the neck over the carotid sinuses are worthy of mention. Shock may be primary or secondary.

Primary shock, a frequent cause of syncope, results from afferent impulses which cause reflex vasodilatation and a resultant serious fall in the blood-pressure due to the actual diminution of the volume of the blood passing through the heart to the arteries. The capillaries of the skeletal muscles are chiefly affected, and in these the blood collects instead of returning to the heart, causing a temporary deprivaton of the blood from the circulation and cerebral anæmia. Secondary shock, or traumatic shock, would appear to result from a reduction in the blood volume due to loss of blood and plasma into the injured tissues, and an increased concentration of the blood in the circulatory system. When the loss of blood becomes such that

it cannot maintain an effective circulation, the vital centres of the brain can no longer obtain a normal amount of blood and death results. Holt and Macdonald,[7] and others, take the view that there is evidence against the acceptance of the " traumatic toxæmia " or histamine theory to account for secondary shock. Holt and Macdonald have been unable to demonstrate the presence of any depressor substance in the blood of the traumatised area, and in no experiment was the blood-pressure reduced to a shock level without there being a loss of plasma and blood into the injured tissues, sufficient in itself to account for the effects observed.

The post-mortem appearances are neither prominent nor characteristic. The cavities of the heart contain comparatively little blood, the organs are pale, and the capillary vessels are congested.

Asphyxia.

Asphyxia may be defined as primarily a state, or series of states, induced by an oxygen supply short of tissue needs.[8] There are two forms of asphyxia. In the first form there is gasping for breath and marked cyanosis with retention of carbon dioxide and depletion of oxygen. In the second form there is no cyanosis, the asphyxia being due to depletion of oxygen, or anoxæmia, without retention of carbon dioxide. Shallow rapid breathing is present which causes the anoxæmia and also an excessive elimination of carbon dioxide, with resultant failure of the respiratory centre. In this form there is no sign of respiratory distress.

Asphyxia may be produced in many ways, including the following :—

Occlusion of the air-passages by foreign bodies, the effects of scalding or corrosives, angioneurotic œdema, acute inflammation, membranous exudations, acute œdema of the glottis, laryngeal spasm, tumours, and abscesses.

Impediment to respiratory function by pressure on the chest wall, for example, by falls of debris, and in lift and pit-cage accidents.

Strangulation, suffocation, hanging, throttling, drowning, and the inhalation of irrespirable gases.

Paralysis of the respiratory nerves or muscles, or of the respiratory centre from injury or disease, or from the action of certain poisons, for example, morphine and barbiturates.

From causes operating from the lungs, or pulmonary circulatory system, for example, lung diseases, pleural effusions, pneumothorax, pulmonary embolism, and circulatory disturbances in the lungs.

In reviewing these examples of the causes of asphyxia, it will be seen that in obstructive asphyxia, the oxygen lack, or anoxæmia, is due to the prevention of breathing, so that an excess of carbon dioxide results. In non-obstructive asphyxia, oxygen is depleted by shallow rapid breathing which produces not only anoxæmia but

an excessive elimination of carbon dioxide, and it is the latter factor which brings about failure of the respiratory centre through lack of stimulation by carbon dioxide. Inhalation of gases may also induce asphyxia in one of two ways—either by stoppage of breathing, and paralysis of the respiratory centre through deprivation of oxygen and lack of carbon dioxide elimination, or by carbon dioxide being eliminated and anoxæmia occurring despite the fact that breathing continues. Poisoning by irritant gases is an example of the first of these causes, and carbon monoxide poisoning illustrates the second, since the carbon monoxide enters into stable union with the hæmoglobin causing oxygen deprivation of the tissues (see p. 582).

When the process of asphyxia is initiated by respiration being suddenly obstructed, either by occlusion of the air-passages or by pressure exerted upon them, external movements of the body, due to breathing, cease within three to five minutes, although the heart continues to beat for some minutes thereafter. The chance of resuscitation of those who though asphyxiated are not yet lifeless depends upon this fact. The phenomena of this type of asphyxia, which is divisible into three stages, are essentially those of carbon dioxide poisoning, namely, stimulation of the respiratory centre with restlessness, increased respiratory action, which gradually becomes more violent, and lividity of lips ; congestion and cyanosis of the face, occasionally convulsions, and unconsciousness ; paralysis of the respiratory centre which is accompanied by slow, irregular breathing, and which merges into death. Interference with the aeration of the blood prevents its oxygenation, and there is an accumulation of carbon dioxide. The arterioles become contracted, with elevation of blood-pressure, and an increased strain is imposed on the heart. The respiratory centre is stimulated, respiratory action is increased, and a further accumulation of carbon dioxide in the blood results. As oxygen starvation continues, both the rate and amplitude of cardiac contraction become lessened and, as the injured capillary vessels dilate, the return flow of venous blood is impeded, blood-pressure falls, and circulation fails. Paralysis of the respiratory centre now occurs, respiratory action is weakened and gradually ceases following increased intervals between respirations.

Post-mortem appearances.

External.

Lividity of the lips and ears and livid colour of hypostasis. In infants and young children, particularly, there is usually lividity of the finger and toe nails. These colorations vary in intensity from a duskiness to a dark-blue tint. The face is congested and livid in colour, although in some cases it may be pale or slightly dusky. Lividity does not necessarily indicate that death has been caused by respiratory failure, and an asphyxial death need not always show lividity. The tongue may lie in normal position, the tip may be pressed against the back of the teeth, or be protruded either between

the margins of the teeth, or beyond them. A frothy and frequently blood-stained fluid may be seen at the corners of the mouth, or at the nostrils, or in both positions. The conjunctivæ are commonly congested, and may show a varying number of small punctate hæmorrhages (see Fig. 56).

Internal.

The larynx and trachea may contain a varying amount of slightly frothy mucus. The lungs are engorged and œdematous. On section, a copious, frothy, dark-coloured, blood-stained exudate will be seen. Petechial hæmorrhages, due to capillary injury, are commonly present on the parietal, pulmonary, and pericardial pleuræ, pericardium, heart muscle, endocardium, and substance of the brain. Some of the marginal portions of the lungs may perhaps show emphysematous changes. The mucous membrane of the trachea is congested.

Microscopically, the lung tissue shows marked dilatation of both capillaries and veins. In infants, interlobar emphysema may be found either in patches, or more or less general in its distribution. The cavities of the right side of the heart are engorged with dark-coloured, imperfectly clotted blood, and the venæ cavæ are also engorged. The cavities on the left side of the heart are often comparatively empty, but this may be due to contraction of the left ventricle during rigor mortis, the blood readily escaping since the arterial system is relatively empty.

The abdominal viscera show marked venous congestion. The brain is often congested, and an excess of serous fluid is found in the lateral ventricles. The cranial sinuses are usually filled with dark-coloured blood. The blood, generally, is dark in colour and is mostly fluid in the large veins.

There are variations, however, in the intensity of the asphyxial signs which may be present, and these are dependent upon the specific circumstances which may attend a given case. When the asphyxial process is slight and prolonged, the congestive element will be diminished; when intense and short, lividity and congestion are marked; and when, during the process of asphyxia, heart-failure precedes respiratory arrest, the asphyxial signs may be less marked, depending at which stage in the asphyxial process cardiac arrest occurred.

A suggested classification of death.

The customary medico-legal classification of deaths into three pathological entities—coma, syncope, and asphyxia, although providing some indication of the body system from which the death is initiated, leaves much to be desired in the light of present-day knowledge.

Gordon,[9] who has introduced a conception for the classification of deaths of medico-legal importance based upon the original investigation of Moon,[10] submits that the cessation of vital functions depends upon tissue anoxia produced in four different ways (see Fig. 47), namely :—

(*A*) By defective oxygenation of the blood in the lungs—anoxic anoxia, caused by obstruction to the free passage of air into the respiratory tract as in suffocation, smothering, and overlaying; by obstruction to the free passage of air down the respiratory tract as in drowning, choking, throttling, strangulation, and hanging; by external compression on the chest or abdominal walls, for example, from falls of earth which produce cessation of respiratory movements; and by breathing in vitiated atmospheres with low oxygen tensions.

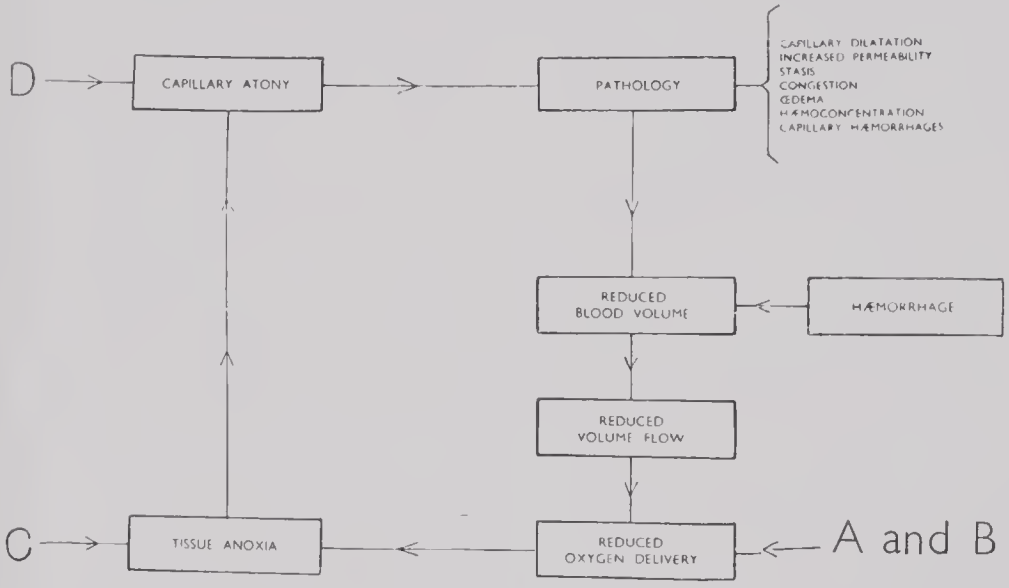


FIG. 47

Vicious cycle initiated by anoxia. (Gordon, based upon the original investigation of Moon.)

(*B*) By reduced oxygen carrying capacity of the blood—anæmic anoxia, for example, in acute carbon monoxide poisoning.

(*C*) By depression of the oxidative processes in the tissues—histotoxic anoxia, for example, in hydrocyanic acid poisoning.

(*D*) By insufficient circulation of the blood through the tissues—stagnant anoxia, for example, in traumatic shock, death due to burns and acute corrosive poisoning.

He states that these four types of anoxia initiate the same vicious cycle, and cause cardiac failure which may lead to death.

Gordon suggests that deaths may be classified in terms of the initial anoxia, and that all post-mortem findings should be divided into two groups, the special features of the particular condition, and the basic pathological changes of circulatory failure. In his view, since anoxia, however caused, produces circulatory failure, the pathological changes associated with this failure vary only in degree in all forms of death, and are otherwise fundamentally similar. He adds that "the intensity of the visceral congestive changes is

relatively slight in the primary cardiac type of collapse as compared with the respiratory form of collapse, since the respiration fails before the heart stops." He directs attention to the term "asphyxia," and points out that asphyxia does not constitute a pathological entity, and cannot clearly be recognised on the basis of morbid anatomical observations alone.

SUDDEN DEATH

Sudden death has been aptly defined as the termination of life which comes quickly under circumstances when its immediate arrival is unexpected.

These deaths arise from a variety of natural causes, of which the more common may be grouped under the following heads :—

Diseases of the cardio-vascular system.

Coronary artery disease and coronary thrombosis.
The sequelæ to coronary thrombosis, rupture of the heart through a softened infarct, and embolism from a thrombus on the endocardial surface of a cardiac infarct.
Valvular disease, especially aortic stenosis.
Rupture of an aortic or other aneurysm.
Acute endocarditis.
Acute myocarditis.
Systemic embolism occurring in bacterial endocarditis.
Fatty degeneration of the heart muscle.
Arterial hypertension with arteriosclerosis.
Pulmonary embolism.
Congenital heart disease in the newly-born.

Respiratory system.

Diseases of the respiratory organs, for example, in cases of pneumonia, especially of the fulminant, influenzal, hæmorrhagic type, acute bronchitis of infancy, rupture of a blood-vessel in pulmonary tuberculosis with cavitation, pulmonary embolism, rupture of an aneurysm into a bronchus, and œdema of the larynx.

The impaction of a foreign body in the larynx, in the case of choking, and regurgitation of stomach contents into the air passages and bronchioles, in an alcoholic state, or in other states of unconsciousness.

Compression of the larynx, or forcible impact against it, with production of cardiac inhibition.

Pressure on the respiratory centre due to tumour or intracranial hæmorrhage.

Central nervous system.

Meningeal hæmorrhage.
Subarachnoid hæmorrhage.
Intracerebral hæmorrhage.

Other causes.

Shock. In such cases, at post-mortem examination, evidence of lymphatism should be looked for, since this condition may have proved a contributory factor.

Extensive rupture of the abdominal organs, especially the liver or spleen.

Ruptured ectopic pregnancy.

Acute infections.

Acute hæmorrhagic pancreatitis.

Hæmorrhage.

Hydrocyanic acid and cyanide poisoning.

Occasionally, epileptic fits.

In a case of sudden death, a medical practitioner should hesitate, unless upon the strongest evidence, to certify the cause of death without a post-mortem examination of the body.

Hamman [11] has published statistics relating to sudden deaths, and has shown that 91 per cent. of sudden deaths from natural causes were due to disease of the cardio-vascular system, and that 65 per cent. of all the cases were due to sudden heart-failure, 21 per cent. to hæmorrhage, and 5 per cent. to arterial embolism and thrombosis. The percentage incidence of lesions in cases of sudden cardiac failure were :—

Disease of the coronary arteries	= 65	per cent.
Valvular heart disease	= 21	,, ,,
Myocardial disease	= 10	,, ,,
Cardiac hypertrophy	= 3	,, ,,

Two instances of sudden death are worthy of comment :—

1. A woman, aged fifty-seven, died suddenly. Post-mortem examination disclosed a rupture of the heart muscle close to the mid-point of the posterior wall of the right ventricle. The rupture, which occurred at the site of an infarction, measured $\frac{1}{8}$ inch in length. The pericardium contained 9 ounces of fluid and clotted blood.

2. A boy, aged twelve years, an excellent swimmer, died suddenly at a private swimming club. Having swam several times across the pool, he left the water and, while standing close to the edge, collapsed and died immediately. Post-mortem examination did not disclose any morbid condition. The lower part of the œsophagus contained two large, undigested portions of potato, the larger measuring $1\frac{1}{2}$ by $\frac{1}{2}$ inches. The stomach contained a considerable quantity of undigested food, including a piece of potato $1\frac{1}{2}$ by $1\frac{1}{4}$ inches in size. The cause of death was cardiac failure, due to inhibition induced by swimming while the process of digestion was in active operation.

References

1. Proc. Roy. Soc. ; (B), Vol. CVII, 214, 1930-31, Smith.
2. "Studies on Muscle," Acta Physiol. Scand., Vol. IX, Supplementum XXV, Stockholm, 1945, Szent-Györgyi. "Chemistry of Muscular Contraction," Academic Press Inc Publishers, New York, 1947, Szent-Györgyi.
3. Jour. Infectious Diseases, Vol. LIV, 395, 1934, Burn.
4. The author is indebted to Dr Alexander G. Mearns, Department of Hygiene, University of Glasgow, for the notes and drawings in connection with the identification of maggots. See also "Medico-Legal Aspects of the Ruxton Case," Livingstone, 1937, Glaister and Brash ; and "Recent Advances in Forensic Medicine," Churchill, 1938, Smith and Glaister.
5. Brit. Med. Jour., Vol. I, 842, 1917, Powell.
6. Police Jour., Vol. XV, No. 2, 140, 1942, Forbes.
7. Brit. Med. Jour., Vol. I, 1070, 1934, Holt and Macdonald.
8. Jour. Amer. Med. Ass., Vol. CI, 261, 1933, Yandell Henderson.
9. Brit. Med. Jour., Vol. II, 337, 1944, Gordon.
10. "Shock and Related Capillary Phenomena," Oxford University Press, London, New York, Toronto, 1938, Moon.
11. Bull. Johns Hopk. Hosp., Vol. LV, 387, 1934, Hamman.

CHAPTER V

DEATH CERTIFICATION AND CREMATION

CERTIFICATION of death is a statutory duty imposed upon members of the medical profession which must be performed without fee. Failure to certify is followed by penalty. It is necessary, therefore, that practitioners should fully understand their responsibilities with regard to certification.

The young practitioner will find it profitable to study the printed matter found in each book of blank forms of certificates issued by local registrars.

It is illegal to dispose of a dead body without a Registrar's certificate or a Coroner's or Procurator-Fiscal's order.

England.

The principal provisions of the Births and Deaths Registration Acts, 1836-1929, are :—

Every certificate of the cause of death required to be given shall be in the prescribed form (see pp. 157 and 158).

The certificate shall be delivered forthwith, by the registered medical practitioner by whom it is signed, to the Registrar.

The practitioner, on signing a certificate, shall give in the prescribed form, to some person required by the Registration Acts to give information concerning the death, notice in writing of the signing of the certificate, and that person shall, except where an inquest is held on the body of the deceased person, deliver this notice to the Registrar (see p 157).

In the case of every still-birth, it is the duty of the person, who would have been required to give information to the Registrar had the child been born alive, to give information to the Registrar of the particulars required to be registered concerning the still-birth, unless there has been an inquest.

Every such person upon giving information shall either deliver to the Registrar a written certificate, that the child was not born alive, signed by a registered medical practitioner or certified midwife who was in attendance at the birth or who has examined the body of such child, or make a declaration in the prescribed form to the effect that no medical practitioner or certified midwife was present at the birth or had examined the body or that his or her certificate cannot be obtained and that the child was not born alive.

It should be noted that the terms " still-born " and " still-birth " are defined as applying to any child which has issued forth from its mother after the twenty-eighth week of pregnancy and which did not at any time after being completely expelled from its mother, breathe or show any other signs of life. It is unlawful to have a still-born child buried without the written sanction of a Coroner or Registrar (see p. 153).

A medical practitioner, unless there are reasonable grounds for suspicion, cannot withhold a death certificate but must certify the cause of death when known, irrespective of the cause.

When the practitioner is unable to decide between symptoms of natural disease and those due to unnatural causes, it is advisable for him, before granting the certificate in such circumstances, to interview the Coroner, in England, or the Fiscal, in Scotland, who will advise the best course to be followed. Deaths certified as directly or indirectly due to violence, or which have been attended by suspicious circumstances, also sudden deaths and every death the cause of which is stated to be unknown, are reported to the Coroner by the Registrar in accordance with his instructions. When a doctor has appended his initials to Statement " A " on the back of the Death Certificate Form, it is the duty of the Registrar of Deaths receiving the certificate to refrain from registering the death until he has been informed by the Coroner what action the Coroner has taken in regard to the death. Failure to certify renders the practitioner liable to a penalty of 40s., and issuing a false certificate makes him liable, on summary conviction, to a fine of £10, or on conviction on indictment, to seven years' imprisonment.

In the case of a death which, on information given by a practitioner or others, has been the subject of an inquest, it becomes the duty of the Coroner to send to the Registrar a certificate of the finding of the jury as to the cause of death within the time required by the Registration Acts.

The following are important points in connection with certification of death in England :—

A medical practitioner may not issue a certificate unless he has been in attendance upon the deceased during his or her last illness.

No other practitioner may sign the certificate on his behalf unless he has also been in attendance upon the deceased during his or her last illness.

With regard to the cause of death, it is desirable that the terms embodied in the Nomenclature of the Royal College of Physicians of London, or in the Registrar-General's Manual of the International List of Causes of Death should be employed. Vague and ill-defined terms should be avoided.

In all cases of sudden deaths or deaths under any circumstances of suspicion, it is the duty of those who are about the deceased to give immediate notice to the Coroner or his officer or to the nearest officer of police, who then communicates with the Coroner.

In certain cases, a practitioner when signing the certificate may have knowledge that a post-mortem or laboratory examination is

to be made, and provision to intimate this information is available on the back of the certificate (see p. 158).

If the certifying doctor has not seen the deceased during the fourteen days before death, the Registrar must notify the Coroner to this effect (see p. 157).

In all cases the practitioner should verify the occurrence of death before granting the certificate (see p. 159).

When the body of a deceased person has to be removed from England or Wales, notice must first be given to the Coroner in whose jurisdiction the body is lying, and prior to its removal the Coroner's permission must be obtained following inquiry.

Scotland.

The enactments bearing upon the subject are the Births, Deaths, and Marriages (Scotland) Acts, 1854-60, and the Registration of Still-Births (Scotland) Act, 1938. Under the Acts of 1854-60 the practitioner who attended during the last illness of a deceased person must give to the Registrar a certificate of the cause of death within seven days following the death (see p. 160). Should he fail to do so, the Registrar must send him a certificate-form partly filled in, accompanied by a request that the completed form be returned to him within three days after receipt under a penalty not exceeding 40s. in case of failure. Although it is the duty of a practitioner to submit the certificate of death to the Registrar, the custom is for the practitioner to hand the certificate to a near relative of the deceased for transmission to the Registrar.

In cases which have been inquired into by the Procurator-Fiscal, this official must inform the Registrar of the result of the inquiry, but there is no defined time within which the information must be returned to the Registrar. Contrasted with England, the relation of the practitioner to the duty of certification of deaths is legally different. In England he is bound forthwith to deliver the death certificate to the Registrar. In Scotland, on the other hand, the practitioner is bound to send it to the Registrar within seven days after death, or within three days after notice by the Registrar, when he has failed to do so.

All deaths are classified by the Registrar-General, and as a rule he classifies each death according to the cause specified as the Primary Disease, line (*a*), in the certificate (see p. 160). It should be noted that in England the term " Immediate Cause " is employed (see p. 157). This should be the cause which initiated the train of events leading to death, or, where two or more independent causes are specified, the condition which the certifier considers as mainly responsible for the death. Where, however, the primary cause is of a general character, for example, arteriosclerosis, the particular manifestation, cerebral hæmorrhage, nephritis, etc., should be specified in line (*b*), as the death will be classified by the Registrar-General to the local manifestation. This is also applicable to certificates issued in both England and Wales, the intention being to

adopt the certifier's preference in each case. It is of the utmost importance that this should be kept in mind by the certifier and that his preference should be clearly stated. Cases sometimes arise in which the practitioner is precluded from stating fully the cause of death and he may have to use a general term. In such instances, the specified term used should be qualified by a word such as "idiopathic" to indicate difficulty in the expression of the full cause. In the certification of death, general terms, for example, "Disease of the Heart," must not be employed, and the nature of the disease must be specifically stated.

The suggestions regarding certification, in each book of forms, should be studied carefully. Books of Certificate Forms (Schedule G) may be obtained by registered medical practitioners from the Registrar of Births, Deaths, and Marriages of the registration district in which they reside.

The Registration of Still-Births (Scotland) Act, 1938, requires that all still-births occurring in Scotland must be registered by the Registrar. The term still-birth is defined under the Births and Deaths Registration Act, 1926, as applicable to England (see p. 154). For the purposes of the Registration Acts, every birth which occurs will fall into one or other of three classes :—

A child who, whatever the duration of pregnancy, breathes or shows any other signs of life after complete expulsion from the mother, is a live-born child and the birth must be recorded in the Register of Births. If the child dies, even within a brief period after birth, both the birth and the death fall to be registered.

The birth of a child before the end of the twenty-eighth week of pregnancy, which did not breathe or show signs of life after complete expulsion from the mother, is not required to be registered.

The birth of a child after the twenty-eighth week of pregnancy which, after complete expulsion from the mother, did not show any signs of life, is a still-birth, and must be registered in the Register of Still-births (see p. 162).

The Act provides that when a still-birth is registered the relative or other person giving the information must either deliver to the Registrar a Certificate of Still-birth, signed by a registered medical practitioner or by a certified midwife, stating that the child was not born alive and, where possible, the cause or probable cause of death, or make a statutory declaration in the prescribed form that no registered medical practitioner or certified midwife was present at the birth, or had examined the body, or that his or her certificate cannot be obtained, and that the child was not born alive.

It is unlawful to have the body of a still-born child buried without the written sanction of a Procurator-Fiscal, or the appropriate Registrar's Certificate.

Under the Children and Young Persons (Scotland) Act, 1937, where a person undertakes for reward the nursing or maintenance of

DEATH CERTIFICATE FORM USED IN ENGLAND. (Front)

COUNTERFOIL

For use of Medical Attendant, who should in all cases fill it up.

Name of Deceased .....

....

Date of Death ..... Age.....

Place of Death .....

.....

Last seen alive .....

Seen* / Not seen* after death.

Cause of death confirmed* / not confirmed* by P.M.

Cause of Death :—

I. *a*.....

due to b.....

due to c.....

II. .....

* Strike out whichever is inapplicable.

BIRTHS AND DEATHS REGISTRATION ACTS, 1836 TO 1929

Registrar to enter No. of Death Entry. .....

MEDICAL CERTIFICATE OF CAUSE OF DEATH

For use only by a Registered Medical Practitioner **WHO HAS BEEN IN ATTENDANCE** during the deceased's last illness, and to be delivered by him forthwith to the Registrar of Births and Deaths direct.

Name of Deceased .....

Date of Death as stated to me.....day of.....19... Age as stated to me...

Place of Death .....

Last seen alive by me .....day of.....19..... Seen* / Not seen* after death by me.

The certified cause of death has* / has not* been confirmed by Post-mortem.

CAUSE OF DEATH

I. Immediate cause†	*a* .....
Morbid conditions, if any, giving rise to immediate cause (stated in order proceeding backwards from immediate cause)	*due to b* ..... *due to c* .....
II. Other morbid conditions (if important) contributing to death but not related to immediate cause ...	....

I hereby certify that I was in medical attendance during the above-named Deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature..... *Qualifications as registered by Medical Council* .....

Residence..... *Date*.....

* Strike out whichever is inapplicable.

† This does not mean the mode of dying, such as *e.g.*, heart failure, asphyxia, asthenia, etc.: it means the disease, injury, or complication which caused death.

SEE BACK

NOTICE TO INFORMANT

I hereby give notice that I have, this day, signed a Medical Certificate of the Cause of Death

of .....

..... deceased

Signature.....

Date.....

This Notice must be given by the Certifying Medical Practitioner to the person who is qualified and liable to act as Informant for the purpose of the registration of the death. As to the person liable to act as Informant, see back.

DUTIES OF INFORMANT

The Informant must deliver this Notice to the Registrar of Births and Deaths of the Sub-District in which the Death took place, bearing in mind that the Death cannot be registered until the medical certificate has reached the Registrar. Failure to deliver this Notice to the Registrar renders the Informant liable to prosecution.

The Informant must be prepared to state accurately to the Registrar the following particulars :—

(1) The Date and Place of Death, and the place of Deceased's usual residence, (2) the full Names and Surname, (3) the correct Age, (4) the Occupation, and (5) whether Deceased was in receipt of a naval or military pension, an old age pension, or other pension or allowance from public funds.

DECEASED'S IDENTITY CARD AND RATION BOOK MUST BE DELIVERED TO THE REGISTRAR.

DEATH CERTIFICATION

DEATH CERTIFICATE FORM USED IN ENGLAND. (Back)

PERSONS QUALIFIED AND LIABLE TO ACT AS INFORMANTS

The following table shows in full the persons, in the order of their successive liability, who are designated by the Births and Deaths Registration Acts as Informants :—

Deaths in Houses:
(1) A *Relative* of the Deceased, *present at the Death.*
(2) A *Relative* of the Deceased, *in attendance* during the last illness.
(3) A *Relative* of the Deceased, dwelling or being at the time of Registration in the Sub-District wherein the Death occurred.
(4) A Person *present at the Death.*
(5) The *Occupier* } of the House in which the Death occurred.
(6) An *Inmate* }
(7) The Person *causing the Body to be buried.*

Deaths not in Houses, or Dead Bodies Found Exposed :
(1) Any *Relative* of the Deceased having knowledge of the required particulars.
(2) Any Person *present at the Death.*
(3) Any Person *who found the Body.*
(4) Any Person *in charge of the Body.*
(5) The Person *causing the Body to be buried.*

Deaths in Institutions :
(1) Any *Relative* of the Deceased who may be available.
(2) The *Chief* (or *Acting Chief*) *Resident Officer.*
(3) A Person *present at the Death.*
(4) The Person *causing the Body to be buried.*

A. Reported to Coroner ?

.....

B. Further information offered ?

.....

N.B.—If either Statement A or Statement B has been filled up, the fact should be noted in the appropriate place above.

Fill up where applicable.

A.

I have reported this case to the Coroner.

Initials of Certifying Medical Practitioner } .....

Fill up where applicable.

B.

I may be in a position later to give, on application by the Registrar-General, additional information as to the cause of death for the purpose of more precise statistical classification.

Initials of Certifying Medical Practitioner } .....

NOTE.—The Practitioner, on signing the certificate, should fill up, sign and date the Notice to the Informant, which should be detached and handed to the Informant. The Practitioner should then, without delay, deliver the certificate itself to the Registrar of Births and Deaths for the sub-district in which the death occurred. It may be delivered by post ; and the Practitioner is supplied by the Registrar, gratis, with postage-paid envelopes for this purpose.

a child under nine years and such child dies, it is the duty of such persons to give notice in writing to the Procurator-Fiscal within twenty-four hours of the death. The Procurator-Fiscal will then hold an inquiry into the cause of death unless a certificate by a duly qualified medical practitioner is produced to him certifying that he has personally attended the child during his last illness and specifying the cause of death (see p. 226).

Since questions may arise as to the disposal of the remains of a child, born dead before the end of the twenty-eighth week of pregnancy, it may be stated that, apart from the general Public Health Aets, there is apparently no law on the subject.

Important points in death certification.

A doctor is bound to give on a death certificate the date upon which he last saw the deceased person alive, and it may be that the date is not very proximate to the date of death, or he may be asked to certify the death of a person whom he has only seen on one occasion. In these circumstances, it is particularly important that the practitioner should verify the fact of the occurrence of death before he grants the certificate.

It is also important that a practitioner should not grant a certificate of death of a person whom he has not seen for a length of time, even although the person is known to him as suffering from a disease which might prove fatal, until he has inquired into the circumstances and examined the body.

Under no circumstances should a practitioner be tempted to sign blank certificates, or to sign a certificate of death of a person while that person, although critically ill, is still alive. This may not only expose him to a charge of false certification, but his action may be deemed "infamous conduct in a professional respect" by the General Medical Council, and may result in the removal of his name from the Medical Register.

CREMATION

The Cremation Act, 1902, and Regulations made thereunder applicable to England and Scotland, deal with the legal aspects of the disposal of dead bodies by incineration. In view of the fact that cremation of a body destroys all evidence of crime, so far as the body is concerned, the procedure, prior to cremation, is more elaborate than that required for ordinary burial.

The procedure in England and Scotland is as follows :—

England.

The following are the principal provisions of the Regulations made in 1930, under Section 7 of the Cremation Act, 1902, and Section 10 of the Births and Deaths Registration Act, 1926 :—

Except where an inquest has been held or a post-mortem examination made and a certificate is given by a Coroner, no cremation is

DEATH CERTIFICATE FORM USED IN SCOTLAND

COUNTERFOIL

No. .....

(Before filling up Certificate, please see "Suggestions" on page ii, especially the first paragraph; and Examples on page v.)

Name of Deceased

Date of Death

Place of Death

When last seen

Cause of Death :—

(*a*)

(*b*)

(*c*)

(*d*)

Pregnancy?

Date of Certification

This Certificate is intended solely for the use of the Registrar of Births, Deaths, and Marriages, and the Registrar-General cautions all persons against accepting or using this Certificate for any other purpose.

MEDICAL CERTIFICATE — WITH CAUSE OF DEATH

No. SCHEDULE (G).

No. of Regn. District.....

No. of Entry in D. Reg.....

Year.....

To the Registrar of the *of* *in the*

of

I hereby certify that I attended *, who died on the*

day of 19 *, at* *, that I last saw the Deceased on the*

day of 19 *, and that the Cause of Death and Duration of Disease were as undernoted :—*

		Cause of Death.	Duration of Disease. Years.	Months.	Days.
Primary Disease	(*a*)				
Secondary Diseases (if any)	(*b*)				
	(*c*)				
	(*d*)				

Witness my hand this *day of* .....19 *Signature*

Professional Title

Residence.....

If the Deceased was a Married Woman, and the death was known to have occurred during pregnancy or within four weeks thereafter, the Certifier will indicate the fact by here inserting the word "Yes": .....

allowed until the death of the deceased has been registered or a certificate has been given, in pursuance of Section 2 of the Births and Deaths Act, 1926, that the death of the deceased is not required by law to be registered in England.

The registered medical practitioner in attendance upon the deceased during his last illness and who can certify definitely the cause of death, must provide a certificate stating the cause of death.

A second certificate or confirmatory certificate must be furnished by another registered medical practitioner, who must be of not less than five years' standing, and who is not a relative of the deceased or a relative or partner of the doctor who has given the first certificate.

When a post-mortem examination has been made by a medical practitioner appointed by the Cremation Authority, a certificate granted by him will be accepted in place of the two certificates previously mentioned. When a post-mortem examination has been made and the cause of death has been certified by the Coroner, these two certificates are not required.

Cremation is not allowed except on the written authority of the medical referee appointed by the Secretary of State on the nomination of the Cremation Authority. The referee, following personal investigation of the cause of death, may grant the confirmatory certificate, or if he has made a post-mortem examination, he may grant a certificate in place of the two certificates usually required. The medical referee will not authorise any cremation except where a post-mortem examination has been made, or an inquest has been held and a certificate given by the Coroner, or after the production of a certificate that the death of the deceased has been duly registered, and he is satisfied that the fact and cause of death have been definitely ascertained. He will refuse cremation if the cause of death assigned in the medical certificates might be due to poison, violence, illegal operation, or privation or neglect until a post-mortem examination has been held. If the post-mortem examination fails to reveal the cause of death, he will decline to allow the cremation unless an inquest is held and the Coroner gives a certificate.

If it appears that death was due to poison, violence, illegal operation, privation or neglect, or if there is any suspicious circumstance, he shall not allow the cremation unless an inquest is held and a certificate is given by the Coroner. The medical referee may permit the cremation of the remains of a still-born child if it is certified to have been still-born by a registered medical practitioner after examination of the body, and if the referee, after such inquiry as satisfies him, considers that it was still-born and that there is no reason for further examination. Before permitting the cremation, he shall, except where an inquest has been held and a certificate has been given by the Coroner, require the production of a Certificate of Registration of Still-birth.

It is unlawful to cremate the remains of any person known to have left a written direction to the contrary or to cremate remains which are unidentified, and cremation will not be allowed unless application has been made and signed by an executor or by the

CERTIFICATE OF STILL-BIRTH FORM USED IN SCOTLAND

COUNTERFOIL

Surname of Father (or Mother) .....

Date of Still-birth .....

Place of Still-birth .....

.....

Cause of Death of child .....

Date of certification .....

This Certificate is intended solely for the use of the Registrar of Births, Deaths, and Marriages.

CERTIFICATE OF STILL-BIRTH

No. of Regn. Dist......

No. of Entry in S. B. Reg......

Registration of Still-births (Scotland) Act, 1938 (1 & 2 Geo. 6, c. 55), Section 1 (2)

The above-mentioned Act defines Still-birth as follows:—"Still-born" and "Still-birth" shall apply to any child which has issued forth from its mother after the twenty-eighth week of pregnancy and which did not at any time after being completely expelled from its mother breathe or show any other signs of life.

(1) This portion of the Form to be used when the person certifying was in attendance at the Birth.

I hereby certify that.....*was delivered of a* * *male* / * *female* *Child on* .....19..., *at*.....; *that I was in attendance at the birth of such child; and that such child was NOT BORN ALIVE.*

(2) This portion of the Form to be used when the person certifying has examined the body of the child, but was not in attendance at the Birth.

I have examined the body of a * *male* / * *female* *child of which I am informed and believe that*.....*was delivered on*.....19..., *at*.....*, and I hereby certify that such child was NOT BORN ALIVE.*

(3) This portion of the Form to be filled up in every case.

I further certify that in my opinion the Cause or Probable Cause of Death of such child was.....

Witness my hand this..... *day of*.....19...

Signature.....

Professional Title (and Registered No. if a Certified Midwife) .....

Residence .....

* Strike out whichever is inapplicable.

NOTICE.—This Certificate must be delivered to the Registrar of Births, Deaths, and Marriages by the person attending to give information of the particulars required to be registered concerning the Still-birth. It is not an authority for the burial of the body of the still-born child in a burial ground or for cremation in a crematorium.

Attention is directed to the provisions of the Cremation Act, 1902, and relative Regulations regarding the disposal of human remains by burning.

A list of the persons required to give information for the registration of a still-birth is given on the top of this form.

Persons required to give information for the registration of a still-born child are:—

(1) the Father (of a legitimate child only);
(2) the Mother;
(3) the occupier of the house in which the birth occurred; or in the case of a birth in an institution the chief (or acting chief) resident officer;
(4) the nurse present at the birth.

REGISTRATION OF STILL-BIRTHS (SCOTLAND) ACT, 1938

SECTION 1 (2) provides as follows:

"Every person upon giving information regarding a still-birth shall

(*a*) deliver to the Registrar a certificate in the prescribed form stating that the child was not born alive, and, where possible, the cause or probable cause of death, which certificate shall, if a registered medical practitioner was in attendance at the birth or has examined the body of the child, be signed by him, and otherwise shall be signed by a certified midwife who was in attendance or examined the body; or

(*b*) make a declaration in the prescribed form to the effect that no registered medical practitioner or certified midwife was present at the birth or has examined the body, or that his or her certificate cannot be obtained and that the child was not born alive."

nearest surviving relative of the deceased. If the application is made by any other person, a satisfactory reason why such application was not made by an executor or nearest surviving relative must be given. The application contains a statutory declaration which must be made before, and signed by, a Justice of the Peace or a Commissioner for Oaths. No cremation shall take place except on the written authority of the medical referee following scrutiny of all documents referred to, and unless satisfied that the fact and cause of death have been definitely ascertained, that there exists no reason for further inquiry and examination, and that all the regulations have been carried out. Without reasons assigned, he is empowered to withhold this authority. He is also authorised to make any inquiries which he may deem necessary before granting the authority to cremate.

Scotland.

The legal provisions with regard to cremation are contained in the Cremation (Scotland) Regulations, 1935, made under Section 7 of the Cremation Act, 1902. The procedure is very similar to that in England (see p. 159). The following are the noteworthy features:—

The death of the deceased must have been registered and a certificate of registration, from the Registrar of Births, Deaths, and Marriages, produced. If death occurred in Northern Ireland, an equivalent certificate of registration must be shown. When death has taken place in England, an acknowledgment by a Coroner that notice of the intention to remove the body out of England has been received by him together with an intimation that he does not intend to hold an inquest, or a certificate, following an inquest, is issued by the Coroner. The application for cremation must have the contained particulars confirmed by a statutory declaration made before, and signed by, a Justice of the Peace, Notary Public, or Magistrate. If a medical practitioner has not been in attendance upon the deceased during his last illness, a registered medical practitioner, who is the ordinary medical attendant of the deceased, may give the first medical certificate provided that he can certify definitely the cause of death. The usual first medical certificate and the confirming medical certificate are dispensed with when a post-mortem examination has been made by a medical practitioner appointed by the Cremation Authority and a certificate is given by him, or if the medical referee has performed a post-mortem examination and furnished a certificate. The customary two certificates are also not necessary when permission for cremation has been given in writing by the Procurator-Fiscal of the district in which the death occurred, or where the death has occurred in England and the cremation of the remains is to take place in Scotland, a post-mortem examination has been made and the cause of death has been certified by a Coroner and a certificate has been given by him, or an inquest has been held and a certificate has been granted by the Coroner. The medical referee is appointed by the Cremation Authority, and the appointment must be

notified by the Authority to the Secretary of State and to the Department of Health for Scotland. The duties of the medical referee in Scotland are the same as in England (see p. 161). In the event of any suspicious circumstances coming to his knowledge, whether revealed in the application or certificates, or otherwise, and in particular if the cause of death assigned on the medical certificates be such as, regard being had to all circumstances, might be due to poison, violence, illegal operation, privation or neglect, or if there is any reason to suspect that death occurred while the deceased was under an anæsthetic, the medical referee must report the matter immediately to the Procurator-Fiscal of the district in which death occurred, and shall not authorise cremation except with the written permission of such Procurator-Fiscal. In the case of the remains of a person who has died in any place out of Scotland, the medical referee may accept a declaration, containing the particulars required, if it has been made before any person having authority in that place to administer an oath or to take a declaration. He may accept certificates if signed by medical practitioners who are shown to his satisfaction to possess qualifications substantially equivalent to those prescribed for each certificate by the Regulations. The medical referee may authorise the cremation of the remains of a still-born child if it has been certified to have been still-born either by the registered medical practitioner who attended at the confinement of the mother or by a registered medical practitioner after a post-mortem examination of the body, and if the medical referee, after such inquiries as he may think necessary, is satisfied that it was still-born and there is no reason for further examination (see p. 162). Where there are any suspicious circumstances in connection with the birth of a still-born child, the medical referee must immediately report the matter to the Procurator-Fiscal of the district in which the birth is alleged to have occurred, and shall not authorise cremation of the remains except with the written permission of the Procurator-Fiscal.

In July 1944, a doctor was fined £50 at Aberdeen Sheriff Court, when he pleaded guilty to nine contraventions of the False Oaths Act, in connection with the signing of death certificates for cremation. The charge alleged that the doctor had wilfully made the false statements in schedules which had to be completed to satisfy cremation regulations, by stating that he had questioned the doctors who had signed the death certificates, when in fact he had not done so. The Procurator-Fiscal said that when a cremation was to take place, a form certifying the cause of death was signed by the deceased's own doctor and, as a further safeguard, a second doctor had to confirm the cause of death, and thus this second doctor was asked to state in the official form that he had questioned the doctor who had attended the deceased. In none of the cases mentioned had the accused done so. It was a serious matter because the safeguard of the second doctor had not been taken. This case was one of a contravention of the False Oaths (Scotland) Act, 1933, Section 2. The president of the General Medical Council announced

that the conviction had been proved and the certificates in question came within the warning notice against untrue, misleading, and improper certificates. The Council did not regard the doctor's explanation as affording any excuse for his laxity, and took a grave view of carelessness in the issue of public certificates. As, however, they were prepared to believe that he had signed these certificates, not from any perversity or desire to falsify, but from a mistaken view of his duty and an error of judgment and that the warning he had received would be sufficient, they did not direct the erasure of his name.

The Cremation Act, 1902, applicable to both England and Scotland, contains a penalty section by means of which the Regulations can be enforced. Section 8 enacts that :—

1. Every person who shall contravene any such regulation, or shall knowingly carry out or procure or take part in the burning of any human remains, except in accordance with such regulations and the provisions of this Act, shall (in addition to any liability or penalty which he may otherwise incur) be liable, on summary conviction, to a penalty not exceeding £50. Provided that any person aggrieved by any conviction may appeal therefrom.
2. Every person who shall wilfully make any false declaration or representation, or sign or utter any false certificate, with a view to procuring the burning of any human remains shall (in addition to any penalty or liability which he may otherwise incur) be liable to imprisonment, with or without hard labour, not exceeding two years.
3. Every person who, with intent to conceal the commission or impede the prosecution of any offence, attempts to procure the cremation of any body, or, with such intent, makes any declaration or gives any certificate under this Act, shall be liable to conviction on indictment to imprisonment for a term not exceeding five years.

PRESUMPTION OF SURVIVORSHIP

When more persons than one die at or about the same time from more or less similar accidental causes, it occasionally becomes necessary to solve the difficult problem of survivorship for the purpose of succession to property. In England, the Law of Property Act, 1925, has done much to overcome the frequent difficulties encountered in such cases. This Act, stated broadly, lays down that where two or more persons die as the result of circumstances which make it uncertain as to which survived the other or others, then, subject to any order of the court, such deaths shall, for the purposes affecting the title to property, be presumed to have occurred in the order of seniority unless evidence to the contrary is forthcoming. The Grosvenor case [1] is worthy of mention since in this connection the words "subject to any order of the court" were construed

to mean that the court could receive evidence, and act upon it in displacement of the new statutory presumption. Two brothers were killed with other people when a bomb fell on a house. Mr Justice Cohen held that, in the absence of proof, the statutory presumption must apply. The Court of Appeal, however, held that the air-raid deaths were to be taken as simultaneous, but the House of Lords reversed this decision.[2] In Scotland, however, there is no similar enactment and thus it is left to the courts to decide matters. With regard to the medical issues involved, the most careful consideration must be directed to each and all of the specific circumstances attending each individual case before an opinion should be expressed. By way of illustration, the following are the facts of one of our cases and the opinions expressed regarding it :—

Two brothers, A and B, were found dead in a gas-filled room and a considerable sum of money was at issue. A was aged seventy-two and B sixty-two.

The following facts, circumstances, and inferences led to the opinion that A had predeceased B :—

A usually slept in the kitchen and B in the bedroom. Both bodies were found in the bedroom. Both persons were found dead thirty-seven hours after having last been seen alive. Post-mortem examinations were made three days and fourteen hours after the men had last been seen alive. The body of B showed marked evidence of putrefaction and suggested that B had died a considerable time before A. Since both men had last been seen alive at a quarter past nine in the evening and were, on account of certain circumstances, assumed dead at ten o'clock on the following morning, the overnight period, amounting to twelve hours, failed to give adequate time for the difference in the two bodies, on a purely time basis, and the reasons must have depended on other factors. The bodies were first examined by a police doctor, thirty-nine hours after the men had last been seen alive, when there was similarity in the post-mortem appearances, therefore it was assumed that the signs of putrefaction on the body of B had manifested themselves between his examination and the dissections of the bodies. Having regard to the average rate of putrefaction, under average conditions, and to the circumstances of the case, the more advanced state of putrefaction of the body of B was held as due to extrinsic and intrinsic causes apart from the time factor. Such factors were :—

B was in bed, above draught level, fully clothed, except for jacket and boots. He wore stockings and his body was covered with one or more blankets and a bed-mat.

On the other hand, A lay on the floor and was attired in shirt, drawers, and undervest. His feet were uncovered.

Having regard to the relative positions of the bed and the window, what sunshine there might have been during the earlier part of July might readily have fallen upon the body of B, and

would have had a hastening effect upon the process of putrefaction. In the case of A, who had been a patient in hospital until just before his death, his bowel movements had probably been carefully regulated and thus his lower bowel was emptier than that of B.

A, when found, had a gas tube in his mouth and had, therefore, been inhaling a high concentration of the gas. The elimination of oxygen from the blood would retard putrefaction. B was found in bed in the room which he normally occupied and was practically fully clothed, which suggested that he had not retired to bed but had merely been resting. A, on the other hand, had apparently risen from bed, and gone into the bedroom, taking his blanket with him, since this blanket was found around his head. It seemed quite certain that A committed suicide by gas poisoning, for the nozzle of the gas tube was still in his mouth and the tube was still grasped in his hand when the bodies were discovered. It was equally certain that B did not have the tube in his mouth after A's death. There was nothing to indicate that B had committed suicide, but, at the same time, his death was fully consistent with accidental gas poisoning, due to the suicidal act of A. There was no doubt that A had sustained a massive dose of gas which must have produced death within a very short period, and that some little time must have elapsed before a sufficient concentration could have been obtained in the room to cause the death of B. If it was assumed that the deaths of both men had resulted from a suicidal pact, it was equally clear that A must have predeceased B. For B to have predeceased A, he must have poisoned himself with gas before A put the tube in his own mouth. This led to the assumption that B turned on the gas, got into bed, and covered his head, a method of committing suicide which is contrary to experience in such cases. It led further to the assumption that after the gas had accumulated in the room in sufficient quantity to render B unconscious, A had come into the gas-filled room, decided on his own destruction, obtained a blanket from the kitchen, and arranged himself on the floor as found. If this had been the true construction, then, in order to allow A sufficient time to arrange matters for his own suicide, the gas concentration must have been relatively low in the room, and the death of B would have been a relatively slow process. In the case of A, however, the tube was placed in the mouth and the inhalation of undiluted gas would encompass his death before the diluted gas in the room could have caused the death of B.

The conclusion reached was that A had committed suicide; B had died as the result of the act of A; and A had predeceased B.

References

1. Grosvenor Case [1944], Lancet, Vol. I, 129; also *re* Grosvenor [1944], 1 All E.R., 81.
2. Hickman v. Peacey [1945], 2 All E.R., 215.

CHAPTER VI

ASPHYXIA

DROWNING, SUFFOCATION, TRAUMATIC ASPHYXIA, HANGING, STRANGULATION, AND THROTTLING

DROWNING

DROWNING is a comparatively common form of death. It is therefore important that what is meant by death by drowning should be clearly defined. A person may be said to have died from drowning into whose air-passages and lungs air has been prevented from entering by any watery, viscid, or pultaceous fluid into which the head has fallen and remained. Death by drowning is an asphyxial death, and the post-mortem signs of asphyxia will be present in the body of any person who dies in water following continued respiration. The measure of their intensity will, however, be determined by the degree and duration of the struggle for breath. In the entire absence of these signs, it may safely be affirmed that death was not due to drowning.

When, however, respiratory action ceases at or before the moment of submersion, the signs of asphyxia will be absent. A person walking along the bank of a river or canal, may be seized with a fatal syncopal attack, and fall into the water, but in such cases, respiration being permanently arrested, water will not be inspired, consequently, the signs of asphyxia will be absent, and death will not be due to drowning. On the other hand, in cases where persons fall into the water in a fainting condition, or as the result of an apoplectic or epileptic seizure, or serious head injury, the result of having fallen from a height, the respiratory function will continue in variable degree, and the death will be an asphyxial one. The post-mortem appearances in such cases will show a difference in degree of the asphyxial signs due to the diminished amount of struggle for breath prior to death, and lesions may be found, for example, cerebral hæmorrhage, and injuries to the body in cases of accident.

For the fulfilment of the act of drowning it is not necessary that either the body or the whole of the head be submerged, since submersion of the mouth and nostrils and inability of the person, so situated, to withdraw himself are the necessary factors. Intoxicated persons, epileptics, and others have been drowned in small pools or shallow puddles of water. We have examined the body of an intoxicated person who fell face downwards in a shallow puddle of water about $1\frac{1}{2}$ feet in diameter and about 3 inches in depth. In

another case a man committed suicide by placing his head in a basin filled with water. Usually, however, the body is submerged.

Before attempting the reconstruction of the likely events prior to death, the most careful and detailed consideration must be directed to the innate circumstances attending the specific case. The absence of asphyxial signs indicates that death took place either before, or almost immediately after, immersion.

The duration of the process of drowning varies from about two to five minutes. Variation in the time depends upon the circumstances of each individual case, for example, whether the person is able or unable to swim, whether presence of mind is or is not retained, and the condition of the person on entering the water. Since the specific gravity of the human body is slightly greater than that of water, when the lungs are ordinarily full of air and the whole of the body is submerged, any little deprivation of air or the raising of a part of the body out of the water is all that is required to cause sinking. Inability to swim, and struggling, are important factors in the submersion of a body.

It must not be forgotten that a person on entering the water may die suddenly before the process of drowning has been properly initiated. This may result from laryngeal spasm due to the sudden entry of water into the larynx. The suddenness of immersion in cold water may cause its impingement on the nasal or post-nasal mucous membrane, and since the glosso-pharyngeal and vagus nerves carry stimuli from the mucous membrane to the central nervous system, death may result from cardiac inhibition.

The first medico-legal question which arises in the examination of a body removed from water is :—

Was death due to drowning ?

From the fact that a large number of deaths are caused by accident during occupation, while bathing or boating, or by set purpose, as in suicide, a presumption of drowning arises. It must not be forgotten, however, that, since a body might have been thrown into the water to mask the commission of crime, presumption is of no value. The examiner should therefore preserve an open mind, since dissection of the body will indicate proof as to whether death was due to drowning or not.

External appearances.

Noteworthy bodily features in death from drowning are :—

Coldness of body.
Pallor of body surface.
Facial appearance.
Appearance and situation of tongue.
Presence or absence of cutis anserina.
Condition and appearance of hands and feet.
Retraction of penis and scrotum.

It should be clearly understood that when putrefactive changes are well developed, many of the foregoing appearances may either be masked or completely destroyed.

Coldness of the body and pallor of the body surface are found in many cases, apart from drowning, and may be dismissed.

Fig. 48
Case of drowning. Note pinkish colour due to exposure to cold.

The face of a drowned person is usually pale, but may show a slight patchy, reddish colour, especially over the cheeks and on the lips, when there has been exposure to cold. This is the result of oxygenation of the hæmoglobin through the capillaries of the skin and mucous membrane. Such coloration may also be present over

Fig. 49

Sodden condition of skin on palmar aspect of fingers and palm of hand in a case of drowning.

Fig. 50

Similar condition to Fig. 49, involving skin on plantar aspect of toes and sole of foot.

other parts of the body (see Figs. 48 and 87). The facial appearance is, however, radically altered following the onset of putrefaction, when it becomes bloated and discoloured. The only sign which may be seen on the face and which may be regarded as presumptive of drowning, is the presence of a fine foam or froth, very occasionally blood flecked, at the mouth, or nostrils, or both (see Fig. 51). Various causes, however, may prevent this from being seen, such as cleansing

of the face, or the froth having been washed away in the process of removal of the body from the water. When originally present and effaced in these ways, it will usually reappear within a short time either as the result of rigor mortis or of putrefaction. The froth is composed of fine bubbles and results from the churning together of air, mucus, and water.

In a number of cases the tip of the tongue may be pressed against the margins of the teeth or protruded slightly beyond them, but in the majority of cases it occupies a normal position.

With respect to the presence of cutis anserina and retraction of the penis and scrotum, nothing more can be shown by their presence than that molecular death of the tissues had not taken place at the time of immersion. Cutis anserina, or "goose-skin," is caused by the contraction of involuntary muscular fibres and the papillæ of the skin can be seen elevated above the normal level.

The condition and appearance of the hands and feet of a body taken from water should always be noted. There are certain appearances found on these parts of every body which has lain in water, whether death has been caused by submersion or not, due to physical causes and apart from the existence of life after entering the water. The changes seen consist of a bleached or sodden condition of the palms of the hands and palmar aspects of the fingers and of the soles of the feet and plantar aspects of the toes. This is caused by the action of the water on the thickened epidermis of these parts. From the puckered, bleached appearance of the hands this condition has been called "washerwoman's hand." This finding only indicates that the body has been in contact with water for some time (see Figs. 49 and 50). There is one sign in connection with the hands, however, which, when seen, is of the greatest importance, namely, the presence, in the firmly clenched hand, of objects such as weeds, sand, stones, or other debris, which may be related to the water from which the body has been taken. This conclusively indicates that the person was alive when in the water, since it constitutes a vital act. The character of sand, mud, or fine gravel under the finger-nails may be identified with the substance from the water-bed or from adjacent banks by some special characteristic. The grasping of articles in the hand or hands is a typical instance of cadaveric spasm (see p. 133).

Of the external signs found on a body taken from water, when drowning has occurred fairly recently, only the following are of substantial importance :—

The presence of a fine foam or froth at the mouth, or nostrils, or both ; and

The presence, in the hand, of objects which are to be found in, or beside, the water from which the body has been taken.

To determine whether death was due to drowning, reliance must be placed upon the internal appearances.

The cause of death being asphyxia, the principal evidence is likely to be found in the lungs and respiratory passages.

Before turning to the description of the lungs, a short explanation of the actual process of drowning should prove of assistance in the interpretation of their condition as found in drowning cases. When a person, who is unable to swim, falls into the water, his body immediately sinks, but it usually quickly rises to the surface again. Expelling the water which has been drawn in, he also expires air from the lungs. When his head again submerges he inspires a mixture of air and water. This initiates further coughing, air and water

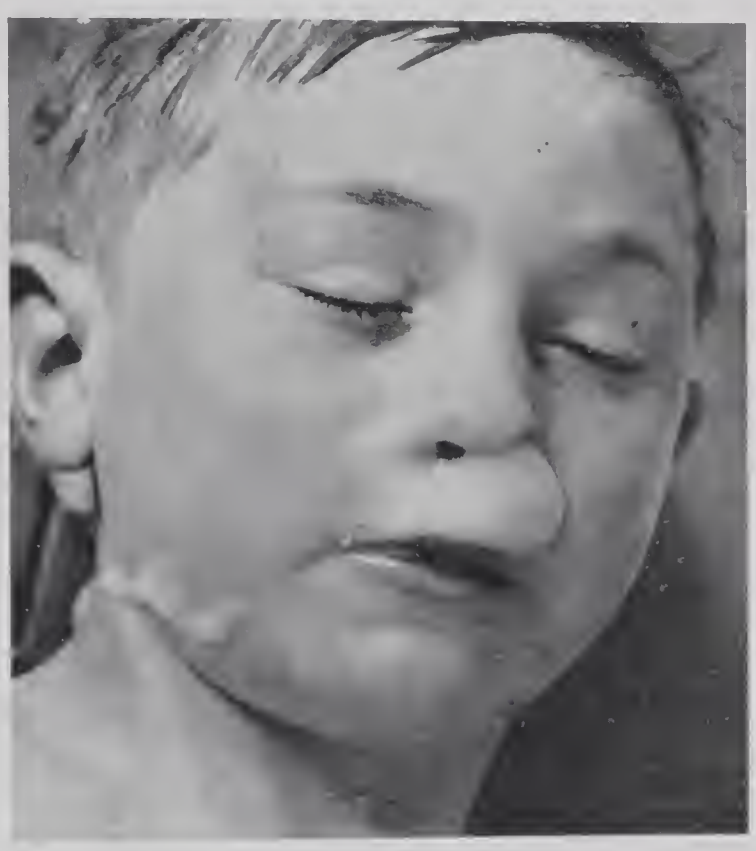


Fig. 51

Froth issuing from nostrils in a case of drowning. (By courtesy of Dr Robert Richards, Aberdeen University.)

are expelled, more water is inspired, and the lungs become progressively heavier, with the result that the tendency to sink becomes greater as this oft-repeated process of expiration and inspiration continues. Finally, the lungs become water-logged and the body sinks to the bottom. During the efforts of inspiration and expiration fluid is swallowed. Prior to the onset of putrefaction, a dead body will sink when placed in a sufficient quantity of water.

Internal appearances.

On opening the thoracic cavity the lungs bulge outwards as if formerly they had been retained under pressure. The term "ballooning" has been applied to this condition. It is the result of increased volume, due to the presence of fluid and air in the bronchi, the latter playing a part since the foam, formed in the process of drowning, acts as a valve which during respiratory effort

permits air entry to the lungs but obstructs air exit from them. Usually the lungs appear to be only moderately congested, and sometimes are very pale, due to air and water which has been trapped in the alveoli, forcing the blood from the lungs, and compressing the vessels in the interalveolar septa. As the result of the increased volume, the lungs often show impressions of the ribs upon their surfaces. On pressure, they feel rather doughy and readily pit. When the lungs are voluminous, red and grey patches may be seen on the surfaces, due to effused blood which has tracked along the interlobular septa to the surface from ruptured alveoli, and to patchy interstitial emphysema, respectively.

In cases of pulmonary fibrosis and when extensive pleural adhesions are present, the degree of ballooning is reduced. When fatal laryngeal spasm has occurred, very little water may enter the lungs, and when the spasm has been intermittent in character the amount may be greatly decreased.

Petechial hæmorrhages are not frequently seen on the surfaces of the lungs due to compression of the blood-vessels in the interalveolar septa by the fluid content of the lungs. On section, an œdematous condition, due to the presence of a copious, watery, frothy, blood-stained exudate will be observed. This fluid is readily expressed on pressure of the cut surfaces or may exude spontaneously from these surfaces. The exudation is usually copious due to the presence of a considerable quantity of water within the vesicles and bronchioles. On examination of the trachea, bronchi and bronchioles, a fine foam or froth will be found on the lining membrane. It may be blood-tinged. Frequently the bronchi are filled with this fluid, but more usually they are less completely full than the bronchioles, which contain a considerable quantity. The presence of this fine, clear, or, occasionally, blood-tinged foamy or frothy fluid unmistakably points to death by drowning. It is accounted for by water entering the air-passages and there becoming intimately mixed with air and mucus. This is essentially due to a vital act, which is proof of interference with the function of respiration, by a liquid, during life.

Particulate matter from the drowning medium may be found in the mouth, pharynx, larynx, trachea, or bronchi, and may permit of comparison.

When putrefaction has become established many of the signs described may be masked. Blood-stained fluid may be found in the pleural cavities either by permeation of the pleura or as the result of its perforation. The lungs are frequently collapsed and shrunken and the typical frothy fluid is absent from the respiratory tract. The general appearance of the heart is often very misleading, owing to the softened character of its musculature, the result of putrefactive change.

In cases of drowning, the chambers of the right side of the heart are usually dilated and contain a considerable quantity of fluid blood, and the associated vessels show congestion. In some cases, the heart is globular in shape, indicative of dilatation of all

the chambers, and is very much relaxed. The abdominal organs and venous system are congested.

During the process of drowning, a varying amount of fluid is usually swallowed, but it will depend upon the character of the contents of the stomach prior to submersion whether this fluid can be identified with that of the drowning medium. Quantities of fluid may have been drunk prior to the act of drowning, but when the stomach is found to contain a disagreeable liquid, which would not be swallowed voluntarily, and which corresponds to the drowning medium, it may be taken as an indication that this was swallowed during the act of drowning. When, for example, liquid manure, muddy water, or water containing debris is found, valuable aid is afforded to the diagnosis of drowning. Experiments have shown that fluid cannot enter the stomach after death, until putrefaction has advanced considerably. The presence of certain fluids in the stomach, therefore, is of importance. Revenstorf [1] is of opinion that the character of the drowning fluid can be shown by the nature of the dissolved and suspended matter which it contains, especially if it is of a distinctive character. The presence of plankton, by which is meant the lower forms of drifting vegetable and animal life in water, in the air-passages and stomach, corresponding to that in the medium of drowning, is significant.

Water entering the lungs passes to the left side of the heart and lowers the chloride content of the blood up to 50 per cent. of its ante-mortem level. Blood from a person who has been drowned in salt water shows an elevation of the chloride content up to 30 to 40 per cent. The chloride chemical test to be of maximum value should be made within a reasonably short period following death since the greater the interval the less reliable are the findings.

The following signs, found in a body taken from water, show that death has been caused by drowning :—

The presence of material held in the hand which could only have been obtained in the water.

The presence in the air-passages of clear, or blood-tinged, foam or froth.

The increased volume, and œdematous condition, of the lungs.

The presence in the stomach of water, or liquid, corresponding to that of the drowning medium, or of substances found in it.

We have had the opportunity to examine twenty-three cases of drowning which occurred at the same time, through the overturning of a large sailing boat. The examinations were made some eight hours after death and the subjects were young adult males. In all, foam or froth was present at the mouth or nostrils or both, and its consistence varied in character from a fluid, slimy, slightly aerated liquid to a fairly dense fine bubbled foam.

Was drowning due to accident, suicide, or homicide?

In many cases an examination of the body will not throw much light upon the answer to this question. There are, however, certain

general indications, attention to which may assist in arriving at an answer.

Among the causes of accidental drowning, may be cited the following :—

Children falling into water.
Persons drowned by the upsetting of a boat.
Persons wandering in a fog and falling into a river, or canal.
Persons drowned while bathing.
Intoxicated persons or epileptics falling into water.

The following case of the accidental drowning of a woman in a rain-barrel is exceptional. She was preparing for a washing, and had evidently been dipping a bucket into the water of the barrel when she lost her balance, fell in, and was drowned.

FIG. 52

A body showing extensive injuries caused by the propeller of a ship.

Another most unusual case reported to the writer was that of a schoolboy who entered the swimming pond of a bathing establishment, during a competition, for the purpose of retrieving diving plates. These were about 6 inches in diameter with a round hole in the centre. He swam along the bottom of the pond picking up as many plates as possible. One of these plates lay on the top of a grating covering a water outlet at the deep end of the pond. In attempting to retrieve the plate he put his finger not only through the centre hole of the plate but accidentally between two bars of the grating. He was unable to extract his finger, and although other swimmers went down to him they were unable to extricate his finger or to raise the grating. At the same time an attempt was made to empty the pond but the boy was dead before this was accomplished.

Several cases of death have resulted from embedding of the head in mud through diving into water.

A woman was tried on a charge that, while in a state of intoxication, she failed to exercise reasonable care of her infant daughter of nine months, who, in consequence, fell out of bed into a pail of water and was drowned. The jury, however, found the accused not guilty.

The question of suicide arises when stones or other heavy articles are found in the pockets, or attached to the person, and in cases of persons who are known to have been depressed or of unsound mind.

An inmate of an asylum committed suicide by drowning himself in a basin of water. When the discovery was made, the upper part of the chest was resting against the front of the basin, the head was sunk just under the level of the brim of the basin, the legs were bent under the body, but the knees did not rest on the floor.

The question of homicide is raised under such circumstances as the following :—

In cases where, upon the banks of the water in which the body has been found, there are evidences of a struggle, and especially when articles belonging to other than the deceased person are found on the banks associated with those known to have belonged to the deceased ; or where, grasped in the hand of the drowned person, there are fragments of clothing or hair, not corresponding to his own.

In cases where, on post-mortem examination, the usual internal signs of death by drowning are absent, and lesions due to violence are found on the body. It is necessary, however, to remember that suicides who have determined to end their lives may have attempted this by one method, but having failed to achieve their purpose, have completed the act by drowning. For example, in one of our cases an elderly man tried to commit suicide by cutting his throat with a razor. From the back room in the house where the razor had been used blood-stains were traced to a swing-bridge over a stream, a distance of about 200 yards, and in a shallow part of the water the body was found.

Although homicidal drowning by pushing the victim into the water may be comparatively easy, it is very uncommon, but occurs occasionally in the case of children or of helpless persons.

In those cases in which marks of violence are found upon a body taken from water, it is necessary for the medical examiner to devote careful attention to detail, since he will probably be asked to express an opinion as to whether or not the lesions were produced during life, and, if so produced, whether they alone account for the death. The differentiation between ante-mortem and post-mortem wounds is discussed under the heading of wounds and their characters (see pp. 248 and 281). The question as to whether wounds upon a body are of suicidal, accidental, or homicidal origin may sometimes be determined by local circumstances referable to the water itself. In navigable rivers or canals, bodies may sustain injuries, not only extensive in character but peculiar in kind, from the propellers of passing ships (see Fig. 52).

Such injuries may cause almost complete evisceration, and fractures with complete or partial removal of a limb or limbs are of frequent occurrence.

In contrast to such cases there are those in which, in consequence of the nature of the injuries found, one can only come to the conclusion that death was due to violence by assault.

When evidence of injury is present upon a body recovered from

the water, the injuries must be compared diligently with objects both in the water or on the banks near it, and special attention must be directed to the question, whether the injuries were sustained before or after death.

When decomposition of a body is far advanced, it will probably be impossible to give any opinion as to whether the lesions were produced before or after death. In every case, when injuries are present, the whole circumstances must be fully considered and weighed before an opinion is given that they are homicidal in origin. The examiner must satisfy himself that the lesion could not have been produced in any other way or accounted for in the light of other evidence.

Occasionally persons are found dead in baths, the result of

FIG. 53

Putrefactive changes affecting a body recovered from water.

natural causes, most usually syncope or cerebral hæmorrhage. Similarly from like causes, accidents may occur in the course of bathing in the sea or river. The possible sudden onset of incapacitating muscular cramp is a predisposing cause of accidental drowning.

The flotation of the body of a person who has died from drowning depends upon the process of putrefaction. When a body sinks, it again comes to the water surface and floats after a variable period. Putrefactive gases play a very important part in flotation, which will therefore be hastened in summer and delayed in winter. If the water contains putrescent material, putrefaction will be hastened and so will flotation. Putrefaction is slower in onset when the body lies in a running stream than when in still water. In winter, a body will probably come to the surface of the water in about a fortnight's time, and this period will be diminished by about a week in summer. Many circumstances may modify this, for example, heavily clothed bodies and lean bodies will take longer to reach the water surface than lightly clothed or fat bodies.

Treatment following submersion.

Treatment of those in whom animation is suspended by drowning may be summarised as follows :—

There should be no delay in commencing treatment. The airway should be cleared so far as possible, care should be taken to ensure that the nostrils and mouth are cleansed, and tight clothing should be loosened.

The person should be placed face downwards to allow the escape of water, and artificial respiration should be initiated immediately, using Schafer's method. This should be maintained for three to four hours. The Drinker combined method of artificial respiration is regarded by some as preferable to Schafer's method since experiment has shown that it affords an increase of 25 per cent. in pulmonary ventilation, and this may just be sufficient to tip the balance in critical cases. Drinker's method combines prone pressure and the arm lift. Eve's method is strongly recommended by many, and consists in using the weight of the abdominal viscera to move the diaphragm by rocking the casualty on a stretcher through a total angle of 60° at the rate of twelve double rocks per minute; 90° rocking at first assists lung drainage. This method should be continued for a considerable period should resuscitation be slow in onset. Tindal [2] has described a convenient method of applying artificial respiration in cases of drowning, especially in the younger subject. The child is picked up and a leg is placed over each shoulder of the operator, with the trunk hanging down and facing him. The chest is then pressed against the operator's body. This method is simple to perform, requires little effort, is effective in emptying fluids, in clearing the airway, in expanding the ribs, in filling and emptying the right heart and increasing the pressure of blood in the brain. The victim can at the same time be walked to nearby shelter and warmth. It can be performed in the sitting position as in a boat.

While artificial respiration is in progress, assistants should remove the wet clothing and apply warmed blankets and covered hot bags. It is of great importance to restore and maintain the heat of the body.

The action of the heart and respiration should be stimulated by appropriate medication.

The patient should be carefully observed for indications of possible complication or relapse.

SUFFOCATION

The term suffocation embraces the causes by which death is produced by impediment to respiration, not due to pressure externally and immediately applied to the windpipe. It does not, therefore, include deaths by hanging, strangulation, or throttling, but comprises deaths by smothering or overlaying, those due to foreign bodies in the larynx, trachea, and bronchi, and those due to irrespirable gases. Suffocation may result from natural disease, accident, or violence suicidally or homicidally applied.

Death by suffocation may be caused :—

By prevention of the action of the muscles of respiration.
By obstruction to the entrance of air through the mouth and nostrils.
By obstruction of the larynx, trachea, or bronchi.
By inhalation of irrespirable gases.

It may arise from :—

Tumours in the throat or larynx.
Bursting of a pharyngeal or tonsillar abscess.
Exudations affecting the mucous membrane of the air-passages, including acute œdema of the glottis.
Accumulation of bronchial secretion, especially in bronchitis of young children or elderly persons.
Rapidly accumulating pleural effusions, especially if bilateral.
Pulmonary œdema.
Severe hæmorrhage in phthisical cases.
Bursting of aneurysms into air-passages or lungs.
Convulsions, especially in young children.

It may likewise result from such accidents as the following :—

Pressure on the chest with fixation of the thoracic walls, as in some cases of overlaying, in falls of material in mines, quarries, collieries and buildings, pit-cage accidents, and lift accidents.
Impaction of bodies in the larynx, trachea, or bronchi. This is by no means uncommon in intoxicated persons. In choking, the impacted material may consist of imperfectly masticated food, bones, partial dentures, and a variety of other things. Objects, such as rubber balloons, may be inhaled by children during play. Regurgitation of fluids into the air-passages may occur in a variety of cases and produce fatal asphyxia. In a case of an infant, aged three months, about a drachm of mucus-like material, contaminated with a white curdy substance, was found in the larynx, and the trachea contained frothy fluid which extended into the large bronchi. Stomach contents were also found in the œsophagus. The stomach contained $3\frac{1}{2}$ ounces of white curdy fluid. In adults, regurgitation may occur during a state of unconsciousness and, not infrequently, in cases of intoxication. In these cases, regurgitated stomach contents, readily recognisable as such, may be found in the pharynx, larynx, trachea, bronchi, and in the bronchioles. Most frequently there is also evidence of regurgitated material around the mouth and nostrils. In one case, a woman, aged thirty-one and addicted to drink, was put to bed in a drunken condition and found dead in the morning. There was no evidence of vomited material on the bedding. Post-mortem examination showed external signs of asphyxia, and the pharynx, larynx, trachea, and bronchi contained a quantity of vinous smelling frothy fluid. All the organs were deeply congested. The

Fig. 54

A unique case of suffocation of two small boys in the driver's box of a disused delivery van. The box, or locker, measuring 29½ inches long, 13 inches wide, and 20 inches deep, was beneath the driving seat. It appears that the boys climbed into it in play, and that the lid came down and held firm. Experiments with the fastening on the locker showed that when the lid was dropped sharply the hasp failed to connect, but it slipped easily over the staple when the lid was let down lightly. (By courtesy of Edward G. B. Fowler, Esquire, H.M. Coroner for the City of Leicester.)

Fig. 55

The driver's box, or locker, on the disused delivery van, showing fastening (see Fig. 54). (By courtesy of Edward G. B. Fowler, Esquire, H.M. Coroner for the City of Leicester.)

urinary bladder was much distended. Inhalation of fluid, or food particles, especially against the epiglottis, may be responsible for fatal laryngeal spasm.

Overlaying of infants.

Pressure of the mouth and face of an unconscious person against a yielding surface, such as a cushion, mattress, or thick rug.

Inhalation of irrespirable gases, such as sulphuretted hydrogen, sulphur dioxide, chlorine, carbon monoxide, smoke in burning buildings, in coal-pits, and other confined spaces; the vapours of iodine, bromine, and ammonia; and the acrid fumes of mineral acids.

We have seen a large number of cases in which death has resulted from burial by falls of sand and by the collapse of buildings. In the former class of case, post-mortem examination very frequently showed inspiration of sand which, in many of the cases, was found in the trachea. In some instances sand particles were found in the large bronchi.

Suffocation may also be caused homicidally in many ways, including the following :—

When pressure is applied over the mouth and nostrils with fixation of the lower jaw, at the same time as fixation of the chest walls by the weight of the body of the assailant.

By placing foreign bodies, such as paper, dough, or other plastic substance into the mouth and larynx of newly-born infants.

By covering the face with a pillow, or applying the hand over the mouth and occluding the nostrils with the fingers.

Post-mortem appearances.

Since the proximate cause of death in suffocation is asphyxia, the reader is referred to a former chapter for the signs of death from this cause (see p. 147). There are, however, certain points to which special attention should be directed, and it must be emphasised that there is no uniformity in the external appearances. Although frequently, in asphyxia, the face is more or less livid in colour and congested in appearance, the conjunctivæ are injected, and the lips and ears are of dark colour, it should be clearly appreciated that these conditions are found only in some cases of death by suffocation. In many cases, the face is pale or only slightly dusky in colour, and there may be an absence of congestion either of the eyes or of the lips. As a rule, however, lividity is well marked in the lips, ears, and finger-nails. In children who have died from asphyxial causes, the livid tint of the nails of the fingers and toes is almost constant.

In certain cases of asphyxia, not only are the conjunctivæ congested, but small petechial hæmorrhages are present. The variation in the external manifestations is due in large measure to the rapidity or slowness with which the asphyxial process is initiated and completed.

From our observations, we are of the opinion that the more rapid

and complete the asphyxiating process, the more marked are the external appearances of lividity. From the external appearances only it may be difficult to say whether death has been caused by asphyxia, except when lividity is well marked.

In addition to the principal signs of death by asphyxia already discussed in a former chapter, some light may be thrown upon the cause of death by noting particularly the following points :—

The colour and consistency of the blood. In deaths by asphyxia the blood tends to be unusually fluid, and is darker in colour. In deaths caused by inhalation of carbon monoxide, however, due to the formation of a stable compound between the gas and the hæmoglobin, carboxyhæmoglobin, the colour of the blood is cherry-

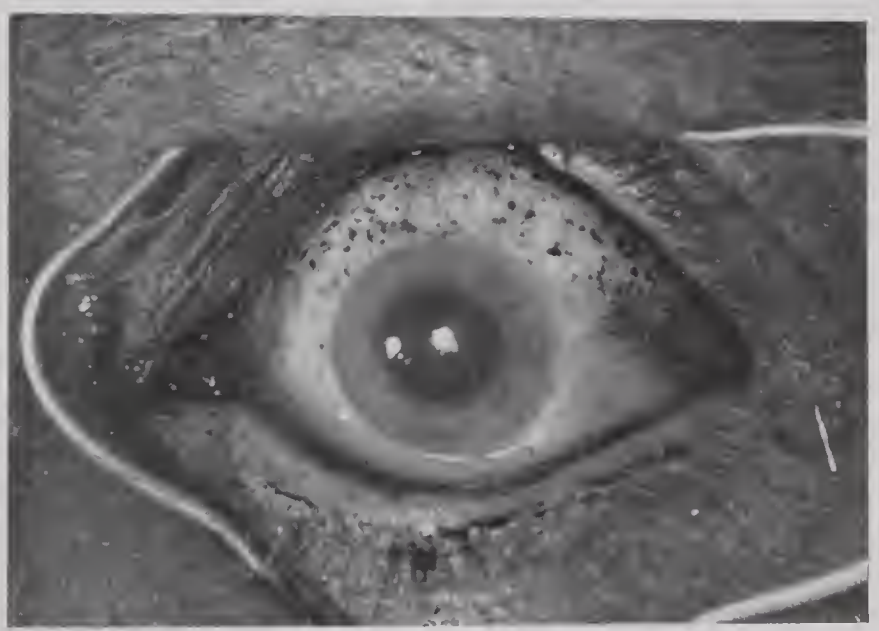


Fig. 56

Subconjunctival petechial hæmorrhages.
(By courtesy of Dr Robert Richards, Aberdeen University.)

red. This is noted not only in the principal blood-vessels, but in all the organs of the body (see Figs. 225 and 226).

In most cases of suffocation in children, and in certain of those in adults, small petechial hæmorrhages may be seen on the serous surfaces of the heart and lungs (see p. 148).

Suicidal suffocation by the insertion of foreign bodies into the back of the throat has occurred in a number of cases, and portions of clothing and other materials have been used for this purpose.

ILLUSTRATIVE CASES

Dr J. Mill Renton [3] has recorded the following case. A man was brought to a workhouse with a history of delusions. In the evening, he suddenly became violent and was placed in a padded room. He quietened down and went to sleep. Twenty minutes later it was observed that his bedclothing had been thrown off, and on closer examination he was found to be dead. Inside the mouth was a

piece of flannel, about 1 foot long by 1 inch broad, and behind it were two strips similar in length. The last of these was so firmly packed over the epiglottis that it was withdrawn with difficulty. He had obtained the cloth by tearing his blanket.

Other instances of placing foreign bodies in the throat show homicidal motive, and include such acts as forcing mud or dough into the throats of newly-born children, or a handkerchief in the case of adults. In one of our cases, the murder of a young woman, a tightly compressed pocket-handkerchief was found impacted in the pharynx. There were also some wounds on the head and face, but death had resulted from asphyxia due to suffocation. The wounds were not of a character to cause death, but were sufficient to have stunned the deceased.

An unusual case of suffocation in a young child was caused through an uncooked haricot bean lodging in the orifice of the right bronchus. The finger and toe nails were dusky. The face was cyanosed. The lungs were pale and sub-pleural petechial hæmorrhages were present on the posterior surfaces of the basal lobes. Death occurred in an ambulance during conveyance to hospital.

Early in 1943, 173 persons died from asphyxia due to suffocation by compression in a crowd, but the cause of death was complex in character. Briefly, the accident resulted from someone falling on a stair leading to a London tube air-raid shelter, with the resultant piling up of persons from behind and above. The victims were pressed together, with restriction of chest movement. It was found that some of the persons on the top had died and some at the bottom were brought out alive. Almost the last person removed from the bottom was a girl of seven who walked without assistance to the first-aid post. There was not a case in which the ribs were broken, and there were only a few cases in which bones were fractured.

Suffocation by the inhalation of coal-gas is the commonest form of suicide. This may be effected by turning on the gas in a room, putting the head in a gas oven, or by placing a gas tube in the mouth. Accidental death also may result readily from the inhalation of coal-gas under a great variety of circumstances (see p. 581).

TRAUMATIC ASPHYXIA

There is a form of asphyxia to which the term "traumatic" has been applied. It is brought about by compression of the chest and abdomen as the result of the victim being crushed between heavy bodies, for example, by a pit-cage, by a fall of coal, or by being crushed in a crowd. When the pressure is great traumatic asphyxia sometimes occurs, the leading features of which are a more or less deep red or violet discoloration of the face, neck, and upper parts of the body and a very injected condition of the conjunctive, with perhaps a varying degree of extravasation of blood. There is usually a well-defined demarcating line between the discoloured upper portion of the body and the lower normally coloured part of the body.

In some cases, the line of demarcation of the lividity may be as high as the clavicles or neck. The skin may show extensive petechiæ. The coloration gradually fades away without the usual gradation of colour exhibited in definitely marked ecchymosis. Proptosis of the eyes may be present, and also fundal hæmorrhages. Gordon and Thomson [4] have described five cases and are of the opinion that the causation of the skin discoloration is due to sudden over-distension of the veins with consequent paralysis of the walls. They are of the further opinion that the occurrences of hæmorrhages into the optic nerve or sheath and damage to the retina by venous back pressure are responsible for the ocular complications. The amount of lividity differs in various cases owing to the period of time, short of producing death by suffocation, in which the mechanical chest pressure is in operation.

Some time ago we examined the body of a young woman whose death had been caused by the effects of traumatic asphyxia and shock due to multiple injuries. The victim, who was seated on the back of a four-wheeled, horse-drawn van, was thrown on to the roadway following a collision between an electric tramcar and the van. The front of the tramcar carried the girl forward for approximately its own length before coming to a standstill. She was pinned beneath the front of the tramcar, her head being clear. The face, ears, and neck were of livid colour and showed multiple petechial hæmorrhages. There were bilateral conjunctival hæmorrhages and the conjunctivæ were œdematous.

HANGING

Hanging may be defined as that form of death which is caused by suspension of the body by a ligature which encircles the neck, the constricting force being the weight of the body. A frequent method is the use of a running noose. The proximate cause of death is asphyxia or comato-asphyxia.

The time occupied in the process of death depends chiefly upon two factors, the severity of the constricting force and the point of application of that force.

The amount of the constricting force depends upon whether or not the body is completely suspended. Complete suspension is not essential since the partial weight of the body is adequate for the purpose. Persons have succeeded in hanging themselves although their feet were in contact with the floor. Only a slight degree of constriction of the neck is required to cause eventual death. When, however, the entire body is suspended, greater weight is thrown upon the ligature and a greater constricting force results. When such a degree of force is applied to the neck, death supervenes more rapidly than in the case of partial suspension with a diminished degree of constrictive force. When the ligature is not tight, death may ensue slowly, chiefly from the effects of coma induced by disturbance of the cerebral circulation as the result of

pressure on the vessels in the neck. The point of application of the force is an important factor not only in the time occupied in dying, but in the subsequent post-mortem appearances.

Experiments have proved that the degree and rapidity of the onset of asphyxial symptoms are greatly influenced by the situation of the ligature on the neck. When the ligature is placed between the lower jaw and the hyoid bone and is moderately tightened, the effect is to disturb breathing, which, however, continues, but at the end of two minutes the subject has to be disengaged from the noose. When the ligature is placed over the larynx, the experiment has to cease at the end of one and a half minutes. When placed over the cricoid cartilage, the experiment cannot be continued for even a few seconds because of embarrassment to respiration. Gosse has shown by personal experiment that unconsciousness may be produced without undue pressure on the air-passages. This is due to obstruction of the cerebral circulation.

The effect of a ligature constricting the neck is primarily the partial or complete occlusion of the air-passages, which obstructs air entry to the lungs. As the result of this constriction, the cerebral circulation is also impeded. The amount of compression exercised on the phrenic and vagus nerves is yet another factor in the causation of death. Apart from implication of respiration a person may die from the effects of suspension. This is well illustrated in the case of a man upon whom tracheotomy had been performed for malignant disease of the throat, and who suspended himself by the neck, the line of ligature being above the site of the cannula. At post-mortem examination asphyxia was absent, but the blood-vessels at the base of the brain, and in the substance of the pons and medulla, were found engorged. In the majority of cases of suicidal suspension the ligature is placed either between the thyroid cartilage and the hyoid bone, or between the hyoid bone and the lower jaw. The constricting force of the ligature causes the tongue, soft palate, and larynx to be pushed upwards and backwards, thus cutting off the passage of air.

Pressure on the vagus nerves may cause cardiac inhibition and sudden death before asphyxial manifestations are established. This possibility should not be overlooked at the post-mortem examination of bodies of persons found hanged.

As in other forms of death from asphyxia, the heart continues to beat for a few minutes after the cessation of respiration, and, therefore, artificial respiration on a suspended person may prove successful if the body is cut down within five or six minutes after the commencement of suspension. After this period, there is little chance of success in the re-establishment of breathing.

In the examination of a body found suspended by the neck, there are three lines of inquiry which demand special consideration :—

External post-mortem appearances.
Local conditions of the neck.
Internal post-mortem appearances.

External post-mortem appearances.

Appearance of the face.

The pupils are usually dilated. The position of the tongue varies from normality to protrusion slightly beyond the line of the teeth. Tongue injury is unusual. The face is usually pale, but may be dusky in hue. The lips are bluish in colour, and the tongue and mucous membrane of the mouth may share in this coloration. Saliva may be seen trickling from a corner of the mouth as the result of increased salivation prior to death, due to stimulation of the salivary glands produced by the ligature. Petechial hæmorrhages may be found under the conjunctivæ and, more rarely, on the eyelids and forehead. The conjunctivæ are frequently congested.

Condition of the genital organs.

Turgescence of the penis, in varying degree, with or without evidence of seminal or prostatic fluid may occasionally be seen, but when present, has no specific bearing on death by hanging. Fluid at the tip of the penis is sometimes present in deaths due to a variety of causes. We have seen it in the case of a man fatally stabbed in the chest, and in several other cases. In some of these, the fluid contained spermatozoa. Only in two instances was marked turgescence of the penis observed. It is most probable that engorgement of the penis may be the result of hypostasis, and that seminal or prostatic fluid may be found by reason of gravitation, or possibly of rigor mortis.

Condition of the rectum and bladder.

The voidance of urine or fæces is by no means infrequently found, and is merely indicative of the loss of power of the sphincters which is common in all deaths by asphyxia as well as in other forms of death.

Local conditions of the neck.

The incidence and character of the marks on the neck will probably indicate the mode of application of the ligature. All marks on the neck should be critically examined to determine whether death is attributable to suspension, or to throttling followed by suspension to cloak the commission of crime. Should scratches be present, they must be carefully considered in relation to whether or not they could have been produced by the ligature. Scratches on the neck or face in the presence of a soft pliable ligature should evoke suspicion, although it must not be forgotten that the dying person, in vain attempt to loosen the ligature, may have produced scratches with his finger-nails.

The line of the ligature must be carefully examined. In suicidal suspension, it usually follows the line of the lower jaw, then passes obliquely upwards behind the ears, where it is commonly

lost. The situation of the mark will, however, be largely influenced by the method used in the application of the ligature to the neck, for example, whether in the form of a loop or a running noose. In the former method, the mark will be most prominent on the part of the neck to which the head has inclined, and less marked over the region of the open angle of the loop. When a running noose has been employed, the mark more or less encircles the surface of the neck, and is most prominent at the point underlying the knot. The reason for the ligature line being close to the lower jaw in the bulk of cases, in which a running noose is used, is that as the body sinks in the act of suspension the ligature gradually slips up the neck until it is fixed by the line of the lower jaw. The relation of the body to the point of suspension and to the ground must also be taken into account, since a variety of angles will be produced and may affect the incidence of the point of constriction. In some cases, the mark may pass over the thyroid cartilage and in others the hyoid bone, but in the bulk of cases it is situated between the hyoid bone and the line of the lower jaw.

A suicide may pass the ligature twice round the neck before he suspends himself, and a double mark, one more or less circular, and the other, oblique, may be seen. A composite ligature, consisting of a series of strands of rope or cord, may be used. A suicide is not particular as to the kind of ligature which he employs, and will probably use what is at hand for the purpose. Ropes, handkerchiefs, braces, leather straps, belts, bed-sheets, and scarves, among others, are frequently employed. All of these factors will produce modifications in the character of the mark or marks found upon the neck. Occasionally, unusual features are found. In the case of a man who hanged himself by a chain, the contour of the individual links was discernible on the skin.

Another case was that of a man who had suspended himself from the knob of a window-shutter about 4 feet above the ground. On the left side of the neck there was a single ligature mark, but on the right side there were three separate furrows, with small intervening ridges of skin of livid colour. The explanation was that the man had used, as a ligature, a series of strands of ordinary hamstring. In cases of suspected infanticide when a mark is found on the neck of the infant, the possibility that it may have been due to constriction by the umbilical cord during birth must not be overlooked. Since, however, the umbilical cord is a soft structure and is unlikely to produce other than limited constriction when situated around the neck, the mark is not usually deep, and does not show the typical appearance of the ordinary ligature mark.

There may be indications in the vicinity of a ligature mark, such as scratches or small bruises, which are suggestive of the nature of the ligature used, when the ligature itself is not available for inspection. These may have been produced by a ligature of coarse texture, or alternatively, by the finger-nails of the victim, or those of an assailant in throttling, prior to suspension of the body to simulate suicide by hanging. In doubtful cases, to eliminate the

possibility of the body having been suspended after death, a piece of tissue from the ligature mark should be excised so that histological examination may determine the presence or absence of vital reaction (see p. 248). When available, the ligature should always be retained for detailed examination.

The colour of a ligature mark may show wide variation and may range from a parchment shade to a purplish hue. Frequently a

Fig. 57

Suicidal hanging. Note ligature mark.

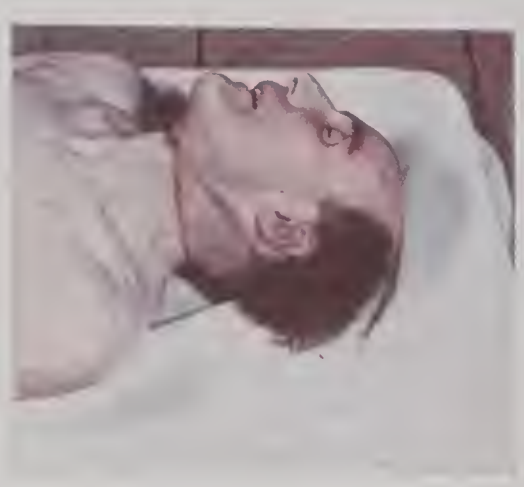


Fig. 58

Suicidal hanging. Note ligature mark.

Fig. 59

Suicidal hanging with a web belt. Note ligature mark.

Fig. 60

Suicidal hanging with a web belt. Note ligature mark.

bluish-red colour may be evident, but sometimes the site of the ligature may be almost devoid of colour. Quite frequently, at first a pale colour may be present, but this subsequently assumes a brownish, dried appearance. The line of application of the ligature is usually depressed, and the degree of depth depends upon the nature of the ligature and the amount of pressure on the tissues.

When bruising is found associated with the mark, its presence indicates that the violence responsible was operative either during life, or was applied immediately after death while there was continuance of molecular life of the tissues. When suspension has occurred

during life, there is usually some degree of bruising of the tissues associated with the site of constriction, but this may be very slight in character.

Internal post-mortem appearances.

The appearances are those of asphyxia. It is unusual to find signs of asphyxia alone, and the surface vessels of the brain, the brain and cranial sinuses show marked congestion. As in death by suffocation, sub-pleural punctiform hæmorrhages may be found, in addition to the signs already described under asphyxia (see p. 148).

A detailed dissection of the tissues of the neck should be made to detect injury of the deeper structures. Sometimes fibres of the platysma may be torn, the sternomastoid muscles injured, the thyro-hyoid ligament damaged, or the processes of the thyroid cartilage fractured.

Petechial hæmorrhages may be found on the epiglottis, in the larynx, and trachea. The trachea will usually show a varying degree of congestion in which the epiglottis is frequently involved.

Accident, suicide or homicide.

When it has been established that death has resulted from hanging, the next question is, whether it was suicidal, accidental, or homicidal ?

It is a statistical fact that death by hanging is a common form of suicide and there is, therefore, a strong presumption in favour of suicide.

In many undoubted cases of suicide some portion of the body is found in contact with the ground. The amount of the body so resting may vary in extent from the major portion of the trunk to merely the tips of the toes. In one of our cases the point at which the rope was attached was only 3 feet from the ground, and the lower part of the body from the hips downwards was resting on the floor. In another, the legs from the knees downwards, were resting.

At the same time, it must not be forgotten that a suicide may give himself a drop. In one case the suicide tied a piece of rope to the banister railing of a stairway, placed his neck within the noose, then jumped into the well of the stairway. The drop was 10 feet, and examination showed that the neck had been dislocated.

The question becomes more complicated, however, when wounds produced by blunt or sharp weapons are found upon the body of the suspended person. Such are sometimes found in suicidal cases. They are well illustrated in the case of a man who tried to suspend himself, but the ligature broke. He next butted his head against the walls and wounded himself. He tried again to hang himself from the ceiling with a bed-sheet fixed to a strap round his neck. This attempt was successful. A suicide may first attempt to cut his throat and then hang himself. Sometimes the attendant circumstances, apart from mere suspension, indicate suicidal action prompted by insanity. As an instance, a man hanged himself from the hook of

a spring-balance weighing machine. Before doing so he dressed himself in female underclothing, and adjusted a mirror so that he might watch the procedure.

Accidental hanging sometimes occurs. A case is recorded of a boy who went into a cowshed in which was a swing attached to a beam. He climbed up to this beam with the intention of sliding down one of the ropes of the swing, but in his descent a woollen scarf around his neck caught on a long nail fastened in the beam, and he became suspended. He was found in this position, and cut down. His face was very cyanosed and he was unconscious, but was resuscitated.

Homicidal hanging is rare. Except in cases of weakness from senility or other enfeebling cause, or under very unusual circumstances, the act cannot readily be perpetrated by one assailant. Usually more than one are involved in its commission. When resistance has been offered, marks of violence are likely to be found.

Judicial hanging.

The method of execution in Great Britain is judicial hanging. Its object is to produce rapid, if not instantaneous, death, either by dislocation or fracture of the upper cervical vertebræ. This is effected by the adoption of two measures, the arrangement of the rope and the drop or fall in which the body at the end of its descent receives a violent jerk. The drop is regulated according to the weight of the person.

The usual lesions which result from judicial hanging are: Dislocation or fracture of the upper cervical vertebræ; partial or complete severance of the spinal cord, close to its junction with the brain; rupture of the cervical muscles; dislocation or fracture of the laryngeal cartilages.

From the lesions produced by judicial hanging it will readily be appreciated that it differs very greatly from ordinary hanging, and that death is caused by fracture-dislocation of the vertebral column. The heart usually continues to beat for a period after the drop. In some cases there may be asphyxial manifestations.

STRANGULATION

Strangulation may be defined as that form of death which is caused by a constricting force applied around the neck by means of a ligature without suspension of the body. Throttling, or manual strangulation, is that form of death which is caused by compression of the throat, the constricting force being the fingers and the hand or hands of the assailant. Ordinary strangulation may be caused accidentally, suicidally, or homicidally. The constricting mark is usually found at a lower level on the neck than in hanging, but may be found at any level, and frequently direct pressure is exerted upon the larynx. The mark more or less completely encircles the neck transversely. In some cases it may be continuous, but in others it may be invisible at some part of the neck. The nature of

the ligature employed plays a prominent part in this respect. The degree and character of injury to the deeper tissues of the neck are dependent on the amount of violence used in the application of the ligature. The underlying muscles frequently show some degree of extravasation, due to rupture of the capillary vessels. The laryngeal cartilages and trachea are usually intact, but in some cases, especially homicidal cases, fractures of these structures are present. Fracture of the hyoid bone is unusual. The injuries in homicidal strangulation are usually more extensive than in accidental or suicidal cases, due to the fact that an assailant frequently

FIG. 61

Suicidal strangulation. The ligature used was a piece of fabric torn from the man's clothing and folded several times. It was retained in position by means of a half-knot.

exerts a greater degree of force than is necessary to cause the death of his victim. It is in such cases that extensive deep-seated injury is likely to be found. When a ligature is suddenly placed around the neck of a person and pulled tightly by another, the assaulted person is rendered unconscious very quickly and is unable to offer appreciable resistance.

Homicidal strangulation is by no means uncommon and is occasionally associated with violation of a female victim. Suicidal strangulation is not infrequent and is usually effected by tying the ligature around the neck. When only a half-knot is employed, the nature of the material composing the ligature will be the determining factor as to whether the ligature will maintain constriction of the neck or loosen after consciousness of the individual is lost. In numerous cases, the ligature is fastened with a double knot. Sometimes a ligature is passed round the neck several times. In differentiating between suicidal and homicidal strangulation, significance

cannot be placed either upon the nature of the knot or its position on the neck. Recent experiments in this connection show some interesting facts: 50 female and 50 male subjects were asked to place a ligature around their own necks and to secure it with a double knot. Next they were requested to carry out the same procedure on the neck of another. When the ligature was suicidally applied by the 50 females, 36 tied a granny knot and 14 a reef knot, 25 placed the knot on the right of the middle line of the neck, 16 on the left, and 9 in the middle line. Of the 50 male subjects, 37 tied a granny knot and 13 a reef knot, 28 placed the knot on the right of the middle line, 15 to the left, and 7 in the middle line. When homicidally applied, 35 females tied a granny knot and 15 a reef knot, 28 placed the knot on the right of the middle line, 14 on the left, and 8 in the middle line. Of the 50 males, 37 tied a granny, knot, and 13 a reef knot, 27 placed the knot on the right of the middle line, 16 on the left, and 7 in the middle line. Only 8 persons tied a reef knot in both tests. One ambidextrous person tied a reef knot on each occasion, and another ambidextrous person did so on one occasion. Strangulation may occur under the most unexpected circumstances. In one case, a woman laid herself on a bed, attached the running noose of a rope round her neck, and tied the other end of the rope to a heavy piece of metal which she threw over the iron bar of the bed-frame. Accidental strangulation occasionally occurs and may happen in a variety of ways, for example, a boy climbed an apple tree and slipped between the branches, his jacket, which was buttoned at the neck, turned up round his neck and strangled him. Recently a boy was strangled by his scarf while playing with a dog.

Strangulation being an asphyxial death, the external and internal signs of asphyxia will be apparent, and what has already been described under this subject applies (see p. 146). The extent and character of the signs will depend in large measure upon the rate of the asphyxial process. When the constricting force has been considerable they will be well marked. It must not be forgotten, however, that in certain cases, signs of asphyxia may be very slight if death has supervened quickly from cardiac inhibition, due to pressure on the carotid nerve plexus.

In cases of manual strangulation or throttling, the situation and extent of the bruised areas on the neck will depend upon the relative positions of the assailant and victim, the manner of grasping the neck, and the degree of pressure exercised upon the throat. The marks are usually found over the lateral aspects of the larynx or trachea, and most frequently correspond to a thumb mark on one side, and a larger mark, or series of marks, on the other, due to compression by the fingers. When the hands of the assailant operate from behind the victim, the compression will be applied practically all round the neck, but certain areas of bruising will be more prominent than others due to the pressure of the finger-tips. As a rule there is extensive bruising of the tissues, which is frequently accompanied by fracture of the laryngeal structures, and, commonly,

the hyoid bone. Further, there may be bruising or laceration of the tongue as the result of it having been pressed against, or caught between, the teeth. In some cases, there may be practically no evidence of bruising on the surface of the neck, although fairly

FIG. 62

Strangulation caused by a scarf held in position by a complete knot.

FIG. 63

The same case as in Fig. 62.

extensive bruising of the deeper tissues may be found, together with fracture of the hyoid bone. The bruising of the deeper tissues is usually discrete in character and frequently the muscular tissues are involved. The extent will naturally depend upon the degree of pressure used. Fracture of the hyoid bone is a common occur-

rence, and when present shows that considerable violence has been applied to the neck. It negatives suicide, and since two

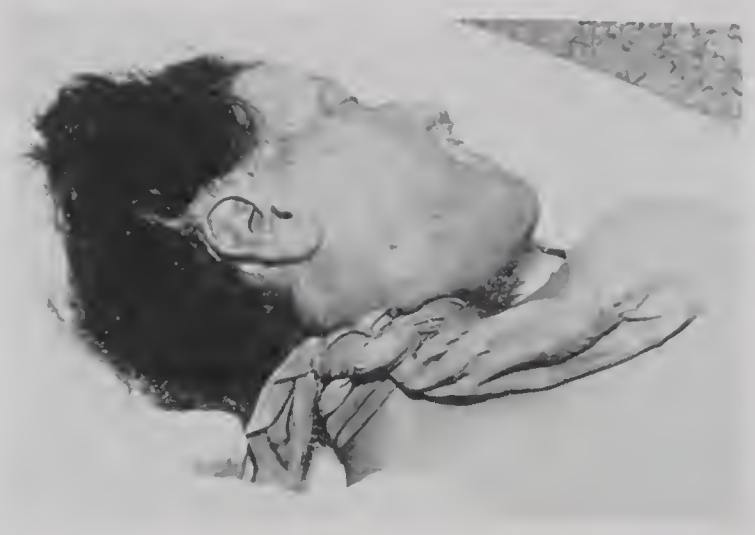


FIG. 64

Case of homicidal strangulation. Note small quantity of foam at nostrils and corner of mouth. (By courtesy of the Commissioner of the Metropolis, Scotland Yard.)

factors are necessary to cause fracture, namely, not only pressure but counter-pressure, the chance of accidental occurrence is very

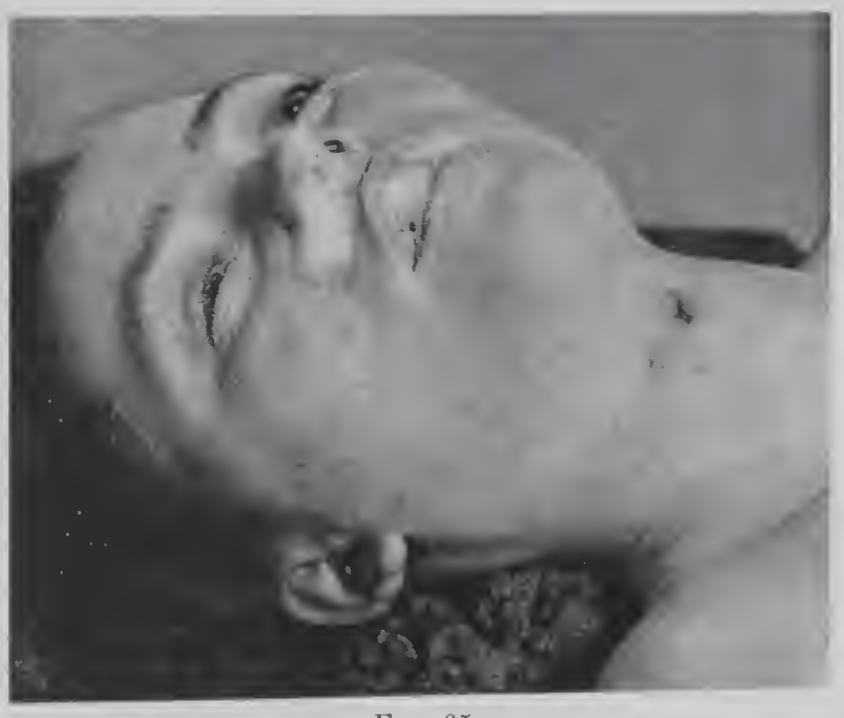


FIG. 65

Homicidal throttling. Note foam at nostrils and mouth, also marks in front of neck.

remote. Forbes [5] reports two cases of fracture of the hyoid bone as the result of pressure and counter-pressure, but unrelated to throttling by the hand. The first of these involved a motor-tractor driver, whose machine had an attached scraper which was controlled

by a wire rope, half-an-inch in diameter. Accidentally, a loop of the rope had got round the driver's neck, which had been pulled up to the winch, thus compressing the tissues of the neck. In the second case, a woman had suspended herself by a dog-lead from a hook with her feet in contact with the floor. The ligature crossed the mid-line of the neck in front at the level of the hyoid bone. It was thought that a spring device, enclosed in a rectangular metal frame on the lead, by its pressure had been responsible for the fracture. The counter-pressure was supplied by the leather part of the lead on the opposite side of the neck. This view was strengthened by an impression on the skin on the right side of the neck just over the hyoid bone. When fracture of the hyoid bone is present there

Fig. 66

Homicidal strangulation with telephone wire cord (see Figs. 165, 170).

is strong indication that the injury has been produced homicidally. When ossification between the body and the horns of this bone has occurred, in subjects of middle age and over, fracture is more easily effected. There may also be fracture of the laryngeal cartilages or of the trachea when considerable violence has been used. To cause fracture of the hyoid bone, the laryngeal cartilages or trachea, post-mortem, a very considerable degree of pressure with the hand is required. Bruising of the tongue, the floor of the mouth, the mucous lining of the larynx, and the epiglottis are common findings in cases of throttling. The areas involved vary from small petechial hæmorrhages to bruises of appreciable size. The skin and underlying tissues at the back of the neck should always be examined carefully for evidence of bruising, the result of counter-pressure exerted by an assailant.

Since asphyxia is the cause of death, the external and internal signs associated with it will be present, usually in accentuated form,

since the asphyxial process is rapid. It must be remembered, however, that if pressure has been applied over the carotid sinus, there may be an immediate vaso-vagal inhibition and a carotid sinus reflex arrest of respiration and circulation. In such cases the asphyxial signs may be either slight or practically absent due to the sudden onset of death. In one of our cases of manual strangulation, although there were surface bruises on the neck, deep bruising was absent, apart from a small, localised hæmorrhage in the tissue clothing the joint between the body of the hyoid bone and its left cornu. There was an abnormal degree of mobility at the left synchondrosis. X-ray examination did not disclose fracture. Although there were certain asphyxial manifestations, it seemed clear

FIG. 67

Abrasions on neck in a case of manual strangulation.

that cardiac inhibition, due to pressure over the carotid sinus, had brought life to a close. The victim of the assault was pregnant, and had been in sub-average health.

The possible effects of trauma in the region of the carotid sinus may be far-reaching, and are thus worthy of respect. Jokl [6] describes fatalities in the boxing ring due to trauma of the lateral regions of the neck with total absence of pathological findings at the autopsy to explain the catastrophe. Another author, whom he cites, has observed a case of sudden death due to a blow over the lateral part of the neck. Jokl also records the case of a knock-out in a fencer caused by a blow to the carotid sinus.

When definite local evidence of violence, such as has been described, is found and asphyxial signs coexist, death has been produced by throttling. Furthermore, these are indications of a homicidal act, since accidental or suicidal throttling cannot be given as a reasonable explanation. The detailed circumstances,

together with the findings, must receive the most earnest consideration in every case before arriving at a definite conclusion.

In throttling there are three impediments to the normal functioning of the body, namely, obstruction of respiration, interference with the blood supply to the brain, and pressure on the carotid nerve plexuses which are composed of fibres from the vagus, sympathetic, and glosso-pharyngeal nerves.

Occasionally a medical witness may be asked in a given case whether the hyoid bone might not have been fractured by artefact. This bone, as a rule, is not easily fractured post-mortem, and such a possibility can be eliminated when extravasation has been found at the site of fracture. He may also be asked to demonstrate how the throat might have been grasped by the assailant. In relation to this point it should not be forgotten that certain marks upon the surface of the neck may not bear an exact and corresponding relationship to underlying deep-seated bruising. This can readily be accounted for by the fact that the mobility of the skin may alter the relationship which normally exists between it and the underlying structures when certain forms of pressure are applied.

The absence of bruising upon the skin, when deep-seated bruising is present, can be accounted for by the maintenance of pressure until death has supervened, since compression of the skin will empty the vessels in it during life, and the heart may have ceased to beat before the pressure has been removed.

Frequently the body may show evidence of a degree of general violence, the result of a struggle. The absence of appreciable injury, however, is not unusual, since throttling quickly renders the victim more or less powerless and unconsciousness supervenes rapidly.

ILLUSTRATIVE CASES

The following case is given at some length, since it is illustrative of most of the injuries commonly found present in death by homicidal throttling :—

The body of a woman, aged fifty-two, was found floating in a canal a short time after death. Post-mortem examination showed that death had not been caused by drowning but from the effects of compression of the throat which had brought about cardiac inhibition during the process of asphyxia. The lips were bluish in colour and a small quantity of fine bubbled foam was present at their margins. The tongue was pressed against the edges of the teeth that remained in the lower jaw and those in the upper denture. There were a few minor injuries on the body, including the face, but the principal injuries were confined to the tissues of the neck. The skin covering the front and sides of the neck was wrinkled and lax and was of an irregular faint bluish hue, but no discrete bruises were visible. The tongue was slightly swollen and the middle third of the anterior margin showed two slightly raised nodular protusions which were faintly blue in colour. On section, these areas showed extensive hæmorrhage throughout the substance of the muscle. There were also two additional areas, of similar but less pronounced

character, on the right of the mid-line of the tongue. The most marked of these hæmorrhagic areas corresponded in position to a gap left by the lateral incisor and canine teeth on the right side of the lower jaw. The epiglottis showed twelve scattered punctate hæmorrhages in the mucosa. The larynx showed similar hæmorrhages immediately below the vocal cords. There was some scanty fine foam at the base of the epiglottis, and a thin film of foam covered the lining of the trachea. The muscles of the neck showed multiple hæmorrhages as follows :—

Diffuse hæmorrhagic staining in the left platysma muscle, 2 inches by 1 inch ;

Blood-clot over the middle of the outer surface of the left sterno-mastoid muscle ;

Two areas of bruising extending throughout the left sterno-mastoid muscle. One area measured 1 inch by $\frac{1}{2}$ inch and was situated on the anterior margin close to the middle of the muscle. The other measured 2 inches by 1 inch and was situated at the lower end of the muscle ;

A bruise in the right sterno-mastoid muscle at the level of the larynx, extending throughout the muscle substance in the region of its anterior border and measuring $1\frac{1}{2}$ inches by 1 inch. There was a similar area, showing evidence of clot, at the lower end of the muscle ;

Bruising at the lower ends of both sterno-hyoid muscles and in the same regions of both sterno-thyroid muscles ;

Two areas of bruising, each the size of a pea, in the muscles at the root of the tongue immediately to the right of the mid-line :

A small bruise over the lower border of the right sub-maxillary gland ;

A bruise in the thyro-hyoid membrane at the point of junction of the left greater cornu with the body of the hyoid bone ;

A bruise in the middle line of the neck immediately above the hyoid bone.

The hyoid bone was fractured through the right greater cornu at a point 1·3 centimetres from the tip of the tubercle. The line of fracture, which was oblique in character, ran from above downwards and forwards. The periosteum in the region of the fracture showed a slight degree of extravasation.

Tissue from the bruised areas was examined microscopically, and from the character of the extravasation and infiltration of blood, it was evident that the lesions had been produced during life.

When all the circumstances of the case were fully considered, it seemed clear that either the woman had been dead when her body entered the water or that death had occurred almost as soon as she had entered the water or within a very short time thereafter, and that it could not have been a case of accident or of suicide.

In another case, pressure had been applied to the upper part of the throat and floor of the mouth. The deceased man was fifty-eight, and at the time of the fatal assault was under the influence of alcohol. The blood alcohol content was 228 milligrammes per

100 millilitres and the urine alcohol content was 340 milligrammes per 100 millilitres. The face showed a large number of petechial hæmorrhages which covered an area extending from the upper part of the forehead to the under surface of the chin (see

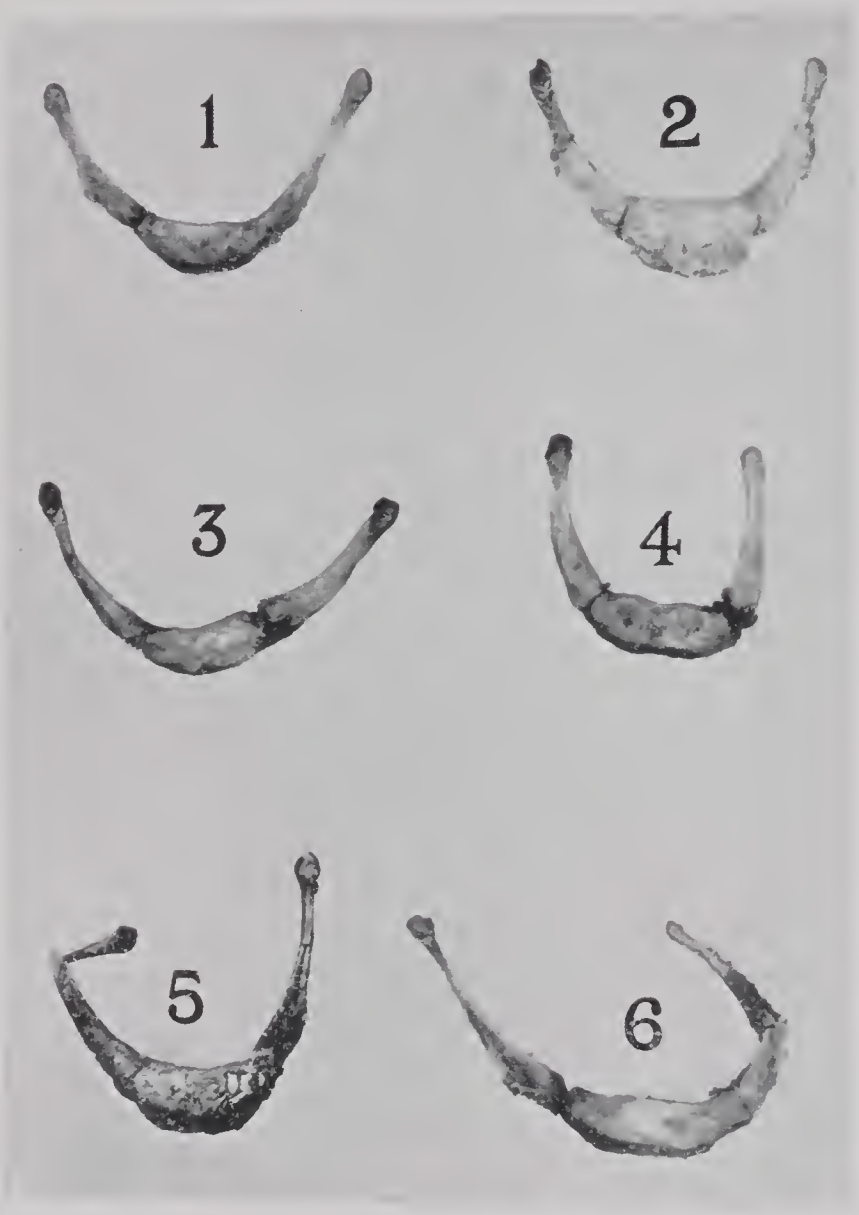


Fig. 68

Hyoid bones.

1 and 2, normal. 3 and 4 show subluxation at left synchondrosis. 5 and 6 show fracture of the right and left cornu respectively.

Fig. 70). The lining of the eyelids of both eyes showed pin-point hæmorrhages and there were also bilateral subconjunctival hæmorrhages. There was no external evidence of bruising on the surfaces of the neck or under surface of the chin. The tongue was congested and showed diffuse extravasation almost throughout its entire substance. The greater part of the lateral margins showed bruising and the front of the tongue was similarly affected apart from a

small portion at the tip. The back of the tongue was the seat of blackish blue discoloration which extended backwards to the anterior surface of the epiglottis, which was slightly œdematous, as were the structures of the upper part of the larynx. The structures, including muscle, in the sublingual and submaxillary regions on both sides showed marked extravasation, but this was more extensive

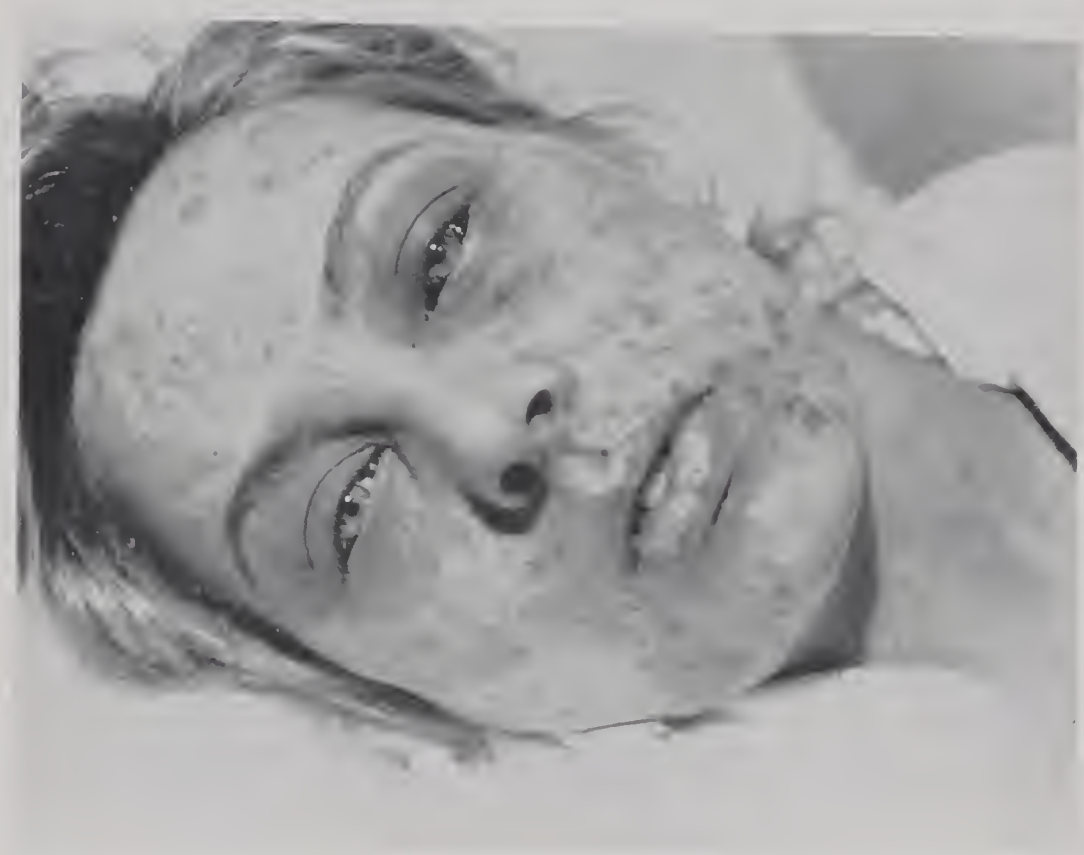


Fig. 69

Petechial hæmorrhages on face in a case of attempted murder by strangulation.
(By courtesy of Dr Gilbert Forbes, Sheffield University.)

on the left side. The tissues in the left submaxillary region were œdematous. The hyoid bone, laryngeal structures, and trachea were uninjured and there was no extravasation of blood into the tissues surrounding them.

One of our cases of throttling was of rather an unusual nature since the victim of the attack survived for about three-quarters of an hour, and was able to return to his home both by bus and by walking. Post-mortem dissection showed marked extravasation in the deeper tissues of the floor of the mouth, more marked on the right side. On the right side of the neck, the tissues around the hyoid bone were bruised, and to a lesser extent on the left side. From the top of the thyroid cartilage down to the upper border of the breast-bone and extending laterally to the deeper tissues on both sides of the neck, there was massive bruising. The bruising extended to the posterior wall of the pharynx and to the adjacent wall of the œsophagus. It travelled laterally from the

posterior border of the thyroid cartilage to the opening of the larynx. The epiglottis was congested, the left anterior part was œdematous and on the right anterior part there was bruising. Bruising was also present on the right lateral and anterior walls of the upper part of the larynx. The right vocal cord was bruised and œdematous with a markedly diminished air channel. Further bruising affected the lining membrane of the œsophagus. The right superior cornu of the thyroid cartilage was fractured together with the left cornu of the hyoid bone. The right cornu was more mobile at its synchondrosis than its neighbour. Death resulted from œdema glottidis.

The following case is one of strangulation by a ligature applied to the neck. The deceased female was aged twenty-three. The finger-nails, toe-nails and lips showed some lividity and scanty pinkish coloured fluid exuded from the nostrils and mouth. A small group of punctate subconjunctival hæmorrhages was present over the upper outer quadrant of the right eye and a few smaller hæmorrhages were situated on the outer corners of the rims of the eyelids. A similar condition affected the left eye. Both conjunctivæ were slightly congested. An artificial silk scarf formed a single ligature around the neck and was held fairly tightly by a reef knot which was firmly tied. The following marks were present on the neck :—Over the front were two principal marks. The mid-point of the lower mark was at a level of $1\frac{3}{4}$ inches above the upper border of the sternum. The mark then passed slightly upwards over the right of the front of the neck, the highest point being 2 inches above the sterno-clavicular joint. The mark also passed slightly upwards over the left of the front of the neck, the highest level being $1\frac{1}{2}$ inches above the clavicle. The maximum level on the right side of the neck was at a point 3 inches below the lobe of the ear, behind which a slightly forked formation of the mark was seen. Close to the mid-line of the back of the neck, these forks united and the mark continued round the back and left side of the neck. The average breadth of the mark was $\frac{1}{3}$ inch. Over the front of the neck and above the ligature mark described, there were several interrupted and less defined pressure marks, due to the ligature, which ran horizontally in relation to the mark which encircled the neck. Running from the immediate left of the middle line of the neck to a point 1 inch below the angle of the right lower jaw was an irregular, curved, parchment-like area which measured $3\frac{1}{2}$ inches by 1 inch. It was of curved contour with convexity towards the jaw. This mark corresponded to the position of the ligature knot and adjacent parts of the ligature when the head was flexed and directed slightly to the right. All the marks upon the neck were of dark red colour and were leathery to the touch. Two punctate abrasions were present, one on the under surface of the left side of the lower jaw and the other on the right side of the chin. Three similar abrasions were present on the right side of the neck. On dissection of the tissues of the neck, two small areas of extravasation associated with the ligature mark were seen. Both

were superficial. The laryngeal structures, the trachea, and hyoid bone were intact. The tongue showed a slight indentation on the under surface of the tip corresponding to the position of the left

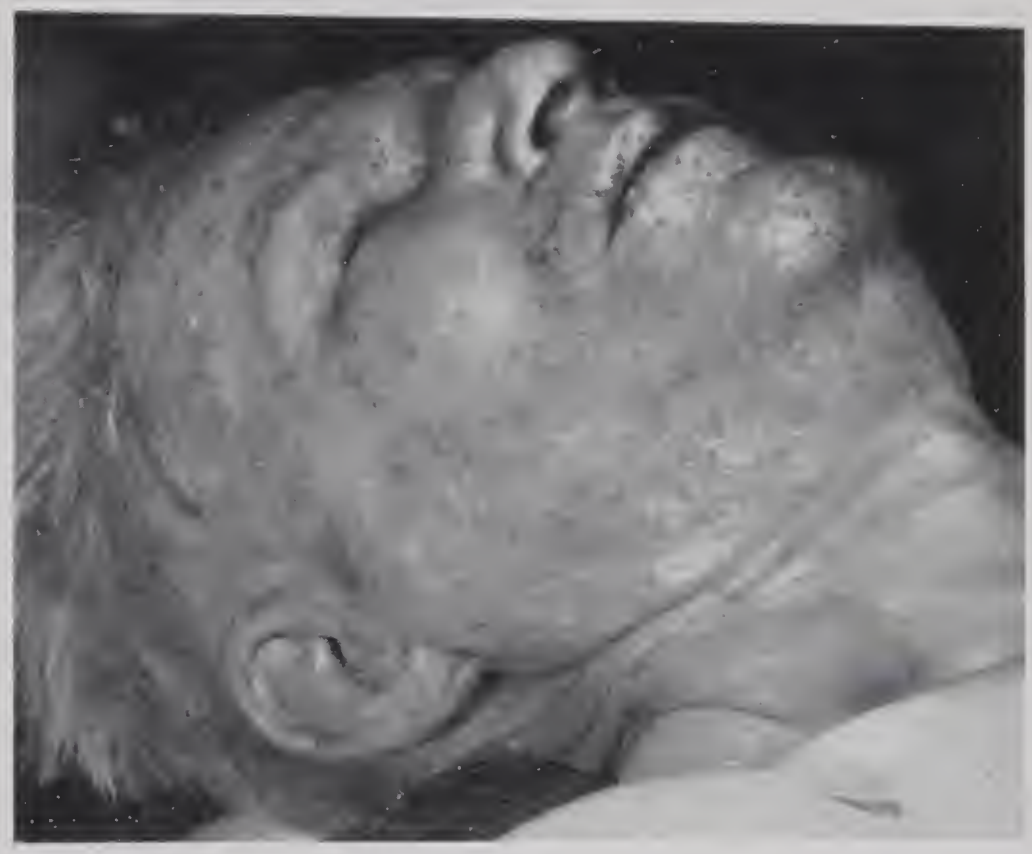


FIG. 70

Multiple petechiæ extending from the upper part of neck and covering the face can be seen.

lower incisor tooth. Incision into this area did not show evidence of extravasation. The epiglottis was slightly congested (see Figs. 62 and 63).

References

1. Jour. State Med., Vol. XIII, 155 and 228, 1905, Revenstorf.
2. "Artificial Respiration Explained," 44. Eve, Livingstone, Edinburgh, 1946.
3. Brit. Med. Jour., Vol. I, 493, 1908, Renton.
4. Glas. Med. Jour., N.S., Vol. CXXX, 180, 1938, Gordon and Thomson.
5. Police J., Vol. XVIII, No. 1, 27, 1945, Forbes.
6. "The Medical Aspects of Boxing," 67-68, Jokl, J. L. Van Schack Ltd., Pretoria, 1941.

CHAPTER VII

DEATH FROM LIGHTNING, ELECTRICITY, AND BURNING

Death and injuries from lightning.

IN Great Britain the annual number of deaths caused by lightning is very small, but such deaths are much more frequent in tropical and sub-tropical countries.

The medical jurist is likely to be consulted in relation to bodies which bear the marks of injury of which lightning may be the probable cause in contradistinction to other forms of violent death.

Jex-Blake [1] states that the lightning flash is a rush of protons and electrons through the air with a liberation of a great amount of energy along a path of two kilometres. The current is direct and of about 1,000 million volts and probably in the region of 20,000 amperes. The duration of a single stroke is about one thousandth of a second. The flash takes meandering channels with secondary flashes branching off. The diameter of the channel down which the lightning travels is estimated as at least eighteen feet. When it hits the earth, objects a hundred feet or more apart may all be struck.

When a person is brought into contact with a flash of lightning, death is usually immediate and is due to involvement of the central nervous system with paralysis of the heart or of the respiratory centre. Spencer [2] holds the view that the shock produces instantaneous anæmia of the brain, due to sudden spasmodic contraction of all the cerebral arteries, the muscular fibres in their coats being firmly contracted. The heart suddenly and forcibly contracts and remains in that state until death supervenes or until stimulated by the return of some blood into it. Cessation of respiration results from sudden anæmia of the medulla and spinal cord.

Even when an individual is more remote from the flash, concussion may result. A flash causes very considerable air-pressure, and its effects are not infrequently responsible for severe injuries to the body.

Lightning-stroke usually causes immediate unconsciousness. The intense disturbance of the air in the immediate vicinity of the flash may either remove the clothing from a body or tear it very extensively. Spencer describes four elements in a flash :—

The direct effect of the current ;
Burning by superheated air ;
Effects of expanded and repelled air around the flash ; and
The " sledge-hammer blow " given by the compressed air pushed before the current.

The most trivial lesions found after lightning-stroke are streaky surface burns which only involve the epidermal layer of the skin. These are frequently characteristic in pattern and have been called "lightning prints" or "arborescent markings" from their tree-like character. They have been likened also to frost designs. They may be found on various parts of the body.

The lesions most frequently seen following lightning-stroke may be summed up as follows: (1) Wounds of almost any description;

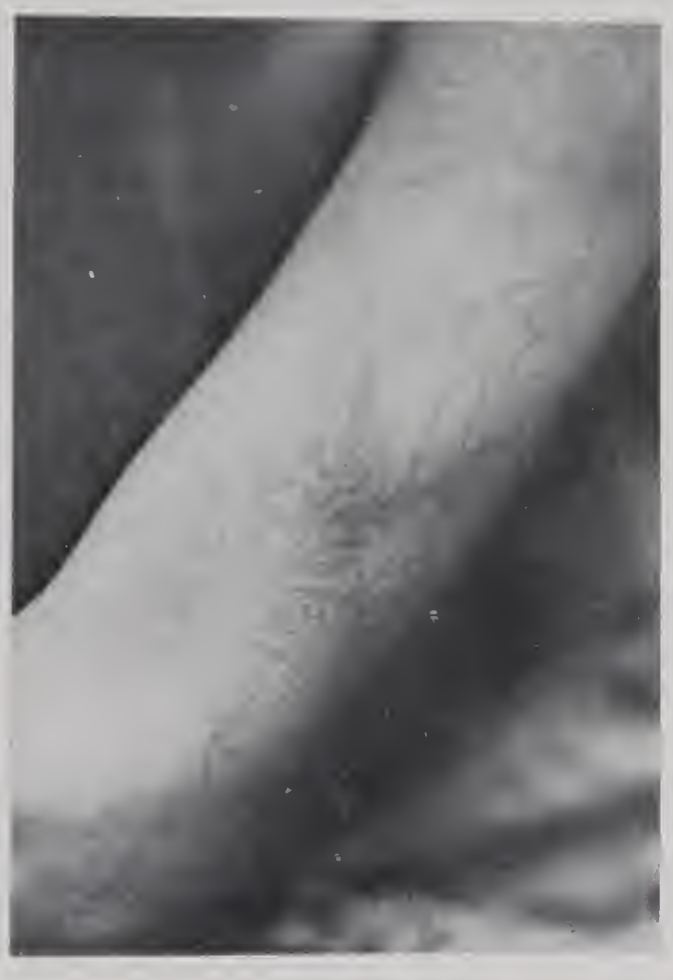


FIG. 71

Arborescent markings on arm. The photograph was taken four hours after accident. Eleven hours after accident, markings had entirely disappeared.

(2) fractures, simple, compound, comminuted, single or multiple; (3) burns of almost any shape, linear, streak, patch, or of arborescent form; (4) ecchymoses; (5) singeing of the hair of scalp, face, or body; (6) impressions of metallic articles upon skin, due to burning, the result of the high temperature attained by these articles.

The view that the electric lesion is not a true burn, but is painless and without inflammatory reaction is expressed by Jex-Blake. Vaso-constriction and vaso-paralysis with subsequent thrombosis account for the pathological effects.

In addition, such conditions as blindness, deafness, paralysis, loss of memory, delirium, and convulsions may result.

A very significant indication of lightning-stroke is the magnetisation of metallic articles found on the person, or of articles carried by the individual, when struck.

The internal post-mortem findings are in many cases remarkably negative. The surfaces of the lungs and pericardium frequently show petechial hæmorrhages. Congestion of the brain, lungs, kidneys, suprarenals and spleen may be found, and hæmorrhage into the pancreas and necrosis of this organ are described. Gangrene of the cæcum after lightning-stroke is recorded.

Critchley [3] has described the histology of the lesions found in the central nervous system following the effects of lightning and electricity. These include focal petechial hæmorrhages scattered throughout the brain, especially in the medulla. When the cranium has been directly hit by lightning, large vascular tears may be found in the cerebrum, or the entire brain may be swollen, softened, and almost diffluent. Chromatolysis of the pyramidal cells and the nerve cells of the medullary nuclei may occur, and the damaged cells show a dark, shrunken and uniformly stained nucleus often eccentric in position.

When a dead body is found after a thunderstorm, and there is an absence of marks upon it, death may not have resulted from lightning-stroke but from syncope due to sudden fright, since many persons are greatly alarmed by a thunderstorm.

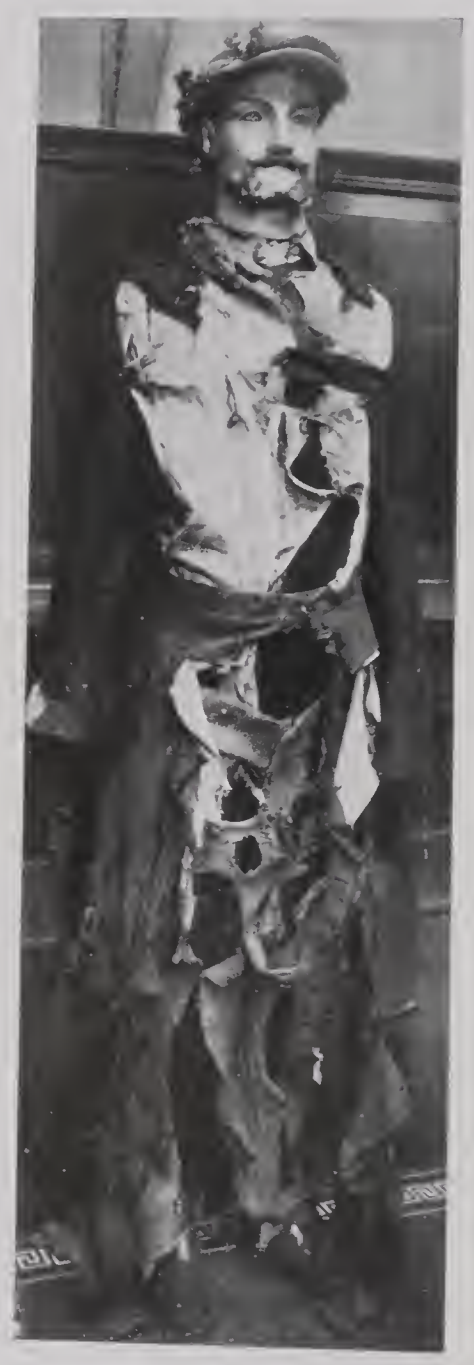


FIG. 72

Effects of lightning-stroke on clothing, which is fitted on lay figure.

Dill [4] reports the case of a man who was killed by lightning while sheltering under an aeroplane during a thunderstorm. A lightning discharge occurred close by. The man's body was found under the wing and close to the body of the plane. Under the body there were camouflage netting and a piece of metal tubing. Some 6 to 8 feet away was a wire earthing, attaching an aerial to a metal rod.

There was a small hole in the victim's cloth cap and a small hairless spot, corresponding to the position of the hole in the cap, on the right parietal region of the head. On each buttock was a circular area, 1½ inches in diameter, having the appearance of a recent burn. The deceased was wearing boots with iron heels and toes.

Lightning fatally injured two soldiers while playing in an Army Football Cup Final replay at Aldershot in April, 1948. Two flashes of lightning, unaccompanied by rain, occurred. One of the flashes was to the right of, and another immediately above, the players. The referee and seven others, including three players, were taken to hospital where two of the players died. Several of the spectators had to receive attention for minor injuries.

Treatment.

Since respiration will be suspended, artificial respiration should be commenced without delay and maintained, for at least four hours if necessary, before abandoning all hope. The patient's face should be closely inspected for the "deglutition sign." Swallowing is said to be the most reliable sign of the return of spontaneous breathing. When it becomes operative, artificial respiration should not be continued. The airway should be kept clear and warmth applied to the body. Oxygen with up to 7 per cent. carbon dioxide should be administered. Cardiac stimulants may be administered as indicated. Warm coffee should be given rectally. The patient should be kept in warm and quiet surroundings. When there is increased tension of the cerebro-spinal fluid, due to œdema of the brain, some of the fluid should be withdrawn by lumbar puncture. Rectal hypnotics may be given to combat delirium should this condition supervene (see p. 211).

Death and injuries from electricity.

The use of electricity is so general, both for industrial and domestic purposes, that accident is almost inevitable. This may occur in a variety of circumstances and frequently causes instantaneous death either from respiratory paralysis or from ventricular fibrillation. Amperage, on account of tissue resistance, is of greater importance than voltage in the production of electrical shock. When accidents from electricity occur there are a number of composite factors responsible, and these include voltage, amperage, resistance, and earthing. The work of Langworthy and Kouwenhoven [5] has contributed important information. They have found that the principal conductors of electrical current in the body are the fluids and that most of the current travels along the blood-vessels. The resistance offered by the various organs differs, since it bears a certain relationship to the morphological and chemical composition of the organs. In their animal experiments it was shown that when a low-tension current was passed through the heart, complete inhibition occurred and fibrillation was produced by the direct action of the current on the muscle fibres and ganglion

cells of the heart. With high-tension current, the heart stopped immediately, but when the current was broken the ventricles

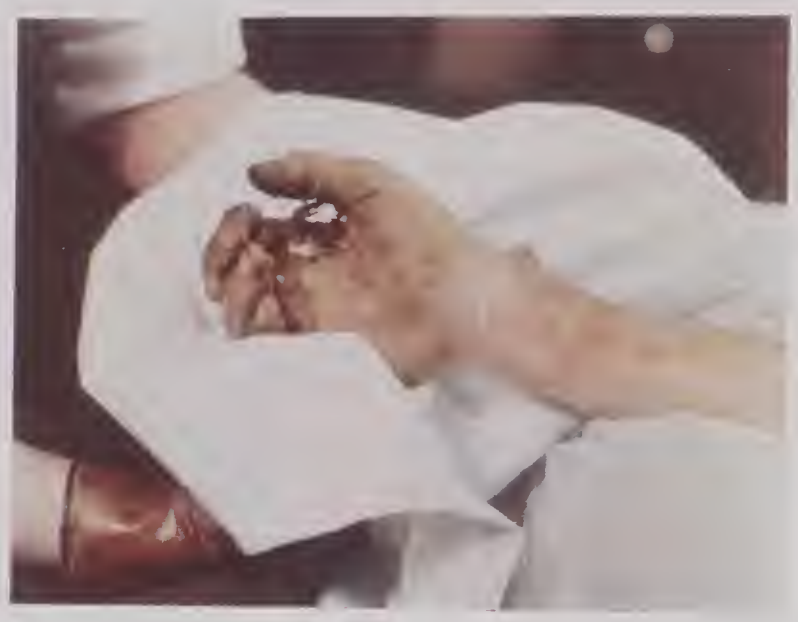


FIG. 73
Electrical burn due to grasping conductor of a pylon carrying 20,000 volts.

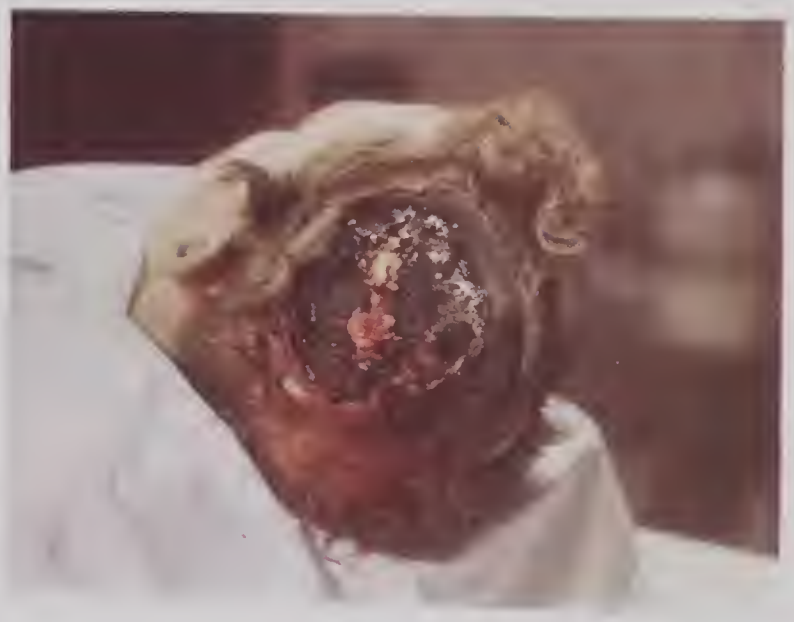


FIG. 74
Same case as Fig. 73. The man fell back and his head contacted the pylon while his hand still grasped the conductor. Note extensive burn.

started to beat rapidly and strongly and the arterial pressure rose. With high voltages, the brain and spinal cord were more often injured, but with low voltages fibrillation of the heart occurred.

The cardiac and respiratory centres in the medulla are very vulnerable to electrical current.

That voltage alone is not the sole factor in producing death is shown by the fact that death has frequently resulted from 110 volts up to 250 volts and that recovery has followed exposure to 2,500 volts. One important circumstance in the likelihood of a fatal accident by electricity is the condition of the hands, feet, and clothing of the person exposed to the electrical discharge. When these are wet or damp, the effects are more serious since the resistance of the skin is lowered by moisture. Weber's personal experiments indicated that there is danger in grasping the conductors of two alternate currents when the difference between their intensity exceeds 100 volts. He believes that the reason why electrical engineers seem to have an apparent immunity from higher voltage is that they are constantly on their guard from this knowledge and are insulated by dry shoes, whereas workmen are more careless and may be working in damp shoes.

The part played by moisture is responsible for the occasional death from electricity which occurs in bathrooms through defective electrical fitments. If a person in a bath switches on an electrical appliance which is defective, through the unearthed wire touching it, he may probably be electrocuted from the current passing through the water and piping, since the remaining wire is earthed, and he becomes a link in the faulty circuit.

Burns caused by electricity.

A burn is frequently present at the point of contact and is caused by the generation of heat due to the resistance of the skin. The site of exit by the current may also show an area of burning. High-tension currents produce considerable localised burning, and in the case of the hands the appearance is blackish-brown and often resembles a dark-coloured glove. Such burns are usually 4th or 5th degree lesions. Wakely suggests the term "electrical necrosis" rather than "electrical burns." Uncomplicated specific electrical skin lesions will only be present when a small amount of heat is generated by a low voltage current with a short duration of contact. He explains that the skin, especially if dry, is a highly resistant barrier, and is by contact the place of entry and exit of the current with resultant liberation of considerable heat. High voltage currents generate much greater heat and produce injuries in which the characteristics of the primary electric lesion are overshadowed by the ordinary burning which is caused by the high temperature, sometimes accompanied by a flash or flame. He states that the electric arc, which may generate up to 7,000° C. is commonly responsible for this type of lesion.

ILLUSTRATIVE CASES

The following cases may be taken as typical of the effects of electricity :—

A man was admitted, unconscious, to an infirmary, his arms and head being severely burned. He was employed in a power station, and had accidently fallen on the "bus bars" connected with the switchboard which had a 6,500 voltage. The following burns were found upon the body. (1) On right arm: (*a*) a burn of 6th degree running from lower ends of radius and ulna on dorsal

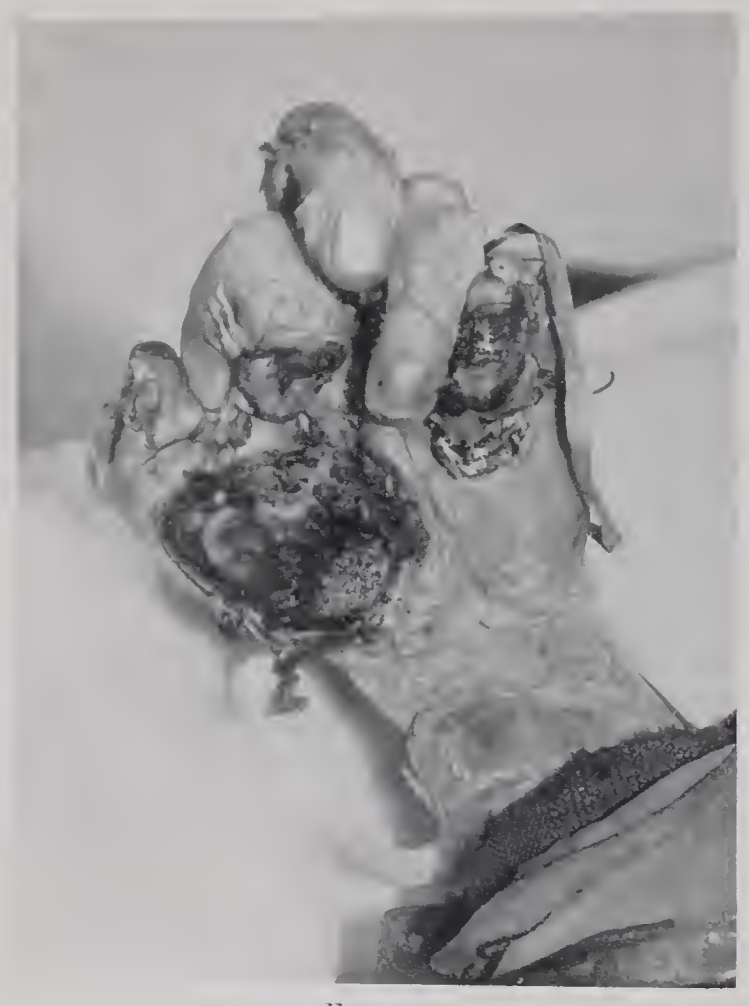


FIG. 75

Electrocution.

Site of entrance of current—11,000 volts—with charring to bone.

(By courtesy of Dr Robert Richards, Aberdeen University.)

surface to lower ends of three outer metacarpal bones; (*b*) a burn of 5th degree all round forearm from wrist to 4 inches up the forearm; and (*c*) a burn of 5th degree in front of elbow-joint. (2) On left arm: (*a*) a burn of 6th degree over whole of palm of hand and palmar surfaces of fingers and thumb; (*b*) a burn of 5th degree along the whole anterior aspect of forearm and in front of elbow-joint; (*c*) a burn of 3rd degree extending for $3\frac{1}{2}$ inches from wrist up the back of forearm; and (*d*) a patch of burning of 2nd degree on the left axilla. On the head, there was an area of burning of

6th degree above and behind the left ear, measuring 4½ inches by 3½ inches. The breathing was of Cheyne-Stokes type. He died eight hours after admission, without regaining consciousness.

A young man died from an electric shock under the following circumstances. Two boys, employed in the same works as the deceased, thought to play a trick on him, and attached a wire from an electric light switch in a room to the handle of the door. The deceased on trying to turn the handle fell to the ground. When picked up almost at once, he was still breathing, though unconscious, and artificial respiration was employed, but without success. One

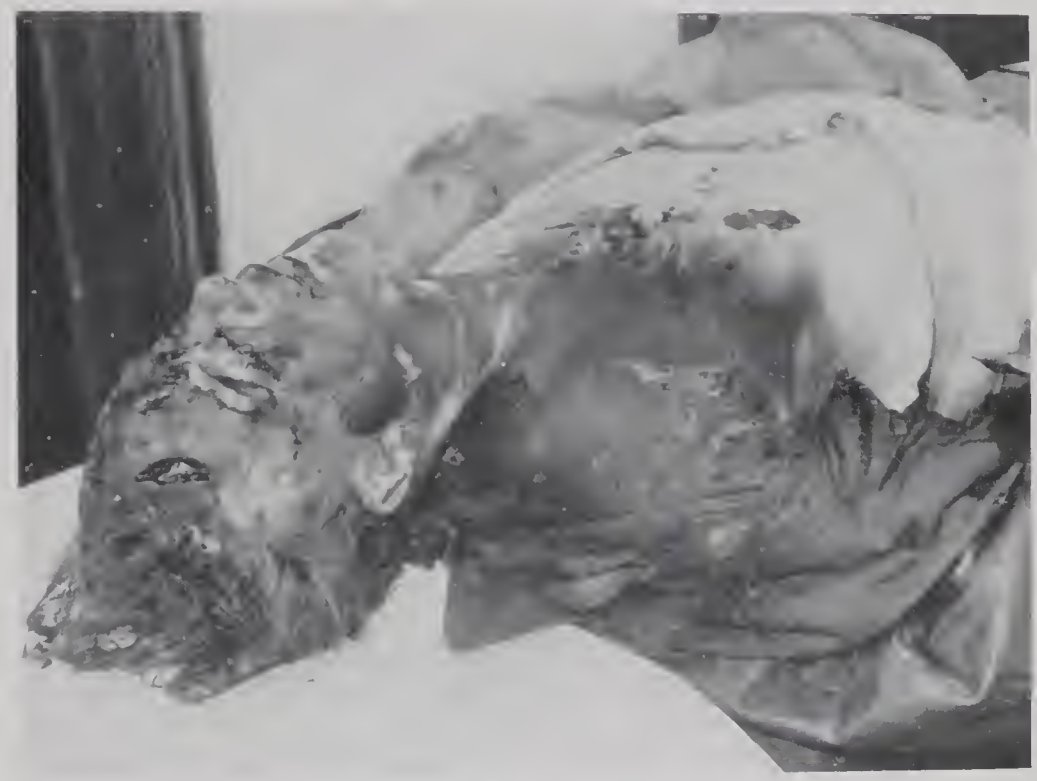


FIG. 76

Electrocution.

Same case as Fig. 75 showing site of current exit on right side of neck where it caused a large excavated wound with charring of chest tissues and flash burns of face.

(By courtesy of Dr Robert Richards, Aberdeen University.)

of H.M. Divisional Electrical Inspectors of Factories said that, from the evidence, the deceased had sustained the full shock of the discharge, about 230-250 volts, in view of the fact that he had been standing on damp soil. There was a wound ½ inch long on the right palm at the base of the little finger.

A lady, aged sixty-one, died of shock from a current said to be alternating, of 50 cycles per second at 240 volts. The deceased was found in bed with wireless headphones still in position, and electric sparks were playing at the points of contact of her metal-rimmed spectacles. Examination showed the deceased was grasping a brass standard electric lamp. Burns were found between the index finger and thumb of the left hand and, of smaller size, on the

face and scalp, corresponding to points of contact of spectacles and headphones. Electrical experts found an exposed wire in the flex of the standard lamp which proved to be the cause of the short circuit. Another bared wire was present in the telephone circuit of the wireless set, causing a second short circuit through the metal body and headbands. It was believed that deceased received the full voltage.

A woman, aged thirty-one, was found dead in her house one morning. A chromium electric table-lamp was lying on the floor close to her right hand. She had been in the act of rolling the flex when one of the wires became detached from the base and came into contact with the metal, thus electrocuting her. There were marks of scorching on the metal at the base of the lamp. The voltage was 250, alternating current. On the skin over the lower part of the upper third of the right forearm there were three small, brown abraded areas. The superficial vessels of the brain and the cranial sinuses were congested. The brain, which showed multiple, petechial hæmorrhages, was œdematous. A few similar hæmorrhages were present over the surfaces of the basal lobes of the lungs. The chambers on the right side of the heart were dilated and the dilatation was more marked in the instance of the right auricle. The right auricle and ventricle, together with the associated great vessels, were very congested. The organs of the body were also congested.

An unusual fatal case resulting from electrical contact has been reported. A training aircraft dropped a strip of tinfoil in a garden. A woman ran to collect it and was electrocuted. The tinfoil in falling had become entangled with overhead electric cables.

In death from electricity, the post-mortem signs will naturally depend upon whether the fatality has occurred primarily from respiratory or cardiac paralysis. When due to the former cause, asphyxial signs will form the predominant features.

Various changes may be observed in the central nervous system, including hæmorrhages in the brain which vary in size, when there has been contact with high voltages. The nerve cells may show various abnormal manifestations. These have been dealt with in relation to the effects of lightning (see p. 205).

Accident and suicide are the commonest causes of death from electricity (see pp. 209-211).

Treatment.

Artificial respiration must be employed with great promptitude and maintained for considerable periods (see p. 178). This should be undertaken in a warm atmosphere. Prolonged artificial respiration may promote recovery in cases of respiratory failure, but there is no effective treatment for ventricular fibrillation.

Maclachlan [6] has examined the reports in 700 cases of injuries by electricity, and believes that the success of resuscitation is greater in cases when the application is carried out within three minutes or less after the shock, than in those in which it is carried out within four minutes or more. It has been asserted that a six-minute delay produces an 80 per cent. increase in the death rate. He also believes

that success may result after many hours of work apparently on a lifeless victim ; 479 of these cases had stopped breathing and 323 were restored to life.

Board of Trade instructions indicate that artificial respiration, in cases of electrical shock, should be maintained for four hours at least. The use of 5 to 7 per cent. carbon dioxide with oxygen when carrying out artificial respiration is advocated on the grounds that it preserves a desirable carbon dioxide level in the body and stimulates the respiratory centre.

An excerpt from the Annual Report of the Chief Inspector of Factories [7] states that the senior electrical inspector reports 13 cases of electric shock with unconsciousness successfully resuscitated by artificial respiration applied for up to half an hour. The majority of "unconscious" shocks occur at pressures below 250 volts and, because of the nature of the apparatus handled, the patient is usually "held" by the live contact. Records suggest that the chances of recovery are slightly greater with higher voltages because contact is seldom maintained, but serious burning is common. Most of those who are going to recover with artificial respiration do so in the first ten minutes, a good proportion recover during the second ten minutes, but fewer after that. It is stated that lumbar puncture, as a useful adjunct in treatment, is not widely enough known. Removal of excess of cerebro-spinal fluid, produced by electric shock, enables the stunned respiratory centre to respond better to artificial respiration.

Stimulants should be exhibited when necessary. The other lines of treatment are those which have been mentioned in relation to the treatment of lightning-stroke (see p. 206).

When a person is in contact with electricity, immediate steps should be taken to free him from the current. The greatest precautions must be taken to prevent further accident ; therefore, if the current cannot be switched off, the rescuers must be fully insulated before putting themselves in contact with the victim and the current which is passing through his body. In the absence of rubber gloves, a thick garment rolled round the hands, or a long, wooden-handled brush, should be employed for disengaging the body. A number of years ago, in France, a boy came into contact with electric cables carrying 10,000 volts and was killed instantly. Two rescuers were killed in the attempt to rescue the body, and it was not until the current was cut off that the bodies of the three victims were recovered.

Capital punishment by electrocution.

In America, this is quite a common method of inflicting the last penalty of the law. The person is strapped to a chair and metal electrodes are placed over the head and around one leg. An alternating current of about 1,700 volts and 7 amperes current strength is passed through the body for about sixty seconds. The current is passed through the body a second time and is continued for a

similar period to ensure that life is extinct. It has been recorded that in death from electrocution, the brain is heated to 140° F. and that vacuolation around the vessels has been noted.

DEATH FROM BURNING

This is by no means an uncommon form of death, and the lesions found upon the body present a variety of appearances. Burns may be produced in the following ways :—

By any agent at a highly elevated temperature, either dry or moist, for example, very hot solid bodies, liquids at or near boiling point, and current or pressure steam,
corrosives,
friction,
lightning, electricity, X-rays, and ultra-violet rays.

A burn may be defined as a lesion which is caused by the application of heat or of chemical substances to the external or internal surfaces of the body, the effect of which is a more or less marked destruction of tissue. This definition, therefore, includes all lesions whether produced by fire, water, or chemical action. A scald may be defined as a lesion which results from the application of liquid at or near boiling-point, or from steam. On a body which has been subjected to the influence of fire, the lesions found may be graduated from the simplest erythematous blush, through the different surgical degrees of burning, to charring of a greater or lesser portion of the body.

The surgical classification of burns is as follows :—

Burns of the first degree.
Erythema, transitory swelling, and subsequent desquamation of the surface layers of epidermis.
Burns of the second degree.
Vesication.
Burns of the third degree.
Partial destruction of the true skin.
Burns of the fourth degree.
Total destruction of the true skin.
Burns of the fifth degree.
Destruction of the subcutaneous tissue and involvement of muscular tissue.
Burns of the sixth degree.
Extension in depth with probably involvement of large blood-vessels, nerve trunks, serous cavities, and bone.

The law does not differentiate between the causes of the lesions, and terms them all as burns. Frequently, bodies taken from a burning building present some appearances of the effects of burning, but it must not be supposed that in all cases death is attributable to this cause. Some persons may have died from asphyxiation, some from shock or fear, and others from injury, either through

falling from a height or from debris falling upon them. In such cases, therefore, although the external appearances may show the effects of burning, the internal appearances may point to another cause of death.

In one of our cases, fire broke out during the night in a lodging-house. The number of the victims was thirty-nine, and most of them bore evidences of contact with fire. The character of the burns, in several instances, showed that some of the lesions were produced before, and some after, death. In certain cases, the area of burning was very extensive. In seven cases there were marked signs of asphyxia, and in all of these the tongue was protruded to varying distances beyond the line of the teeth, and in each it was blackish in colour, partly from lividity and partly from deposited soot.

The medico-legal points which demand consideration in connection with bodies exposed to burning are the following :—

Are the lesions found due to burning ?
Were they produced before or after death ?
Do they account for death ?
Are there lesions, other than those due to burning, which might alone account for death ?
Are the burns accidental, suicidal, or homicidal ?

Are the lesions found due to burning ?

The lesions and appearances caused by fire may be summarised as follows :—

Vesicles, more or less widespread.
Roasted patches of skin, or of deeper parts of the body.
Singeing or burning of the hair of the body or of the clothing.
Deposits of carbonaceous material on the body.

Those caused by scalding are :—

Erythema.
Vesication.

Those caused by corrosives are :—

Inflammatory redness of skin.
Ulcerated patches of skin.
Discoloration and staining of skin and clothing.

It should be noted that vesicles are rarely found when corrosive acids or alkalis have been used. When phosphorus, dissolved in carbon disulphide has been employed, vesication will probably be found, because the phosphorus becomes oxidised by the air and is ignited when the carbon disulphide evaporates.

The diagnosis, therefore, between burning by fire and by scalding may be established by the presence or absence of certain lesions upon the body. Wherever singeing of the hair or clothing,

deposition of carbonaceous material on the skin or clothing, and charring of tissue are found, fire has been the cause of the lesions. In such cases, some degree of vesication will also be found. If vesication is extensively present and the other signs are absent, it may be affirmed that the injuries have resulted from scalding.

The lesions found on a body when corrosives have been applied are characteristic. The differentiation between lesions due to corrosives and those due to fire or heated fluid or steam, may be established by the following points :—

The absence of vesication.
The presence of coloured stains on the skin or clothing.
The presence of chemical substances in the stains (see p. 223).

Marks of burning may be found upon a body and on the clothing which may not have been caused by fire, but by friction, as is shown in the following case.

A man employed in a saw-mill disappeared from his usual place during working hours. The workmen employed in the neighbourhood of the shaft, in which the driving-belt of the machinery revolved, had their attention attracted by the odour of burning which came from this shaft. The body of the man was found at the bottom of the shaft, between the wall and the driving-belt. Extensive marks of burning were found both upon the body and the clothing.

Were the lesions produced before or after death ?

The answer to this question depends upon the presence or absence of vital reaction in the lesions. The presence of vesication, the character of the fluid in the vesicles, and the inflammatory changes in and around the vesicles are highly important. If the vesicles show the following characters, vital reaction has occurred :—

Serous fluid which contains both albumin and chlorides ;
An inflammatory areola around the vesicle ;
Inflammation of the base of the vesicle ;
The presence of pus.

When pus is present it indicates that the person has lived for at least thirty-six hours after the injury. On the other hand, it may be affirmed that the vesicle was of post-mortem origin if the following characteristics are present :—

If the vesicle is limited in size, and its contents are scanty ;
If it contains air, or only a small quantity of non-albuminous fluid with an absence of chlorides ;
If inflammatory reaction is absent.

Vesicles may be produced shortly after death, when molecular life of the tissues is still present, but in this event the vesicles do not contain albumin.

As a general principle it may be stated that a vesicle which shows marked evidence of vital reaction, has been produced during

life, but that a vesicle which contains air or watery vapour, and which shows no evidence of vital reaction, has been produced after death.

On making a dissection of a body which may have been exposed to fire, attention should be specially directed to the air passages where carbonaceous material may be present. A sample of blood should also be examined for carboxyhæmoglobin. These lines of examination may afford important evidence as to whether the person was alive or dead when the fire originated.

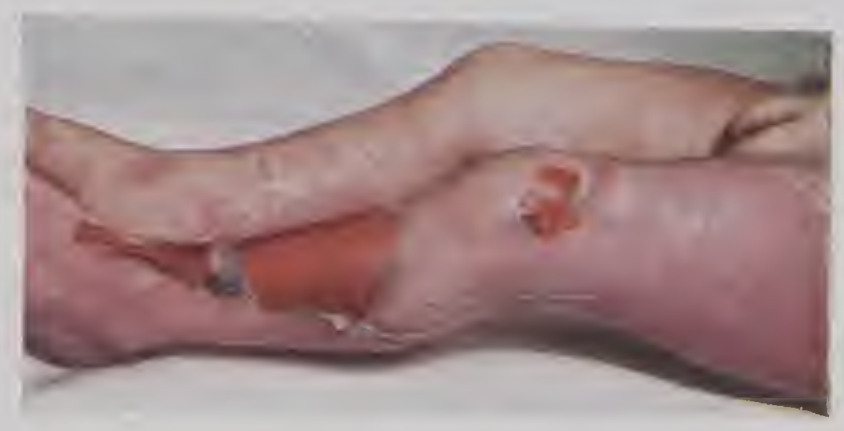


Fig. 77

Extensive burns.

Do the lesions account for death?

This question raises several points, including the age and general health of the deceased person together with the area of the body involved by the burns. In broad terms, it may be stated that children succumb quickly to the primary effects of shock due to burning, and to the secondary effects from inflammation of the serous membranes, such as pleurisy, peritonitis and meningitis, from perforating ulcer of the duodenum, and from septic absorption.

The danger to adult life from burning is usually in direct proportion to the extent of the body surface involved rather than to the severity of the burn itself, when limited in area. Burns, although comparatively superficial, which involve one-third to one-half of the surface of the body, are generally fatal. As the result of many observations, we have noted that death has usually ensued in cases in which one-third of the body surface has been implicated.

In individual cases, however, recovery may follow even when large areas have been affected. Involvement of approximately 10 per cent. of the body surface, or even a smaller area, has caused death in young children. Rosenthal [8] states that the modern conception of the pathogenesis in burns is that there is a local accumulation of fluid which begins immediately or shortly after the injury and an increase in the blood concentration. When this process becomes too severe, the blood-pressure falls and death results from cardiac failure or secondary shock. Autopsy findings in thirty fatal cases showed that eleven deaths occurred after shock had been overcome. In five of these death was attributed to "sepsis, hypoproteinæmia,

and anæmia." The earlier theory of the existence of a burn toxin is not now largely supported. Blackfield and Goldman [9] believe that death from the later effects of burning arises from concentration of the blood with certain biochemical reactions, namely, an elevation of the non-protein nitrogen and a lowering of the blood-chloride level, together with toxæmia. The origin of the toxæmia remains uncertain, but is probably due to the presence

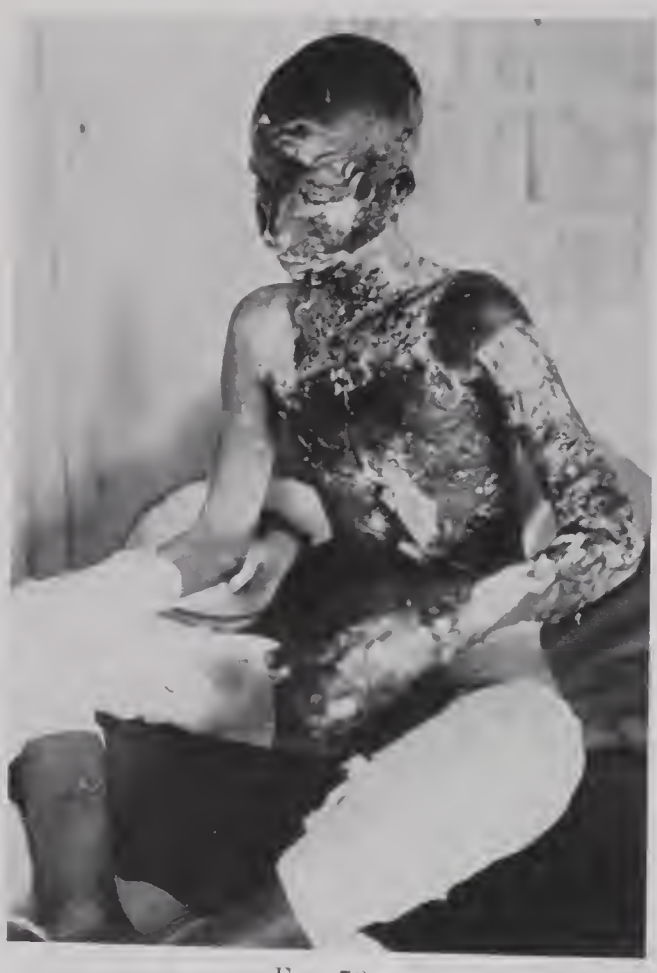


FIG. 78

A case of recovery from burning affecting an extensive area of body surface.

of a split protein, which arises from the damaged cells of the deeper layers of the skin. This is not a universally accepted view. Some suggest that acute toxæmia is probably due in part to absorbed products from the burned area, and in part from septic infection. Results of research have shown that there is a marked catabolism of protein and that the nitrogen loss in the urine may be the equivalent of as much as four times the normal daily protein intake of the patient. Protein may also be lost from the surfaces of the burned areas in the exudate. The nitrogen equivalent of this may be as high as a quarter of the total being lost in the urine. Thus a diet with high protein content is necessary for the patient. Belt [10] has reported four cases of extensive superficial burns, death having

occurred within four days, which presented severe liver damage with mid-zonal necrosis. Erb, Morgan, and Farmer [11] have published an account of a number of fatal burns, all examined by the same pathologist, between 1920 and 1942. Forty-one cases had been tanned and twenty-five of these showed definite hepatic necrosis at autopsy, while this lesion was completely absent in twenty-one untanned patients. Liver damage is chiefly coagulation necrosis of the centres of all lobules.

Are there lesions, other than those due to burning, which might alone account for death?

This question arises when there is a suspicion that fire has been used to conceal crime, and it will depend entirely upon the condition of the body, together with the attendant circumstances, whether a definite answer can be given.

It must be remembered in this connection that fissure fractures of the skull are sometimes found following exposure to fierce heat and that extra-dural hæmorrhage sometimes accompanies heat fractures. X-ray examination of charred bodies may prove of importance in detecting or eliminating the presence of a bullet or bullets.

Are the burns accidental, suicidal, or homicidal?

It may safely be asserted that by far the largest proportion of deaths from burning and scalding occurs by accident, that such deaths are rarely suicidal in this country, and that deaths by homicide are uncommon. Accidents account for a great variety of cases, as for example, old, helpless, or intoxicated persons falling into the fire, children falling into vessels of scalding water, and adults falling into vats of scalding fluids or molten metal in the course of industry. Children are prone to burning through accident befalling them in a variety of ways, including the upsetting of vessels containing boiling liquids, playing with matches or lighted material, and interfering with the fire. Buildings going on fire, bursting of boilers, explosions in mines, or of gas or gunpowder, and the occasional upsetting of paraffin lamps are also some of the general causes of accidental burning.

FIG. 79

Effects of nitric acid throwing. Severe burning of upper part of body, and loss of vision of right eye.

With regard to legislative precaution against the exposure of

children to risks of burning, the Children and Young Persons (Scotland) Act, 1937, enacts that: if any person who has attained the age of sixteen years has custody, charge, or care of any child under the age of seven years, and allows that child to be in any room containing an open fire-grate not sufficiently protected to guard against the risk of the child being burnt or scalded, without taking

FIG. 80

Fatal case of burning due to clothing having been set on fire.

reasonable precautions against that risk, and by reason thereof the child is killed, or suffers serious injury, he is liable on summary conviction to a fine not exceeding ten pounds. This provision does not affect such person's liability to be proceeded against by way of indictment (see p. 227).

Homicidal burning or scalding is not common, but from time to time deaths are caused by the wilful application of fire. It has been accomplished by throwing a lighted paraffin lamp, by saturating the clothing of the victim with inflammable liquid and setting it alight, by a violent attack with a red-hot implement, by throwing boiling water, and by other methods.

ILLUSTRATIVE CASES

An elderly woman was charged with having assaulted and caused the death of two children, aged three and four years respectively, by putting them on a fire. Post-mortem examination showed that the children had died from shock consequent upon extensive burns.

A mother was charged with having, while she was in an intoxicated condition, put her infant of four months into a scalding bath. Both legs were scalded. On the left leg, extending from the knee to the little toe, was an area devoid of epidermis, and on the right cheek, an oval-shaped vesicle $\frac{1}{2}$ inch in diameter. The cause of death was shock.

Of the many fatal cases of burning and scalding which we have seen, some were of unusual character. In one case, for example, a man fell into a cauldron of boiling maize in a distillery, and when the body was recovered it was in a condition of heat-stiffening, and had assumed the "pugilistic attitude" frequently seen in such cases. The arms were extended from the shoulders, and the forearms partially flexed. The legs also were partly flexed at the knees. Heat-stiffening occurs in bodies exposed to intense heat by burning

or immersion in hot liquids and is due to coagulation of the albuminates in the muscles. Rigor mortis does not supervene in such cases and the primary rigidity persists until the onset of putrefaction. From the position assumed by the limbs, which simulates the general attitude of a boxer, the term " pugilistic attitude " has been applied to this condition (see Fig. 81).

From an analysis of our cases we find that some of the causes of burning were, by gunpowder, by molten metal, by boiling porridge, by heated metal, by a red-hot poker, by scalding fluid, by falling into a tub of boiling water, by setting bedclothes on fire, by accidental incineration of a motor car, by fires attributable to different causes, by steam, by scalding in a railway carriage, and by accidents with lamps.

The facts of an unusual case of burning due to accident are worthy of record :—

The deceased man had been a passenger in a motor car which was involved in a collision with another car. At the time, he occupied the near rear seat. After the impact, the car, out of control, mounted the pavement, where it struck an electricity section box, before falling over on its near side. Petrol flowing from the tank at the rear of the car became ignited by sparks from the electric cables in the section box, and within a few minutes the car was ablaze. Four of the occupants, including the driver, were released, but it was found impossible to extricate the deceased, as the rescuers had to retreat on account of the intense heat. When the fire brigade had extinguished the blaze, the body of the trapped man was removed from the burnt-out car.

Post-mortem examination showed that the body was in a condition of heat-stiffening, and the pugilistic attitude of burning was well demonstrated. The entire head, face, and upper part of neck were charred. The chest and abdomen showed 3rd and 4th degree burns, and the back, together with parts of both buttocks, showed 3rd degree burns. The arms and hands were burned, the lesions varying from 2nd to 6th degree. The pubic hair was singed, and the penis and scrotum were charred. The legs showed 1st to 5th degree burns, but both feet were practically unaffected, having been protected by footwear (see Fig. 81).

The pharynx, larynx, trachea, and main bronchi showed the presence of carbonaceous material. The blood did not show the presence of carboxyhæmoglobin.

In cases where a body has been exposed to fierce heat, lesions which suggest infliction by a sharp instrument may sometimes be found. These are due to the bursting open of the tissues, and on close examination can be differentiated from incised wounds produced by a knife (see pp. 240 and 420).

Suicide by burning is rare in this country, but cases are on record of persons who have thrown themselves into baths, vats, or cauldrons of boiling fluid or molten metal.

In the East, we have seen many suicidal cases in which persons have saturated their clothing with petrol and set it on fire.

Fig. 81

The effects of burning caused by accidental incineration of a motor car. The "pugilistic attitude" is shown.

Post-mortem appearances.

While the external appearances of burning have been described, something remains to be said regarding the internal appearances. These will depend upon whether death has resulted from shock or asphyxia or whether the individual has survived the immediate effects of burning and has died from subsequent complications such as inflammation of the serous surfaces, septicæmia, pneumonia, or nephritis. Occasionally duodenal ulceration may develop following burning. In asphyxial cases, blood taken from the heart should be examined for the presence of carboxyhæmoglobin (see p. 712). The nasal passages, pharynx, larynx, trachea, and bronchi should be carefully inspected for the presence of carbon particles. When these are found, there is indication of smoke inhalation.

In delayed death from burning a toxæmic condition develops probably due to combined causes, including septic absorption, and

Fig. 82

Burning, first to third degree.

is indicated, post-mortem, by the presence of cloudy swelling and other changes in certain of the organs, including the liver. This condition has already been described (see p. 217). The changes in the blood may lead to occlusion of the small vessels, and this has been suggested as a cause of such conditions as duodenal ulcer. The post-mortem findings will therefore depend on many factors.

Burning by corrosives.

Cases which involve the throwing of corrosive fluids occasionally occur, and the chemicals often used are either acids or alkalis. These fluids are frequently thrown upon the face, with the object of destroying vision or causing facial disfigurement.

In Scotland, it is a crime for any person wilfully, maliciously, and unlawfully to throw at, or otherwise to apply to, any other person any sulphuric acid or other corrosive substance, calculated by external application to burn, injure, murder, maim, disfigure, disable, or with intent to do some other grievous bodily harm.

Should death result, the crime is a capital one, provided that under the circumstances the act committed amounted to murder.

The law of England on this subject is embraced in the Offences against the Person Act, 1861, where it is enacted that: Whosoever shall unlawfully and maliciously . . . put or lay at any place or cast or throw at or upon or otherwise apply to any person, any corrosive fluid or any destructive or explosive substance, with intent . . . to burn, maim, disfigure, or disable any person, or to do some grievous bodily harm to any person, shall, whether any bodily injury be effected or not, be guilty of felony.

The diagnosis of lesions due to burning by corrosives depends upon the following points: (1) The character of the lesions; (2) the marks of staining, due to chemical action; (3) the absence of signs produced by fire or scalding liquids; (4) the identity by analysis of the corrosive used. The lesions do not present the same variation in appearance as those by fire, or the characteristic vesication of scalding. The colour of the stains upon the skin or clothing may afford a guide to the nature of the corrosive. Nitric acid produces a yellowish stain, sulphuric acid and caustic alkalis leave reddish-brown or dirty-brown stains, while hydrochloric acid and carbolic acid cause whitish or greyish-yellow stains on the skin and mucous membrane. In some cases the colour of the stain upon the clothing may be vitiated by the colour of the fabric. Sulphuric acid, or oil of vitriol, is probably the most commonly employed acid, but other acids and alkalis are also used (see p. 702 for appropriate qualitative tests for the various corrosive fluids).

The soiling fluid may be extracted from contaminated clothing or removed from the surface of the stained skin. Removal of corrosive substances from the skin may be effected, in most cases, by the use of pledgets of cotton-wool moistened with distilled water.

ILLUSTRATIVE CASE

The following case is illustrative of serious injury produced by the use of nitric acid. The neck and chest, which were involved extensively, showed yellow discoloration of the skin, superficial corrosion, and necrotic areas.

Discrete yellow spots were scattered over the face, the corneæ were opaque, and the conjunctivæ markedly congested and œdematous. The acid had run down both arms and the front of chest on to the upper half of abdomen. Twelve days after the assault, the burned areas had sloughed and many deep sloughs were separating, chiefly from the regions of the left shoulder and side of chest. The temperature fluctuated between 102° and 104° F. Five days later all the sloughs had separated, and the temperature was normal. Vision of the right eye was lost, there being necrosis of the lower half of the upper eyelid, while the tissues of the eye were destroyed. Three months after the sloughs had separated, the burns had healed (see Fig. 79).

Spontaneous combustibility.

Present-day opinion is entirely opposed to the possibility of spontaneous combustion, for the simple reason that the human body contains such a high percentage of fluids.

Preternatural combustibility.

It is well established that the action of certain micro-organisms upon composite organic substances may generate inflammable gases.

Whether or not the subtle processes of physiological chemistry can effect changes which increase the general inflammability of the tissues of the human body is a question which is still in the region of conjecture, and, therefore, any medico-legal bearing which it may have should be disregarded.

BURNING BY X-RAYS

Repeated exposure to X-rays continued over long periods may produce lesions, both extensive and serious, which prove refractory to treatment. Radiologists and others who have been so exposed have suffered from severe dermatitis, affecting the hands, especially the region of the finger-nails, which has caused degenerative changes including warty growths. Ulceration is not infrequent in such cases, in which there is always the possibility of the development of epithelioma. The chronicity of the lesions, together with the possible risk of malignancy, has led to amputation of the fingers or portions of them in a number of cases. The nature of the lesions produced as the result of a single exposure in excessive dose depends upon the extent of over-exposure, and may vary from slight erythema to ulceration and necrosis of tissue which is difficult to heal and which may take many years to do so. When blistering results from overdosage, the skin is very friable and ulceration is a frequent advent. The difficulty in the healing of these lesions probably results from a condition of endarteritis obliterans in the smaller vessels, with a consequent diminution in the blood supply. Ulcerative lesions cause a considerable degree of pain and discomfort. Scott [12] describes a type of X-ray reaction, a "late reaction," which he explains cannot be classified as an indication of overdosage, although little is known of its etiology. In this condition the changes do not originate primarily in the skin and the reaction may not develop for months or even years following the last exposure to the rays. He states that a blue discoloration, resembling a bruise, is seen below the skin and that this gradually becomes the seat of extensive and deep sloughing; after a long delay healing takes place with a considerable degree of atrophy of the skin. He holds the view that this reaction is rare and that blame cannot be attributed to the radiologist, since there is no known method of guarding against such accidents. Certain persons are thought to be more

susceptible to X-rays than others, especially those who are fair-skinned.

In respect, therefore, that such mishaps have given rise to actions for damages against operators,[13] it is necessary that more than ordinary caution be exercised in giving an opinion respecting implied culpability or carelessness on the part of an operator; indeed, an opinion should be given only after full knowledge of all the facts of the case, and even then with much prudence.

BURNING BY ULTRA-VIOLET RAYS

The local effects of over-exposure of the skin to sunlight usually vary from erythema, mild or severe, to vesication which may be of limited or extensive character. The use of artificial sun baths has now become fairly general, but overdosage, particularly in those unaccustomed to them, may produce severe erythema, and a case of severe dermatitis following exposure has been reported by MacCormac and M'Crea.[14]

References

1. Lancet, Vol. I, 351, 1946, Jex-Blake.
2. "Lightning, Lightning Stroke, and its Treatment," Spencer. Baillière, Tindall & Cox, 1932.
3. Lancet, Vol. I, 68, 1934, Critchley.
4. Brit. Med. Jour., Vol. II, 426, 1942, Dill.
5. Jour. Industr. Hyg., Vol. XII, 31, 1930, Langworthy and Kouwenhoven.
6. Ibid., 291, Maclachlan.
7. Annual Report of the Chief Inspector of Factories, 1945. H.M. Stationery Office.
8. Ann. Surg., Vol. CVI, 111, 1937, Rosenthal.
9. Journ. Amer. Med. Ass., Vol. CXII, 2235, 1939, Blackfield and Goldman.
10. Jour. Path. and Bact., Vol. XLVIII, 493, 1939, Belt.
11. Ann. Surg., 234, 1943, Erb, Morgan, and Farmer.
12. "Radiology in Relation to Medical Jurisprudence," 1931, Scott.
13. Brit. Med. Jour., Vol. II, 1660, 1910.
14. Ibid., Vol. I, 693, 1925, MacCormac and M'Crea.

CHAPTER VIII

DEATH FROM STARVATION AND NEGLECT AND FROM COLD AND EXPOSURE. EFFECTS OF HEAT.

CASES of death from starvation occur in one or other of the following circumstances : (1) famine ; (2) entombment in pits, mines, or landslides ; (3) neglect on the part of parents or guardians ; (4) wilful withholding of food ; (5) wilful refusal to take food.

The law takes cognisance of those cases which are due to wilful neglect and unintentional neglect. In addition to wilful starvation being an offence at common law, it falls, especially in the case of children, under statute law, which shows close similarity in both England and Scotland. The Children and Young Persons (Scotland) Act, 1937, consolidates in its application to Scotland certain enactments relating to persons under the age of eighteen years. It contains provisions which are of importance in connection with the prevention of criminal neglect and starvation.

Part I of the Act, which deals with Child Life Protection, contains certain provisions for preventing objectionable traffic in child life and for the protection of children under nine years taken for reward.

If a person undertakes for reward the nursing and maintenance of a child under nine years, apart from its parents or having no parents, he must give notice in writing thereof to the local authority, and should the child die or be removed from his care he must within twenty-four hours thereof give notice in writing of the death or removal to the local authority and to the person from whom the child was received. In the case of death he must within a like period give similar notice to the Procurator-Fiscal of the district within which the body lies. On receipt of such notice the Procurator-Fiscal must hold an inquiry into the cause of death, unless there is produced to him a certificate by a duly qualified medical practitioner certifying that he personally attended the child during its last illness and specifying the cause of death, and the Procurator-Fiscal is satisfied that there is no ground for holding an inquiry. Failure to give any of these notices is an offence.

Power is also given to the local authority to fix the number of children under nine years who may be kept in any dwelling, and to order the removal of any child kept in unsuitable premises or by unsuitable persons.

Part II enacts certain provisions for the prevention of cruelty to,

and exposure to moral and physical danger of, children and young persons. It makes it an offence for a person who has attained the age of sixteen and has the custody, charge, or care of a child or young person under that age wilfully to assault, ill-treat, neglect, abandon, or expose him in a manner likely to cause him unnecessary suffering or injury to his health, or to fail to provide adequate food, clothing, medical aid or lodging for him, or to take steps to procure such under the Acts relating to the relief of the poor where he is unable to provide this himself.

If a person goes to bed under the influence of drink and a child under three years of age in bed with him is suffocated, such person is deemed to have neglected the child in a manner likely to cause injury to his health.

It is an offence for a person who has attained the age of sixteen years and who has the custody of any child under the age of seven years, without taking reasonable precautions, to allow the child to be exposed to the risk of burning or scalding whereby he is killed or seriously injured. This provision does not affect such person's liability to be proceeded against by way of indictment (see p. 219).

The Adoption Act, 1939, which is applicable to England, protects both the adopted child and the adopter. By this Act, payment for adoption is no longer permitted and no body of persons other than a registered adoption society, or local authority, may arrange for the adoption of a child. The society or local authority investigates the full particulars regarding the proposed adoption. A medical report on the state of the health of the child proposed for adoption must be obtained.

Wilful neglect.

Wilful neglect of children is accompanied by some measure of imperfect nutrition, but the degree of malnutrition usually stops short of starvation. It may be said that wilful starvation of a child or young person to the extent of causing death is comparatively rare. The Children and Young Persons Act, however, definitely lays down what derelictions of duty on the part of parents or guardians come within the category of neglect or cruelty to children. The Act provides that a parent or other person legally liable to maintain a child or young person shall be deemed to have neglected him in a manner likely to cause injury to his health if he fails to provide adequate food, clothing, medical aid, or lodging for the child or young person, or if, being unable otherwise to provide such food, clothing, medical aid, or lodging, he fails to take steps to procure the same to be provided under the Acts relating to the relief of the poor.

In looking for the evidences of neglect, the medical examiner must take cognisance of the following points :—

The condition of the child with respect to cleanliness of person and of clothing ;

The condition of the child with respect to bodily nutrition.

Dirty, unkempt, imperfectly clad children offer clear indications of the absence of proper parental care, and a filthy condition of skin and clothing, a verminous state of body and hair, with marks of scratching or ulceration, or even disease of the skin, assist to complete the picture. These conditions, when found, may be taken as sufficient evidence of general neglect.

Special care, however, must be exercised in the consideration of the state of nourishment of children. Imperfect nutrition, as

FIG. 83

The bodies of two aged persons who were found dead, the result of starvation. (By courtesy of Dr Robert Richards, Aberdeen University.)

shown by relation of growth to age, and of height to weight, may arise from different conditions, such as inherited or acquired disease, or feeding insufficient or unsuitable as to quality and kind of food.

Careful investigation should be made for the existence of disease, or of evidence of causes of malnutrition whenever serious disproportion between the actual and average age, height, and weight is discovered in any case, or group of cases. In order, therefore, to establish evidence of imperfect or inappropriate nourishment, as to quantity, quality, or kind of food, it is not only necessary to eliminate diseases or conditions causative of malnutrition, but to review such evidence as there may be of the feeding. This is especially necessary in cases in which accused persons are charged with homicidal starvation, for the lines of defence most usually adopted are either the existence of disease or causes of malassimilation or malnutrition. In post-mortem dissection, therefore, all evidences of disease, if present, must be noted with relation to their effect upon the pro-

duction of emaciation. In the absence of disease, there would be grounds for inferring that death was due to inadequate nourishment. It must not be overlooked that diminished nutrition may be a determining factor in the causation of certain diseases, and the difficult question of cause and effect may arise occasionally.

Of the diseases which present externally apparent signs of starvation, tuberculosis, malignant disease, and diabetes are probably the most common.

In all cases, and especially those of children, the examiner should make inquiry regarding the mental condition of the person.

Signs of starvation.

The following clinical pictures are based on observations of prisoners-of-war returning to this country during the spring of 1945, of Dutch persons shortly after liberation, and of the inhabitants of Belsen concentration camp. The author acknowledges reference to the monograph of Dr Janet Vaughan.[1] Cases of starvation may be divided into two groups, namely, dry cases and wet cases. In the first of these groups there was extreme emaciation, the body weight deviating from the normal by 39 to 50 per cent. In severe cases, the blood-pressure was not obtainable and the pulse was impalpable. Gross cyanosis was evident and the feet showed slight œdema. In the second group, œdema involved the face, arms, legs, and feet in marked degree. Frequently there were ascites and pleural effusions. Pyrexia was common and watery diarrhœa was the rule. In Belsen, the men were eunuchoid and the women acquired male characters. Anæmia was usually present.

FIG. 84

A subject showing general effects of advanced malnutrition.

Burger, Sanstead, and Drummond [2] referring to starvation in Western Holland state that three types of death were observed, namely, sudden, unexpected, or slow. In most cases, broncho-pneumonia affecting both lower lobes was found at autopsy.

In concentration camps in Holland, living skeletons were seen in whom the weight of the bones, usually 15 per cent. of an adult's body weight, accounted for 50 per cent. of the four to five stones that these people weighed.

It has been shown that the limited amount of protein, which would suffice if the calories are adequate, is devoted to the production of energy. The plasma thus becomes deficient in proteins, its osmotic pressure falls, and the fluid leaks into the tissues. Children do not develop typical œdema, but are pale and doughy-looking.

Post-mortem appearances.

The body shows an absence of fat and the skeletal muscles gross wasting. The organs are frequently pale, and the heart, liver, kidneys, and spleen are atrophied. The omental and mesenteric

FIG. 85
Emaciation.

FIG. 86
Same case as in Fig. 85.

tissues are lacking in fat and are atrophied. The intestines are also atrophied and a non-specific ulceration may affect the large bowel. The gall-bladder usually contains dark-coloured bile, gall-stones may be present, and the urinary bladder is practically empty.

An adult may survive complete privation of food and fluid for a period of from seven to ten days. If water is taken, this period will be greatly lengthened and the person may survive for many days. Cases have been reported in which survival continued for as long as fifty-eight and sixty-one days. Special regard must be paid to the age and state of the particular person. Young subjects usually succumb more readily than adults, and healthy young adults will probably survive much older persons.

Black [3] has carried out experiments on human subjects who were given an adequate diet and salt intake, but were deprived as much as possible of their water intake. The body weight began to fall as soon as the subject stopped taking water and the loss of weight was greater on the first than on subsequent days. In three to four days, the weight loss amounted to about 6 per cent. of the body weight, corresponding to 10 per cent. of the body water. Despite this considerable loss, general symptoms were not severe, although huskiness of the throat and inability to swallow caused considerable discomfort. Black gives his estimate of the amounts of fluid in the different compartments in a normal man weighing 70 kilograms as follows :—

	Total Body Fluid. Percentage of Body Weight.	Litres.
	65	46
Plasma . . .	5	3
Interstitial fluid .	20	14
Intracellular fluid .	40	29
TOTAL .	65	46

DEATH FROM COLD AND EXPOSURE

Death from cold and exposure in this country is most infrequent. In such cases, the primary cause of death is attributed to a decreased dissociation of oxygen from the hæmoglobin to the tissues which have a diminished capacity for utilising it. The liability to this form of death is less in robust persons than in the case of newly-born infants, young children, and aged or debilitated subjects. The factor of intoxication plays an important part in many cases, since in drunkenness the body temperature readily becomes subnormal. The symptom of drowsiness, which is often experienced by persons who have been exposed for considerable periods to rigorous weather, is probably attributable to the effects of a progressive oxygen starvation of the brain.

Post-mortem appearances.

External.

Irregularly-shaped red patches are present over limited areas of the exposed surfaces of the body (see p. 170). The situation of these is determined by the areas of the skin exposed to the air, since they are not found upon covered portions of the body. They must not be confused with hypostasis which is found only on the dependent parts, or with the cherry-red coloration of the skin in carbon monoxide poisoning, which is found equally upon parts exposed to, and covered from, the air. The coloured areas, due to cold and exposure, are usually well marked on the cheeks and on the mucous membrane of the lips. These areas may be attributed to the marked degree of oxygenation of the hæmoglobin in the blood within the capillary vessels which has occurred directly through the skin and mucous membrane (see Fig. 48).

Fig. 87

Note bright red colour of skin.

Kreyberg [4] states that the underlying cause of all types of pathological reaction to cold is an acute aseptic inflammation.

Frost-bite, immersion feet, and trench feet for practical purposes may be regarded as synonymous terms. Ungley [5] who has made an extensive study of immersion hands and feet is of the opinion that the essential cause of the condition is exposure of the limbs to cold, insufficient to freeze the tissues, and that immersion has no specific action apart from its effect in maintaining the low temperature of the parts. The hands, though not immersed, may be damaged as much as the immersed foot. During the exposure period the skin at first becomes red, later pale yellow, then mottled, and later, blue or black. Even if the whole foot is black, tissue loss may be confined to the ends of the toes. Ungley found that men over forty and those under seventeen died from the effects of exposure sooner than those of intermediate age.

In true frost-bite there is a condition of arteritis obliterans not found in immersion foot.

Internal.

The blood generally has a bright-red colour in bodies seen within a comparatively short time after death, due to the lack of dissociation of oxygen from the hæmoglobin by the action of the cold temperature to which the body has been exposed.

The internal organs and the large vessels show a marked degree of congestion. The brain and meningeal vessels are also congested.

It will, therefore, be seen that the post-mortem signs are not distinctive in character, and in arriving at a conclusion in any given case that death was due to exposure and cold, it is essential that all the circumstances must be investigated with care. In view of the similarity of the coloured marks on the skin to those in carbon monoxide poisoning, a spectroscopic examination of the blood should be made when there is any element of doubt (see p. 335).

Sometimes evidence of freezing will be found upon the body. We have seen two cases of death from cold and exposure in which there was audible crackling on flexing the knee joints, apparently due to the breaking down of frozen synovial fluid. It is not inconceivable that the body of a murdered person might have been carried from a house and laid in the open in order to divert suspicion. Should the body have become frozen, some indication of the time of exposure, following death, may be afforded by observing whether or not rigor mortis supervenes after thawing of the body. In such a case, the place where the body was found should be carefully examined, especially when wounds are present upon the body, before the balance of evidence is finally adjusted in the determination of the cause of death (see p. 282).

Gardener [6] quotes a personal communication from Rabinowitch, McGill University, regarding several deaths which the latter ascribes to the action of intense cold on the carotid sinus. In winter, when the temperature was many degrees below zero, babies that had been placed out-of-doors to sleep were sometimes found dead with nothing to account for death at the post-mortem examination. In these cases, it was discovered that there was a gap between the head covering and the blankets. Experimentally, Rabinowitch has been able to produce sudden death on laboratory animals by allowing a stream of cold air to play on their necks over the position of the carotid sinus. In the children and animals, post-mortem examination showed similar findings, namely, congestion of the organs and no diminution of the blood volume in the right side of the heart or in the veins leading to it.

THE EFFECTS OF HEAT

Heat exhaustion and heat stroke.
Heat cramps.

Heat exhaustion and heat stroke.

Morton [7] has suggested that there is no hard-and-fast line between severe heat exhaustion and heat stroke and, since we are in complete agreement with this view, these conditions will be described under one heading. He suggests that it is advisable to adopt only one term, heat hyperpyrexia. This condition is prone to develop in those who have to work in abnormally high temperatures, for example, stokers and engineers and, contrary to past belief, neither is exposure to sunlight nor the presence of humidity in the

atmosphere necessary. Contrary to the view that ultra-violet and violet rays play an important part, it is now held that the infra-red and yellow rays are responsible for the condition. Obese subjects, debilitated persons, and chronic alcoholics are prone to heat hyperpyrexia. Morton believes that there is a prodromal stage of from one to three days' duration, during which symptoms of exhaustion accompanied by a reduced excretion of urine are shown, and that collapse may supervene, when the condition suggests profound shock.

When heat exhaustion is associated with well-marked acidosis, the condition may merge into one of heat hyperpyrexia. Heat hyperpyrexia may become established without the prodromal stage of heat exhaustion, and the condition of heat exhaustion may not merge into one of hyperpyrexia.

The symptoms and signs most frequently associated with exhaustion include those of collapse, in varying degree, and those of depression of the nervous system. Nausea, vomiting, diminished urinary output, subnormal temperature, or elevation of the temperature seldom exceeding 101° F., muscular cramps, and mental depression or anxiety are frequent manifestations. The clinical picture of hyperpyrexia becomes established when the temperature reaches about 107° to 110° F. Mental excitement, headache, lack of co-ordination, delirium, convulsions, and coma form the picture.

Lee [8] states "that hyperpyrexia is reached when the mean temperature of the body is such that the continued life of some vital tissue is endangered. In practice, it is the nervous system which is vitally affected. Should the magnitude of the external heat be too great, or the resources of the body for promoting heat loss be insufficient, the body temperature will rise to the critical point before thermal equilibrium between the body and its environment is re-established."

Ladell, Waterlow, and Hudson [9] describe the effects of heat due to desert climate, as ascertained from a hundred and nine cases. Twelve hyperpyrexial cases had a rectal temperature of 107° or more, but in none of them was a history of excessive exposure to sun, unusually hard physical work, or lack of sleep, elicited. In only one case was there an excessive intake of alcohol. In all cases a copious amount of urine was passed, and the skin all over the body was hot and dry. There was also partial or complete loss of consciousness. The heat exhaustion type of illness began when the daily maximum temperature was above 115° F. for three to four days on end and the clinical picture included giddiness, anorexia, headache, and constipation in varying combinations. The predominant features were vomiting and cramps in the legs and thighs followed by arms, abdomen, feet, and jaw. There was profuse sweating and the rectal temperature was 100·6° F. on an average. Another type of illness manifested itself, during the second half of the summer, by defective sweating, polyuria, and a moderately severe prickly heat. In these cases, the blood and plasma chlorides were diminished despite the presence of chlorides in the urine.

Post-mortem appearances.

These are atypical as a rule. In some cases, petechial haemorrhages may be found in the brain and heart.

Treatment of heat exhaustion.

Treat as for shock. An intravenous administration of 2 per cent. sodium bicarbonate in saline will prove rapidly beneficial. In less severe cases, alkaline solutions by the mouth should be administered. In all cases, a purgative should be given at the outset and regular bowel action maintained.

Treatment of heat hyperpyrexia.

The body temperature should be reduced by cold sponging, which must be repeated at frequent intervals until the temperature is lowered to about 102° F. It should be maintained at this level for a period and then stabilised at about 99° F. The patient should be kept quiet and a prolonged period of convalescence should be insisted upon.

According to Ladell, Waterlow, and Hudson, the treatment should consist of liberal administration of fluids by the mouth, about 16 pints daily. In certain cases, a salt intake orally, in daily doses of 1 ounce, is advocated. When marked dehydration is present, intravenous saline should form part of the treatment. Dehydration is secondary to lack of salt rather than lack of water and both the blood and urine show a gross salt deficiency.

Heat cramps.

Various terms, including "miner's cramp," and "stoker's cramp," have been applied to this condition, which is characterised by painful cramps or tonic spasms affecting the muscles of the body. The condition occurs in those who work in hot atmospheres and is caused by the excretion of large quantities of sodium chloride through excessive loss of perspiration.

Treatment.

The condition is quickly relieved by the administration of sodium chloride by the mouth, a level teaspoonful of salt dissolved in a quart of water, or rectally, a 1 per cent. solution of sodium chloride. Sodium chloride taken in tablet form is a valuable and efficient prophylactic measure. For the primary relief of excessive pain sedatives may be given.

References

1. Medical Annual, 326, 1946, Vaughan.
2. Lancet, Vol. II, 282, 1945, Burger, Sanstead, and Drummond.
3. Ibid., Vol. I, 298, 1945, Black.
4. Ibid., Vol. I, 338, 1946, Kreyberg.
5. Ibid., Vol. I, 681, 1943, Ungley.
6. Clinical Journal, Vol. LXXII, No. 2, 66, 1943, Gardener.
7. Proc. Roy. Soc. Med., Vol. XXV (2), 1261, 1931–32, Morton.
8. Trans. Soc. Trop. Med., Vol. XXIX, 11, 1935–36, Lee.
9. Lancet, Vol. II, 491, 1944, Ladell, Waterlow, and Hudson.

CHAPTER IX

THE MEDICO-LEGAL ASPECTS OF WOUNDS

ALL lesions of the body, external or internal, caused by the application of violence may be designated as wounds.

A wound is therefore a solution of continuity of any of the tissues of the body caused by injury.

By the Offences Against the Person Act, 1861, which applies to England, provision is made for the punishment of those who inflict injury upon others. The sections of special importance in regard to wounding are :—

Wounding or causing any grievous bodily harm to any person by any means whatsoever with intent to murder is a felony.

Wounding unlawfully and maliciously by any means whatsoever or causing any grievous bodily harm to any person or shooting at any person or attempting to discharge any kind of loaded arms at any person with intent in any of these cases to maim, disfigure, or disable any person, or doing some other grievous bodily harm to any person is a felony.

Wounding unlawfully and maliciously or inflicting any grievous bodily harm upon any person either with or without any weapon or instrument is a misdemenour.

The terms "bodily harm," "injury," and "grievous bodily harm" are not defined in the Act.

In Scotland, there is no Act corresponding to the Offences Against the Person Act, but such offences are criminal at common law.

WOUNDS IN RELATION TO DANGER TO LIFE

In order to convey some meaning to the minds of others with respect to the relative gravity of wounds, certain descriptive terms have been applied to them, and medical practitioners should have clear and definite conceptions of what are meant by these terms.

A slight wound means one in which the lesion is neither extensive nor serious, which heals rapidly, and which leaves no deformity behind it. A dangerous wound is one that is either extensive or serious with relation to the organ or part wounded, which presents surgical difficulty in its cure, but which is not attended usually by fatal consequences. A mortal wound is one which almost immediately after its causation or within a short time thereafter causes death by interfering with the function of a vital organ, or with the general functions of the body.

Wounds involving the following structures are the cause of many deaths within short periods after their infliction :—

The heart and large blood-vessels ;
The brain and upper part of the spinal cord ;
The lungs ;
The stomach, liver, spleen, and intestines.

From time to time unexpected recoveries occur, for example, Singleton [1] records a case of wounding which involved the inter-ventricular septum of the heart with survival for forty-five days after operation. He also describes further cases in which recovery followed the suture of a wound in the wall of the left ventricle, wounding of the pericardium, without operation, and the lodgment of a bullet in the right ventricle, the patient remaining well after a period of two years.

In another of his cases, a bullet had perforated the wall of the left ventricle, and without operation the patient lived for eleven days. Many other cases could be cited to illustrate recoveries which were not anticipated.

Frequently, we have seen homicidal deaths which were caused either by penetration of one of the cavities of the heart, the aorta, the pulmonary artery, the common carotid artery, the jugular vein, or the femoral vein. In these cases death followed almost immediately.

With regard to wounds of the abdominal viscera from applied violence, serious wounding may be present without any visible external mark of violence on the abdominal wall.

A wound is dangerous to life only when the danger is imminent. The term should not be employed to designate a wound which, originally simple in character, becomes dangerous from unexpected complication such as erysipelas, or septicæmia, even although the wounded person may die as the result of the complication. Grievous bodily harm is inflicted when the injury causes some measure of pain or inconvenience to the assaulted or injured person, and so affects the health of that person.

WOUNDS IN RELATION TO CULPABILITY

Homicide is the killing of a self-existent human being by another human being. The destruction of an unborn child or a partially born child is not homicide (see p. 416).

The death must not be too remote from the injury, otherwise the presumption is in favour of another cause of death.

Justifiable homicide is the term applied to homicide which is justified by the circumstances which led to the killing of a person, for example :—

A woman who kills a person who attempts to rape her ;

A person who kills another in self-defence, provided that the force or means used is not more than is necessary for the purpose of self-defence, that there is reasonable apprehension that the attacker intends to inflict serious injury or to kill, and that there is no opportunity for the person attacked to apply to the public authorities for protection or to make his escape.

To constitute the crime of murder, there must be wilful and malicious intent to kill, or a wicked recklessness as to the consequences. In Scotland, the term culpable homicide is applied to the act of one who kills another while doing an unlawful act, or by his gross and wicked negligence, without any wilful and malicious intent to kill, or by a wicked recklessness as to the consequences.

There are three groups into which culpable homicide may be classified :—

1. Where there is intent to kill, and the homicide is neither murder nor justifiable homicide, for example, when there is gross provocation.
2. Where there is no intent to kill, but death results from unlawful conduct by the person responsible.
3. Where homicide is caused by negligence or rashness in the performance of a lawful act, for example, driving a motor car.

In England, the analogous term for this crime is manslaughter.

In England, suicide, or self-murder, is a crime. In the event of the act being unsuccessful, it is punishable. If two persons agree to commit suicide together, and one of them recovers, the survivor may be charged with the murder of the other. In Scotland, the general practice has been to limit proceedings to those cases in which the attempt constitutes a breach of the public peace.

In the crime of murder, the accused must accept the risk of the state of health, age, and sex of the deceased when the injury was inflicted, and he is also responsible for the result of the treatment of the injured person. In England, if the injured person survives the injury for a year and a day, and death supervenes thereafter, the crime committed ceases to be one of murder.[2]

When skilful surgical operative procedure is required, following the infliction of a wound, and after it the injured person dies, the person who caused the wound is chargeable either with the crime of murder, or culpable homicide, or manslaughter. In such circumstances, however, it would be competent for counsel on behalf of the accused to lead evidence to prove that the treatment was not skilfully applied. If the person who applied the treatment is a registered medical practitioner and if it has been applied for the purpose of cure and in good faith, the assaulted person would be considered to have been skilfully treated, unless it is proved that the original injury was not in itself dangerous to life, or that improper treatment caused death.

Remote causes of death in homicidal cases.

In the case of R. v. Pym,[3] Mr Justice Erle ruled that where a wound is inflicted which, in the judgment of competent medical advisers, is dangerous, and the treatment which they bona fide adopt is the immediate cause of death, the party who inflicted the wound is criminally responsible. In the case of R. v. Holland,[4] Mr Justice Maule ruled that it made no difference whether the wound was in its own nature instantly mortal or whether it became the cause of death by reason of the deceased not having adopted the best mode of treatment. The real question was whether, in the end, the wound was the cause of death.

In November 1938, at Denbigh Assizes, during the trial of a

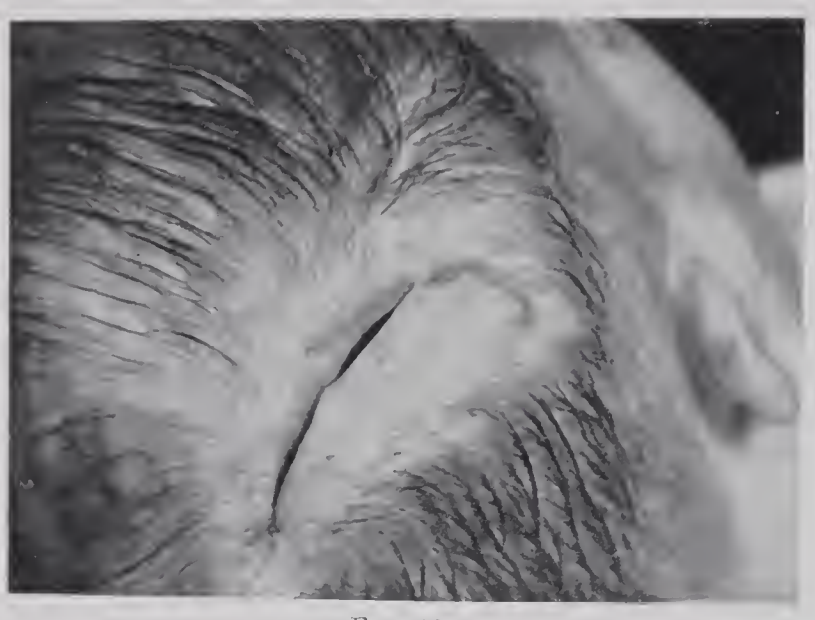


FIG. 88

Incised wound of scalp. Note curved abrasion close to anterior angle of wound.

man on a charge of manslaughter, it was stated that the death of the deceased was not the result of the injury inflicted by the accused, but on account of the prolonged period of anæsthesia resulting from the surgeon's stool having slipped when the located bullet had been about to be removed. This accident had caused delay, and when the operation had been resumed, the location of the bullet had changed. The patient died under the influence of the anæsthetic. Post-mortem examination showed the presence of an enlarged thymus gland. Medical evidence was led to the effect that if the surgeon had not sustained this accident, the bullet would have been removed and recovery would have resulted. Death was due to chloroform poisoning. The accused was found not guilty.[5]

CHARACTERS OF WOUNDS

In cases of wounding, it is important that the character of the wound, in relation to the class of weapon by which it was produced,

should be properly designated. For this purpose we classify wounds as follows :—

Incised wounds.
Lacerated wounds.
Contusions or bruises.
Abrasions or scratches.
Firearm wounds.

Incised wounds.

An incised wound may be defined as a solution of continuity without loss of substance. Such a wound is produced by a sharp-edged instrument, such as a knife, scissors, glass, the sharp edge of a metal vessel, or earthenware, among others. Such instruments may be used either to cut or stab.

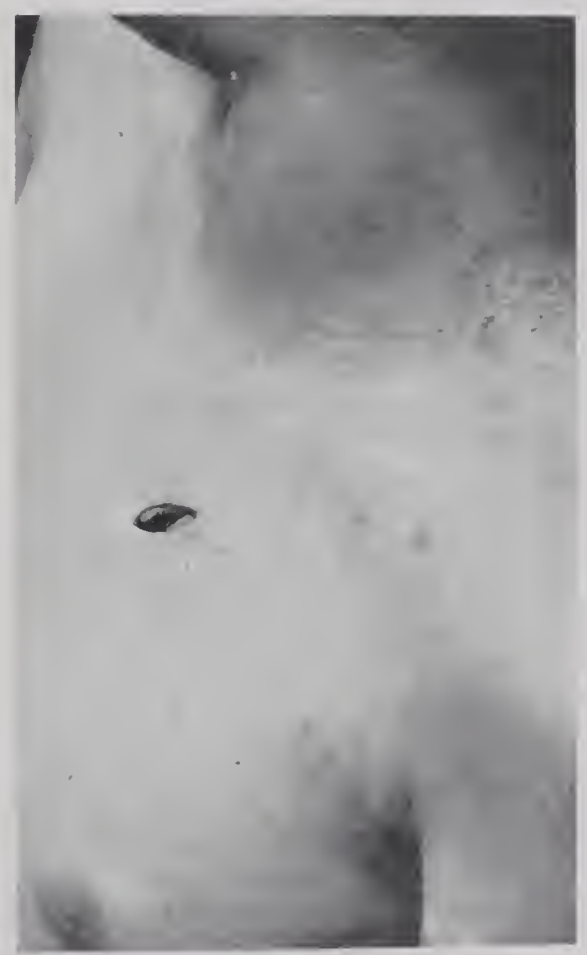


Fig. 89
Stab wound of chest.

The form of an incised wound depends upon the method of using the instrument. When it is used as in stabbing, the form of the wound is fusiform or spindle-shaped, due to the greater gaping of the tissues in the central part of the wound, but when it is used for cutting, the wound will show a more or less linear contour. The degree of gaping or retraction depends upon the amount of elasticity of the tissues severed, and upon the direction of the wound. A wound made in the direction of the muscle fibres will gape less than one made at right angles to them.

The edges of an incised wound are clean-cut, regular, and well-defined, begin abruptly, show maximum retraction in the centre, and terminate gradually. An incised wound usually causes copious bleeding. With relation to the class of weapon which produces stab wounds, the wounds are broader than the thickness of the blade which causes them, and their length is usually somewhat less than the breadth of the instrument. This is caused by gaping of the edges with a resultant slight decrease in the length of the wound. When an incised wound has passed through a limb, the wound of exit is likely to be smaller than that of entrance, because of the common

tapering character of instruments capable of producing wounds of such depth. It may happen, however, that by reason of uniformity of breadth of blade, exit and entrance wounds are practically indistinguishable in size.

Under certain circumstances, and in certain situations on the body, wounds produced by a blunt instrument may simulate the appearances of an incised wound. These wounds are usually found over bone which is thinly covered with tissue, in the regions of the head, forehead, eyebrow, cheek, and lower jaw, among others. When such a wound exposes hair-bulbs at its edges, it is possible by examining these carefully to decide whether they have been cut or crushed and thus establish whether the wound was caused by a sharp or blunt instrument. As a rule, especially in the living subject, a wound produced by a blunt instrument will disclose some degree of bruising and swelling of the edges and the deeper tissues will be less cleanly severed than when divided by a sharp-cutting instrument.

Lacerated wounds.

These wounds are caused by forcible contact with blunt instruments, and their edges are irregular, with a varying degree of tearing.

FIG. 90

Homicidal injuries. Note lacerated wounds on head and right hand. The wrists are tied together.

As a rule, there is an absence of correspondence between their shape and that of the instrument which produced them. If the violence has not severed the tissues completely, on separation of the edges, small irregular bridges of connective tissue will probably be seen stretching across the gap. Oblique impact with a blunt instrument

may produce a flap-like tear. Lacerated wounds are usually accompanied by a varying degree of contusion at the edges, and when death has not been immediate, the margins frequently show a degree of swelling. The extent of contusion and swelling found will depend upon the degree and incidence of the concussive force, and the resistance of the tissues. When the site of injury involves tissue overlying bone, as on the scalp and face, bruising may be marked. Lacerated wounds may or may not bleed much. This depends upon whether the blood-vessels are wholly or only partially severed; in the latter case, because of the inability of the vessels to retract, bleeding may be profuse, but in the former, the severed ends may retract and seal with clot and, consequently, there may be relatively little hæmorrhage. Lacerated wounds usually show some loss of substance, the process of healing is slow, and an extensive scar

FIG. 91
Multiple lacerated wounds caused by kicking.

FIG. 92
Multiple lacerated wounds of the face due to kicking.

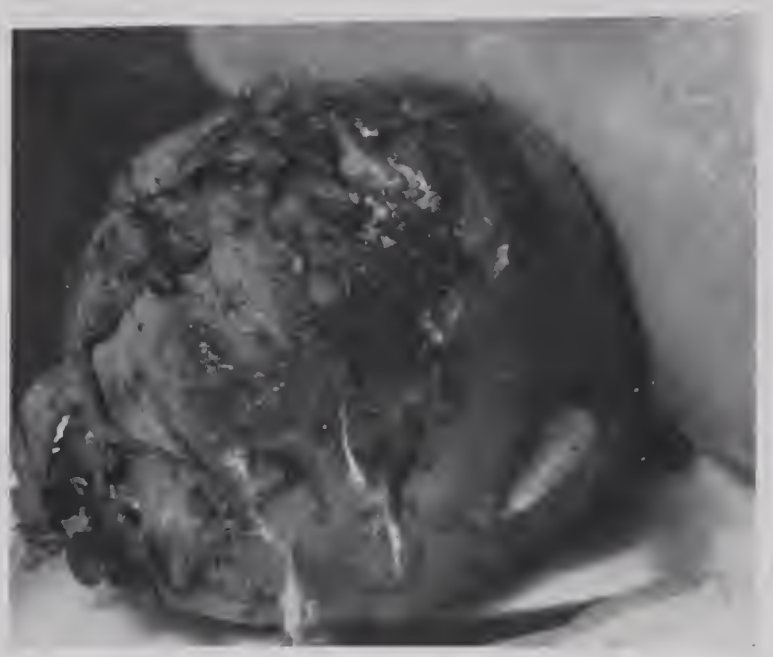


FIG. 93

Multiple lacerated wounds on the back of the scalp produced homicidally.

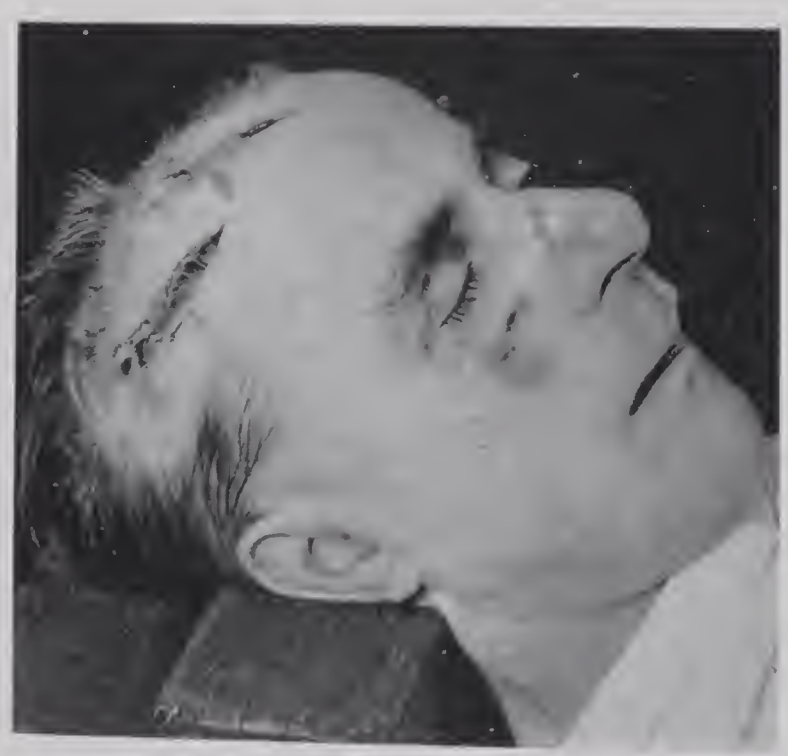


FIG. 94

Multiple lacerated wounds of the scalp produced by two blows with a shovel. Contusions are present on the face.

may result. It must not be forgotten that in certain instances the edges of a lacerated wound may appear quite regular when examined by the naked eye, and the use of a magnifying glass may be necessary to disclose the existing irregularities.

FIG. 95

Transfixation by a spiked railing. The boy fell from a window on the first floor and the metal rod had to be severed at its base to permit the removal of the patient to hospital. He survived the injury for a period of fourteen hours.

Incised and lacerated wounds may puncture the tissues, penetrate a cavity of the body, or perforate or transfix a limb (see Fig. 95). More usually such wounds are incised, but comparatively blunt-pointed, thin weapons may also produce them. An element of bruising of the edges may be present, depending upon the nature of the weapon used. A punctured wound is one that pierces the tissues. The term "penetrating" should be used to designate a wound which, passing through the tissues, enters a cavity of the body, such as the thorax or abdomen. A perforating wound is one which transfixes the tissues, for example, by passing through a limb. The depth of such wounds is greater than the length or breadth. To indicate the general character of the instrument which inflicted such wounds, the terms "incised" or "lacerated" should be included in the designation of the wounds, for example, punctured-incised, punctured-lacerated, perforating-incised, and penetrating-lacerated.

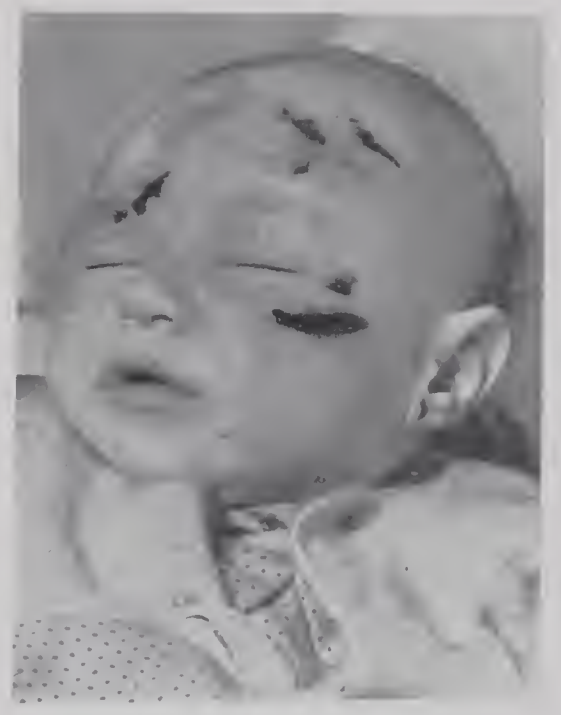


FIG. 96

Lacerated wounds of the head and face caused by repeated blows against the wall of a room.

FIG. 97

Multiple lacerated wounds of the scalp.

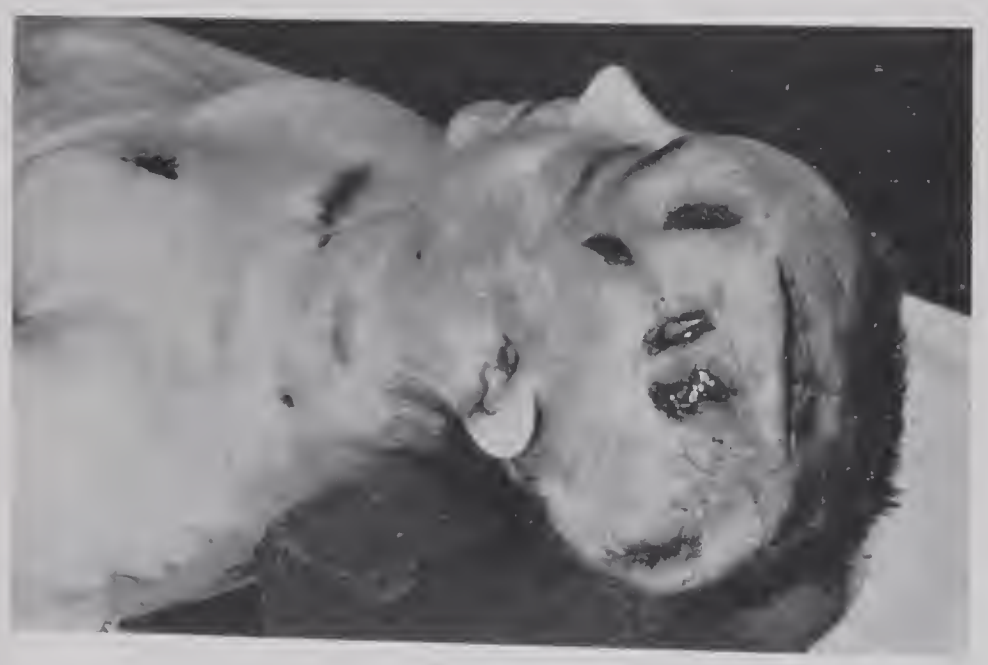


FIG. 98

Multiple lacerated wounds of the head due to blows by an iron bar.

Contusions or bruises.

Some degree of contusion is frequently found in or around the edges of lacerated wounds, and firearm wounds. The term contused wound should be abolished, since wounds are either caused by sharp or blunt instruments. If the edges of an incised or a lacerated wound show contusion, the fact ought to be noted, but the nature of the wound will determine the group to which it belongs, and indicate the class of weapon which has produced it. The term "incised" ought to be used only to signify a wound caused by a cutting instrument, and the term "lacerated" to a wound caused

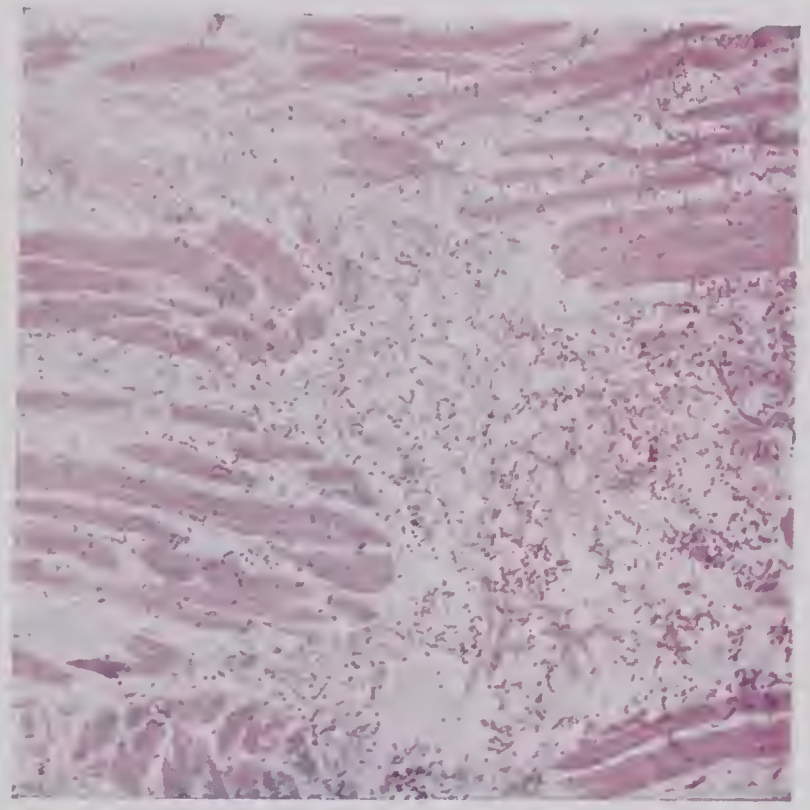


FIG. 99

Bruising. Extravasation of blood and infiltration of the muscular tissue by corpuscles. ×65.

by a blunt instrument. The element of contusion, when present, should be treated only as an incidental fact, suggestive of the probable amount of violence used, and the likely type of weapon employed with relation to the injured part.

A contusion may be defined as a breach of continuity of the structure of the true skin or subjacent tissues without loss of external continuity, caused by the application of a blunt instrument. As the result of the injury, blood extravasates from the damaged vessels and infiltrates the surrounding tissues, thus giving rise to the condition sometimes termed ecchymosis. Contusions or bruises vary in size from a small discoloration to an extensive swelling, as in hæmatoma. The extent of rupture of the subcutaneous vessels depends, under normal circumstances, upon two

principal factors, namely, the severity of the concussive force and the vascularity of the part struck. The extent of a bruise may increase considerably after infliction, due to continued extravasation of blood and infiltration of the tissues. This may also be seen after death as the result of the diffusion of blood pigment. When bruising is deep-seated, the surface appearance may prove to be misleading, since the colour change may be slight in comparison with the amount of blood which has collected in the deeper tissues. Lax tissue frequently bruises more readily than firm tissue, for example, the eyelid as opposed to the palm of the hand. The time of onset of discoloration varies in different circumstances, and the colour depends upon the amount of blood extravasated together with its distance from the surface of the skin. After bruising has commenced, the bruising tends to deepen in colour until the blood ceases to be effused. The part shows some swelling. Later, the blood separates into serum and coagulum, and as the serum is absorbed the swelling becomes reduced. Gradually the clot also becomes absorbed, and during this process the bruise undergoes changes in colour—from violet to blue, from blue to green, from green to yellow, until, at last, the skin assumes its normal hue. Hæmoglobin in an enclosed space or cavity is acted upon by tissue enzymes. These are responsible for the formation of hæmatoidin which produces a chocolate colour. The infiltrated blood cells, through the action of histiocytes, ultimately produce bilirubin which imparts a yellow colour to the bruised area.

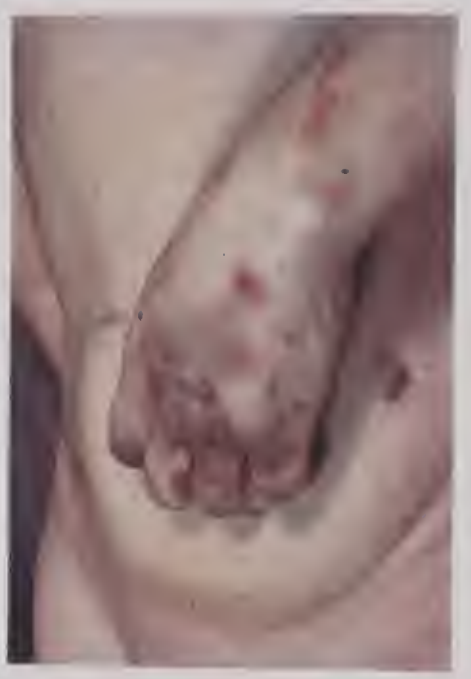


Fig. 100
Bruising.

Only in a very general way, and after consideration of all the circumstances, can the period at which the original violence was applied be inferred from the colour-tone of the contusion. Usually in a bruise of average size the dark-blue colour appears about the third day, the greenish, from the fifth to the seventh day, the yellowish, from the eighth to the tenth day, and the normal tint will probably be restored to the part between the thirteenth and eighteenth day.

When bruising is extensive and deep-seated, the colour takes a longer time to appear externally. It should also be remembered that the part where the external discoloration appears does not in the case of deep-seated extravasations necessarily indicate the exact point of application of the violence. Owing to the disposition of the planes of areolar tissue and muscles in the limbs and other

parts of the body, the discoloration, due to infiltration of blood, may show itself either above or below the part struck.

Severe internal injury may result from the forcible application of a blunt instrument without any apparent surface bruising.

In scorbutic, purpuric, and hæmophilic subjects the amount of discoloration present in bruising may be out of all proportion to the amount of violence used, and if the presence of such a factor is not recognised, very erroneous opinions may be formed.

Under certain circumstances, ecchymosis may result from causes other than application of external violence. In cases of whooping-cough, on account of the explosive nature of the cough, ecchymosis is quite commonly found in the conjunctivæ, and, less frequently, in the face and neck. When through embarrassment of breathing there is increased blood-pressure, small and spontaneous extravasations of blood may result, as, for example, in asphyxial cases.

Ante-mortem and post-mortem bruises.

The signs which are indicative of ante-mortem production of bruises are swelling of the tissues, discoloration of the skin, extravasation of blood into the true skin and subcutaneous tissues, with infiltration. When a bruise is well developed, an examiner is justified in assuming the view that it was produced during life. Nevertheless, for medico-legal purposes, a microscopical examination should be made to verify the presence of infiltrated blood. Since infiltration is possible only while the heart is beating, this sign is conclusive that the injury was produced during life (see Fig. 99). While molecular life remains in the tissues, considerable violence applied to a dead body with a blunt instrument will produce a slight degree of blood extravasation, but never to the same extent as during life, and infiltration of the tissues will be absent.

Suspected areas of bruising should always be incised to differentiate them from colour marks due to hypostasis, since both conditions may coexist in the same region of the body. In bruising, extravasated blood is present, but in hypostasis the severed small vessels are filled with blood and extravasation is absent.

Abrasions or scratches.

An abrasion is the most trivial form of wound, being restricted to skin injury. Such injuries may provide very significant indications in certain cases. The causative agent is frequently a finger-nail, and the scratches so produced may be found on many parts of the body. The face, neck, arms, thighs, and female genitals are fairly common sites. They may be associated in minor degree with ligature marks upon the neck and with cases of throttling. The regions of the inner sides of the thighs and genitals may be abraded in rape cases where there has been considerable resistance. Their form, extent, and location will depend on the circumstances of each case. They are commonly found in vehicular accidents, more particularly when the body has been dragged over a rough surface.

FIG. 101
Multiple abrasions and contusions.

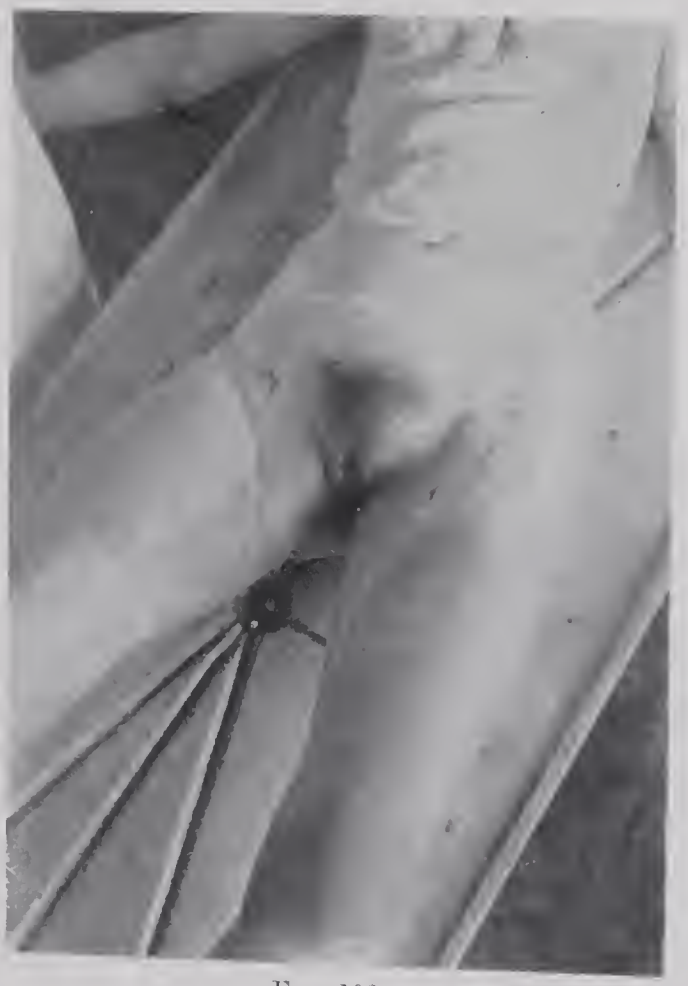


FIG. 102
Abrasions on thighs due to finger-nails.

With abrasions the effusion of blood is scanty, and where the lesion is uninfected, healing is rapid. Abrasions of post-mortem origin present a brownish-yellow, dried, parchmented appearance, and

Fig. 103

Abrasions on the face. Contusion on right shoulder.

there is no evidence of bleeding. A similar condition may occasionally be found on certain areas of a dead body due to the obliteration

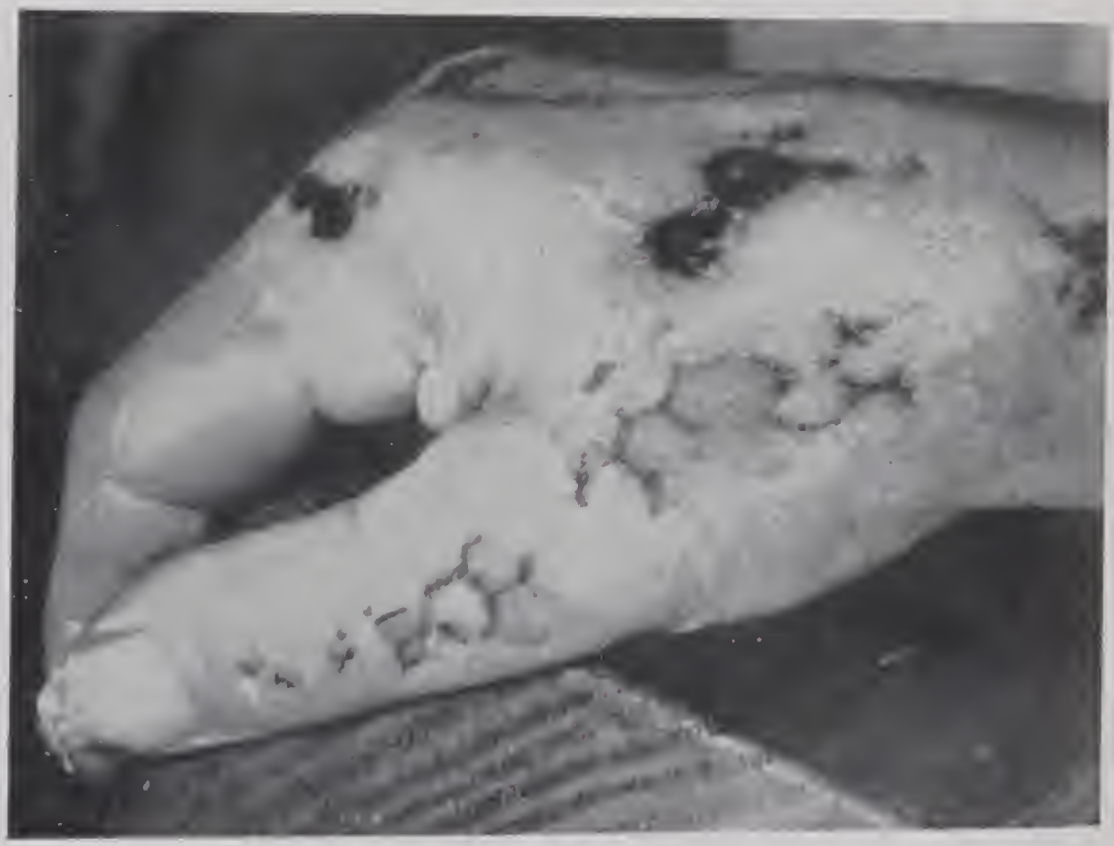


Fig. 104

Abrasions showing pattern of motor car radiator. The subject was run down by a motor car and died from head injuries.

(By courtesy of Dr Robert Richards, Aberdeen University.)

of fine capillary vessels. Those inflicted some time before death will show some evidence of vital reaction, including congestion of the vessels in the dermis under the site of injury.

Firearm wounds.

The appearances of these wounds must, in considerable measure, depend upon the exact conditions of the individual case under examination. Some of the conditions which may affect the appearances are :—

The nature of the weapon which fired the shot.
The shape and composition of the missile.
The range at which the weapon was fired, as affecting its velocity at the moment of impact.
The part of the body struck in relation to the amount of obstruction experienced by the missile in its passage through the tissues.
The direction of fire.

The types of firearms which will receive consideration are :—

The revolver.
The automatic pistol.
The rifle.
The shot gun or sporting gun.

The revolver.

This has been so designated on account of the fact that the weapon has a cylindrical magazine situated at the rear of the barrel which is capable of a revolving motion, and which has accommodation for five or six cartridges, each of which is housed in a separate chamber. After a shot has been fired, the circular magazine is rotated by the cocking of the hammer, and in this way the next cartridge is brought into proper position for firing. The cartridge of a revolver bullet has a projecting rim around the edge of the base, and the bullet is composed of lead or lead mixed with an alloy. Revolver ammunition is sometimes charged with black powder, although smokeless powder is usual. This type of weapon has a muzzle velocity of about 600 feet per second or less, and is termed a low-velocity weapon.

When a revolver is fired at very close range, almost in

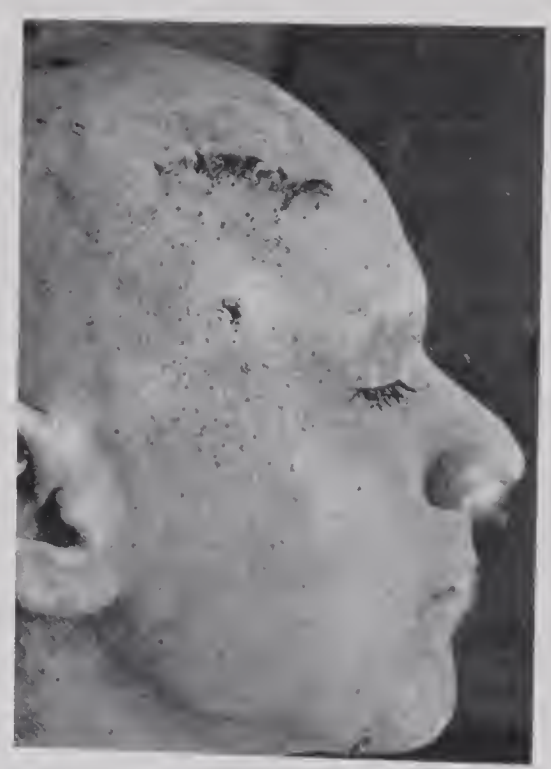


FIG. 105

Entrance wound of revolver bullet which lodged in brain. Tattooing by powder particles is seen around wound. The darkened area above is due to scorching.

1. The bullet strikes the block.

2. The bullet has penetrated into the block. Note how quickly the fibres of the wood tend to close the hole.

3. The bullet has been slightly deflected by the grain of the hard ash block.

4. The bullet breaks through the block.

5. The bullet emerges, shattering the wood and being partly disintegrated itself.

FIG. 106

X-ray study of calibre ·22 bullet in wood block. (Exposure time 1 microsecond.) (By courtesy of *Life* magazine, New York, and the Westinghouse Laboratories, Bloomfield, N.J.)

contact with the body surface, the wound of entrance is not circular in contour, but is cruciform or stellate with lacerated edges. This results from laceration of the tissues with some excavation due to expansion of the liberated gases. When the site of the injury is

Fig. 107

Entrance wound of revolver bullet which passed through head. Size of wound is indicated by circular disc of paper.

Fig. 108

Exit wound, in same case, larger than entrance. The edges are everted.

hair-covered, the hairs will show evidence of singeing, and when the ammunition has been charged with black powder there will be burning, in varying degree, around the entrance wound, together with an irregular ring of carbon deposit, usually about a quarter

Fig. 109

Exit wound of revolver bullet. Note underlying haematoma.

of an inch or more in diameter. In addition, the skin will show tattooing with powder particles driven into the skin. This description of a close-range wound is by no means similar to that of a wound produced at longer ranges, from several inches upwards, since at these ranges the entrance wound is no longer

of cross-shaped or stellate lacerated appearance, but is more or less circular or oval and corresponds approximately to the size of the projectile. Such wounds show marginal bruising and some inversion of the edges. There may also be an area of tissue bruising around the wound. The edges may show a grease collar. When the range reaches about 6 inches there is usually an absence of burning, although there will probably be some evidence of bruising and of powder marks. At a range of about 12 inches and over, the skin around the wound does not, as a rule, show evidence of powder marks.

When the weapon has been fired at very close range, almost in contact with the surface of the body, the wound of exit is usually of smaller size than the wound of entrance. With increased range this finding is usually reversed and the exit wound is frequently larger than the wound of entrance, due to laceration of tissue by the bullet being deviated in its passage through the body as a result of deflection in varying degree. When the projectile encounters bone the extent of deflection may be very great and the behaviour of the bullet may display many vagaries in the terminal part of its course. Should splintered bone be carried before it the tissues are lacerated, and the resultant wound is frequently very large. Except in contact shots, the wounds of entrance and exit may be of similar size when obstruction has not been encountered by the bullet.

It is unwise to dogmatise on the characters of revolver wounds, especially in regard to wounds of entry, since so much depends upon the pattern, calibre, and condition of the weapons which may produce them ; there are also many other factors which must be taken into consideration. With the view of determining the likely range at which a wound was inflicted, firing experiments, with the suspect weapon, should be made and the results of the tests carefully examined in relation to the known ranges. Microscopical comparisons can also be made between the particles of carbon removed from the region of the entrance wound and from the various experimental targets.

The automatic pistol.

This type of firearm is so named because when a cartridge is fired, the empty cartridge case is ejected, and a new cartridge slips into the breech automatically, as the result of the recoil. The cartridges are contained in a vertical magazine in the stock of the weapon, which usually holds six to seven cartridges. This class of firearm is better termed " self-loading " than automatic, since, as Burrard points out, an automatic firearm is one which will continue to fire, and go on firing, so long as the trigger is held back, whereas in all " self-loaders " the trigger must be pressed for every shot that is fired. The cartridge case, which is almost rimless, contains smokeless powder. Since this type of weapon fires a

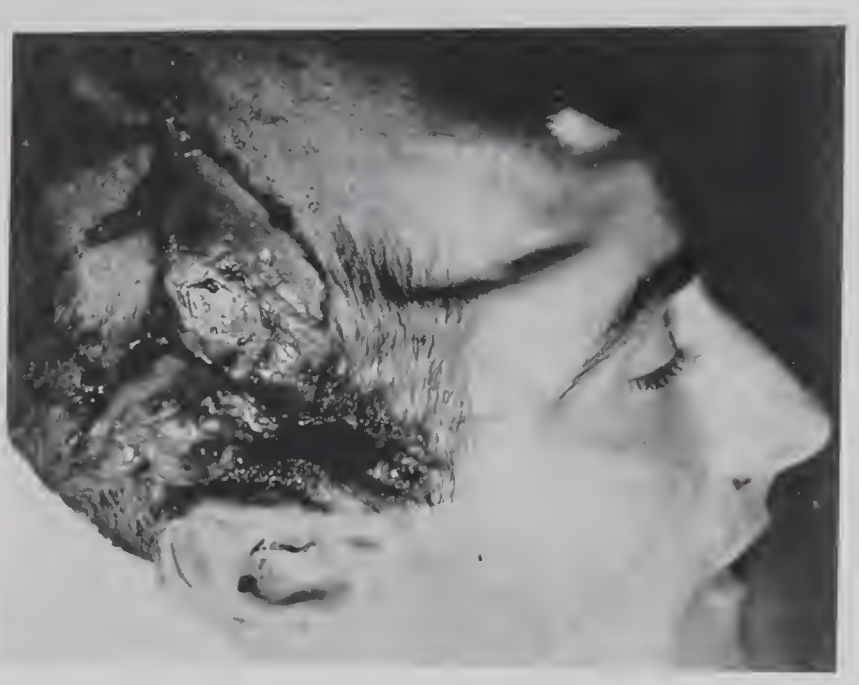


Fig. 110

Suicidal wounding by service rifle bullet at contact range. Note extensive laceration around entrance wound and depression of frontal bone due to fracture.

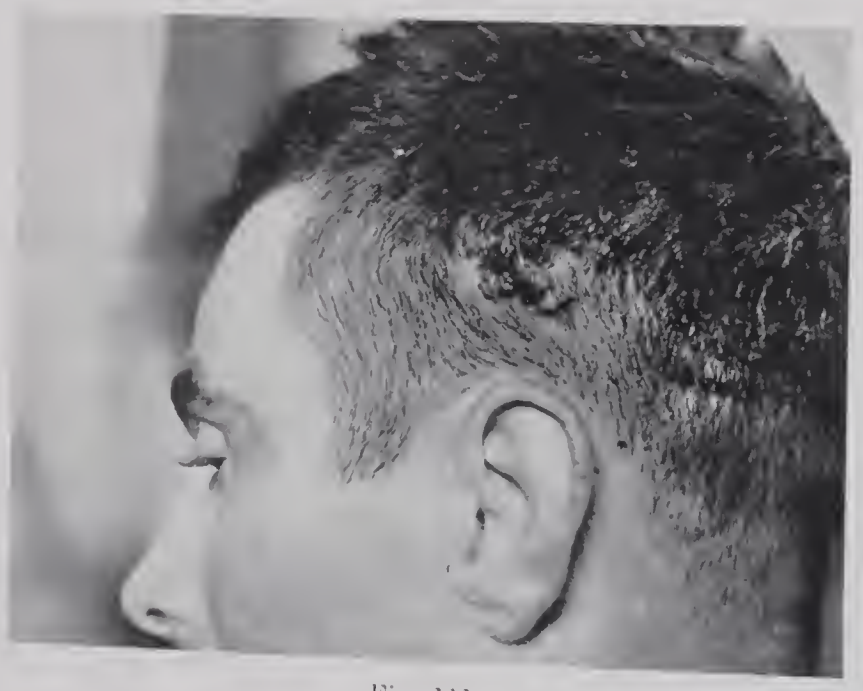


Fig. 111

Exit wound in case illustrated in Fig. 110.

bullet with a muzzle velocity up to 1,200 or more feet per second, the outer casing of the projectile is composed of hard metal such as nickel, steel, or cupro-nickel, although the inner core is composed of lead. The wounds produced have the characters of those already described under revolver wounds.

FIG. 112

X-ray study of calibre ·22 bullet passing through bone.

(Exposure time 1 microsecond.)

Radiograph showing the destructive action of a bullet passing through a piece of beef shin-bone. The pressure has forced the marrow out of the bone and the splintering of the bone and broken particles of the bullet may be seen.

(By courtesy of *Life* magazine, New York, and the Westinghouse Laboratories, Bloomfield, N.J.)

Propellant powders.

Black powder, or gunpowder, is composed of potassium nitrate, sulphur, and charcoal, and grey, or smokeless powder, of nitrocellulose, or of a combination of nitrocellulose and nitroglycerine, gelatinised, to retard the rapidity of the explosion and to maintain a uniform pressure on the projectile. Smokeless powder is grained into solid cylindrical patterns. The combustion of smokeless powder produces much less flame and smoke than combustion of black powder, since the process is more efficiently performed with the former than with the latter. The powder deposit around the wound, even at close range, is much less when smokeless powder is used, and around wounds inflicted at a range of a few inches, burning and tattooing are much less marked.

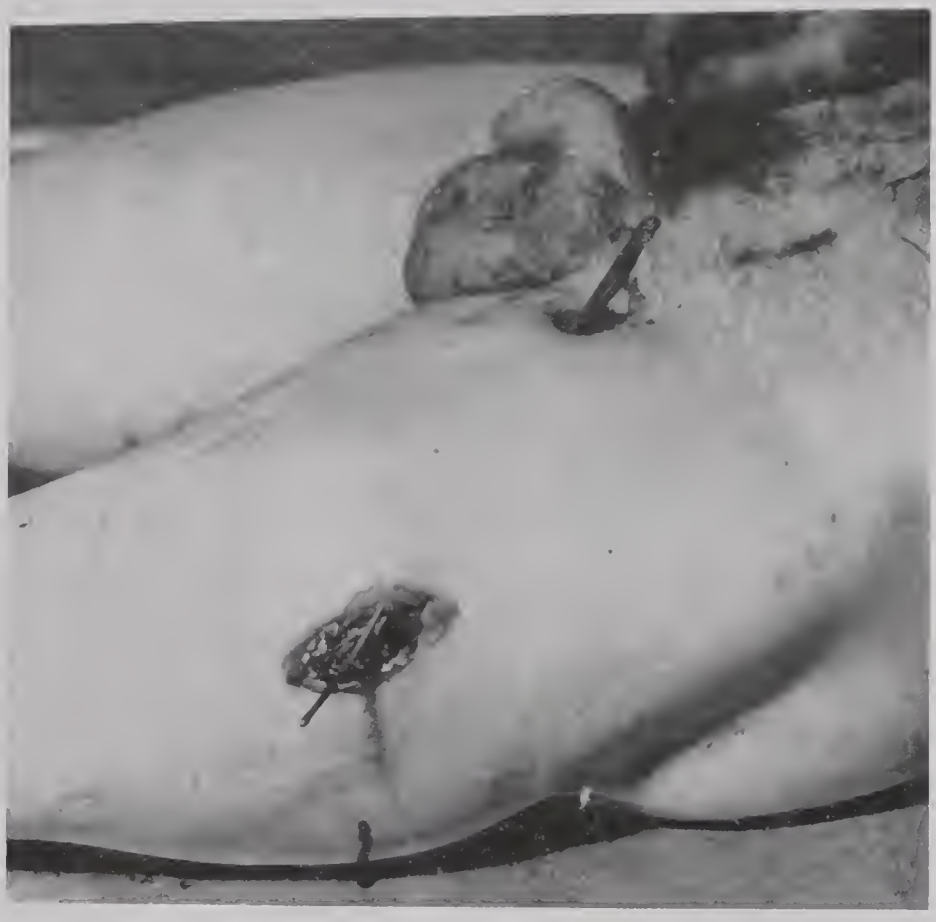


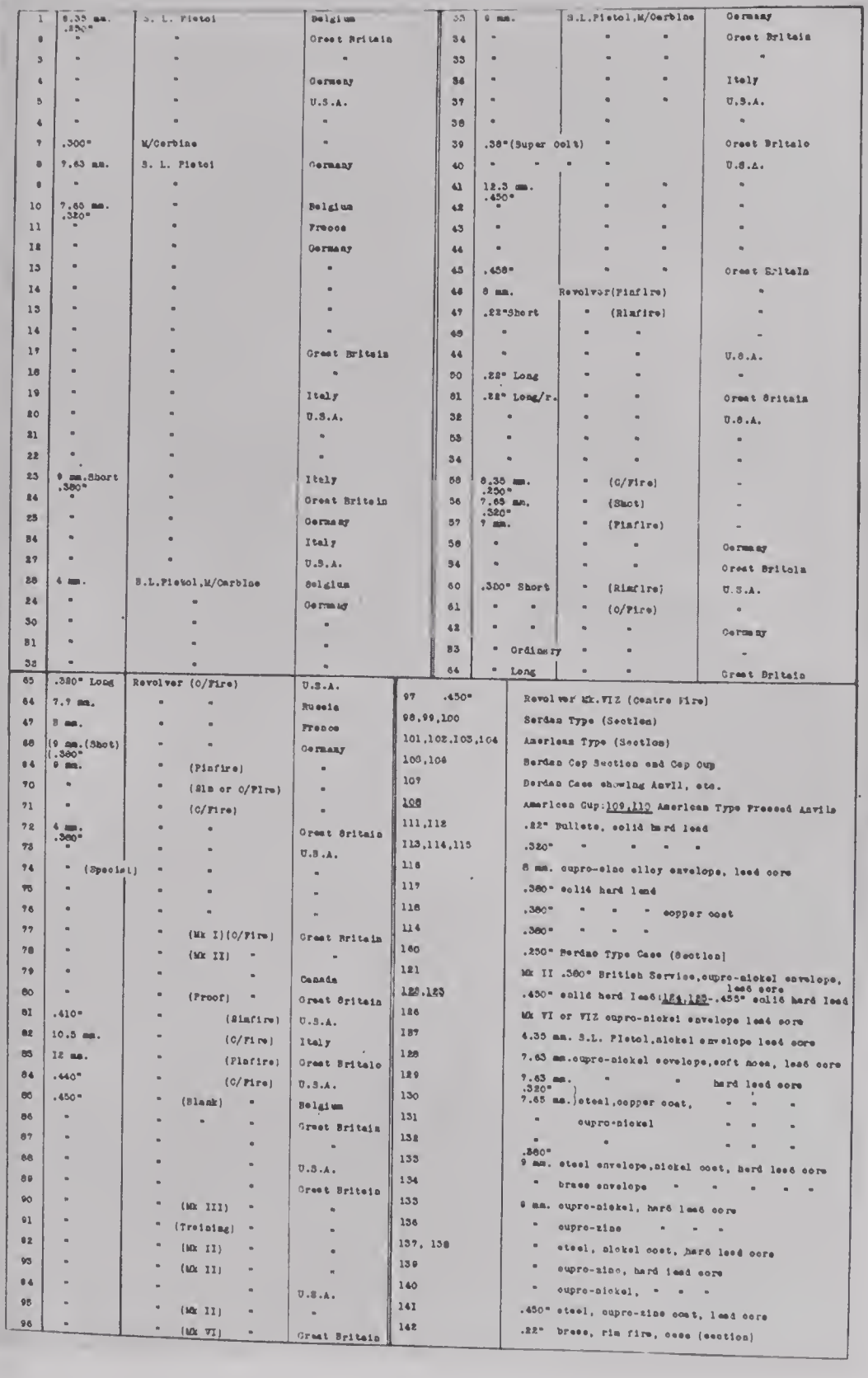
FIG. 113

Entrance and exit wounds by service rifle bullet. Abrasion in pubic region caused by impact of bullet with stem of button on great-coat. The femur was fractured.

FIG. 114

Exit wound of service rifle bullet.

Fig. 115
Ammunition for revolvers and automatic pistols.
(By courtesy of the City of Glasgow Police.)



1	6.35 mm. .250"	S. L. Pistol	Belgium
2	"	"	Great Britain
3	"	"	"
4	"	"	Germany
5	"	"	U.S.A.
6	"	"	"
7	.300"	M/Carbine	"
8	7.63 mm.	S. L. Pistol	Germany
9	"	"	
10	7.65 mm. .320"	"	Belgium
11	"	"	France
12	"	"	Germany
13	"	"	"
14	"	"	"
15	"	"	"
16	"	"	"
17	"	"	Great Britain
18	"	"	"
19	"	"	Italy
20	"	"	U.S.A.
21	"	"	"
22	"	"	"
23	9 mm.Short .380"	"	Italy
24	"	"	Great Britain
25	"	"	Germany
26	"	"	Italy
27	"	"	U.S.A.
28	4 mm.	S.L.Pistol,M/Carbine	Belgium
29	"	"	Germany
30	"	"	"
31	"	"	"
32	"	"	"

33	9 mm.	S.L.Pistol,M/Carbine	Germany
34	"	" "	Great Britain
35	"	" "	"
36	"	" "	Italy
37	"	" "	U.S.A.
38	"	"	"
39	.38"(Super Colt)	"	Great Britain
40	" " "	"	U.S.A.
41	12.3 mm. .450"	" "	"
42	"	" "	"
43	"	" "	"
44	"	" "	"
45	.456"	" "	Great Britain
46	8 mm.	Revolver(Pinfire)	"
47	.22"Short	" (Rimfire)	"
48	"	" "	"
49	"	" "	U.S.A.
50	.22" Long	" "	"
51	.22" Long/r.	" "	Great Britain
52	"	" "	U.S.A.
53	"	" "	"
54	"	" "	"
55	8.35 mm. .250"	" (C/Fire)	"
56	7.65 mm. .320"	" (Shot)	"
57	9 mm.	" (Pinfire)	"
58	"	" "	Germany
59	"	" "	Great Britain
60	.300" Short	" (Rimfire)	U.S.A.
61	" "	" (C/Fire)	"
62	" "	" "	Germany
63	" Ordinary	" "	"
64	" Long	" "	Great Britain

65	.380" Long	Revolver (C/Fire)	U.S.A.
66	7.9 mm.	" "	Russia
67	8 mm.	" "	France
68	(9 mm.(Shot) (.380"	" "	Germany
69	9 mm.	" (Pinfire)	"
70	"	" (Sim or C/Fire)	"
71	"	" (C/Fire)	"
72	4 mm. .360"	" "	Great Britain
73	"	" "	U.S.A.
74	" (Special)	" "	"
75	"	" "	"
76	"	" "	"
77	"	" (Mk I)(C/Fire)	Great Britain
78	"	" (Mk II) "	"
79	"	" "	Canada
80	"	" (Proof) "	Great Britain
81	.410"	" (Simfire)	U.S.A.
82	10.5 mm.	" (C/Fire)	Italy
83	12 mm.	" (Pinfire)	Great Britain
84	.440"	" (C/Fire)	U.S.A.
85	.450"	" (Blank) "	Belgium
86	"	" " "	Great Britain
87	"	" "	"
88	"	" "	U.S.A.
89	"	" "	Great Britain
90	"	" (Mk III) "	"
91	"	" (Training) "	"
92	"	" (Mk II) "	"
93	"	" (Mk II) "	"
94	"	" "	U.S.A.
95	"	" (Mk II) "	"
96	"	" (Mk VI) "	Great Britain

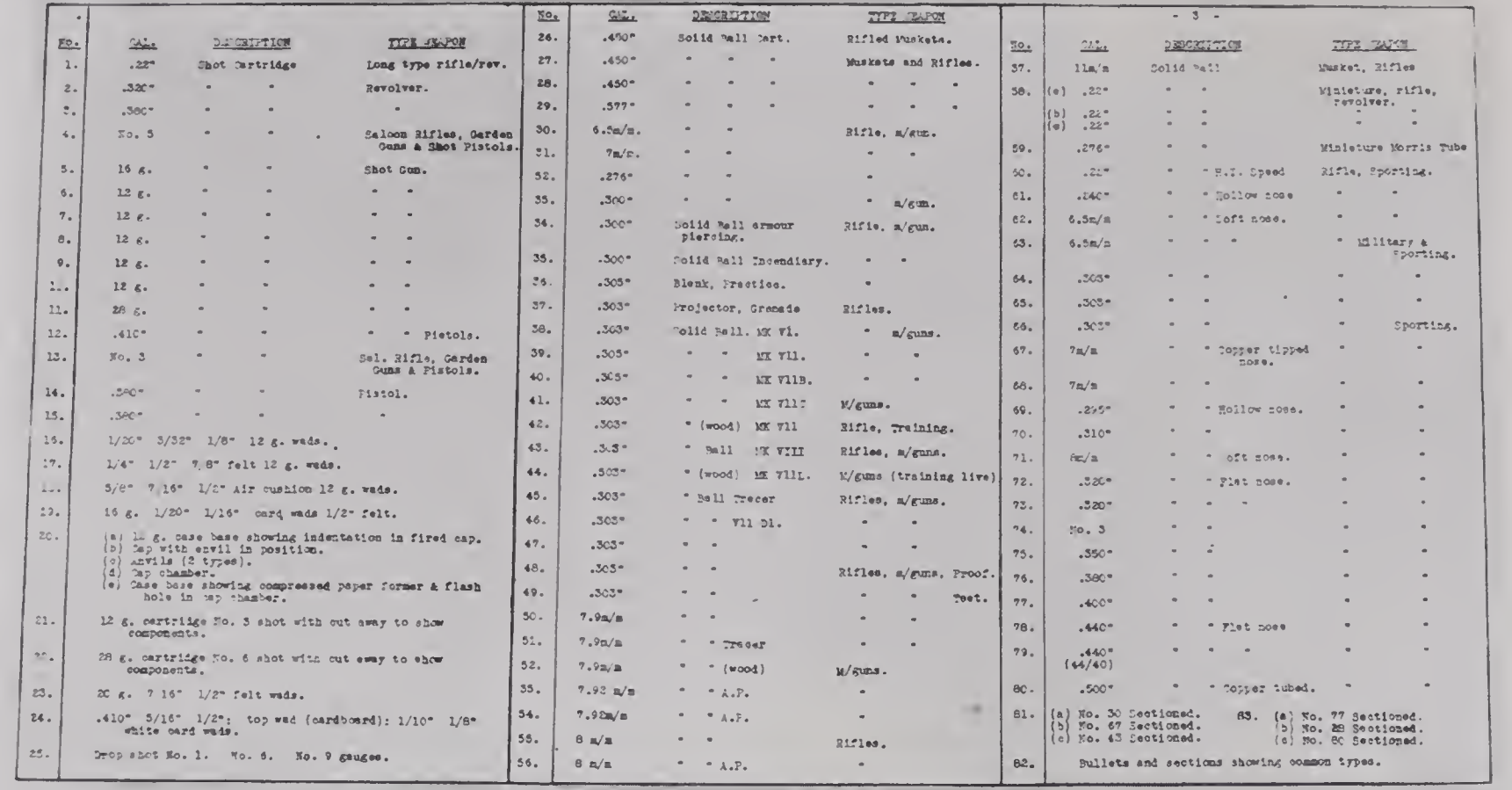
97	.450" Revolver Mk.VIZ (Centre Fire)
98,99,100	Berdan Type (Section)
101,102,103,104	American Type (Section)
105,106	Berdan Cap Section and Cap Cup
107	Berdan Case showing Anvil, etc.
108	American Cup:109,110 American Type Pressed Anvils
111,112	.22" Bullets, solid hard lead
113,114,115	.320" " " " "
116	8 mm. cupro-zinc alloy envelope, lead core
117	.380" solid hard lead
118	.380" " " " copper coat
119	.380" " " "
120	.250" Berdan Type Case (Section)
121	Mk II .380" British Service,cupro-nickel envelope, lead core
122,123	.450" solid hard lead;124,125-.455" solid hard lead
126	Mk VI or VIZ cupro-nickel envelope lead core
127	4.35 mm. S.L. Pistol,nickel envelope lead core
128	7.63 mm.cupro-nickel envelope,soft Aces, lead core
129	9.63 mm. " " hard lead core
130	.320" 7.65 mm.)steel,copper coat, " " "
131	" cupro-nickel " " "
132	" " " " "
133	.380" 9 mm. steel envelope,nickel coat, hard lead core
134	" brass envelope " " " " "
135	9 mm. cupro-nickel, hard lead core
136	" cupro-zinc " " "
137, 138	" steel, nickel coat, hard lead core
139	" cupro-zinc, hard lead core
140	" cupro-nickel, " " "
141	.450" steel, cupro-zinc coat, lead core
142	.22" brass, rim fire, case (section)

Fig. 116

Explanatory table for Fig. 115.

Fig. 117

Ammunition for rifles and shot guns.
(By courtesy of the City of Glasgow Police.)



No.	Cal.	DESCRIPTION	TYPE WEAPON
1.	.22"	Shot Cartridge	Long type rifle/rev.
2.	.320"	- -	Revolver.
3.	.380"	- -	-
4.	No. 5	- - .	Saloon Rifles, Garden Guns & Shot Pistols.
5.	16 g.	- -	Shot Gun.
6.	12 g.	- -	- -
7.	12 g.	- -	- -
8.	12 g.	- -	- -
9.	12 g.	- -	- -
10.	12 g.	- -	- -
11.	28 g.	- -	- -
12.	.410"	- -	- - Pistols.
13.	No. 3	- -	Sel. Rifle, Garden Guns & Pistols.
14.	.380"	- -	Pistol.
15.	.380"	- -	-
16.	1/20" 3/32" 1/8" 12 g. wads.		
17.	1/4" 1/2" 7/8" felt 12 g. wads.		
18.	5/8" 7/16" 1/2" Air cushion 12 g. wads.		
19.	16 g. 1/20" 1/16" card wads 1/2" felt.		
20.	(a) 12 g. case base showing indentation in fired cap. (b) Cap with anvil in position. (c) Anvils (2 types). (d) Cap chamber. (e) Case base showing compressed paper former & flash hole in cap chamber.		
21.	12 g. cartridge No. 3 shot with cut away to show components.		
22.	28 g. cartridge No. 6 shot with cut away to show components.		
23.	20 g. 7/16" 1/2" felt wads.		
24.	.410" 5/16" 1/2"; top wad (cardboard): 1/10" 1/8" white card wads.		
25.	Drop shot No. 1. No. 6. No. 9 gauges.		

No.	Cal.	DESCRIPTION	TYPE WEAPON
26.	.450"	Solid Ball Cart.	Rifled Muskets.
27.	.450"	- - -	Muskets and Rifles.
28.	.450"	- - -	- - -
29.	.577"	- - -	- - -
30.	6.5m/m.	- -	Rifle, m/gun.
31.	7m/m.	- -	- -
32.	.276"	- -	-
33.	.300"	- -	- m/gun.
34.	.300"	Solid Ball armour piercing.	Rifle, m/gun.
35.	.300"	Solid Ball Incendiary.	- -
36.	.303"	Blank, Practice.	-
37.	.303"	Projector, Grenade	Rifles.
38.	.303"	Solid Ball. MK VI.	- m/guns.
39.	.303"	- - MK VII.	- -
40.	.303"	- - MK VIIB.	- -
41.	.303"	- - MK VIIC	M/guns.
42.	.303"	" (wood) MK VII	Rifle, Training.
43.	.303"	" Ball MK VIII	Rifles, m/guns.
44.	.303"	" (wood) MK VIIL.	M/guns (training live)
45.	.303"	" Ball Tracer	Rifles, m/guns.
46.	.303"	- " VII Di.	- -
47.	.303"	- -	- -
48.	.303"	- -	Rifles, m/guns, Proof.
49.	.303"	- -	- - Test.
50.	7.9m/m	- -	- -
51.	7.9m/m	- " Tracer	- -
52.	7.9m/m	" " (wood)	M/guns.
53.	7.92 m/m	- " A.P.	-
54.	7.92m/m	- " A.P.	-
55.	8 m/m	- -	Rifles.
56.	8 m/m	- " A.P.	-

No.	Cal.	DESCRIPTION	TYPE WEAPON
57.	11m/m	Solid Ball	Musket, Rifles
58.	(a) .22"	- -	Miniature, rifle, revolver.
	(b) .22"	- -	- -
	(c) .22"	- -	- -
59.	.276"	- -	Miniature Morris Tube
60.	.22"	- " H.V. Speed	Rifle, Sporting.
61.	.240"	- " Hollow nose	- -
62.	6.5m/m	- " Soft nose.	- -
63.	6.5m/m	- - -	" Military & Sporting.
64.	.303"	- -	- -
65.	.303"	- -	- -
66.	.303"	- -	- Sporting.
67.	7m/m	- " Copper tipped nose.	- -
68.	7m/m	- -	- -
69.	.295"	- " Hollow nose.	- -
70.	.310"	- -	- -
71.	8m/m	- " Soft nose.	- -
72.	.320"	- " Flat nose.	- -
73.	.320"	- - -	- -
74.	No. 3	- -	- -
75.	.350"	- -	- -
76.	.380"	- -	- -
77.	.400"	- -	- -
78.	.440"	- " Flat nose	- -
79.	.440" (44/40)	- - -	- -
80.	.500"	- " Copper tubed.	- -
81.	(a) No. 30 Sectioned. 83. (a) No. 77 Sectioned. (b) No. 67 Sectioned. (b) No. 28 Sectioned. (c) No. 43 Sectioned. (c) No. 80 Sectioned.		
82.	Bullets and sections showing common types.		

FIG. 118

Explanatory table for Fig. 117.

The rifle.

Common examples of this weapon are the military rifle and the miniature rifle. The former has a magazine and bolt action, the latter is usually a single-loading weapon. The British service rifle has a muzzle velocity of about 2,500 feet per second, and can probably kill at a range of about 3,000 yards. The pressure in the firing chamber is about 20 tons per square inch. As the bullet leaves the barrel it rotates at about 3,000 revolutions per second. With such a high initial velocity the bullets frequently pass through the body, and if no resistance to their passage has been encountered, the size and shape of the entrance and exit wounds may be very similar except, perhaps, for some inversion of the edges in the former and eversion of the edges in the latter. The tissues through which the

FIG. 119

Entrance wound, on upper lip, produced by bullet from military rifle. Note tattooing of face with powder particles.

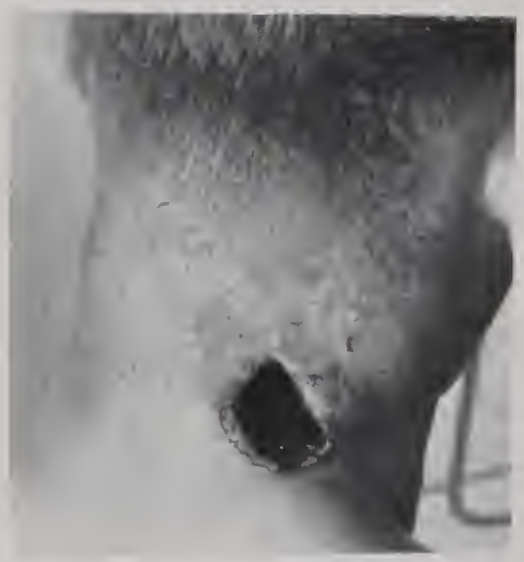


FIG. 120

Exit wound produced by the same bullet as in Fig. 119, and by splintering of bone.

projectile has passed are usually the seat of bruising in varying degree. Those who have had war experience are able to appreciate the wide range of variation and character which such wounds may present. The effects of high velocity projectiles show many vagaries and most unexpected injuries may be found. The entrance wound, as a rule, approximates the size of the bullet which has produced it. If, however, bone is encountered by the missile in its passage through the body, there may be considerable deflection, or splintering of bone or both. When the bullet thus deviated from its path ploughs through the tissues to force an exit, or when fragments of splintered bone are forced out through the tissues, the wound of emergence will be large and of lacerated character. It may measure several inches in diameter (see Figs. 111, 113, 114, 120). When the head is the seat of wounding, at, or near, contact range, the entrance wound may be of large size due to extensive laceration of tissue which is frequently accompanied by diffuse fracture of underlying bone (see Fig. 122). It has been noted

FIG. 121

Infra-red photograph of entrance wound on back of head. Projectile was of ·22 calibre. Note cross-shaped contour of wound due to close range.

FIG. 122

Suicide with a military rifle. The butt end of the weapon was rested on the ground, the forehead applied to the muzzle, and the trigger released by means of a piece of wood.

that, at short ranges, when a rifle bullet has passed through more than one limb, for example, the thighs, although the entrance wound of the limb first struck is frequently fully consistent with expected characters, the exit wound is lacerated and large, despite

FIG. 123

Suicide with a military rifle. The skull was comminuted and the brain, seen suspended in the undergrowth, was forcibly ejected from the cranial cavity.

the absence of bone damage. The entrance wound on the second limb may show very extensive laceration, several inches in diameter, due to alteration in the path of the flight of the bullet experienced by traversing the first limb.

The shot gun or sporting gun.

These weapons have for their projectile collections of small shot which vary in size, depending upon the type of cartridge employed.

The lead pellets are held in position by wads, one placed between the powder and shot charges, the other on the top of the shot charge, and held in position by the end of the stout paper cartridge case turned over against it. After firing, the pellets disperse soon after their exit from the barrel, and this dispersion increases with the range. The degree of dispersion can be controlled to some extent by a "choking" device near the termination of the barrel. This takes the form of a slight constriction which varies in degree in different weapons. To describe this, such terms as full choke and half choke are used. In certain weapons there is no "choke" device. If a shot is fired close to the body surface, up to a few inches, the shot enters as a mass, and in addition the liberated gases and flame lacerate the tissues, which

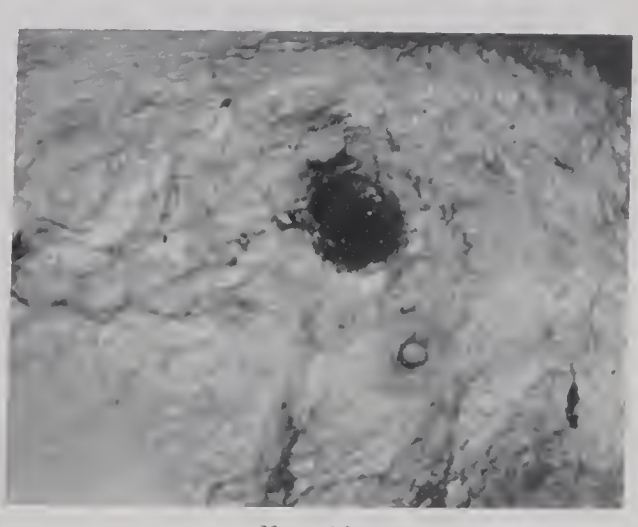


FIG. 124

Suicidal wound on left side of chest by sporting gun fired close to clothed body surface.

FIG. 125

Multiple perforations of clothing by pellets from sporting gun (see Fig. 126).

show evidence of burning, carbon deposit, and powder tattooing. The wads may be forced into the wound, and this may prove an important clue to the class of cartridge used. When the gun has been fired at from 1 to 3 feet from the body, a more or less irregularly circular wound about $1\frac{1}{2}$ to 2 inches in diameter will be produced (see Fig. 124). There will be evidence of some degree of scorching,

carbon deposit, and tattooing. So far as dispersion is concerned, with a "half-choke" gun the pellets will show a spread of about

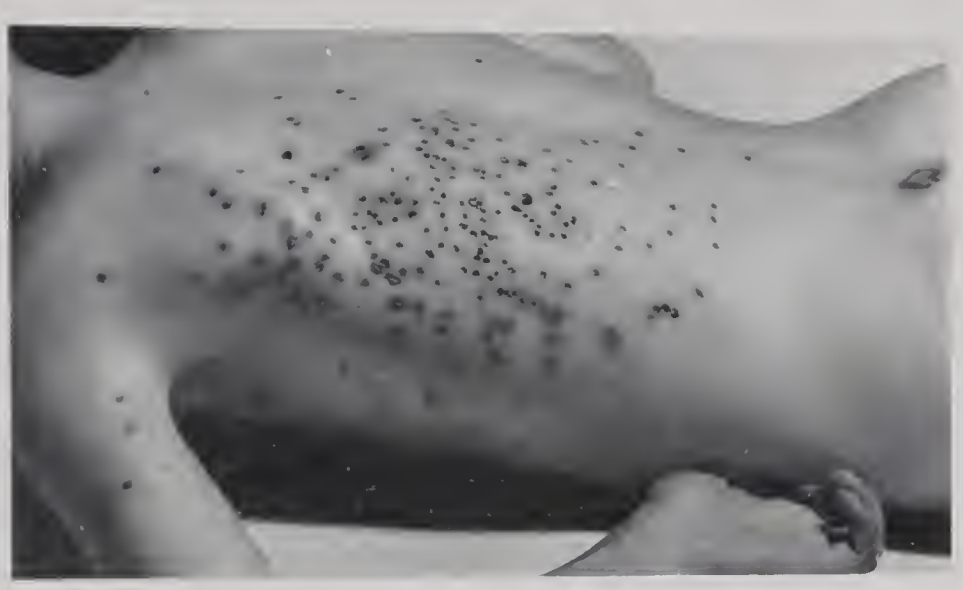


FIG. 126

Wounds produced by pellets from sporting gun fired at about 10 yards. Some 207 pellet wounds present, covering maximum spread of about 12 inches. Injury on left buttock is a bruise. (Figs. 125 and 126 by courtesy of Chief Constable of West Riding of Yorkshire, Wakefield, and Dr P. L. Sutherland, Wakefield.)

5 inches in diameter at a range of 5 yards, about 12 inches in diameter at 10 yards, about 16 inches in diameter at 15 yards, and about 20 inches in diameter at 20 yards. To determine the dispersion at the same ranges with a gun of "full choke," an approximate method is to deduct a quarter from the measurements of these diameters of spread. At a range of over a yard and up to about 3 yards, evidence of burning disappears and probably only faint tattooing will be found. Beyond a yard, the entering shot produces an irregular wound, and, as the result of commencing dispersion of the pellets, individual pellet holes may be detected.

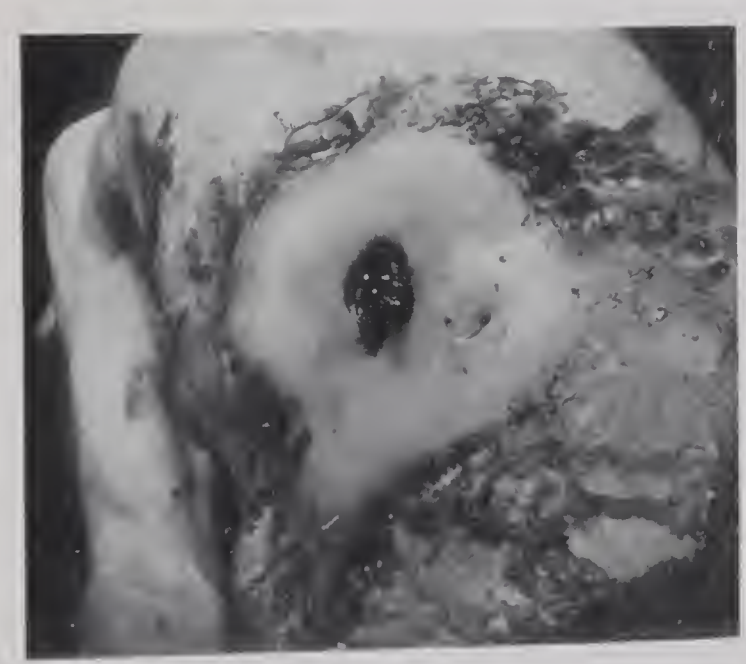


FIG. 127

Abdominal wound by sporting gun.

When smokeless powder is used, blackening and tattooing will be less marked at all ranges than with black powder.

Walker[6] recommends the use of infra-red photography for the determination of powder marks on clothing and finds it of considerable assistance in the estimation of range. Smokeless powder residues do not give a dense black pattern of individual spots when photographed with infra-red light, but black powder residues do. By this method a permanent graphic representation of the powder residue pattern is produced without destruction or alteration in the fabric. Blood-stains do not greatly interfere.

Accurate estimations of the pellet patterns at different ranges are not possible, since so much depends upon the idiosynerasies of individual cartridges. This is due to the fact that the cardboard wad is so frequently dislodged in an oblique fashion that turbulence of the shot occurs within the barrel and thus affects the pellet patterns at other than close ranges. With regard to the size of the area of wounding produced by a sporting gun almost irrespective of choke, an approximate estimate, at different ranges, may be obtained by using this simple formula :—

If X = range in yards, then the diameter of the wound = (X + 1) inches.

It should be clearly understood, however, that the dimension of the area of wounding, as calculated in the foregoing, is that of the cone of the shot spread, measured on a plane perpendicular to the line of fire and upon a relatively flat surface.

Identification of firearms.

Since the revolver, the self-loading pistol, and the rifle, fire a

Fig. 128

Top—Striker pin impressions on fired cartridge cases.
Foot—Rifling marks on fired bullets.

single bullet, it is necessary to make provision to maintain the bullet in a state of stability during its passage through the muzzle

1. The charge just before entering the wood block. The rear end of the charge is spread by the pressure action of the wad.

2. The charge has entered the block. Note the pressure wave in the wood block travelling ahead of the charge.

3. The charge has passed through the block.

FIG. 129

X-ray study of a shot-gun charge passing through a wood block.
(Exposure time 1 micro-second.)
By courtesy of *Life* magazine, New York, and the Westinghouse Laboratories, Bloomfield, N.J.)

of the weapon and during its flight. This is effected by the rifling on the interior of the barrel from breech to muzzle, which consists of a number of grooves which are cut longitudinally. Although parallel to each other they assume a spiral formation. Between the grooves are elevated surfaces termed lands. As the bullet passes along the barrel of the weapon at high speed its surface comes into intimate contact with these lands, forcing it into a spin which, when the bullet emerges from the muzzle, exerts a type of gyroscopic action. A fired bullet will thus show indentations of a series of slanting parallel grooves, varying in number from four to seven, made by the lands. The pattern of the rifling in weapons of different manufacture varies both in number of grooves and direction of the riflings. The markings upon fired bullets thus become of high value in the identification of the weapon from which they have been fired.

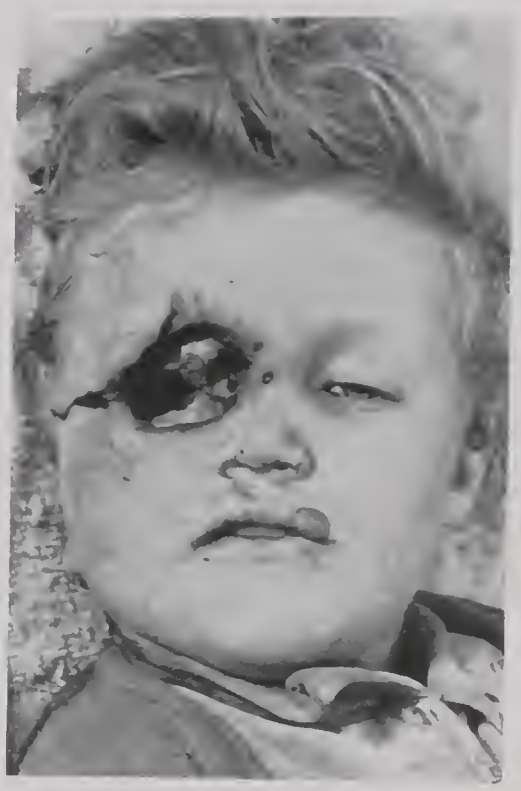


FIG. 130

Entrance wound caused by sporting gun at close range (see Fig. 131).

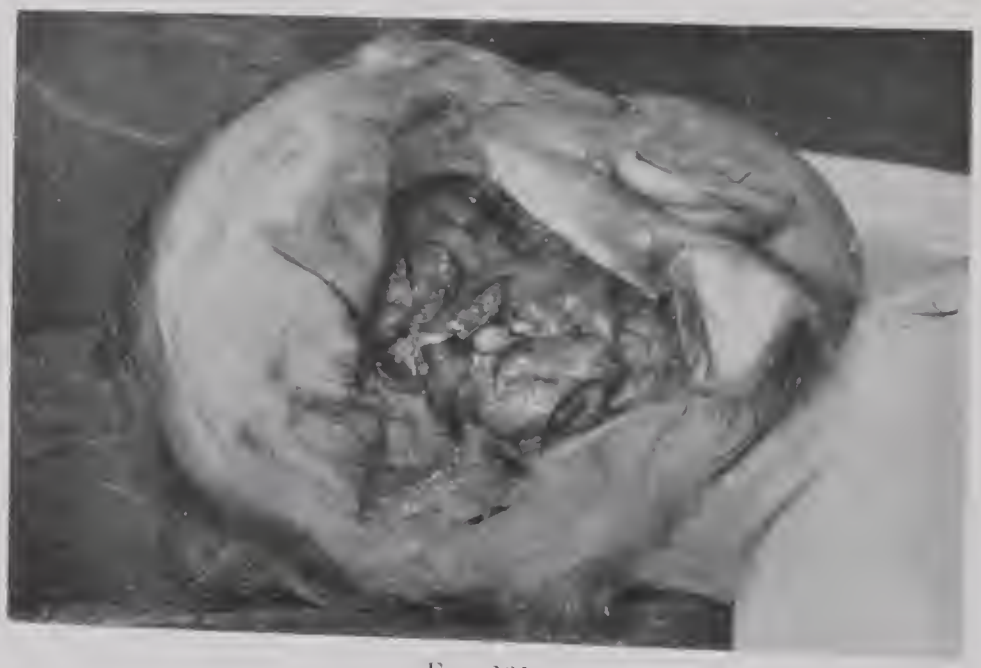


FIG. 131

Exit wound in the same case (see Fig. 130).

Fired cartridge cases also may be identified with the firearm from which they were ejected. Such investigation is based on the facts that an individual firearm will impart to the cartridges which it fires, its specific markings on the breech face and striker. In self-loading and automatic weapons, the ejector mark upon a fired cartridge may be compared with the characters of the metal block of the firearm which causes the ejection of the cartridge. For comparison purposes, test rounds should be fired from the suspect weapon and the cartridges compared in detail with those under examination. It is more difficult to establish identity by means of a fired bullet than by means of a fired cartridge case, since there is more chance of a static pressure mark on the latter than sliding pressure on

FIG. 132

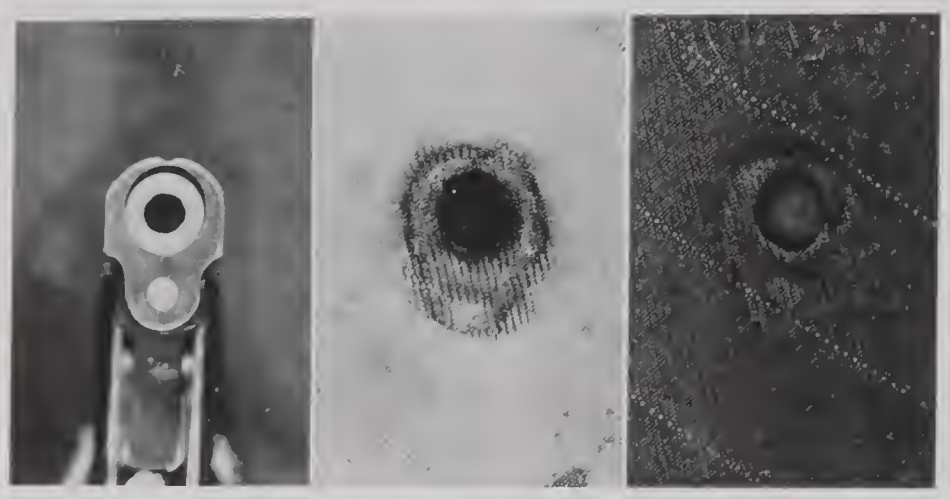
Comparison photomicrograph. Matching of land engraving marks on crime and test bullets.

(By courtesy of the City of Glasgow Police.)

the former. Cartridge case markings are also more easy to interpret. The greatest care must be exercised in removing a bullet from a body so that marks due to artifact, such as scratches, are not produced on the bullet. Such markings may vitiate subsequent identification of the projectile. When necessary, the use of forceps, the gripping arms of which have been covered with rubber tubing, is recommended. When the bullet has been extracted an identification mark should be engraved upon its base.

Since the identification of firearms, and their projectiles should always be undertaken by firearm experts, the necessary technique has been omitted. The reader who desires such information should consult standard works on the subject.[7]

The use of a mine detector for the recovery of bullets has been reported. The Winnipeg City Police, by this means, has met with success in two murder cases, and long, tedious searches have been abbreviated. In one of the cases, the bullet was recovered from a great pile of clay and mud excavated from a building foundation.[8]



A *B* *C*

FIG. 133

A, Muzzle of self-loading pistol. *B*, Infra-red photograph of scorched area on clothing showing contour of *A*. *C*, Ordinary photograph of scorched area on clothing. The outline showed that strong pressure must have been applied to the surface at the moment of firing. Such contours are of importance in the reconstruction of crime.

(By courtesy of the City of Glasgow Police.)

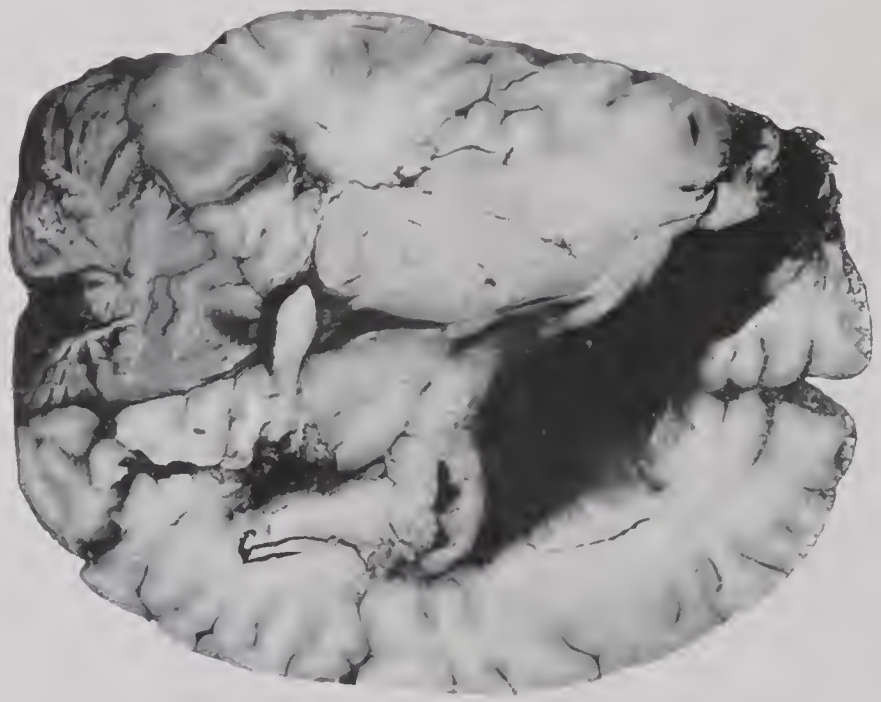


FIG. 134

Bullet track through cerebral hemispheres.
Note laceration of brain tissue.

Bullet wounds of the head.

When a bullet enters the skull and emerges, an examination of the entrance wound will show that the aperture in the bone differs in relation to the outer and inner tables: more bone is splintered from the inner than from the outer table. The converse is found in the exit wound in which a greater amount of bone is removed from the outer than from the inner table. The reason lies in the fact that there is an expanding impulse transmitted through the bone. The exit aperture is as a rule larger than that of the entrance. In certain cases, the possibility of a bullet ricochetting after entering the cranial cavity must not be overlooked, and in the odd case in which it occurs the deviation in direction of travel may be such that the point of emergence may occur in the most unexpected place. A rifle bullet on encountering the skull may, on account of its high velocity, probably cause an extensive fracture of bone due to sudden liberation of energy. When a bullet strikes the head at a very narrow angle to the surface, and glances from it, a gutter wound of the scalp and skull will result. When the skull is involved, there may be severe splintering of the inner table.

Bullet wounds on other parts of the body.

In performing a post-mortem examination in a case of shooting, certain precautions must be adopted. If there is a single wound upon the body, it must be a wound of entrance, and therefore the bullet must be recovered. Should the entrance wound be soiled with blood, great care must be exercised in sponging it away so that any tattooing of the skin may not be disturbed. When the bullet has been fired through the palate, unless it has emerged from the skull, no wound will be visible on the exterior of the body. Exit wounds never show evidence of carbon deposit, burning, or powder tattooing. It is frequently found that the track of the bullet is a devious one due to its deflection by bone, and the projectile may ultimately be found in a most unexpected situation. This point is of practical significance, when the question arises in the case of two wounds being found on the body not corresponding to the likely line of traject, as to whether they have been produced by one or two bullets. The bullet usually takes a straight path through the body, and in this event the relative positions of the entrance and exit wounds will indicate the direction of fire. When a cavity has been penetrated and a quantity of blood has collected, a careful search for a bullet in the effused blood should be made. The possibility of the bullet being overlooked in such a case might result through its removal in a measuring vessel with the blood. In isolated cases an X-ray examination may prove necessary for the localisation of a bullet within the body. The writer recalls a case in which the missile was ultimately shown to be lying within

the hip-joint although the wound of entrance was situated on the chest. When the bullet penetrates the skin obliquely, the contusion collar is more pronounced on the aspect of the wound corresponding to the direction of entry of the missile, and thus indication of the direction may be given. Sometimes the bullet strikes the body at a tangent, and instead of penetrating the tissues merely grazes them, producing a furrow of varying depth. The fact that a single bullet may cause several wounds on the body, for example, perforation of the arm followed by entrance into, and exit from, the chest, must not be overlooked. Disintegration of a portion of the projectile, fragmentation of bone, or the emergence of foreign bodies, such as pieces of metal button, may be responsible for multiple wounding.

A spent bullet may cause surface injury only, in the form of bruising or laceration, and it may be very difficult to arrive at an opinion as to cause and effect unless the history of the case is clear.

The vagaries exhibited by wounds from firearms are so numerous that it is prudent to avoid expressions of opinion regarding the nature and course of the missile, unless and until the whole facts of the case have been investigated and the results carefully considered. The following cases will illustrate many of the points which have been touched upon.

ILLUSTRATIVE CASES

Fatal wounding by bullets fired from revolvers and automatic pistols.

Revolver bullet.

We examined the body of a man alleged to have been accidentally shot in a private shooting club. An ovate wound, measuring $\frac{5}{8}$ by $\frac{3}{8}$ inch, was situated on the left temporal region. The eyelids were deeply bruised. The scalp tissues, over an area of 6 inches in diameter, around the wound contained extravasated blood. In the left temporal bone was an ovate aperture of the same size as the wound of entry. Over the surface of the brain was a thin layer of blood. On the outer surface of the left frontal lobe was an area of laceration. The bullet had passed through the substance of both frontal lobes, been deflected backwards through the right half of the cerebrum, and had finally lodged in the right occipital lobe. The roof of each orbital cavity was the seat of fracture. The bullet, which was distorted, measured $\frac{9}{16}$ by $\frac{5}{16}$ inch.

It appeared that both accused and deceased were members of the club, and that both were expert shots. The deceased in friendly rivalry challenged the accused to hit a clay pipe which the deceased man was to hold in his mouth, and it was during the act of shooting at the pipe that the deceased was killed. The bullet had struck

the pipe, been deflected at an angle, and had entered the head of the victim (see Fig. 135).

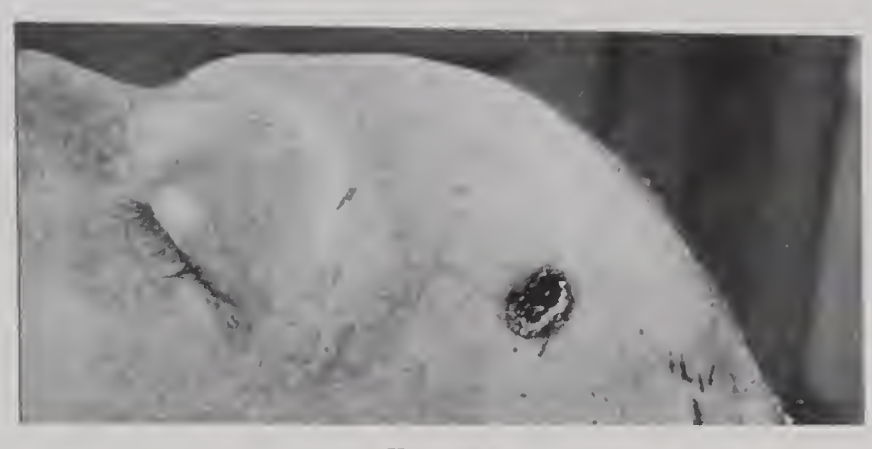


FIG. 135

Entrance wound of bullet, extracted from the brain. Bullet entered head sideways due to ricochet.

Multiple revolver bullets.

This case is illustrative of the occasional multiplicity of small calibre revolver bullet wounds in suicide.

FIG. 136

Suicidally inflicted revolver bullet wounds of chest, five in number.

Over the third and fourth intercostal spaces, covering an area of 3 inches in diameter and at a distance of $1\frac{1}{2}$ inches from middle line of chest, was an area of scorching (see Fig. 136). Within this area were five punctured wounds, the edges of which were black. Each wound was $\frac{1}{5}$ inch in diameter. Multiple firearm wounds suggest homicide rather than suicide, but in a number of suicidal cases such wounds may be found. In these suicidal cases, bullets of small calibre, ·22 or ·25, are usually employed. A number of instances have been met with where several bullets of this type have penetrated the heart.

Automatic pistol bullet.

The entrance wound, situated on the left side of chest, $1\frac{1}{4}$ inches external to, and in line with nipple, was circular, and measured $\frac{1}{8}$ inch in diameter. The edges showed a grease collar and underlying bruising which measured $\frac{3}{8}$ inch in diameter. On right side of chest, 3 inches external to nipple, and in line with it, was a faintly bruised area $\frac{1}{2}$ inch in diameter. In the centre a small ill-defined linear abrasion was situated, and palpation over this area disclosed the presence of the bullet. On the inner surface of

middle-third of right upper arm, corresponding in position to the abrasion on side of chest, was a faint bruise, approximately ½ inch in diameter. Dissection of the body showed that the bullet had passed through the tissues of the chest wall, had fractured the fourth rib, and penetrated the left lateral surface of pericardium, and interior surface of the heart, immediately to the left of the interventricular septum, at a point 1½ inches above apex of heart. It emerged from the heart on the right side, 1 inch below auriculo-ventricular border, passed through the right lateral surface of pericardium, the inner surface of the lower portion of the middle lobe of right lung, and into right chest wall, in fourth interspace, where it lodged immediately beneath the skin. Both pleural cavities contained blood-clot, which amounted to 3 pints in the left cavity and 2½ pints in the right.

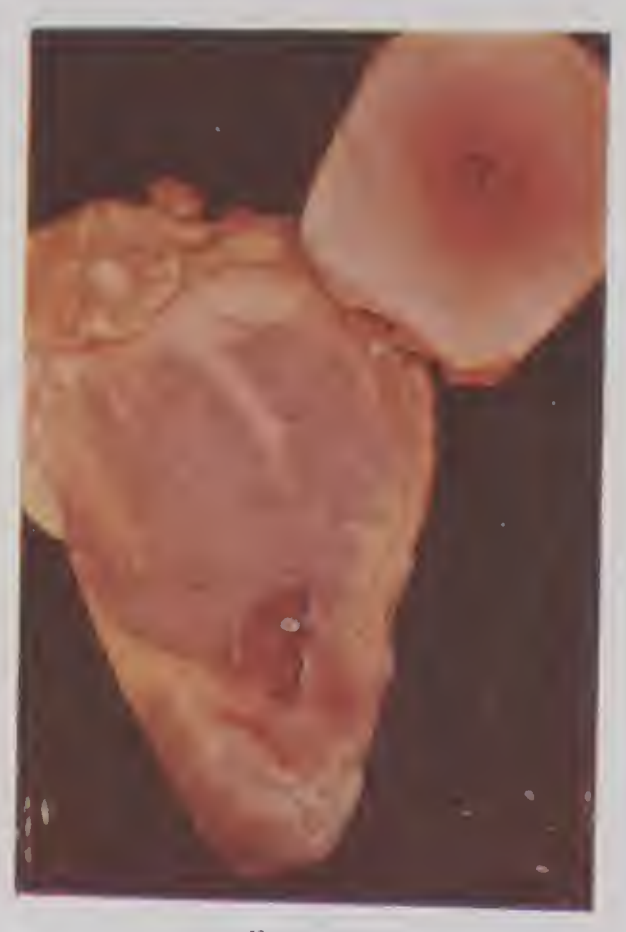


FIG. 137

Firearm wound of chest with penetration of heart. Wound on body surface seen on top, right.

Service rifle bullet.

The entrance wound, which was situated 5 inches above the anterior superior spine of left ilium and 2½ inches external to it, penetrated the abdominal cavity between eleventh and twelfth ribs. There was no scorching or tattooing. The exit wound, which was lacerated and measured 5 by 2 inches, was situated on the back, immediately to the left of the middle line of lumbar region of vertebral column. There was marked loss of muscle tissue and laceration. The cavity formed barely admitted the closed fist, and at its base the upper pole of left kidney could easily be palpated. There was a quantity of free blood in both flanks and the tissues around left kidney were bruised and lacerated. The kidney was severely lacerated, and its lower pole had almost been severed. The left transverse processes of third and fourth lumbar vertebræ were comminuted.

Sporting gun injury.

The body was found on a farm road with a large wound in the abdomen, and the sporting gun of the deceased man lay by his

side. On the front of abdomen there was an almost circular wound, measuring $1\frac{5}{8}$ by $1\frac{3}{8}$ inches, through which a small mass of intestine protruded. The wound was immediately above, and to right of, the umbilicus. There were no marks of tattooing around the wound, but the edges were scorched. The abdominal cavity contained 20 ounces of blood. On the inner surface of the abdominal wall the wound measured $3\frac{1}{2}$ by $2\frac{3}{4}$ inches. In the wall of the intestines there were many small shot. In the lower part of lumbar portion of vertebral column the bodies of the vertebræ were peppered with shot, and the surrounding tissues, including the main abdominal vessels, were severely injured. Lying in blood in the left side of the pelvis were the entire wad of a cartridge and fragments of clothing, in which were several pellets of shot (see Fig. 127).

FIG. 138

Suicidal rifle wound of face. Site of entrance wound close to right eye. Exit wound on outer side of left eye.

Unusual case of volitional movement following automatic pistol wound of brain.

Kerr [9] records a most unusual case. The facts put briefly were that an elderly man shot himself with a ·45 Colt automatic pistol. The wound of entry was situated under chin, 2 inches from point, and immediately to left of mid-line. The wound passed into skull, just behind roof of left orbit, passed through frontal and temporal lobes of brain, which were lacerated and pulped, then passed through skull on left side of frontal bone making an exit, $1\frac{1}{2}$ inches in diameter, and severely lacerating scalp, $3\frac{1}{2}$ inches directly above left eye. Following the injury, the man walked from a garden shelter, near a hotel where he lodged, to the grass in front of the shelter and continued in a circle for about 165 yards. He returned to the shelter, where he rested upon a seat and finally returned to his hotel. On his arrival there, he rang the

FIG. 139

Decapitation due to firing an explosive charge placed in the mouth. This was a case of suicide, and a detonator was employed to fire the charge.

bell, spoke to the servant, hung up his umbrella in the hall, took off his overcoat, and walked upstairs to the bathroom where he collapsed and lost consciousness. The brain showed considerable laceration and hæmorrhage. Portions of brain had been blown through the top of the skull on to the roof of the shelter. The man lived for three hours after his injury.

General data upon which the kind of instrument used may be inferred from the nature of the wound.

The forms of wounds are very variable, and frequently their shape bears no relationship to the weapon which has produced them. At the same time, there are certain features which may assist in arriving at a conclusion.

Incised wounds which show sharply defined edges are, in practically all cases, the result of injury caused by a sharp instrument. The rare exceptions are wounds which have been produced over bony ridges or prominences by forcible contact with a blunt instrument. Differential diagnosis between the use of a sharp or blunt weapon in these cases can be established by careful examination of the condition of the hair-bulbs at the wound edges with a hand-lens and by the facts that a wound caused by a blunt instrument will probably show some evidence of bruising and swelling at the edges and a less clean severance of the deeper tissues.

When a sharp-pointed, edged instrument, used perpendicularly, enters the body, the wound is less in length than the breadth of the instrument, and broader than the thickness of the blade.

When the same instrument enters the body obliquely, the edges of the wound may appear unequal, one edge being straight and the other curved, and, not infrequently, owing to the bevelled shape of the wound, beads of subcutaneous fat may appear between the edges.

Injuries caused by glass and earthenware present different appearances. Some closely simulate wounds by an instrument of the dagger type, others by instruments used as in cutting.

In wounds of the scalp caused by pieces of glass or crockery, small fragments of these substances may be found either in the tissues, or embedded in the bone itself, from which the nature of the weapon used can be determined. We have found this in several cases.

Injuries by glass very frequently show a parallelism of the edges of the wound with a sudden tapering at one end, to form a superficial scratch or abrasion.

Wounds with ill-defined, irregular, or ragged edges, which may be linked by small bridges of connective tissue, are always caused by forcible contact with a blunt instrument. When found on the scalp, whole or crushed hair-bulbs may be picked out of the edges of the wound.

Wounds of mixed character may be found upon the body, most commonly the head, when a bottle has been the weapon. In these cases co-existent bruises, lacerated wounds, and incised

wounds may be found and the general appearance of the lesions may suggest that different types of weapons have been used. The explanation lies in the fact that the unbroken bottle has been the cause of the contusions and lacerations, and that while these injuries were being inflicted it has broken, with the result that incised wounds are added to the seat of injury. When a sharp-bladed knife strikes a convex surface of the body obliquely, for example, the arm or leg, the wound produced may be curved in shape.

Contusions or bruises found upon the body are the result of forcible application of a blunt instrument. In most cases they do not afford any further clue to the character of the instrument used, but occasionally from their defined nature and from the presence of more than one mark of similar shape, a reliable opinion may be possible. A series of contusions in the form of stripes, the breadths of which closely correspond to one another, would be indicative of the application of a stick, a leather strap, or similar weapon. In bruising, the amount of extravasated blood is not always a reliable indication of the severity of the violence used, since certain parts of the body, because of their vascular character, for example, the genitals, may bruise readily. Extensive bruising may result from the application of a very moderate degree of violence in persons of purpuric, hæmophilic, or scorbutic tendency. It must also be noted that under rare and exceptional circumstances ecchymosis may develop spontaneously. In offering an opinion as to the amount of violence used from the extent of the extravasation present, the preceding factors must receive due consideration.

Wounds which are cruciform, stellate, circular, or oval in shape, and to which on other parts of the body in a more or less direct line other wounds correspond, are most frequently produced by traversing missiles. Single wounds of this character may either be due to a missile which has entered the body and is retained or to sharp-pointed instruments. Usually the history of the case will determine the difference, and in dead bodies dissection will clear up any difficulty. The recognition of firearm wounds becomes obvious, when around the wound there is evidence of scorching or engraining of the skin with particles of carbon, or with the products of smokeless powder.

Significance of situation, shape, direction and dimensions of wounds.

In dealing with wounds which form the subject of medico-legal inquiry, great accuracy in their description must be observed. In describing their dimensions, it is insufficient to state approximate measurements, since they must be exact in every particular. The following points with reference to wounds require to be noted carefully :—the **situation, shape, direction** and **dimensions**.

Situation of the wound.

Sometimes from the position of a wound upon the body some light may be thrown upon the likely mode of its production. Certain

parts of the body are chosen sites for suicidal wounds, as, for example, the throat, chest, forearm, groin, and thigh. Wounds on certain other parts oppose, if they do not absolutely negative, the suggestion of suicide, by reason of their inaccessibility, for example, wounds

Fig. 140

Self-inflicted wound of scalp caused by an axe.

on the back. Not only do wounds on the vertex frequently negative suicidal production, but also accidental production, since it is rare to injure the vertex by a fall from the standing position (see Fig. 169). Nevertheless, an opinion in such cases must not be lightly given, as the following case, for which we are indebted to Dr W. McWilliam, will clearly demonstrate. A patient in a mental hospital attempted to commit suicide by using an axe. He seized the shaft with both hands and brought the cutting edge up towards his head, thus inflicting a scalp wound which also destroyed the periosteum and scored the outer table of the parietal bones in three places, to a depth of $\frac{1}{16}$ inch. He did this without rendering himself unconscious. If the patient had succeeded in his attempt, and if he had been found dead, it is highly probable that the injury might have been regarded as the result of a homicidal act (see Fig. 140). In cases where wounds are found upon the throat of a dead body, it is advisable always to examine the hands carefully. In this way it may sometimes be possible to distinguish between homicidal and suicidal cut-throat wounds, for in the former class of case,

Fig. 141

Homicidal stab wound of the neck.

unless the victim is taken unawares from behind, or is otherwise unable to offer resistance, the hands when used in defence are liable to be injured. Homicidal wounds are usually inflicted over some vital region of the body, either on the front of the chest near the region of the heart, on the neck, on the head, or on the back between the shoulders.

Shape of the wound.

This may afford important information in relation to the class and type of weapon which may have caused the injury. The subject has already been discussed (see p. 277).

With regard to the expression of an opinion as to whether a specific instrument has caused a particular wound, the opinion should be limited to the statement that the wound could or could not have been inflicted with such an instrument, and that the appearances of the wound are consistent or inconsistent with its infliction by such an instrument or class of instrument.

Direction and dimensions of the wound.

These factors have an important bearing on the relative positions of the assailant and the assaulted person when the wound was inflicted. Such information is of special importance in cases of wounding by firearms, but the direction of the wounding is also of importance in many other cases, for example, in cases of stabbing. When the relative positions of the assailant and assaulted person are in question by reason of absence of direct evidence as to the manner of the infliction of the wound, the medical examiners may be expected to offer some opinion on the point from the characters of the wound or wounds.

Not only must the recorded measurements of the wound or wounds be accurate, but the relative positions between the sites of the wounds, when there are more than one, must be carefully noted. The direction and extent of deep wounds also demand close attention and detailed description. Any incisions made post-mortem should avoid interference with the site of previous injury.

Should it prove necessary to insert a probe, care must be taken to select one with a blunt end and force must not be used. Unless these precautions have been adopted, it may subsequently be alleged by the defence that at least part of the injury was produced by the investigation. Broadly speaking, it is better to defer the use of a probe until dissection has reached a stage when its passage can be seen and controlled.

When wounds are found upon a clothed part of the body, it is important to examine the different superimposed garments both together and separately. All evidences of the passage of instruments through the fabrics must be carefully noted and measured, together with their relation to the situation of the lesions on the body. In this way a lead may be given as to the position of the assaulted person at the moment of the assault, a point of considerable importance, especially in penetrating wounds of the chest. It occasion-

ally happens that on examining a loose under-garment, more marks of perforation are discovered upon it than there are wounds on the part of the body corresponding to the position of the garment, or on the outer clothing. This is due to wrinkling or folding of the underwear.

In the examination of a dead body upon which there are several wounds, it is necessary to assess their individual importance in relation to the fatal issue.

It must not be forgotten that death may result from a comparatively small wound.

We recall a case in which a boy of between seven and eight was stabbed on the back of the neck by another boy. The wound proved fatal, and post-mortem examination disclosed a partial severance of the spinal cord. The knife used had a narrow, thin blade which had insinuated itself between the third and fourth cervical vertebræ, leaving an almost imperceptible wound at the site of entry (see p. 290).

Ante-mortem and post-mortem wounds.

In medico-legal practice the differentiation between wounds inflicted before and after death is most important. In arriving at an opinion, a detailed examination is necessary. If signs of vital reaction are present, the injury has been produced during life.

The signs of vital reaction will naturally depend on the period of survival following injury, and these may be swelling, effusion of lymph, pus formation, or evidence of repair. When inflammatory signs in any degree are found, they constitute definite evidence of infliction of the injury during life. When there is an element of doubt, microscopical examination of tissue sections must be made before arriving at an opinion as to whether the wound was of ante-mortem or post-mortem origin. Even if the victim has survived the injury only for a period of some hours, leucocytic infiltration will probably be evident on microscopical examination.

It should not be forgotten that, so long as molecular life remains in the tissues, some retraction of the edges of a wound inflicted after death may occur.

A word of caution is appropriate in connection with hæmorrhage and the inferences to be drawn as to whether it is significant of ante-mortem or post-mortem origin. As a rule, a profuse hæmorrhage is indicative of the injury having been produced during life, but, on the other hand, absence of copious blood cannot always be regarded as a sign of post-mortem injury since shock or rapid death following an extensive or mortal wound might readily account for its absence. When gross post-mortem injury occurs a considerable quantity of blood may collect as the result of oozing from dilated vessels. Such factors should receive careful consideration.

Age of the wound.

The question as to the age of a wound may arise under a variety of circumstances, and the following data will assist in arriving at an

opinion, although the nature and extent of the wound are highly important qualifying factors :—

Cellular infiltration will be well marked microscopically in twelve to eighteen hours after the injury.

During the first twenty-four hours the margins of the wound will be temporarily adherent by blood and serum, and will show vascular congestion ; swelling and leucocytosis are present.

New capillaries will probably have formed in about thirty-six hours.

In forty-eight to seventy-two hours, spindle-shaped cells which run at right angles to the vessels will be present.

Pus may be seen in septic wounds after forty-eight to seventy-two hours, or sometimes earlier.

In five to six days fibrils which run parallel to the vessels will have become established.

When a wound heals by granulation, as it always does if there is loss of substance and the edges cannot be brought into surgical continuity, the stage of granulation exists for so variable a period, depending upon so many factors, that an answer, when ventured upon, should be given with great caution.

Granulation tissue is subsequently transformed into young scar tissue, its cellular elements disappearing as the fibrous tissue increases in amount. While these changes are taking place, the surface is being covered by epidermis which grows from the margins of the wound, and, by proliferation gradually covers the granulations with a thin pink pellicle. As it increases in thickness, it assumes a bluish hue and when cornified becomes whitish in colour (see p. 96).

Is the wound accidental, suicidal, or homicidal ?

This question is bound to arise in every case in which a dead body, with marks of violence upon it, is found. While in many cases the attendant circumstances may give clear indication of accident, suicide, or homicide, there are numerous instances in which a reliable opinion can be formed only after the most meticulous investigation of the scene of the occurrence of the injuries, and of the injuries themselves, together with all the circumstances.

The criminal authorities expect that when a medical examiner is called to examine a body at the scene of possible crime, he shall note every point on and around the body which might throw light upon the circumstances attending the injuries. Among the most important points to be noted at the locus are the position of the body, with relation to prominent objects of furniture, and to weapons, if these are present, and the incidence of blood-stains on or near the body and upon objects or weapons.

The general appearances of the surroundings of the body, for evidence suggestive of a struggle should never be overlooked, together with the physical appearances of effused blood. The importance of

the latter observation is considerable, since the formation of pools, splashes, smears, trails, or jets frequently plays an important part in the reconstruction of events (see p. 324). The direction of the jets or splashes and their location in relation to the surfaces of the body are likely to throw possible light upon the position of the deceased at or about the time of the infliction of the injuries. The hands should be inspected for blood-staining, weapons, hairs, pieces of torn clothing, and other objects. The suspect weapon, when

FIG. 142

Photograph showing scene of murder. Note many details which should be recorded prior to disturbance of body.

present, should be scrutinised for blood-staining and its applicability for the infliction of the injuries found should receive detailed consideration. This examination, so far as handling is concerned, should be deferred until the finger-print experts have concluded their investigation (see p. 74).

By careful examination of the scene of crime, valuable information regarding the reconstruction of events may be possible.

ILLUSTRATIVE CASE

In a case of culpable homicide in which an old man, who was brutally attacked by a man of twenty, died as the result of the violence, an examination of the locus made it clear that the assault had been a prolonged one. It had commenced at the entrance to a cottage and had been continued along the passage and in the kitchen. The instruments of attack were pieces of a wooden curtain pole. Human tissue was present on the floor of the passage, on the leaf of a kitchen table, on the walls, and on a gilt frame of a picture. Several of the pieces of the pole showed attached hair, and hairs were also adherent to different articles. A comparison

between these specimens and the hair on the head of the victim showed that all were generally consistent with a common source. Blood-staining, which was copious and diffuse, was present on the front door of the cottage, in the passage, and in the kitchen. The assaulted man survived for some twenty-eight hours. His injuries, which for the greater part involved the posterior half of the scalp, over an area of 7½ by 6 inches, were lacerated in character. Multiple wounds were present, and the majority which extended to bone had caused separation of the scalp from the skull. Several flaps, in irregular form, were attached to the posterior margin of the front half of the scalp. Considerable areas of scalp tissue were missing from points where the skull had been completely denuded of tissue (see Fig. 93). Other lacerated wounds were present on the face, and there was fracture of the bone of the nose and the left malar bone, together with two linear fractures of the skull. The irregular surface of one end of a piece of curtain pole, thought to have been used, was considered responsible for the scattering of the small portions of tissue during the attack.

Cut-throat wounds.

Passing now to the consideration of homicidal incised wounds, those of the throat are of frequent occurrence. Homicidal wounds may be found on the throat at any level, and their direction is dependent upon the relative positions of the assailant and his victim. For this reason, these wounds may assume a variety of directions in different cases, more especially when a struggle has taken place and the ultimate wounding has been inflicted by the assailant in a frenzied state. It is unwise to attempt differentiation between homicidal and suicidal cut-throat solely on the grounds that the former has a more gross appearance and shows greater severance of tissues than the latter. It is true that in a number of suicidal cases the wound may have a rather less pronounced character, that there may be tentative and trivial wounds on the neck, and that the severance of tissues may not be so extensive as in homicidal cases, but in others the injuries may be very extensive. In one of our cases, the suicide, who was insane, inflicted a wound which was 8 inches long and severed completely

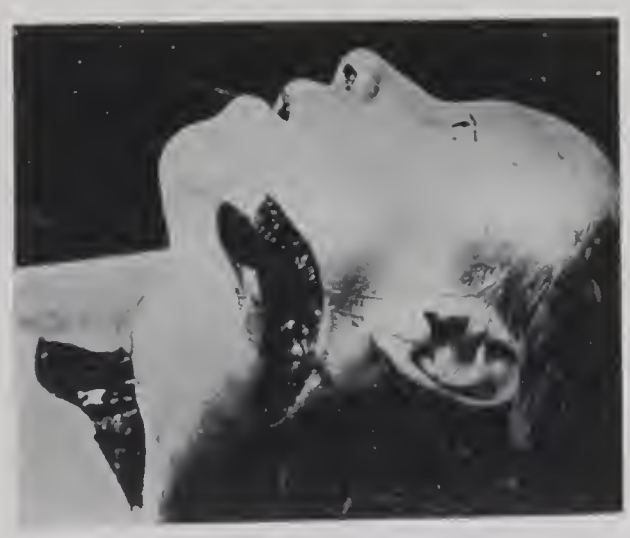


FIG. 143

Case of homicidal cut-throat. The lower wound, on slightly higher level than clavicles, severed muscles, blood-vessels, and trachea.

all the tissues of the neck down to the vertebral column, the anterior aspect of which showed a surface cut caused by the razor used. In homicidal cases there may be multiple deep wounds inflicted in different directions, and their multiplicity, together with the injuries produced, might clearly indicate that the wounding had been continued after volitional movements of the victim had ceased.

All the circumstances must be placed under strict review in certain cases, before a differential diagnosis between homicidal and suicidal cut-throat can finally be made. Defensive incised wounds upon the hand or hands of the victim are reliable indications of the hands having been interposed between the weapon and the victim's body, and are thus presumptive of homicidal attack (see Figs. 144 and 145). Such wounds should not be confused with wounds made over the wrist in a previous attempt at suicide by severance of arteries.

FIG. 144

Incised defensive wounds on back of hand.

In suicidal cases, when a right-handed person cuts his throat, the wound is inflicted from left to right and, as he makes the wound, considerable pressure is applied to the cutting instrument. When the wound has been well established, the pressure is decreased until finally the blade is withdrawn from the surface of the neck. As a result, the part of the wound which has been inflicted initially is of greater depth than the terminal part, made during the gradual withdrawal of the blade, which becomes progressively more shallow in character until the extremity of the wound is reached. The tailing of the wound is therefore indicative of the site of the withdrawal of the blade. The line of the wound is usually from left to right and usually from slightly above downwards and across the neck. If, however, the instrument is held in the left hand there will be a reversal of most of these features. While in some cases of suicide, tentative wounds may be present on the neck, they are by no means common to all. Since a suicide usually extends the head preparatory to inflicting the wound, its line may be more or less transverse, and the great vessels of the neck may escape serious injury as the result of the protection which is afforded by the sternomastoid muscles and the prominence of the larynx. The air-passages are frequently injured. In order of frequency, the sites of injury are over the thyroid cartilage or the thyro-hyoid membrane, over the cricoid cartilage, above the hyoid bone, or over the upper part of the trachea.

In cut-throat injuries, when death results, the cause of death is hæmorrhage, or septic pneumonia brought about by aspiration, or the effects of local infection. Aphonia is a frequent manifestation due to injury of the recurrent laryngeal nerve or nerves or of the vocal cords.

FIG. 145

Case of homicidal cut-throat. Multiple wounds on neck. Wound on right hand caused by the victim attempting to shield herself.

In certain cases of cut-throat wounds, the body of the wounded person may be found either close to the spot where the wound was inflicted or at some distance from it. In the latter event, there will probably be a trail of blood leading to the place where the body is found. The distance between these points will vary very greatly in different cases.

In one case a man, after inflicting a wound on his throat, walked a distance of 200 yards before he finally ended his life by drowning.

In another, where a young man killed his sweetheart on the public road by cutting her throat, it was evident from the commencement of the blood-marks on the footpath to the point where her body was found that she had travelled a distance of 105 yards.

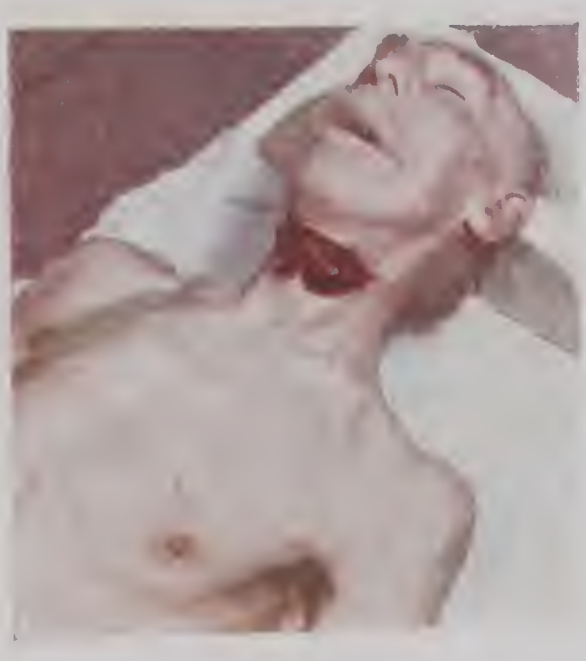


FIG. 146

Suicidal cut-throat.

The possible distance which a person may walk after receiving the wound will largely depend upon the rapidity of the loss of blood. In those cases of suicidally inflicted throat wounds in which the suicide has ended his life in some other way, it is usually found that the injuries to the throat are not extensive and that important vessels have not been cut. When the large blood-vessels have been severed, the rapid and extensive loss of blood will quickly induce collapse, and therefore the distance travelled after the infliction of the wound will probably be short. When the question of distance traversed becomes one of some importance, the answer must be obtained from a careful examination of the wound or wounds on the throat and consideration of the general vigour during life of the person wounded.

Illustrative case of homicidal cut-throat.

On the front of the neck there was a large, gaping wound, which extended from ear to ear. The right extremity was $1\frac{1}{4}$ inches below

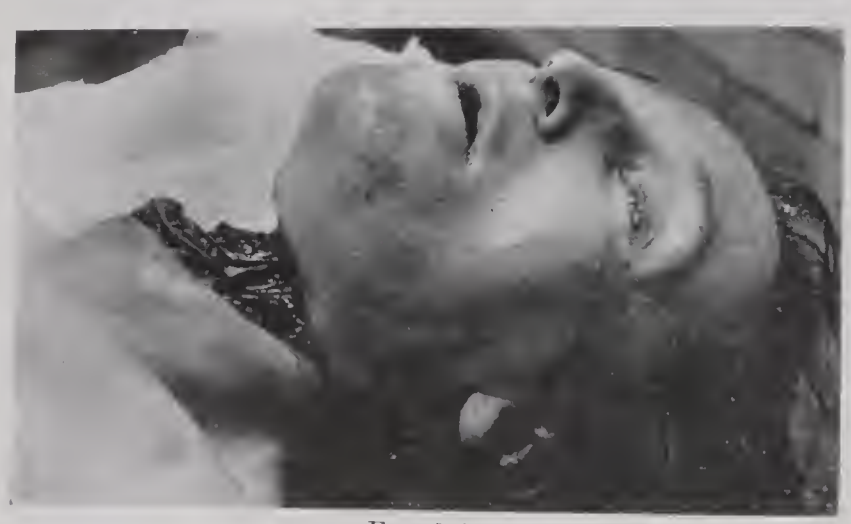


FIG. 147

Homicidal cut-throat.

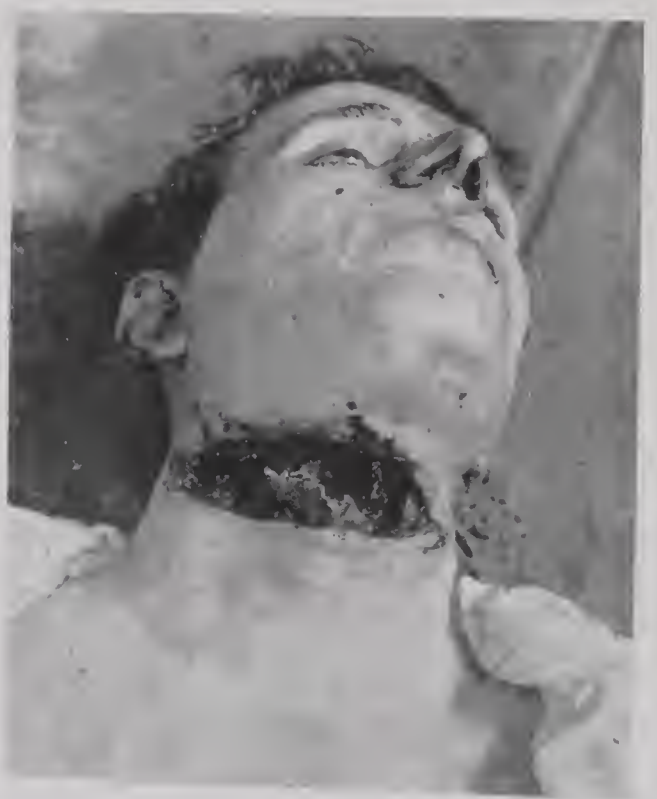


FIG. 148

Case of suicidal cut-throat. Thyroid cartilage was divided, together with all the structures down to the deep vessels on both sides. The left internal jugular vein was the only one of these vessels injured.

the level of the lobe of the right ear and $1\frac{1}{2}$ inches behind it. The left extremity was situated $2\frac{1}{2}$ inches below the level of the lobe of

left ear and directly below it. At each extremity there were three distinct tails or gashes. The wound measured 8 inches in length, and its gape was 3 inches. All the muscles in front of the neck, the important blood-vessels and nerves, together with other structures, were severed down to the cervical spine, and the bodies of the third, fourth, fifth, and sixth vertebræ showed surface injury by the weapon. Lying in front of the vertebral column there was a sharp-pointed, sharp-edged piece of steel, measuring $\frac{1}{4}$ inch long and $\frac{1}{8}$ inch broad, which filled, accurately, a gap in the razor which was found. Between the under surface of the point of the chin and the principal wound were numerous superficial scratches running at different angles. On the back of the right, middle, and ring fingers there were superficial wounds caused by the weapon.

Stab wounds.

Homicidal stab wounds are most frequently found on the neck, chest, and back. Suicidal wounds, as the result of stabbing, are

FIG. 149

Stab wounds, eleven in number, on front of chest and abdomen caused by pocket-knife. These wounds penetrated heart, lung, and left lobe of liver.

comparatively rare. The homicidal infliction of such wounds on the abdomen are not so frequent, in our experience, as those on the chest or neck. Hæmothorax is a frequent complication of penetrating wounds of the chest, and the effusion of blood varies widely in quantity. The amount may be relatively small, but in some cases the pleural cavity may contain 2 or 3 pints of blood. Injury of the intercostal vessels or of the lung, or both, is usually the source of the bleeding. Pneumothorax, hæmo-pneumothorax, or subcutaneous emphysema, are conditions which often result from such injuries of the chest. Stab wounds of the chest frequently pierce the heart or large vessels. The aorta is often injured at some point situated close to the heart. In many cases of homicide the wounds are multiple and their number will often give a clear indica-

tion of the determined character of the attack (see Fig. 149). When the weapon has been directed to the abdomen, a variety of injuries may result, and almost any of the abdominal organs may be

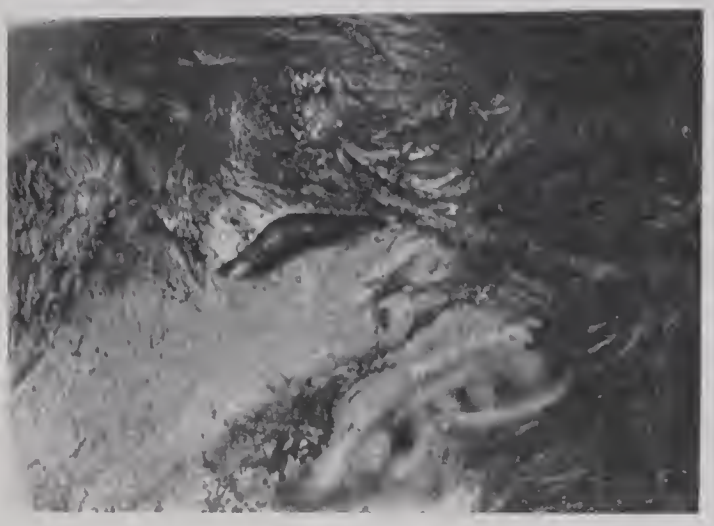


FIG. 150

Stab wound on back of neck, which penetrated neural canal between base of skull and atlas, with severance of right vertebral artery.

FIG. 151

Stab wounds on chest and abdomen. Note incised, defensive wounds on wrists.

implicated, although the liver, stomach, and intestinal tract are commonly involved. Occasionally there may be a protrusion of bowel through the wound (see Fig. 151). Stab wounds of the neck commonly result in injury to the carotid vessels and jugular veins. Certain stab wounds may be almost imperceptible on the body surface if fine weapons have been employed, for example, narrow-bladed knives, needles, hat pins, etc. When exploring wounds produced by stabbing, it is sometimes noted that disparity exists between the length of the peccant weapon and the depth of the wound track. This is due to the impact of the weapon compressing the tissues struck (see p. 292 and Fig. 153). Volitional movements, following serious injury, may be retained for some time (see p. 276). In one of our cases a vigorous young man ran after his assailants for a distance of between 40 and 50 yards before collapsing, despite the fact that post-mortem examination showed an incised wound which passed through the wall of the left ventricle and measured $\frac{1}{4}$ inch in length.

ILLUSTRATIVE CASES OF STAB WOUNDS

Suicidal stab wounds by hat-pin.

One night a woman arrived by train at a town near Glasgow, she was obviously ill and the police were called. On being taken to the police station, a long hat-pin was found sticking through her neck from front to back. After medical attention she was conveyed to hospital, but died soon afterwards.

On the left side of the neck, $2\frac{1}{2}$ inches below the lower jaw, and $\frac{1}{2}$ inch to left side of mid-line of the neck, was a small punctured wound. On the back of the neck to right side of spine was a dark reddish point which, on being incised, exuded some blood. One and a half inches below the left nipple, and $\frac{3}{4}$ inch to inner side, was a small punctured wound, and $\frac{1}{2}$ inch above it were two superficial puncture marks.

On dissection of the tissues of the neck, it was found that the perforating wound had passed through the neck from front to back, but had not entered any important blood-vessel or seriously injured any important structure. The puncture wound on the left breast had entered the intercostal muscles between the fifth and sixth ribs, the pericardial sac, and the left ventricle. The left pleural cavity contained 17 ounces of fluid blood. It was apparent that the weapon had been moved after insertion, either deliberately or by the act of breathing.

Homicidal stab wound of left eye.

The upper lid of the left eye was bruised and swollen. An incised wound, measuring $1\frac{1}{2}$ inches, was present on the upper eyelid close to eyebrow and perforated the eyelid. Examination of the interior of the skull showed a considerable amount of clotted blood covering the left half of cerebrum. At the base of the brain

and in a line with the back of the left orbit was a wound which had severed the pons for about two-thirds of its breadth. Further examination showed that the weapon had passed between the upper part of the eyeball and the roof of the orbit in a backward, inward, and slightly upward direction, had slightly injured bone at the back of the orbit, and had severed the internal carotid artery within the skull cavity. The entire length of the wound from the eyelid inwards was almost 4 inches. The weapon, on which were found blood-stains, was an ordinary table-knife. The blade measured $5\frac{1}{2}$ inches, and its breadth, which was uniform, was $\frac{7}{8}$ inch.

Homicidal stab wound of neck.

A punctured incised wound, $\frac{5}{8}$ by $\frac{3}{16}$ inch, was situated $1\frac{1}{2}$ inches below the lobe of left ear, and almost parallel to lower surface of the lobe. The wound penetrated the internal jugular vein and pharynx.

Homicidal stab wound of neck with broken bottle.

An incised wound, $2\frac{1}{2}$ inches long, was situated on the left side of the neck. It commenced at the base of the anterior attachment

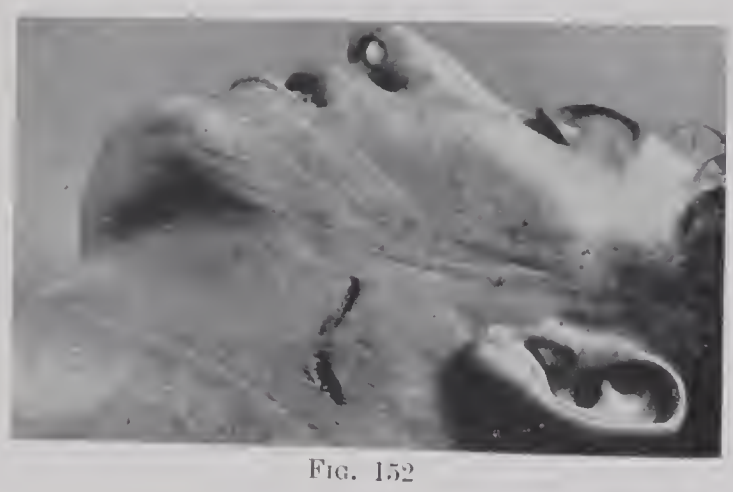


FIG. 152

Stab wounds on neck.

of the ear and extended downwards on to the neck. The upper part of the wound involved the lobe of the ear from which a partially severed portion was still attached to the side of the face by a slender strip of skin. The wound on the neck increased in depth from above downwards and attained a maximum depth of $1\frac{3}{4}$ inches. The internal jugular vein had been penetrated.

Homicidal stab wound of back.

A spindle-shaped, incised wound, measuring $\frac{1}{2}$ by $\frac{1}{8}$ inch, was situated at a point $1\frac{1}{4}$ inches to right of middle line of vertebral column and over tenth interspace. A second wound was present on the immediate right of the vertebral column and 3 inches above the first wound. It measured $\frac{3}{8}$ by $\frac{1}{8}$ inch and presented similar

characters to the first wound. There were also incised, defensive wounds on the right index and middle fingers, and on the left thumb, together with a linear wound of the epidermis covering the front of the right wrist. The right pleural cavity contained 4 pints of blood, and there was an incised wound on the lateral aspect of the right lung, 2 inches above its basal margin. The injury to the lung, which measured $\frac{1}{2}$ inch in length and $\frac{5}{8}$ inch in depth, had been caused by the entry of a knife through the lower of the two wounds on the back.

Homicidal stab wound of chest.

A wound was situated over the sterno-clavicular articulation of the left clavicle, measured $\frac{3}{4}$ inch in length, and entered the common carotid artery. Death was practically instantaneous.

Unusual fatal stabbing.

The wound was on the front of the chest, $\frac{1}{2}$ inch below the level of the left nipple and $\frac{1}{4}$ inch to the left of the mid-line. It penetrated

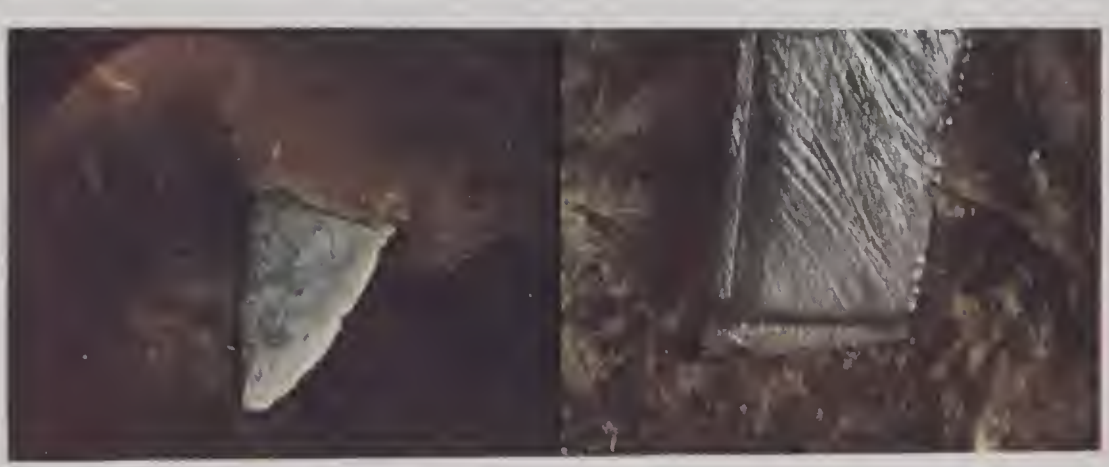


FIG. 153

Posterior surface of sternum showing projection of embedded portion of blade which passed inwards for a distance of $\frac{5}{16}$ inch, causing laceration of anterior wall of heart (see Fig. 154).

FIG. 154

Broken end of pocket-knife blade embedded in sternum. Photograph of anterior surface of the bone. Note base of broken part of blade of suspect knife placed almost in contact with embedded portion of blade (see Fig. 153).

the sternum opposite the inner end of the fourth left costal cartilage. The broken end of a pointed pen-knife blade was embedded and projected through the inner surface of the bone for $\frac{5}{16}$ inch. The blade had been sheared on the outer surface of the bone with which it was flush. The anterior mediastinal tissues showed extravasation, œdema, and emphysema. The wound pierced the pericardium and also the underlying anterior wall of the heart, just below the right auriculo-ventricular border which showed a very irregular, excavated lacerated wound $\frac{3}{4}$ inch long. Although the wound passed deeply into muscle it had failed to effect entry through the endocardial surface. The character of the wound was due to repeated contact

between the heart wall and the point of the embedded broken blade caused by combined action of the heart and breathing prior to death. A suspect's knife was submitted for examination when it was found that the larger blade had been broken. Comparison between the embedded portion of the blade and the remaining part of the blade in the knife was fully consistent with the two pieces having been part of one blade (see Figs. 153, 154).

Before proceeding to further consideration of various forms of wounding, homicidal, suicidal, or accidental, it is expedient to deal with the subject of fractures of the skull and other bones, since many of the injuries to be described include such fractures.

FRACTURES OF SKULL AND OTHER BONES

Fractures of skull.

Such fractures may implicate either the vault or base of the skull, or both, and may be caused either by direct or indirect violence. When direct violence is applied to the vault, as, for example, by a blow, the lines of fracture radiate from the point of impact, and, speaking generally, fractures of the skull usually occur at the point or points at which the greatest force has been applied. The views of Rowbotham [10] are that the precise manner in which the bone breaks is determined by the fact that its tensile strength is less than its power to resist compression and therefore whichever table of the skull happens to be on the convexity of a bend, and thus subjected to stretch, will be the one to fracture first. A counter force must act on the skull at the same time as the injuring force, if a fracture is to occur, and the rigid support which the skull receives at its occipital condyles is one of the most important anatomical features concerned in the mechanisms of fracture of the skull and injury to the brain. Fractures of the vault often result also from the effects of indirect violence transmitted from the base, or as the result of compression of the skull which has exceeded the degree of its temporary expansion. When the vault has been the seat of injury, the fracture may be simple or compound, linear, or comminuted, with or without depression. The degree of compression, when present, will naturally vary and it may be slight in degree or extreme, as, for example, when fragmented bone is driven inwards, perhaps into the substance of the brain. In rarer cases, the fractured area may show some elevation. Fractures of the skull may be extensive yet unaccompanied by gross injury of the brain, but it must not be forgotten that serious intracranial injury may result from a simple fracture, or perhaps without fracture, as the result of contrecoup and consequent meningeal hæmorrhage or laceration of the brain substance.

With regard to fractures of the base, here again these may follow either direct or indirect violence. Direct violence is not commonly a cause, but one must not overlook the type of case in which there has been the passage of an instrument through the orbital cavity

or where a firearm wound has perforated the palate (see p. 302). Indirect violence is the common cause of basal fractures which are frequently produced by falls from a height when the feet or buttocks of the injured person strike the ground first. Fractures of the

FIG. 155

Comminuted fracture of skull with slight separation of fronto-parietal suture.

FIG. 156

Comminuted fracture of skull.

vault often extend to the base. When compression of the base is the cause of fracture, the line follows the path of least resistance, but is deflected from the thicker and more resistant portions of bone. Apart from penetrating wounds of the skull, the brain may also receive injury by distortions of the skull or by movements of the brain within the cranial cavity.

In all medico-legal post-mortem examinations, the membranes should be stripped thoroughly from the base so that the presence of fracture may be verified or excluded.

When violence is applied to the skull, certain factors play a part in the determination of the type and character of the resultant fracture. These include the position of the head when struck, the degree and nature of the violence applied, the area affected, whether the force applied is general or localised, and the thickness of the bone which varies considerably in different individuals. Turning to depressed fractures of the vault, in those produced by a blow with a heavy instrument of limited striking surface, or by a lighter instrument of similar striking area used with considerable force, the shape of the depression in the bone may bear an approximate resemblance to the shape of the striking area of the weapon used. Further, the inner table of the bone will be more extensively splintered than the outer table. Comminuted fractures are produced by violence from heavy objects with a fairly large striking surface or as the result of repeated blows, more or less over the same area, by an instrument of limited striking head. Surrounding the area of comminution, linear fractures, radiating in different directions, will usually be observed.

Complications of fracture of the skull.

Common complications of fracture of the skull include concussion, compression, and contusion or laceration of the brain. Several of these complications are often concurrently present. Concussion, in mild or severe degree, is a usual accompaniment of head injury, and there are several theories as to its cause. Trotter assumes the condition to be induced by a hyperacute compression of the skull which causes an instantaneous anæmia of the brain, the result of momentary diminution of the intracranial capacity. He defines concussion as "a condition of widespread paralysis of the functions of the brain which comes on as an immediate consequence of a blow on the head, which has a strong tendency to spontaneous recovery, and is not necessarily associated with gross organic change in the brain tissue. Gross anatomical changes are not necessary and the petechial and other hæmorrhages found are not the causes of concussion, but may sustain the paralytic manifestations produced by the concussion."

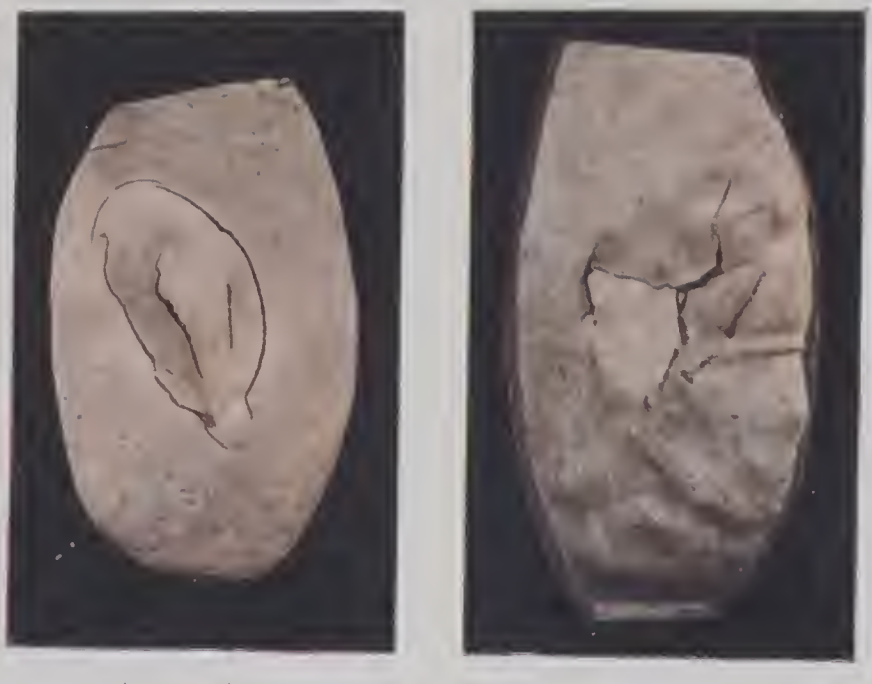
Denny-Brown and Russell[11] hold the view that to cause concussion, no deformation of the skull is necessary, and that it is a direct traumatic paralysis of nervous functions, without vascular lesion, persisting for a varying period according to the type and severity of the blow. The paralytic phenomena are due to a direct generalised physical injury of the neurone. In their experiments they found that an important factor was that the head should be free to move and that the velocity of movement necessary requires to be about 28 feet per second in "acceleration concussion." When the head was fixed and was unable to move, concussion did not result from blows delivered at this velocity. The suddenness of

acceleration was the factor of importance. They attribute concussion to reflex paralysis of the respiratory and vasomotor mechanism and of the motor mechanism of the pons and medulla. "Deceleration concussion" occurs when the head in movement is suddenly brought to rest. Compression concussion can occur as the result of crushing of the head. They did not find contrecoup injuries when the head was fixed at the moment of impact. Their view is that there is a cleavage between pia and cortex, strongly suggestive of a suction effect at the moment of injury, due to a momentary vacuum under the membranes opposite to the point of impact, and that a hæmorrhage will occur from the rupture of an artery into an area of surrounding lowered tension. The experiments of Holbourn [12] on the mechanics of head injury are of interest. He found that injury to the brain may result from distortion of the skull or without distortion. He used models, consisting of a gelatin "brain" inside a paraffin-wax "skull" and analysed the forces which could result in cerebral injury. He contends that shearing strain is the chief agent, and that the idea of coup or contrecoup injuries resulting from alternate compression and rarefaction due to free movements within the skull must be abandoned, since there is no appreciable relative movement between the parts as the result of linear acceleration. A certain amount of shear may occur below the point of impact, particularly if the skull is broken, and this would account for coup. A much greater shear strain develops as the result of rotation of the skull, and the fact that changes in the rotational velocity are usually greater at the pole opposite to the point of impact would account for the more extensive injury of contrecoup. His observations refute the notion that polar contusion is caused by movement of the brain in a straight line till it bumps on the far side of the skull, since he shows that contrecoup is the result of shear due to rotational injury. Practically all blows on the head rotate it, and the damage done to the brain depends on how firmly it is gripped by the meninges and internal conformation of the skull. Strains are called into play by the change in the velocity of the head caused by a blow. The principal motion is the sliding of the pia relative to the arachnoid and of the arachnoid relative to the dura, the latter being presumably greater. Thus the vessels which drain the cortical veins into the venous sinuses will be stretched and may rupture at any point, causing subdural or subarachnoid hæmorrhage. Jefferson suggests that the unconscious state characteristic of concussion is not primarily a cortical matter, but dependent on intraneural changes in the brain stem.

Intracranial hæmorrhage.

Extradural and subdural hæmorrhage are very frequently associated with cranial fracture. Extradural hæmorrhage is the result of rupture of the middle meningeal artery, diploic veins, or dural venous sinuses, and subdural hæmorrhage follows injury of the dural sinuses, cortical vein if arachnoid has been torn, or lacera-

tion of the brain with tearing of the pial vessels. When the dura has been torn, co-existent extradural and subdural hæmorrhage will be found, but both forms may be present without such injury to the



Outer table. Inner table.

FIG. 157

Depressed fracture of skull, showing outer and inner tables.

FIG. 158

Large extradural hæmorrhage.

dura mater. Subdural hæmorrhages may give rise to a large extravasation of blood which may fill the subdural space. Such hæmorrhages are relatively slow. Rarely a subdural hæmatoma may result from pathological causes apart from trauma, for example, inflammation.

Subarachnoid hæmorrhage may be caused by injury or result without trauma. Such hæmorrhages are found at the base of the brain, and the spread through the subarachnoid space may be very extensive in character. Frequently the hæmorrhage covers almost the entire surfaces of the brain. In some cases there may be rupture into the subdural space or into the brain substance, and the blood will find its way into the lateral ventricles. Bleeding is most commonly due to rupture of a vessel entering into the formation of the circle of Willis, but may be caused by rupture, through stretching or injury, of arachnoid trabeculæ due to a sudden and excessive movement of the head. The severity and the site of hæmorrhage, within the circle of Willis, vary greatly. Rupture of a healthy artery may occur as the result of violence, for example, a fracture of the skull associated with laceration of the brain. Intracerebral hæmorrhage may originate a secondary subarachnoid hæmorrhage. Spontaneous subaraehnoid hæmorrhage is also a well-recognised condition and is of frequent occurrence without the advent of recognisable trauma. It may occur in individuals of all ages, and the symptoms may be slight or the condition may prove rapidly fatal. The most frequent cause is rupture of a developmental miliary aneurysm, or developmental defect in the media of the vessels, in younger subjects, or arterio-sclerotic changes in the media, associated with high blood-pressure, in older persons. When rupture occurs there is usually no ascertainable determining factor. Physical strain, a heightened blood-pressure, a blow on the head or a knock on the face may precipitate rupture of a defective vessel. It is generally accepted that, from the medico-legal aspect, fatal cases with a history of slight violence require the most painstaking investigation in order to apportion the effect of the injury in relation to the fatal termination. To arrive at the opinion that trauma is the cause, objective evidence of mechanical injury should first be obtained. Such investigation necessitates the removal of clot with the greatest care to avoid injury by artifact, together with dissection of the vessels entering into the formation of the circle of Willis. A solution of hydrogen peroxide is very helpful in aiding the removal of clot, and dissection of the vessels is facilitated when performed under a shallow covering of water following suspension of the brain in a hammock of gauze. When the vessels have been

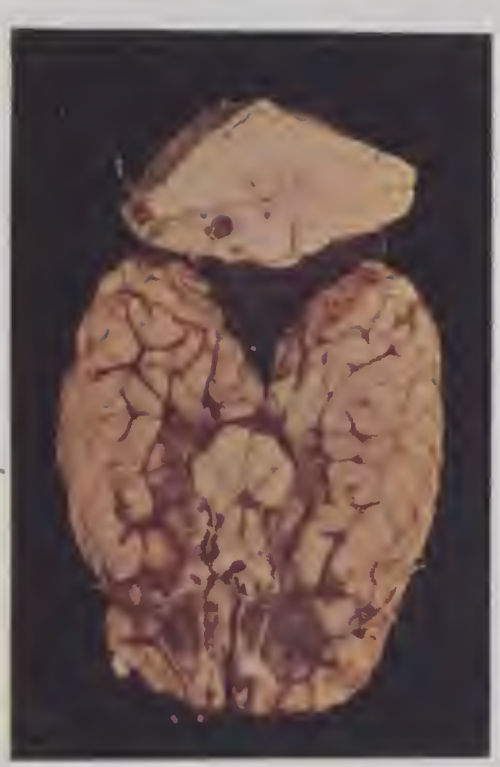


Fig. 159
Subarachnoid hæmorrhage.

dissected out, a glass plate should be placed under the surface of the water in the container in which the dissection has been performed, and the vessels floated upon its surface. The plate should then be gently withdrawn leaving the vessels spread upon it. To detect latent perforation, the injection of a coloured solution, for example, methylene blue, will be found valuable. When internal defect is being sought, microscopical examination of serial sections may prove necessary. We have had to investigate a number of cases of this character. Magee [13] has published a review of 150 cases of spontaneous subarachnoid hæmorrhage and has shown that the main incidence of aneurysmal rupture fell in early, or early middle, life. Autopsy was carried out in 58 cases and in 43 a ruptured aneurysm was found. In the remainder, the hæmorrhage was definitely confined to the subarachnoid space, but no aneurysm could be detected. In this series there was a high incidence of aneurysmal dilatation on the right side of the circle of Willis, and on the anterior offshoots.

It is interesting to note that out of forty-three boxing fatalities submitted to post-mortem dissection in only one instance was subarachnoid hæmorrhage found.

Intracerebral hæmorrhage is usually due to disease of cerebral vessels. When close to the surface, it may be the result of laceration.

Amnesia following head injuries.

Amnesia, or loss of memory, following head injuries is quite common and is usually associated with concussion. Russell,[14] who investigated several hundred cases, states that after head injuries, accompanied by immediate loss of consciousness, certain disturbances of memory occur, that on full recovery there is little or no recollection of events during the period of confusion following injury, and that after recovery there is no memory of the moment of injury. "Permanent" retrograde amnesia may vary from a period of seconds up to seven days, the latter being the longest period found in the investigation of over two hundred cases. He adds that, in over five hundred cases, he has never known a "permanent" retrograde amnesia to cover a period of more than a week before the injury, and that in his view it seems probable that cases in which it appears to last for years are really cases of hysteria. He stresses the further point that a retrograde amnesia of several years' duration may appear to be present before consciousness has fully recovered, because the memory for distant events tends to return before the memory of more recent occurrences. In cases recovering from concussion, events which occurred immediately before the injury are occasionally remembered indistinctly during the period of confusion, even though there will be complete amnesia for these events after consciousness has returned in full. This may result in the patient making false accusations. McConnell [15] states that prolonged amnesia, in certain cases, after cranial injury is

due to the presence of subdural fluid associated with reduction in the volume of the brain.

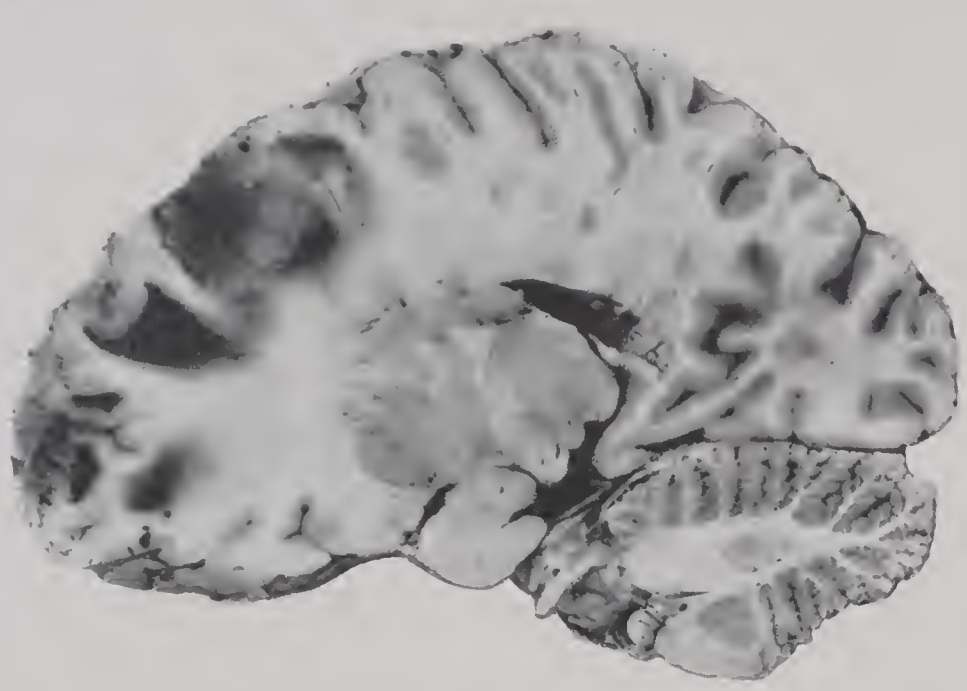


FIG. 160

Contusion of cerebral hemisphere showing multiple areas of extravasation.

Fracture of the spine.

The frequent sites of injury are the upper or lower part of the cervical, and the lower part of the dorsal or upper part of the lumbar, regions of the vertebral column. When direct violence has been applied, however, any portion of the spine may be the seat of fracture. The principal factors in the production of these fractures include direct crushing, compression due to a fall from a height, either the feet or buttocks striking the ground first, and over-flexion or extension. The act of judicial hanging produces fracture of the upper part of the cervical region, and the atlas or axis may be involved in this type of injury. When fracture occurs in the upper part of the cervical region and the cord is injured, death occurs rapidly, but when the lower regions are implicated, death may not occur, although paralysis of the parts of the body below the site of fracture supervenes rapidly, especially when displacement of the fragments occurs. The extent and character of the paralysis will obviously depend upon the position of the fracture. When the fracture is complete, due to either direct or indirect violence, separation is usually effected through an intervertebral disc to which is attached a piece of the body of the vertebra situated immediately below the site of separation. We have seen this type of lesion in a number of cases. Fractures which are incomplete assume many forms and include compression of the vertebral body, separation of the transverse process or spine, together with linear fracture of the vertebral body. In wounding by firearms, marked fragmentation of one or more of the vertebræ is sometimes

found. In a case of fatal wounding by a rifle bullet, both the third and fourth cervical vertebræ were severely comminuted and fragments of bone had been extruded through the wound of exit.

Fracture of other bones.

Fractures of other bones of the osseous frame may cause death on account of their situation, extent, or complications. Fractures of the ribs are of considerable importance in certain medico-legal cases, more especially when due to crushing, since adjacent and

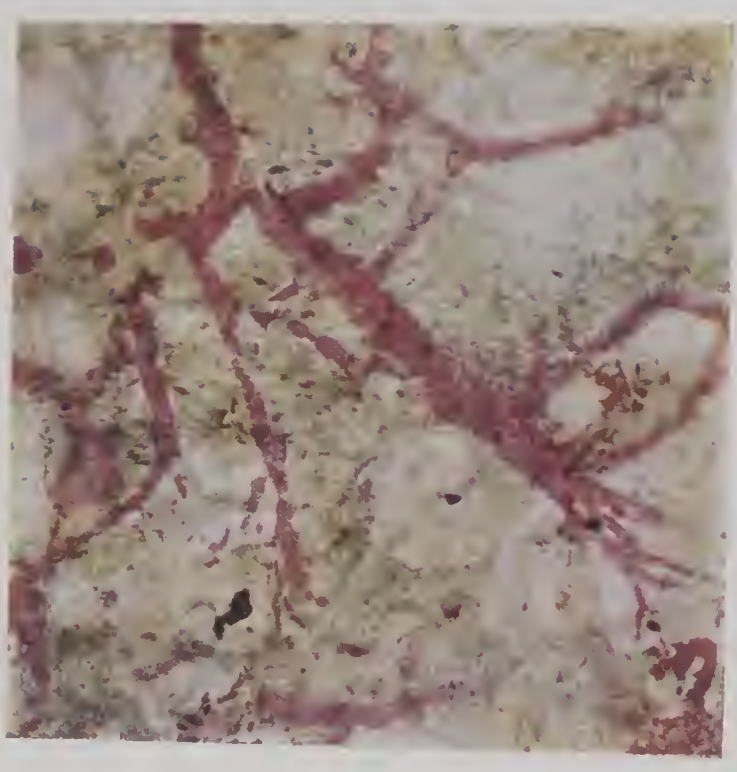


FIG. 161

Fat embolism of the lung following multiple fractures. × 50 diameters.

important structures are readily injured, including frequently the lungs and less often the heart. Laceration of the lung is often associated with fracture of one or more ribs, and hæmothorax, pneumothorax, interstitial emphysema, intrapulmonic and intrapleural hæmorrhage, pleurisy, pneumonia, pulmonary abscess or air embolism are complications to be considered. Injury to lung, for example, bruising or laceration, may result from crushing or impact without rib fracture. We have examined the bodies of many persons, especially of those run over by vehicles, in which such injuries have been found. Rib injury sometimes occurs in the course of restraining violent insane persons, and occasionally a suspicion may arise that greater physical force than necessary may have been employed with ensuing fatal results. In the investigation of such cases, the fact

that certain asylum patients, especially those suffering from general paralysis of the insane, may be affected by excessive fragility of bone should not be forgotten. In these cases the ribs are readily fractured, and the examiner making the post-mortem examination should test the frangibility of the unbroken ribs and, if thought necessary, retain a specimen for pathological investigation. When bones are fragile, forcible movements of a body after death are occasionally responsible for fracture.

Possible fractures of the pelvis in accidental injuries due to falls from a height, crushing, and vehicular accidents, should always be looked for.

Fat embolism is an occasional fatal complication of fractures of the long bones. Post-mortem examination of tissue microscopically, with hæmalum and Sudan III staining, will show fat globules in the vessels of the lungs, brain, or renal glomeruli (see Fig. 161). Robb-Smith [16] reported 115 fatal accidents, in 35 per cent. of which fat embolism occurred, and in 25 per cent. of the series this condition was regarded as a major factor in the causation of death.

Head injury.

Head injuries are most usually accidental or homicidal, and more rarely suicidal. In the differentiation of cause, the attendant circumstances are of great importance, since it is by no means difficult to conceive cases in which, from the examination of the body, it would be almost impossible to distinguish with accuracy between accident and homicide.

ILLUSTRATIVE CASES

The following cases of head injury illustrate some of the different types of lesion which may be found.

Caused by poker with rounded head.

Twelve lacerated wounds were present on the scalp. An extensive compound comminuted fracture involved the right frontoparietal region of the skull. Many pieces of bone, mixed with lacerated brain tissue, were present in the site of injury.

Caused by sharp-pointed poker.

Both eyes were bruised. In the left lower eyelid, towards inner canthus, was a lacerated wound, $\frac{7}{8}$ inch in length, which passed through tissues of lid, penetrated tissues on inner side of orbit, and fractured orbital plates of upper maxillary and ethmoid bones. The surface of right lobe of cerebrum was covered by a thin layer of extravasated blood, which was more marked on front and under surfaces of right frontal pole. A laceration of brain-substance, which admitted tip of little finger, was situated on under surface of right frontal pole into which the crista galli of ethmoid bone had been driven. The assaulted man lived for five days. These injuries were caused by a violent thrust of the instrument.

Caused by a fall on kerb of pavement following assault.

A fracture involved the base of skull, extended to the vault, and measured $3\frac{1}{2}$ inches in length. A subdural hæmorrhage was present. In addition, on vault of skull was a depressed fracture composed of irregular bone, which had been partially rounded off, thus indicating that it had been present for at least several years.

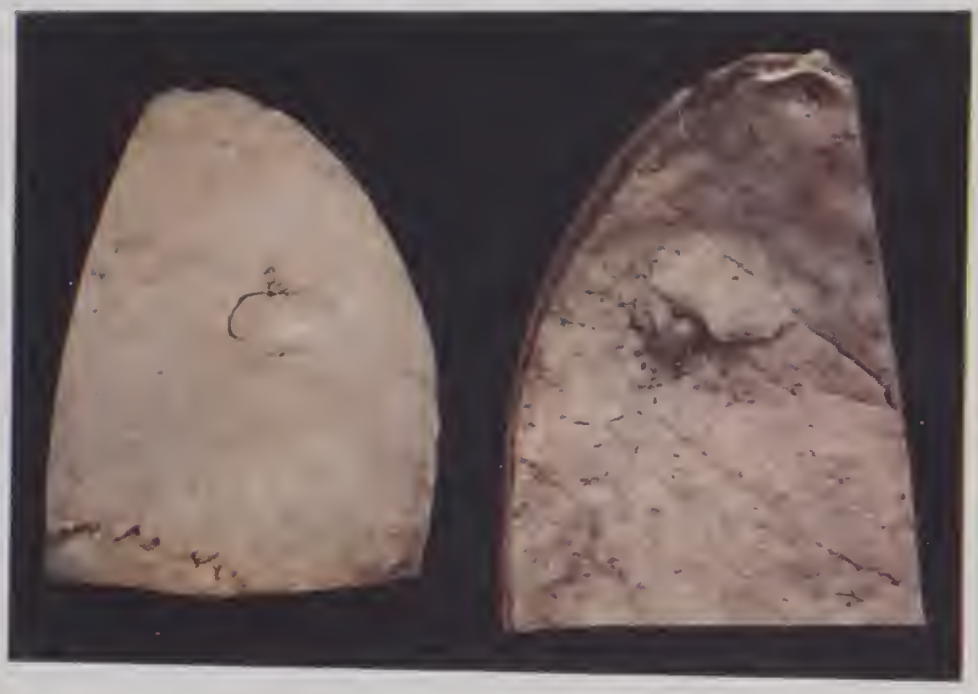


Fig. 162

Localised, depressed fracture of skull, outer and inner tables, showing evidence of repair.

It was ascertained that twenty-one years before, the deceased had been struck on the head with the pointed end of a slater's hammer (see Fig. 162).

Caused by corner of axe.

Two and a half inches above upper level of lobe of left ear was a clean-cut wound, measuring $1\frac{1}{2}$ inches, which passed to bone. Immediately beneath the wound was a depressed fracture of skull, $\frac{7}{8}$ inch in length. This involved inner table of parietal bone, which was broken into four pieces, over an area of $3\frac{1}{2}$ by 4 inches. There was an extensive extradural hæmorrhage. The brain tissue was contused over an area of 2 by $1\frac{1}{4}$ inches.

Following the injury, the deceased left his house, proceeded to the police station to get his injury dressed, but left because he would not await the arrival of the surgeon. He continued drinking during the course of the afternoon and evening, and late at night went to sleep in an empty house, with a companion, where, in the morning he was found dead.

Homicidal injuries caused by unknown weapon.

The following wounds and fractures, among others, were present : A crescent-shaped, incised wound, 1½ inches, exposing frontal bone 2 inches above level of right eyebrow. The frontal bone was fractured at base of wound. On the scalp, 1½ inches behind wound described and 4 inches above lobe of right ear, was a second incised wound, 1 inch long, which penetrated to bone. Farther back on the scalp was a third incised-wound, gaping in character, from which brain-tissue emerged and which measured 3 inches. In addition, there were two incised wounds of small size which penetrated to bone. There had been bleeding from left ear.

The deep tissues on right side of head showed extravasation over an area, 9 by 4 inches. The bones forming right side of skull were comminuted, and detached portions of bone varied in size from the finger-nail to the palm of the hand. Lacerated brain-tissue mixed with bony debris protruded from the skull opening.

On the surfaces of both lobes of cerebrum was an extensive subdural hæmorrhage. There was also a fracture of base of skull on right side of anterior fossa over roof of right orbital cavity. The suture between occipital and parietal bones was separated. This was more marked on right side.

Unusual injury by broken bumper of motor car.

The victim, while at a distance of 30 feet from a collision between two motor lorries, was struck on the head with a piece of metal, weighing half a pound, which, having been broken off the front bumper of one of the vehicles, shot in the direction of the fatally injured man. The piece was impacted in the right frontal region of skull. The bone was comminuted over an area of 3 by 4 inches. Extending backwards from posterior margin of this area was a linear fracture, almost parallel to middle line of vault and at 1¼ inches to right of it, which terminated at anterior border of occipital bone. Two further linear fractures forked downwards from the first. The area of comminution of frontal bone was continuous with a wedge-shaped, and almost completely detached, portion of bone which had its apex in the central part of anterior fossa of skull. There was fracture of both orbital plates and of

FIG. 163

Localised, depressed fracture of skull.

cribriform plate. The sides of the wedge measured $2\frac{1}{4}$ inches, and the base, which was directed upwards, measured 2 inches. An extensive subdural hæmorrhage was present. The superior articular process of seventh cervical vertebra was also fractured and there was partial separation of sixth and seventh cervical vertebræ with exposure of intervertebral disc and surrounding bruising.

Caused by kick on face.

The nose was slightly flattened with some deviation to right, and there was bruising of both upper eyelids over the inner halves. Entire lower lids were similarly involved. Bruising affected bridge of nose. Other minor facial and body injuries were also present. Examination of the brain showed generalised purulent meningitis. There was fracture of fronto-nasal junction, basal fracture of crista galli of ethmoid and two fractures of the cribriform plate. Fracture of the cribriform plate is often associated with laceration of the dura and thus the risk of meningitis is introduced since the fracture communicates with the nasal passage.

Alleged fist blow on face and fall on stone floor.

Death occurred in an aged man eight days after the injury. There was a small localised extradural hæmorrhage, at base of brain, on floor of posterior fossa to the left of, and just behind, foramen magnum. A small localised subdural hæmorrhage was situated over right orbital plate and was related to an area of surface laceration on under surface of frontal pole of right cerebral lobe. The laceration continued backwards over the under surface of the lobe for a distance of $4\frac{1}{2}$ inches. There was also a laceration on under surface of left lobe of cerebellum. A smeared, subdural hæmorrhage was present over surfaces of brain, together with a similar hæmorrhage on under surface of cerebellum.

Severe head injury with recovery.

A man of thirty-one was attacked with a hammer. Three lacerated wounds were present on left side of head. They were roughly parallel, horizontal in direction and 1, 2, and 3 inches, respectively, above the external auditory meatus. The uppermost wound was 2 inches long and the lower wounds 1 inch. The surrounding tissues were pulped and fragments of bone and brain were evident on the surface between the wounds. On removal to hospital, the wounds were cleaned, excised, and stitched. Some bone fragments were removed. Patient remained unconscious for five days. A right-sided facial paralysis developed two days after the injury and persisted for twelve days. He was fed by nasal catheter. He was able to say such words as "yes" and "no" and to write legibly twelve days after the attack. He also appeared to understand all that was said to him and was able to feed himself. His condition had improved sufficiently thirty days after sustaining the injuries to allow adequate exploration

of the damaged brain. A scalp flap was turned down and many indriven fragments of bone with pulped brain tissue were washed out. About a cubic inch of brain tissue seemed to be missing. The dura was too damaged to allow suture. During the latter half of the operation a blood transfusion was maintained. The patient made satisfactory progress but aphasia persisted. He was discharged eight weeks after admission, walking quite normally. On the morning of the assault we inspected the scene of crime. The blows had been struck while the victim lay in bed. The pillow, densely stained with blood which had soaked through to the bolster beneath, showed two small pieces of brain tissue adherent to its surface. The wall at the head of the bed was considerably bespattered with small blood-stains, also the back wall and ceiling. Adherent to the ceiling were, what appeared to be, small pieces of brain tissue. We also examined a blood-stained joiner's claw hammer which had been found in the house. Following discharge from hospital, the man reported at intervals. At first the bone gap was very pulsatile, but gradually developed a dense fibrous protective layer. When seen two years later, he had been back at his former work for some months. He was instructed to report occasionally in case a "dural cyst" should develop. The writer is indebted to Professor J. A. G. Burton for the clinical notes.

Further examples of head injury will be found in the section, Vehicular and other Accidental Injury (see p. 310).

Causation of injuries.

On the general question of wounding in relation to accidental, suicidal, or homicidal causation the field is too wide, both as to the types of wounds and their attendant circumstances, to permit of a dogmatic statement of guiding principles which would be of absolute value to the student. It is in this matter that experience, powers of observation and interpretation are brought into operation, more especially when the evidence is of a purely circumstantial nature. An examiner will be well advised to form conclusions with caution, since circumstances may prove deceptive. In many cases, however, there is no reasonable doubt that the wounds have been caused by homicidal infliction.

Should circumstantial evidence lead to the apprehension and trial of a suspected person, while there may be no doubt regarding the wounds on the body of the deceased, there may be, and frequently is, doubt regarding the manner in which they were produced, or the type of weapon used.

One of our cases illustrates the point. A woman was found dead, lying on her back in a bedroom with a number of bruises and some abrasions on the body, scalp, forehead, left eye, arms, and legs. Among several blood-stains upon the carpet, the principal one, which had saturated the material, was some inches to the right of the head. Two upper teeth in a denture were broken, pressure had been applied to the tissues of the neck, and some of the abrasions had the appearance of having been caused by finger-nails. Her husband was

charged with murder. The evidence, on behalf of the Crown, was to the effect that death was due to cardiac arrest during the process of asphyxia, and that the character and distribution of the injuries were inconsistent with self-infliction, although some were consistent with accident. The woman had taken a large quantity of alcohol, and analysis of the blood showed that the quantity was equivalent to almost a bottle of whisky. Evidence for the prosecution was that this amount might have rendered the woman liable to sudden cardiac collapse, that the pressure to the neck had been applied by human hands, and that the injuries might have been caused by a person endeavouring to restrain the woman, although the amount of alcohol consumed would probably have caused a condition of coma rather than one of violence. Evidence regarding the broken

FIG. 164
Localised, depressed fracture of skull produced by the tooth of a rake.

teeth was that the fracture was consistent with violence, and that if they had been broken by striking the edge of the kerb around the fireplace, they would have been more shattered. Medical evidence, on behalf of the defence, was that the woman might have died from the effects of alcohol, suffocation might have been caused by regurgitation of stomach contents into the larynx and trachea, that the bruising could have been caused by a fall, and that the scratches might have been caused by an attempt to lift the limp body, which would be heavy. The charge of murder was reduced to one of culpable homicide. The jury returned a unanimous verdict of not guilty.[17]

Sometimes there are strange vagaries in the manner of suicidal wounding, and when a suicide is suffering from insanity, well-developed in form, the wounds are often very extensive in character.

With regard to injuries in general, all that can be said is that the

whole circumstances of the wounding, and the environment of the body when found, must be completely observed, considered, and weighed before an opinion is expressed regarding accidental, suicidal, or homicidal causation.

Homicidal injuries.

The two cases which follow are illustrative of typical homicidal injuries :—

A woman was found dead in her shop. The wrists had been firmly bound together by strong, cord-like material, and beneath

Fig. 165

Homicidal strangulation. Position of body as found (see Fig. 66).

the body was an axe, with its wooden shaft broken into two pieces. Lying on the floor was an empty glass siphon-bottle.

The forehead and top of head showed multiple wounds. No other marks of violence were present on any other part of the body. The vault of the skull was severely comminuted and pieces of broken bone lay in the scalp tissues. Some of these fractures extended into base of skull. The frontal lobes of brain were severely lacerated.

and the surface and base of brain were covered with effused blood. From the appearances of the wounds it seemed likely that they had been caused by the weapon found, the head of which showed both axe and hammer-head formations.

In the second case, the face, trunk, arms, and thighs of the woman's body were covered with dirt mixed with blood. There were several wounds on the left side of face, all lacerated in character, the left ear was almost completely torn from the head, and the cartilage split in several directions. The wounds on the right side of the face were fewer in number, but the upper half of the right ear was also torn from the scalp. The upper lip on its inner surface was pulped, and protruding from between the lips was a fractured portion of the upper jaw to which some teeth were attached. Most of the wounds, almost uniform in size and shape, measured $1\frac{3}{4}$ inches in length, and were crescentic in form. The front of the chest and neck were extensively bruised, and on the left side there was a crescentic-shaped wound of the same length as the wounds on the face. The thighs and arms also were extensively bruised.

Dissection of scalp showed a pulpy condition of left temporal muscle with extensive extravasation and similar, but less marked, bruising of right temporal muscle. Dissection of the face showed extensive bruising of tissues, especially on left side, together with the following compound fractures : (*a*) floor of left orbit, (*b*) zygomatic process (comminuted), (*c*) upper maxilla (comminuted), (*d*) mandible, on right and left sides (comminuted), and (*e*) nasal bone. There were fractures of second, third, and fourth ribs on right side, each rib in two places, and of second and third ribs on left side. The appearances, together with the uniform size and shape of the wounds and bruises, were consistent with their infliction by a booted foot.

Visceral injuries.

Penetrating wounds of the abdomen may be homicidal, suicidal, or accidental. Danger to life arises from three principal causes, namely, shock, hæmorrhage, and sepsis. The application of violence by a blunt instrument may readily produce rupture of the internal organs without leaving any visible external mark of violence. Similarly, crushing of the abdominal wall with resultant visceral injury may leave practically no evidence on the surface, especially when death has been almost instantaneous. Such injuries are described under Vehicular and other Accidental Injury (see p. 310). The organs which may be injured by blows include the liver, spleen, small intestine, and the urinary bladder. The bladder in a distended condition, not uncommon in intoxicated persons, may be ruptured by a kick. We have seen cases of this character, in one of which the tear, situated on the upper surface, measured 2 inches. Rupture of the bladder may also occur from penetrating wounds or as the result of fracture of the pelvis. An interesting case of rupture of the liver, which resulted from external violence by a

forcible blow from the head of a second person, was examined by us. There was no evidence of external injury. The deceased man, who was able to return to his home, survived the injury for only some hours. Post-mortem examination disclosed a rupture of the liver at the junction of the right and left lobes. The abdominal cavity contained $1\frac{3}{4}$ pints of blood. Kicks and falling on hard objects, together with crushing, are responsible factors in the production of liver rupture. The right lobe is the common site of injury.

In another case a boy of fifteen, who was standing on a bunker in order to reach a shelf, fell on account of collapse of the bunker. He soon became ill. The abdomen became tender and distended, and the abdominal respiratory movements were restricted. There was dulness in the left flank which extended up to the left costal margin. Pain was referred to the left shoulder. Operation disclosed rupture of the spleen accompanied by a large internal hæmorrhage. The boy died while under the anæsthetic.

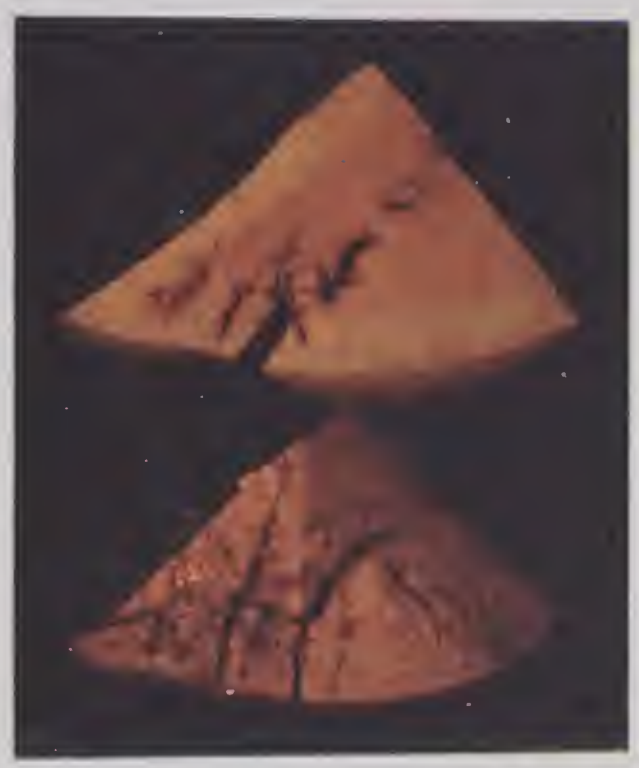


Fig. 166
Rupture of liver.

Cases of spontaneous rupture of the spleen have occurred in malaria.

Weston [18] describes the case of a girl, aged nine, who was running along a garden when she tripped and fell, striking her left costal margin and epigastrium on the edge of a step. Operation disclosed that the spleen was almost completely divided. Recovery took place.

Rupture of the stomach by external violence is uncommon. Kidney injury usually results from crushing, but sharp blows or forcible kicks may be responsible factors.

Osborne [19] has reviewed his findings in 262 fatal accidents with the following order of frequency of injury to viscera, namely, lungs, liver, spleen, heart, large bowel, small bowel, kidney, bladder, and pancreas. He also describes contusions of the lungs and heart.

Vehicular and other accidental injuries.

Injuries following vehicular accidents, especially in cases of running down by motor cars, are commonly in the nature of crushing injuries, with rapidly fatal results. Fractures of the skull

and ribs are of frequent occurrence, together with fractures of the vertebral column, pelvis, and any of the long bones. When the chest has been crushed and there are multiple and extensive fractures of the ribs, resultant injury to the lungs, heart, and great vessels is frequently seen. Rupture of the lung may be found without fracture of the ribs, as the result of compression.

In many cases of extensive visceral injury, there is no visible evidence of external injury, and this can be accounted for by the rapid advent of death. Recently, we examined the body of a man who had been killed through being wedged between a motor vehicle and a stone wall. The injuries included crushing of the chest wall, with multiple fractures of ribs on both sides, fracture of sternum, and severance of vertebral column in mid-dorsal region,

FIG. 167

Comminuted fracture of the skull due to vehicular injury.

with marked over-riding at the site of fracture. The visceral injuries included laceration of right lung with irregular separation of the three lobes, laceration of left lung, diaphragm, pericardium, and apical portion of heart which was detached. The thoracic aorta was severed at the atrium. The greater part of the right lobe of liver was extensively lacerated. Although there were a number of abrasions present on the surfaces of the body, there was no evidence of bruising externally.

In vehicular accidents, the lesions commonly found comprise multiple fractures of the ribs, with accompanying laceration of the lungs or heart. Hæmothorax and hæmopericardium are often present. Fractures of the skull, with laceration of the brain, are very frequent injuries, together with fractures of the pelvis and the long bones. In one case, which involved two small boys who were coming downhill in a home-made barrow when it collided with a commercial motor vehicle proceeding along the street, all these

injuries were present in both bodies. In addition, one of them also showed a fracture through the seventh dorsal vertebra with over-riding. The bodies did not show any evidence of bruising on the outer surfaces of the chest or abdominal walls.

Fractures of the mandible are commonly found and are often associated with extensive injury of the skull. In one of our cases the mandible was fractured in three places, the posterior part of the body of the sphenoid bone was detached, and there was rupture of the contiguous cavernous sinuses. In addition, there was an almost complete transverse severance of the medulla and pons. The fracture of the sphenoid bone extended laterally in different directions across the middle fossa to the temporal and the parietal bones on the right side, and to the basal portion of the temporal bone on the left side. A further fracture extended forwards from the sphenoid bone and led to an area of fragmentation of the roof of the right orbital cavity before passing upwards on to the frontal bone. The right occipitoparietal suture was mobile.

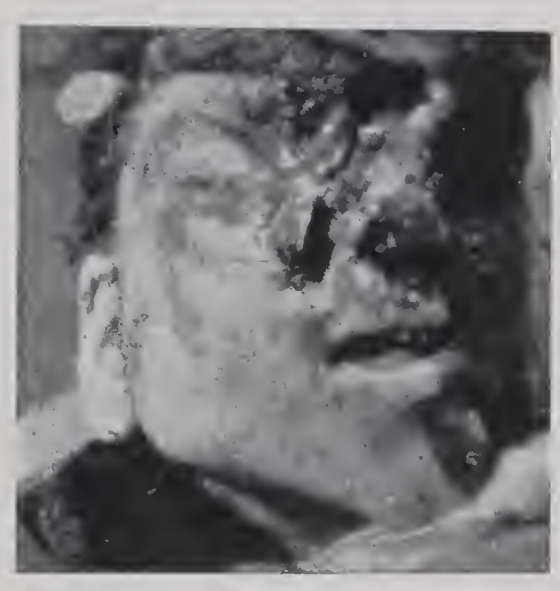


FIG. 168

Multiple lacerated wounds of the face, with fracture of underlying bones, due to crushing by an electric train.

In many cases a varying degree of protrusion of brain substance, the result of comminution, is present. Extradural, subdural, and subarachnoid hæmorrhages may be found together or alone, depending upon the nature and extent of the head injury (see p. 296). Especially when the head is involved, the hair and scalp should be examined carefully for glass particles and, any found, should be retained for subsequent comparison with glass from a suspect vehicle. A specimen of hair close to the site of wounding on the scalp should also be preserved for comparative examination with any hairs subsequently found adherent to the car thought responsible for the injury. In one of our cases, material taken from an offside door of a suspect car consisted of fourteen small pieces of tissue which varied in size from an ordinary pinhead to that of a glass-headed pin; histological examination established the fact that the fragments consisted of muscular, connective, and epidermal tissue. Serological examination showed that the tissue was of human origin.

A sample of blood should be taken at the post-mortem examination of the body of the victim, so that it may be analysed and the amount of alcohol present, if any, estimated, since the question of contributory negligence may subsequently arise. The blood should also be typed to facilitate subsequent comparison with suspect staining (see p. 342).

Crush injury and renal function.

Allusion should be made to the condition of crush syndrome. This condition may follow injury due to the pinning of a limb or limbs beneath heavy material for a period of several hours. The first signs are erythema or blisters of the compressed part with loss of sensation and paralysis. Swelling of the underlying necrotic muscle follows, due to the plasma leaking through the injured capillaries, and haemo-concentration results. Renal damage due to the substances absorbed from the necrotic muscle is probably produced as soon as the circulation to the damaged part is re-established, although often not noticeable clinically until some days later. The urine which may either be of a reddish or smoky hue, is acid in reaction and contains albumin. The damaged limb, or limbs, becomes progressively swollen and tense for four to five days. There may be vomiting and abdominal rigidity. The first week is the critical period. Skin and muscle may slough away and sepsis may supervene. Death may be sudden in those affected by this syndrome. The probable underlying cause of the condition is that myohæmoglobin from muscle produces renal failure by blockage of the tubules. Prognosis largely depends on the degree of muscle necrosis.

Unusual fatal accident.

A girl, aged thirteen, was found lying on the road. It was raining and becoming dark. Later she died in hospital from the effects of a comminuted, depressed fracture of the skull. The principal external injury was a lacerated wound of stellate shape, with three limbs measuring $1\frac{1}{4}$, $\frac{1}{2}$, and $\frac{3}{4}$ inches, situated immediately to the right of the mid-line of the crown of the head. The wound communicated with the interior of the skull, and the surface of the brain was exposed. The comminuted, depressed fracture, from which eight small bone fragments had been removed by operation, measured $1\frac{1}{4}$ by 1 inch. The inner surfaee, which showed the same measurement, had two small pieces of bone still partially attached to the edges, and four similar pieces had been completely detached. Underlying this area was a surface laceration of brain. Subsequently a request was made to examine the wheel of a motor bus in relation to the injury. A specimen of hair was removed from the edge of the scalp wound. On a solid disc of the wheel, ten six-sided nuts were mounted, and on one of them some staining was observed, together with a few adherent hairs.

The approximate measurements of this nut were as follows :—

Transverse measurement between angular points of sides = $\frac{7}{8}$ inch.
Measurement between parallel edges of sides = $\frac{7}{8}$ inch.
Length of side of nut and projecting bolt = $1\frac{1}{2}$ inches.

Certain surfaces of the nut, when impinged on plasticine, produced a stellate impression similar to the shape of the wound on the head of the girl. The stained material on the nut could not be identified either as mammalian or human blood, although

presumptive tests for blood were positive, since not only was the staining scanty but it had the appearance of having been diluted with moisture.

Fig. 169

Comminuted, depressed fracture of vault of skull. Nut of wheel is shown in situ.

The hairs, when compared with those from the head of the girl, showed characters which were fully consistent with a common source.

Examination of the footwear of the girl gave clear indication of marked and irregular wear of both soles and heels. These areas had been reinforced with material in a very amateurish fashion and would readily have caused the girl to stumble forward, more especially if she had been running with wet shoes and had tried to stop quickly. The injury upon the head was fully consistent with infliction by the wheel which had been examined, and could have been produced by the head falling forward and striking the nut while the wheel was in motion (see Fig. 169).

Unusual injuries resulting from lift accident.

A hydraulic goods lift became defective and fell from the fifth floor to the basement of a warehouse. At the time, the lift was carrying a load of carpets, and a porter was in charge. He was killed as the result of the following injuries :—

The fourth rib, on left side, was fractured in two places, 1½ inches to left of sternal border and at junction of middle and posterior thirds. The pericardium was penetrated, and the wall of the right auricle was perforated in two places. The anterior surface of the upper lobe of the left lung had been penetrated, the lung was collapsed, and the pleural cavity contained 4½ pints of blood. There was also a small perforation of the wall of the lower part of the ileum.

Injuries of genitals and genito-urinary and intestinal tracts.

Crane and Moody [20] describe the case of a boy who fell from a roof and landed astride a fence. He sustained a lacerated wound on inner

aspect of upper part of left thigh. Two weeks later pus was present in the urine. A month after the accident he suffered from pain and difficult micturition which were accompanied by swelling of the penis. A piece of wood, 3·5 by 0·5 centimetres was removed from pendulous part of urethra. Six days later cystoscopic examination showed a larger fragment in the bladder and, on left lateral wall, the site of entrance was observable. This fragment measured

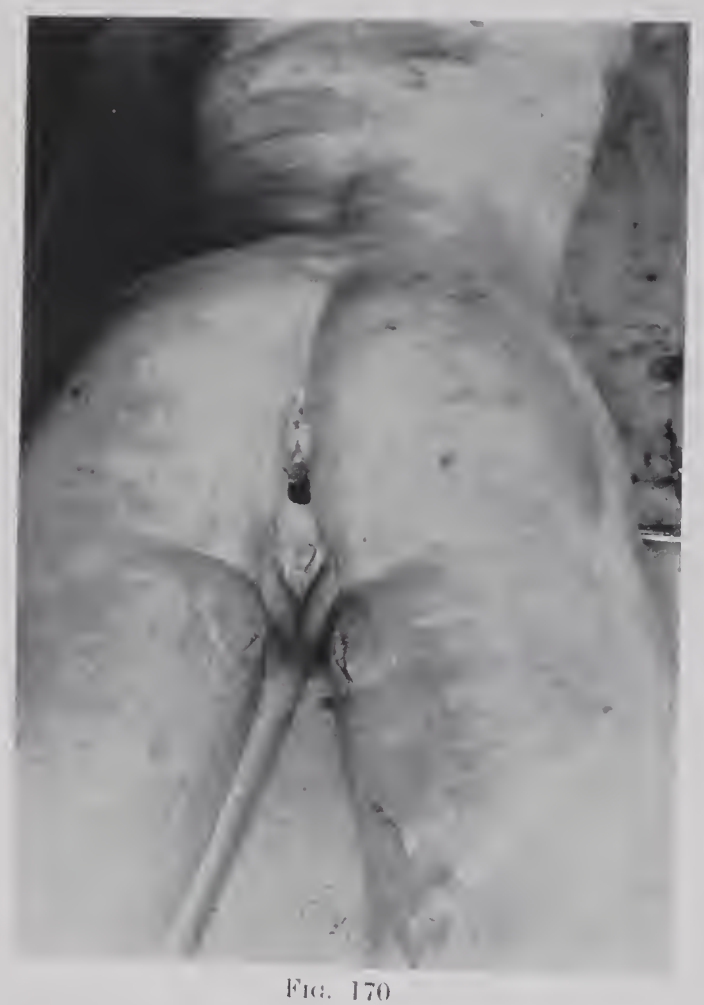


Fig. 170

Body shown in Fig. 165. Marked dilatation of anal orifice due to the insertion of a blunt object following death.

6 by 0·5 centimetres. The splinter of wood had been driven up the inner aspect of thigh, through the obturator foramen, and into the bladder, where the fragment had split. No evidence of extravasation of urine or of hæmorrhage occurred at any time.

Injuries of the urethra in the male, including rupture, with extravasation of urine, may follow a kick or blow on the perineum or a fracture of the pelvis. The male genitals may be the seat of trauma by blows, kicks, machinery or vehicular accidents, and those of the female, more commonly by the first two factors.

Vernon and Kelly [21] describe the case of a man aged twenty-five, a steward, who, while lying in his bunk, became involved in an argument with a stronger man. In the scuffle that followed he was pulled by the penis. The skin was torn through at a level ½ inch from the junction of the penis with the pubic region, the tear involving nearly the whole circumference of the organ, less than 1 inch of skin being left intact on the posterior surface. The suspensory ligament of the penis was exposed, but had escaped injury.

Delprat [22] records the case of a tractor-driver whose tractor bumped into a ditch and whose clothing became entangled in the mechanism. The man's penis was torn out in the gears.

Harden [23] reports a case of mutilation of the external genitalia

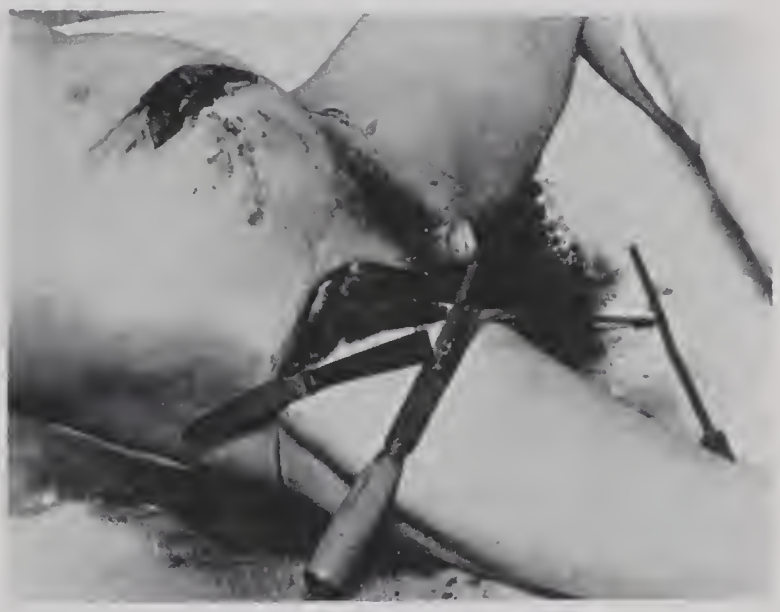


Fig. 171

A case of murder with mutilation of vulva, and wounding of abdomen and right groin. (By courtesy of the Commissioner of Police of the Metropolis, Scotland Yard.)

by an angry paramour. There was a large pear-shaped avulsion of pubic region, extending from line of hair on mons veneris, including clitoris and upper third of vulva. In the lower part of right hypogastrium was a deep cut with a protusion of several inches of omentum. Recovery followed operative treatment.

Psychopathic states, in which there is sexual perversion, have sometimes led to self-mutilation of the genitals. Such cases are usually found among male subjects. Injuries to the sexual parts are not infrequently homicidal, and it is more than probable that many of these are the result of insanity.

Apart from mental derangement of the perpetrator, vulvar injuries are sometimes the result of brutal assault by responsible persons. We have seen a few cases of women who have died as the result of severe hæmorrhage following vulvar injuries by kicking. In these cases the wounds were situated either within the labia minora or on the under surface of the symphysis pubis. In all cases

of vulvar woundings, caused by sharp instruments, there is a grave danger of severe hæmorrhage owing to the great vascularity of the parts.

Intestinal trauma is most commonly produced by penetrating injury, although a kick upon the lower part of the abdomen may be responsible. In one of our cases, a kick on the abdomen caused rupture of the ileum, which is the commonest site of injury from this cause.

James [24] describes the case of a young farmer who jumped backwards from a waggon and received an impalement wound of the rectum from the handle of a pitchfork stuck in the ground. Laparotomy showed an irregular, peritoneal tear in the retrovesical pouch, and two hay seeds were found lying on the anterior surface of the stomach. A left iliac colostomy was performed, was closed a month later, and recovery followed.

Hambly [25] reports details of a boy of fifteen who dropped from a beam and, as he dropped, one of his companions made a move at him with a 5-foot steel rod which pierced the perineum. The rod was pulled out and a piece of string was tied round it to mark the depth of entry. Operation showed that the wound passed under rectal mucosa, through the pelvic mesocolon, abraded the transverse colon at its lower border, completely transfixed a portion of the small intestine, and made an aperture in the great omentum at the lower border of the stomach. The wound was closed, without drainage, and uninterrupted recovery followed.

Baumgarten and Cantor [26] report the case of a woman who fell on a small table supported by an iron rod. The top of the table broke and the woman became impaled upon this rod, which passed through the vagina and bladder and entered the peritoneal cavity, where it penetrated a loop of ileum and almost completely cut the mesentery free from its attachment. The patient recovered and was discharged from hospital on the fourteenth day.

Animal bites.

It must not be forgotten that certain injuries found upon bodies may have been caused, post-mortem, by carnivora, such as rats, mice, dogs, and other animals. In these cases, it is most important to differentiate these injuries from ante-mortem wounds (see pp. 248, 281).

CASES ILLUSTRATIVE OF UNUSUAL INJURY

Removal of head from body by dog.

A man of sixty-three had not been seen by any of his friends for four days. The police broke into the locked house and found the fully clothed but headless body lying in front of the fireplace. A collie dog also in the room was most hostile and had to be shot.

Examination of the body showed that death had been due to coronary disease. The head had been severed close to the level of the upper borders of the sternum and clavicles, in front, and the line of severance had been continued to the back, at a fairly uniform level. The wound of severance, which showed serrations, was of the lacerated type (see Fig. 172). The cervical vertebræ, with the exception of the seventh, which showed irregular damage, were missing. The arch of the aorta, part of the trachea, most of the œsophagus, and the upper lobe of the left lung had been removed. The condition of the body was consistent with death having

Fig. 172

Decapitation, by a collie dog, following death.

occurred some four or five days prior to examination, and the nature of the injuries was consistent with having been caused by the teeth of a dog. All the injuries had been made after death. A post-mortem examination of the dog showed that the stomach contained portions of skin, under-tissue, muscle, bone, lung, brain, trachea, and aorta. A piece of scalp tissue, with greyish-white hair, and three bundles of greyish-white hair, were also found. In all, twenty-seven pieces of bone were recovered. Vomited matter, on the floor of the room, contained fourteen pieces of lung tissue, the largest measuring 5 by 3 inches. On the floor of the room, the greater part of the denuded vault of the skull was found. The lower half of the occipital bone, the entire base of the skull, and bones of the face, except the mandible, were missing. The major part of the mandible was found near the dog. The edges of severance

of the skull were very irregular. The dog, driven to desperation by hunger and thirst, had attacked the exposed part of the body.

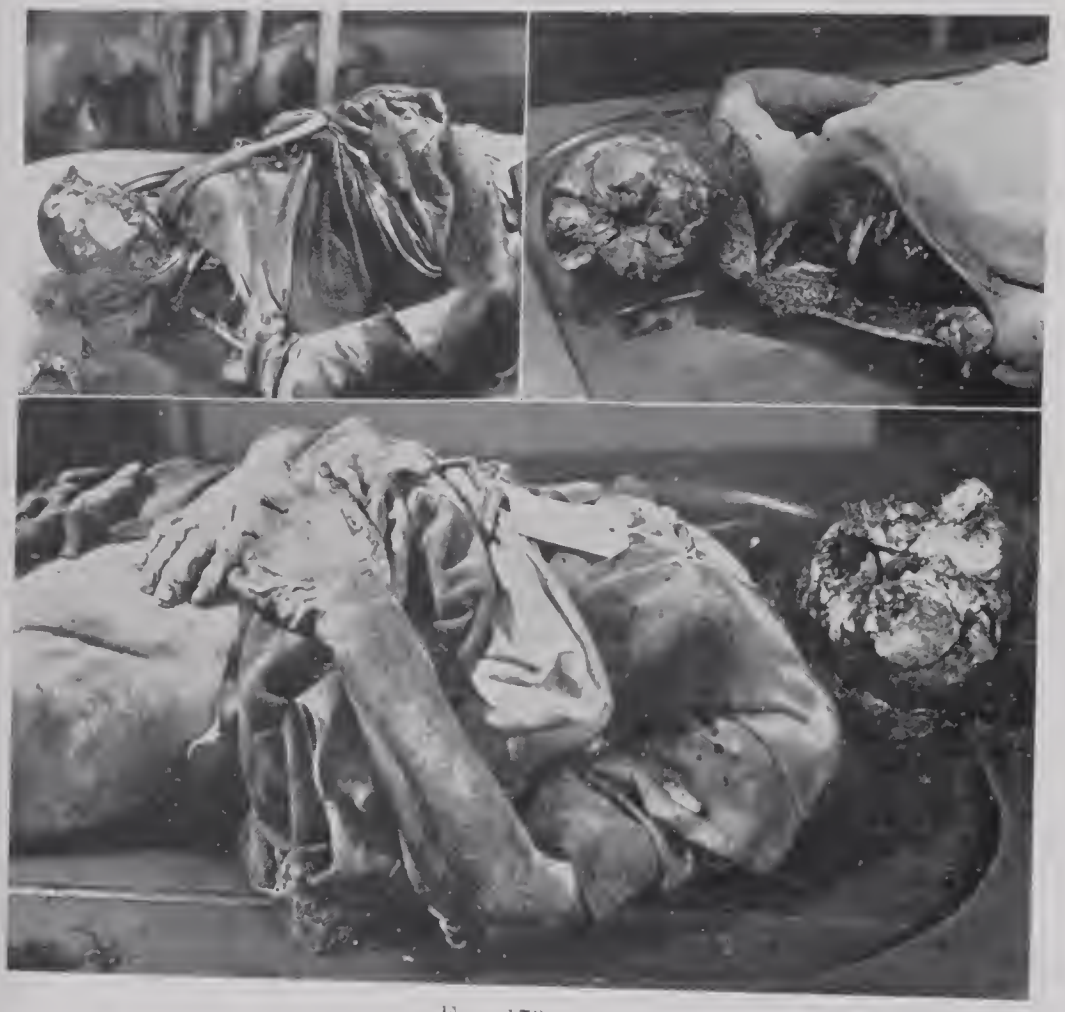


FIG. 173

Post-mortem injuries caused by a Scots terrier dog.
(By courtesy of Dr Robert Richards, Aberdeen University.)

Incised wound of thorax and abdomen.

A boy, aged four, while walking along a street, was attacked by a stranger, who used a packer's knife. The principal wound extended downwards and slightly to the right of the middle line of the body, from a point close to the upper border of the sternum to the upper part of the abdomen. The incised wound, which measured $6\frac{3}{4}$ inches, severed the second to the sixth ribs on the right and penetrated the right pleural cavity. Apart from slight surface bruising, the lung was uninjured but collapsed. The diaphragm was divided for a distance of 2 inches, and there was a small incision on the anterior surface of the left lobe of the liver. The assailant, who was arrested and charged with murder, proved to be an epileptic, and was confined.

Foreign bodies in the stomach, rectum, and bladder.

Foreign bodies, of very varied character, are often found in the stomach. Not only are children prone to swallow such things but adults are by no means exempt and may do so either accidentally or deliberately. Intentional swallowing of objects is not uncommon among inmates of mental hospitals. Wheeler [27] relates the case of a male patient in an asylum from whose stomach, by gastrotomy performed on three occasions within a period of some six years, 1,300 foreign bodies were removed. These consisted of staples, pieces of glass, a nail file in three portions, and a host of miscellaneous metallic foreign bodies. Radiological examination also disclosed foreign bodies in the ileum, colon and rectum. On one occasion two nails, two staples, and two pieces of glass were removed from the intestine by enema. As the result of his habits, the patient suffered from subacute and acute hæmorrhagic gastritis. The submucosa was diffusely hæmorrhagic, œdematous, and moderately inflamed. He recovered.

Foreign bodies are occasionally found in the rectum, urethra, and bladder, commonly as the result of perverted sexual practices. Miscellaneous articles have been found in the rectum, as the result of erotomania, and these have included small bottles, and in one case, a cold cream jar with a lemon inserted into its open end.

Cook [28] describes the case of a youth, aged eighteen, who was mentally retarded. He was admitted to hospital complaining of pain over the lower abdomen, dysuria, and frequency of micturition of some five weeks' duration. There was suprapubic tenderness and the prostate was enlarged, soft, and tender. A pencil was removed from the bladder by the suprapubic route. It measured $1\frac{3}{4}$ by $\frac{5}{16}$ inches and was covered with phosphates. The boy first stated that he had swallowed the pencil, but later admitted that he had pushed it along the urethra, some five weeks before, and that he had taken twenty minutes to accomplish the act.

Death from air-pressure.

A man was accused of having discharged a pneumatic air-pressure pipe at a boy. A blast of air which entered his mouth caused almost instantaneous death. Post-mortem examination showed that the face was markedly swollen, the tongue was protruded, and the upper lip was bruised. The whole surface of the body, with the exception of the feet and ankles, was emphysematous and crepitant. The lungs were collapsed.

Brown and Dwinelle [29] record three examples of rupture of the colon, with gaseous distension of the abdomen, due to the application of a compressed-air jet to the anus. In one of these, the jet was merely thrust inside the clothes through a tear in the leg of the trousers. It has been reported that more than half of sixty-five cases on record have died.

Effects of blast.

In air.

Blast is the result of the abrupt release of a large volume of gas formed by detonation of a high explosive, usually trinitrotoluene (see p. 608).

Three definite waves are recognised :—

1. Wave of positive pressure.
2. Wave of mass movement of air (displacement).
3. Wave of negative pressure.

The pressure and air displacement are the causal factors in injury. The positive pressure is followed by a negative one which initiates marked suction effects. The results on the surroundings and persons in the vicinity of the explosion may be uniform. Often, however, freak effects of force, distribution, and direction are observed, probably due to such causes as uneven rupture of the bomb casing and to reflection and deflection of the blast from oblique and irregular surfaces.

In addition to the trauma caused by the impact of primary and secondary missiles, together with the effects of forcible propulsion of the subject against solid objects, injury may also result from an abrupt change in pressure upon the soft tissues, which most frequently affects the chest and abdomen.

Pulmonary concussion, varying in degree, is almost invariably present in those who have received injuries when in buildings which have sustained damage through a direct hit by a bomb or parachute mine. This condition is not so frequently present when bombing has occurred in the open. Mechanical factors are present in enclosed spaces, since pressure increases until the walls and roof of the building give way. Contusion and rupture of both hollow and solid viscera are frequently found, without any accompanying bone injury under such conditions. Secondary injuries may be superimposed by falling debris.

When injury from blast has been sustained, hæmorrhage into the air-passages and a consequent blood-stained froth at the mouth are common. Should the victim survive for a sufficiently long period, signs of pneumonia appear. At post-mortem examination, rib markings may be found on the surface of a lung or lungs. There may be signs of pneumonia with hæmorrhage into the substance of the lung, liver, spleen, kidneys, adrenals, bladder, and sometimes the brain. Fat embolism may also be present.

When death takes place immediately, the lesion in the chest is mainly hæmorrhage from the alveolar capillaries. There is no massive external hæmorrhage, although bleeding into large portions of lung, usually in the costo-phrenic region, is often found.

Multiple petechial hæmorrhages of the lining of the auditory passages, and sometimes also of the tympanic membrane, are of frequent occurrence due to the effect of near blast. The condition may be accompanied by rupture of one or both ear drums.

In water (immersion blast).

The explosion under water of mines, bombs, depth charges and torpedoes gives rise to somewhat similar lesions, and although the destructive waves of mass movement and suction are absent, the primary impact is conveyed over a wide area of surrounding water.

The effects are most marked on tissues or viscera containing air. The large intestine, with its contained gas, thin walls and scanty muscle is frequently injured. The small intestine is less commonly affected. The muscular, elastic stomach suffers least. Retroperitoneal hæmorrhages are almost constant, and laceration and rupture of bowel walls common. Internal damage is more severe when the abdomen is facing the explosion.

As with blast in air, solid viscera are affected in degree varying from contusion to multiple rupture. Spinal concussion, often transient in character, is frequently observed. Death may result with little or no external evidence of violence.

References

1. Amer. Jour. Surg., Vol. XX (N.S.), 515, 1933, Singleton.
2. "Outlines of Criminal Law," Kenny, 15th Edition, 162, 1944, Cambridge University Press.
3. R. v. Pym (1846), Vol. I, Cox 339.
4. R. v. Holland (1841), Vol. II, M. and Rob., 351.
5. Med.-Leg. Rev., Vol. VII, Part I, 78, 1939.
6. New Eng. J. Med., 216, 1024, 1937, Walker.
7. "Textbook for Small Arms," H.M. Stationery Office, 1929. "The Identification of Firearms and Forensic Ballistics," Burrard, 1934. "Identification of Firearms," Gunther, 1935. "Firearms Investigation, Identification and Evidence," Hatcher, 1943, Small Arms Technical Publishing Company, U.S.A. "Forensic Chemistry and Scientific Criminal Investigation," Lucas, 1937. "Recent Advances in Forensic Medicine," Smith & Glaister, 1938.
8. Police J., Vol. XX, No. 2. 158, 1947, Smith.
9. Lancet, Vol. I, 467, 1945, Kerr.
10. "Acute Injuries of the Head," Rowbotham, 1942. Livingstone.
11. Brain, 64, 93, 1941, Denny-Brown and Russell.
12. Lancet, Vol. II, 438, 1943, Holbourn.
13. Ibid., Vol. II, 497, 1943, Magee.
14. Ibid., Vol. II, 762, 1935, Russell.
15. Ibid., Vol. I, 273, 1944, McConnell.
16. Ibid., Vol. I, 135, 1941, Robb-Smith.
17. R. v. Watson, Med.-Leg. Rev., Vol. IX, Part III, 128 and 176, 1941.
18. Brit. Med. Jour., Vol. II, 1142, 1932, Weston.
19. Lancet, Vol. II, 277, 1943, Osborne.
20. Jour. Amer. Med. Ass., Vol. CIV, 1702, 1935, Crane and Moody.
21. Brit. Med. Jour., Vol. I, 925, 1935, Vernon and Kelly.

22. J.A.M.A., Vol. CXXV, 274, 1944, Delprat.
23. Amer. Jour. Surg., Vol. XXIX (N.S.), 453, 1935, Harden.
24. Lancet, Vol. I, 326, 1939, James.
25. Ibid., Vol. II, 672, 1938, Hambly.
26. Amer. Jour. Surg., Vol. XXV (N.S.), 343, 1934, Baumgarten and Cantor.
27. Med.-Leg. Rev., Vol. IX, Part II, 109, 1941, Wheeler.
28. Amer. Jour. Surg., Vol. XXIX (N.S.), 449, 1935, Cook.
29. Ann. Surg., 115, 13, 1942. Brown and Dwinelle.

CHAPTER X

EXAMINATION OF BLOOD-STAINS

CLOSELY associated with the subject of wounds is that of blood-stains (see p. 283). In every case in which a dead body, with wounds upon it, is inspected, examination should be carefully made for the presence of blood-stains, and for their incidence upon the body and in its vicinity. An examiner will meet a wide variety of blood-stains, in respect of character, incidence, and size, namely, as drops, spirts, splashes, trails, smears, and pools.

FIG. 174

Blood-stains of various shapes.

In the above figure varieties of staining, namely, drops, splashes, or "exclamation mark" stains, and smearing, are seen. The broad portions of the splashes indicate the points at which the blood has first impinged obliquely upon the surface and thus show the direction from which the blood came. The circular stains are due to blood having dropped vertically downwards on to the surface.

The incidence and character of stains in relation to the position of a body, and to the vicinity, should be carefully noted on a rough plan, together with measurements, since this procedure will prove most helpful by indicating the position of the person at the time of wounding, or the place at which the injury may have been

inflicted. It is the routine practice of the police to take photographs of the scene of crime, and these are of service to the medico-legist not only in refreshing his memory, but in the reconstruction of a crime, more particularly in relation to the likely manner in which wounds have been caused. Such photographs are of special importance when, in a case of homicidal assault, a plea of accident is raised (see Fig. 142).

When hairs or other objects are found adherent to blood-stains, they should be preserved for examination, since they may prove of value in the elucidation of certain points.

Fig. 175

Blood-stained scene of murder.

The detection of blood-stains on clothing and on other articles is of high importance, as by this means a suspected person may be associated with a crime (see p. 53).

Nail clippings from the fingers of a person suspected of assault should be examined, when possible, since the presence of blood may prove significant.

Colour of blood-stains.

Blood-stains vary in colour, depending upon a number of circumstances, the principal of which are :—

Age of stain.
Amount of blood composing it.
Nature and colour of material on which it is placed.
Treatment to which stain has been subjected, for example, washing or drying.

Fresh blood-stains on a light-coloured surface are of red colour, but after a variable interval they become reddish-brown or dark brown.

The alteration in colour may commence within twenty-four hours or within a shorter period if the stains are exposed to a warm temperature. Even after short periods it is not possible from their colour to express a reliable opinion as to age with any degree of precision. If the blood has been long exposed to air, hæmatin is produced by decomposition changes which occur in the pigment. As time elapses, the blood becomes more fixed.

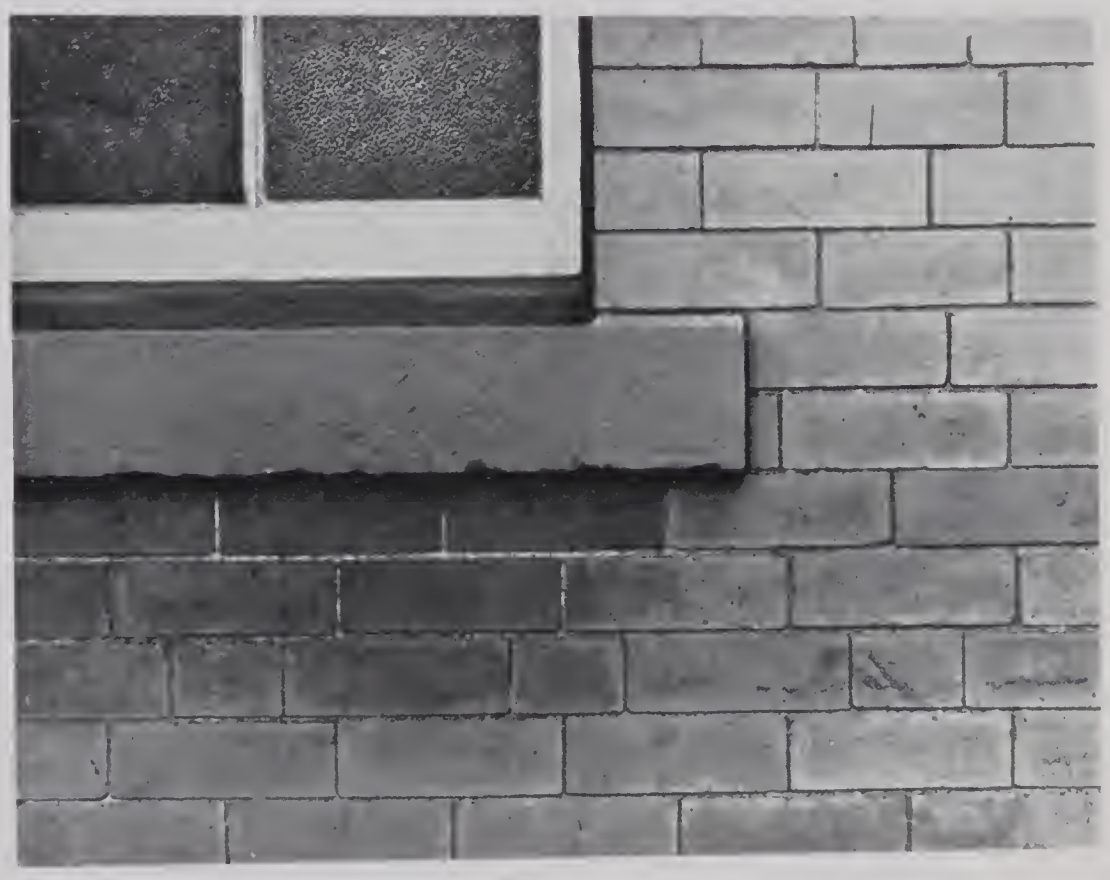


FIG. 176

Blood-staining on a wall at the scene of crime.

On certain fabrics blood-stains are less visible in daylight than in artificial light, as, for example, on brown, blue, and dark-coloured materials. On light-coloured fabrics and on leather, wood, metal, or stone, they are more evident in daylight. By reason of their albuminous composition, when dry they impart a stiffened feeling to thin fabrics, such as cotton, wool, silk, or linen, and even to thick worsted or woollen tweeds, since blood seals the fibres together. On many metallic articles blood-stains appear as dark shiny spots or smears, and when desiccated, frequently show fissures and cracks.

The fact that a stain is of reddish or brownish colour goes no further than to raise a suspicion that it may be due to blood, and detailed examination is imperative in order to establish its true character. There are many substances, apart from blood, which

produce coloured stains, such as jams, jellies, cochineal, vegetable colouring matter, dyes, rust, snuff, fæcal material, organic salts of iron, and various solutions of chemical substances.

No one should undertake the examination of stains unless fully experienced in this line of investigation. Should the result be doubtful it should be regarded as negative.

Physical examination.

The shapes of suspect stains and their position on a garment should be noted. For this purpose it will be found helpful if the clothing is placed on a tailor's model, so that the exact positions of the stains may be noted.

Presumptive chemical tests should be applied (see p. 328). When a positive result is given, the solubility of the stain should be tested.

A portion of the stained fabric should be excised, leaving when possible some of the material untouched for the use of another examiner. The solubility of a blood-stain depends largely upon the age of the stain and the type of material on which it is found. In certain cases, the treatment to which the stain has been subjected may prove an important influencing factor in relation to its solubility or insolubility, for example, washing or ironing. As a general rule, the more recent the blood-stain, the greater its solubility. The rapidity or slowness of solution must, however, depend on so many factors, irrespective of age, that no definite rules for the determination of the precise age can safely be laid down. In recent stains the hæmoglobin is readily soluble, but the older a stain becomes the more difficult it is to effect solution, since the soluble hæmoglobin gradually becomes converted into insoluble hæmatin. The best solvent for blood is normal saline, which contains 9 grammes of sodium chloride per litre of distilled water. The excised portion should be moistened with a few drops of normal saline and allowed to steep for a period of from half an hour to twenty-four hours or more, depending upon the degree of solubility or insolubility. A watch-glass is the most suitable receptacle for the purpose, and when steeping has to be prolonged it should be covered with a bell-jar to limit the evaporation of fluid. Difficulty, if not indeed failure, to achieve solution of a stain by the use of an ordinary solvent may be experienced if the garment upon which the staining is present has been ironed, if on a dyed garment, the dye has acted as a mordant, or if the garment has been washed with soap and water.

When the stain is on wood, metal, leather, plaster, or other firm surface, some of the material may be removed by gentle scraping over a watch-glass.

In the case of a rusty weapon on which blood may be mixed with rust, a preliminary test, based upon the behaviour of blood toward hydrogen peroxide, may be employed. To a portion of the rusty deposit in a watch-glass, placed on top of a piece of black paper,

a drop of water, made feebly alkaline, and a drop of hydrogen peroxide, should be added.

If blood is present, even in small amount, numerous comparatively large bubbles will develop and gradually increase in number. When the rust contains blood only in some of the particles, the bubbles will show only at those particles.

When the bubble reaction is entirely absent, blood is not present. This is a negative test, since other fluids of the body, when mixed with rust, such as saliva or pus, give a similar reaction. When a positive response is given, the presence of blood must be demonstrated by the application of tests for blood.

Presumptive chemical tests.

The benzidine and phenolphthalein tests detect the presence of a substance in the blood-colouring matter, assumed to be a peroxidase, which in the presence of hydrogen peroxide, oxidises colourless bases to coloured salts. These tests are merely preliminary, and are not specific for blood, since several other substances also give the reaction. They are, however, of great value as negative or sorting tests, as the stains which fail to react may be discarded.

Benzidine test.

Two reagents are required, namely, a 10 per cent. solution of benzidine in glacial acetic acid, and hydrogen peroxide (20 volumes). Both should be freshly prepared. To one part of the benzidine solution, two parts of hydrogen peroxide are added. If on the addition of one drop of the mixture to the extract of a suspected stain a greenish-blue colour immediately appears, the reaction is positive. Should it be undesirable to scrape or excise a part of the stained material, the test can be carried out equally well by rubbing the stain firmly with filter paper and adding a drop of the mixed solution to the paper. In addition to blood, a similar colour reaction is given with pus, nasal secretion, gluten, several plant juices, hypochlorites and formalin. The test, therefore, is not suitable for employment with tissue or fluids which have been preserved with formalin.

Phenolphthalein test.

Two reagents are also required for the performance of this test. The first is prepared by boiling 2 grammes of phenolphthalein and 20 grammes of potassium hydroxide in 100 millilitres of distilled water, and adding about 20 grammes of powdered zinc during the process. Boiling should be continued until the solution becomes colourless. A quantity of powdered zinc, to ensure reduction, should be left at the bottom of a dark glass bottle in which the solution should be kept. The second reagent is hydrogen peroxide (20 volumes).

To perform the test, a few drops of stain extract should be placed in a watch-glass and a drop or two of the first reagent added.

When several drops of the solution of hydrogen peroxide are next added, the appearance of a pink colour, more or less intense, shows that the reaction is positive. The test is extremely delicate and gives a reaction with dilutions of one part of blood in five to six million parts of water.

We have submitted this test to critical investigation.[1] Ninety substances were tested to ascertain whether any of them would give a positive response. These included saliva, urine, perspiration, seminal fluid, leucorrhœal discharge, albumin, human milk, colostrum, cow's milk, normal fæces, soap solution, aniline dyes, together with a large number of chemical compounds. In none of these experiments was a positive reaction shown. The presence of cochineal, on account of its colour, in the absence of blood tends to simulate a positive response, but when cochineal and blood coexist, the reagents produce an orange colour. Copper salts also simulate a positive reaction in the absence of blood. This test should not therefore be employed when the fluid under examination may have been in contact with copper pipes or fittings.

Microscopic examination.

The presence of recognisable blood cells microscopically indicates that the blood is insufficiently old for the disorganisation of these bodies. When blood clot is present, the cells, by the use of proper technique, may be detected microscopically for considerable periods after the stain has been formed. The conditions under which the stained material has been preserved play an undoubted part in the preservation or destruction of the cells. Previous contamination, of several kinds, of the material on which the blood has been shed tends to produce fairly rapid disintegration of the cells. Laboratory tests have shown that corpuscles can be demonstrated in blood-stains upon various articles and materials after several weeks, when kept under suitable conditions.

It must be clearly understood that differentiation with certainty between the blood corpuscles of man and those of other mammals, with odd exception, is not possible for medico-legal purposes.

In the blood of man, and of mammals, the corpuscles are circular and non-nucleated, with the exception of the camel family, Camelidæ, in which the corpuscles are lanceolate-oval or elliptical in shape, and are non-nucleated.

In birds, fish, reptiles, and amphibians the corpuscles are oval in shape, and contain nuclei.

The employment of the serological or precipitin test, although not a test for blood, but a specific protein test, will, when the presence of blood has first been established by other tests, enable the source of the blood to be determined by disclosure of the specific protein which it contains (see p. 339).

Occasionally the determination of the character of corpuscles in blood composing a stain is of importance. In several cases of alleged killing of hens and their theft, we obtained conclusive evidence of

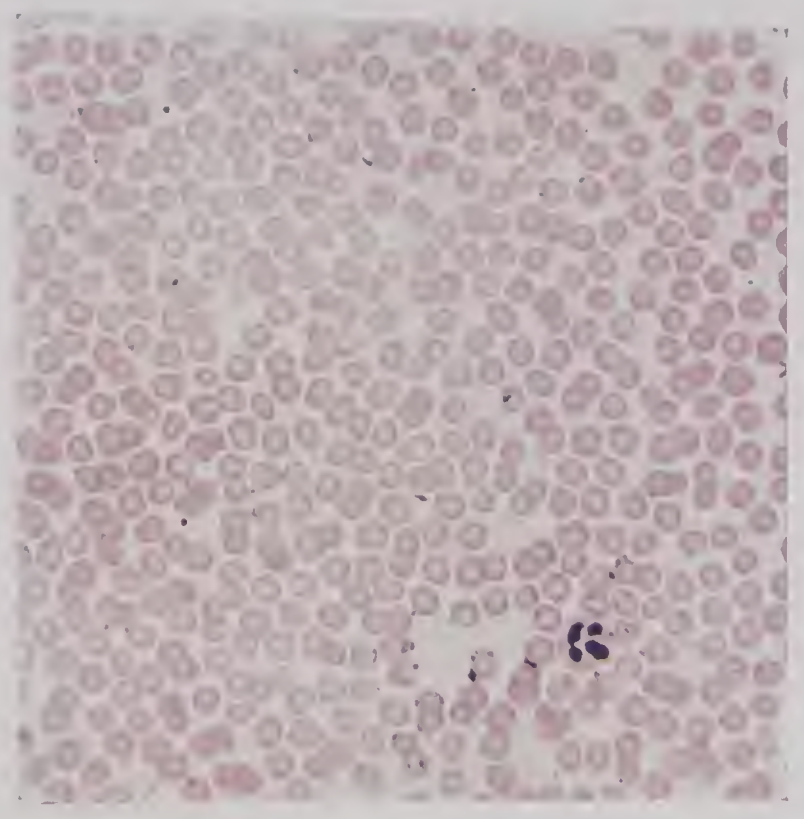


FIG. 177
Human blood corpuscles. ×400.

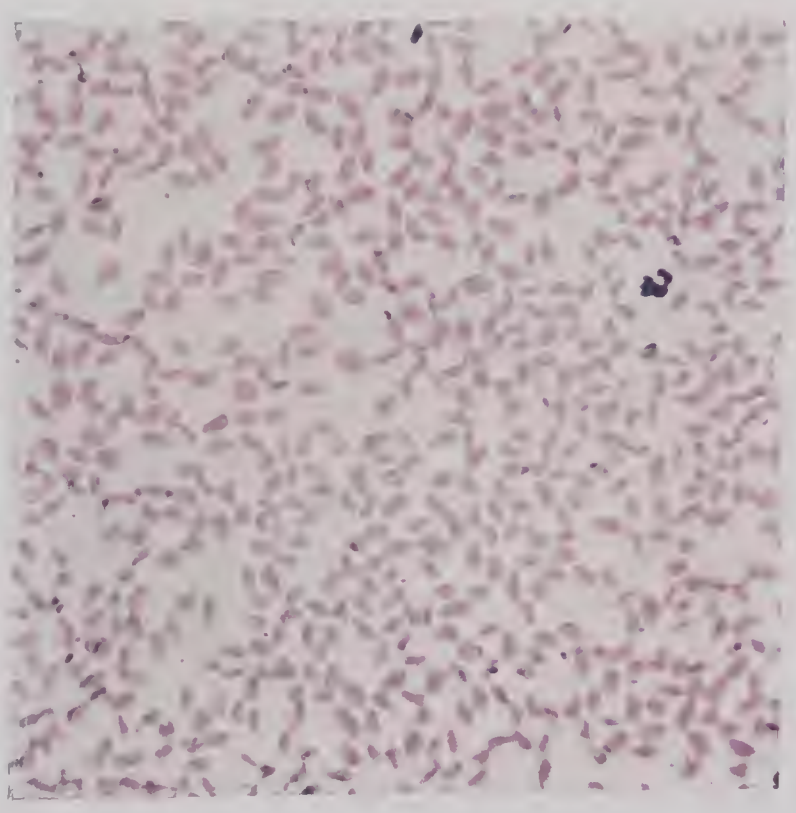


FIG. 178
Blood corpuscles of camel. ×400.

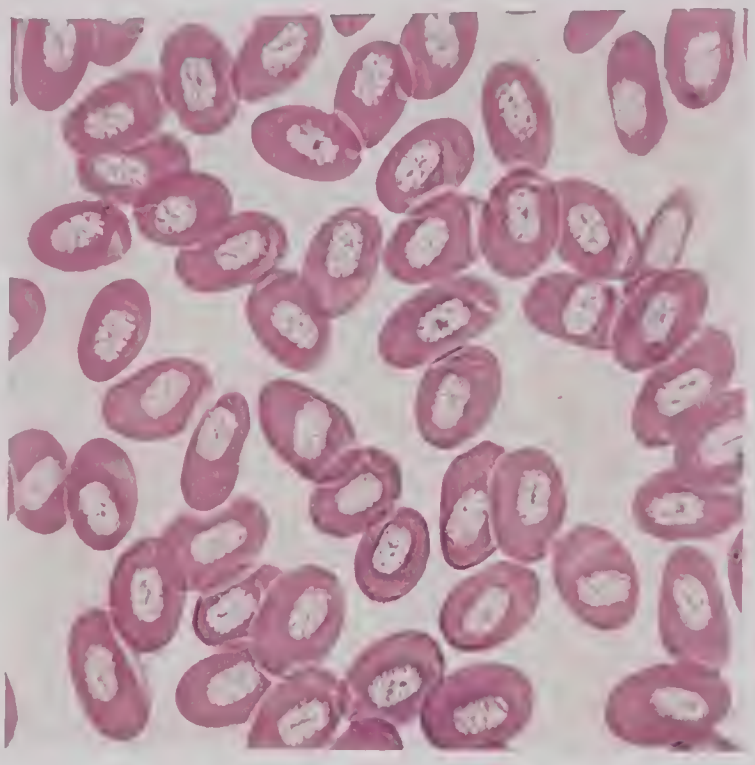


FIG. 179
Blood corpuscles of frog. ×400.

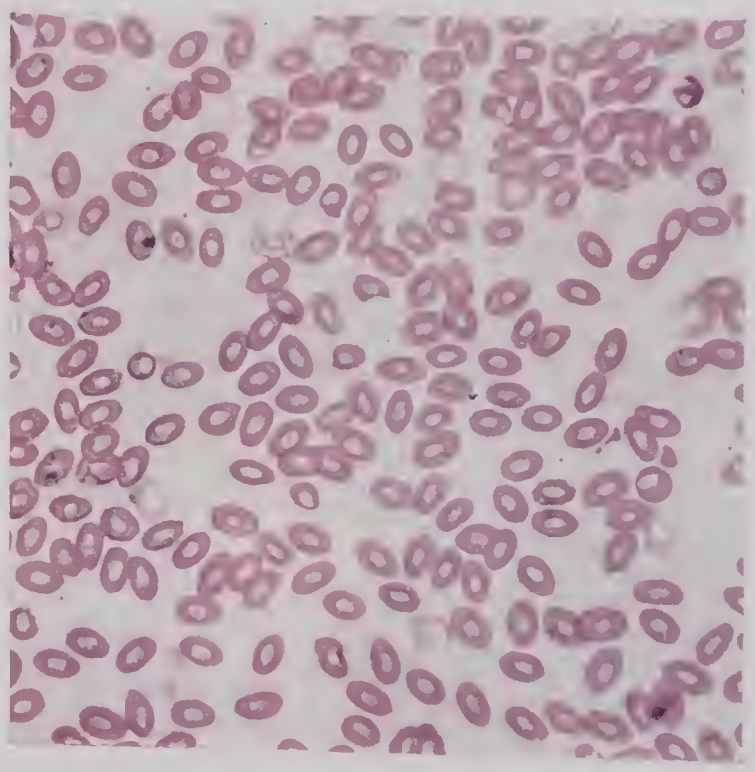


FIG. 180
Blood corpuscles of hen. ×400.

the presence of avian blood in stains upon various articles, by finding nucleated, oval corpuscles and by obtaining a positive response to the precipitin test with the use of anti-fowl serum.

In certain cases of alleged rape, it may become a question of importance whether the blood-stains found on the underclothing of the female alleged to have been assaulted were caused by hæmorrhage from violence to the sexual parts, or by menstrual blood. The answer to the question will depend upon the presence or absence of wounding of the parts and upon the special character of the blood, since menstrual blood frequently shows the presence of endometrial cells.

Chemical examination.

Hæmin crystal test or Teichmann's test.

Concentrate a portion of the coloured solution, place a drop upon a microscope slide, add and mix a drop of glacial acetic acid

Fig. 181

Hæmin crystals. ×85.

and a small crystal of sodium chloride, heat gently to dryness, and examine microscopically. If blood is present, crystals of hæmin, or the hydrochloride of hæmatin, will be found. These may vary in colour from faint yellowish-red to brownish-black, may be rhomboidal in shape, and may be found as separate crystals, in clusters or in masses. It is preferable to use some of the dry powdered stain, rather than the extract from the stain. An alternative reagent, which yields more constant results, is composed of potassium

iodide, chloride, and bromide, each 0·1 gramme, dissolved in 100 millilitres of glacial acetic acid. The test has been superseded by the hæmochromogen crystal test, on account of the uncertainty of obtaining hæmin crystals.

Hæmochromogen crystal test.

Probably the best results in obtaining these crystals are given by the Takayama method.

The composition of Takayama's solution is as follows :—

Sodium hydroxide (10 per cent.) . . .	3 ml.
Pyridine	3 ,,
Glucose (saturated solution)	3 ,,
Distilled water	7 ,,

The first-named reagent acts as an alkali and blood-solvent, and the glucose as a reducing agent.

The solution, when kept in a yellow glass-stoppered bottle, will remain effective for a month or two.

An alternative solution,[2] which gives excellent and much more rapid results, although the crystals tend to disappear more quickly, is prepared from the following ingredients :—

Saccharose	3 grm.
Lactose	3 ,,
Potassium hydrate (10 per cent) . . .	5 ml.
Pyridine	5 ,,
Distilled water	10 ,,

For permanent preparations, Takayama's solution is recommended.

Two or three drops of either of the above solutions are added to some of the powdered material scraped from the stain, or to some of the dried stain extract, on a microscope slide. If blood is present, pink, feathery crystals of hæmochromogen, or reduced alkaline hæmatin, usually in sheaves or clusters, but sometimes in several other forms, will be seen microscopically. If this test is performed in the cold, and with the first solution, crystals should not be looked for until after the lapse of half an hour, but if the slide is heated

Fig. 182

Hæmochromogen crystals. ×75.

gently, the formation of the crystals will occur more rapidly. On examining the crystals with a microspectroscope, the typical spectrum of hæmochromogen will be seen (see Fig. 184).

This test gives good results even with old stains, and should be employed as one of the routine tests for blood, since it is delicate, reliable, and easy to perform.

Spectroscopic examination.

In extracts from fresh blood-stains, hæmoglobin, either in an oxidised or reduced form, depending upon the conditions in which the stain has been exposed, will be found. If oxyhæmoglobin is exposed to a vitiated atmosphere it will become deoxidised hæmoglobin or reduced hæmoglobin. In a very impure atmosphere, after a lapse of time methæmoglobin will probably be formed. By examining a solution of the stain spectroscopically, the form of the hæmoglobin will be indicated by the spectrum shown, since hæmoglobin and its derivatives have the capacity of absorbing certain light-rays after they have passed through a prism. The following spectra are characteristic of the various states in which hæmoglobin may be found :—

Oxyhæmoglobin.

Two distinct bands are present between the Fraunhofer lines D and E, the one nearer D being about half the breadth of the other and more defined.

Reduced hæmoglobin.

One broad band between D and E is visible.

By adding a few drops of Stokes' reagent, or a few drops of ammonium sulphide, to a solution of oxyhæmoglobin, the spectrum of reduced hæmoglobin will be seen on spectroscopic examination. Stokes' reagent may be made by using a particle of ferrous sulphate, a particle of Rochelle salt (sodium potassium tartrate), and a few drops of ammonia.

Methæmoglobin.

Four absorption bands are seen. Two of these resemble the bands in oxyhæmoglobin, the third is situated in the red between the C and D lines, and the fourth, which is placed between the E and F lines is more indistinct (see Figs. 183 and 184).

Hæmatin.

Hæmatin is found in old blood-stains. The spectra of both acid and alkaline hæmatin are of little diagnostic value on account of lack of definition. These are seen in Fig. 183. When alkaline hæmatin is treated with ammonium sulphide, hæmochromogen is produced.

Hæmochromogen or reduced alkaline hæmatin.

This spectrum consists of two bands. The first is a dense narrow band in the yellow part of the spectrum, almost midway between the D and E lines, the second is broader and less dense, and lies in the green part of the spectrum. Part of the purple is also absorbed. Since this is one of the most delicate spectra, it is the most frequently

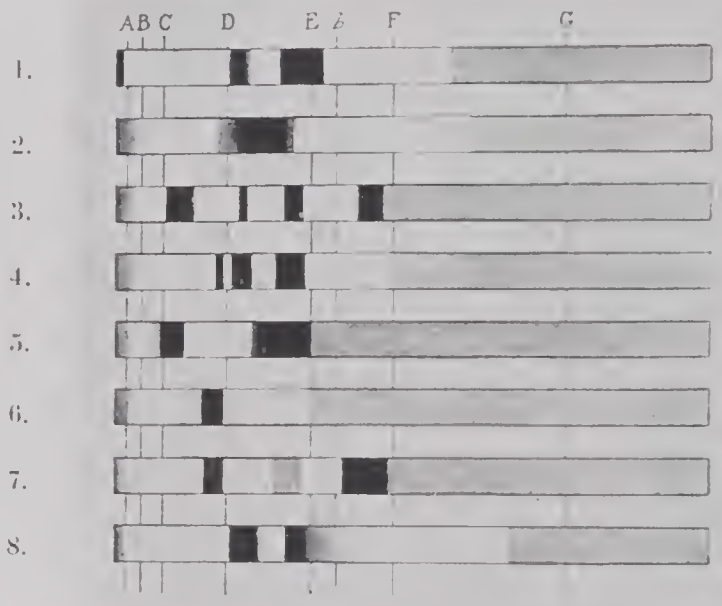


FIG. 183

Blood spectra.

1. Oxidised hæmoglobin or oxyhæmoglobin.
2. Reduced hæmoglobin.
3. Methæmoglobin.
4. Alkaline methæmoglobin.
5. Acid hæmatin.
6. Alkaline hæmatin.
7. Sodium-fluoride hæmoglobin.
8. Carboxyhæmoglobin.

used in the routine examination of blood-stains. Spectroscopic examination either of a fluid preparation or of the crystals may be made. For routine purposes, to establish the presence of blood it is sufficient to obtain hæmochromogen crystals, and thereafter to detect the presence of the spectrum of hæmochromogen by examining them through a microspectroscope (see Fig. 182).

Carboxyhæmoglobin.

Two bands, resembling those of oxyhæmoglobin but nearer the violet end of the spectrum, are present.

Hæmatoporphyrin or iron-free hæmatin.

This may be obtained by adding some strong sulphuric acid to a very small quantity of blood solution, and thereafter gently warming the mixture. When the solution is diluted with some absolute

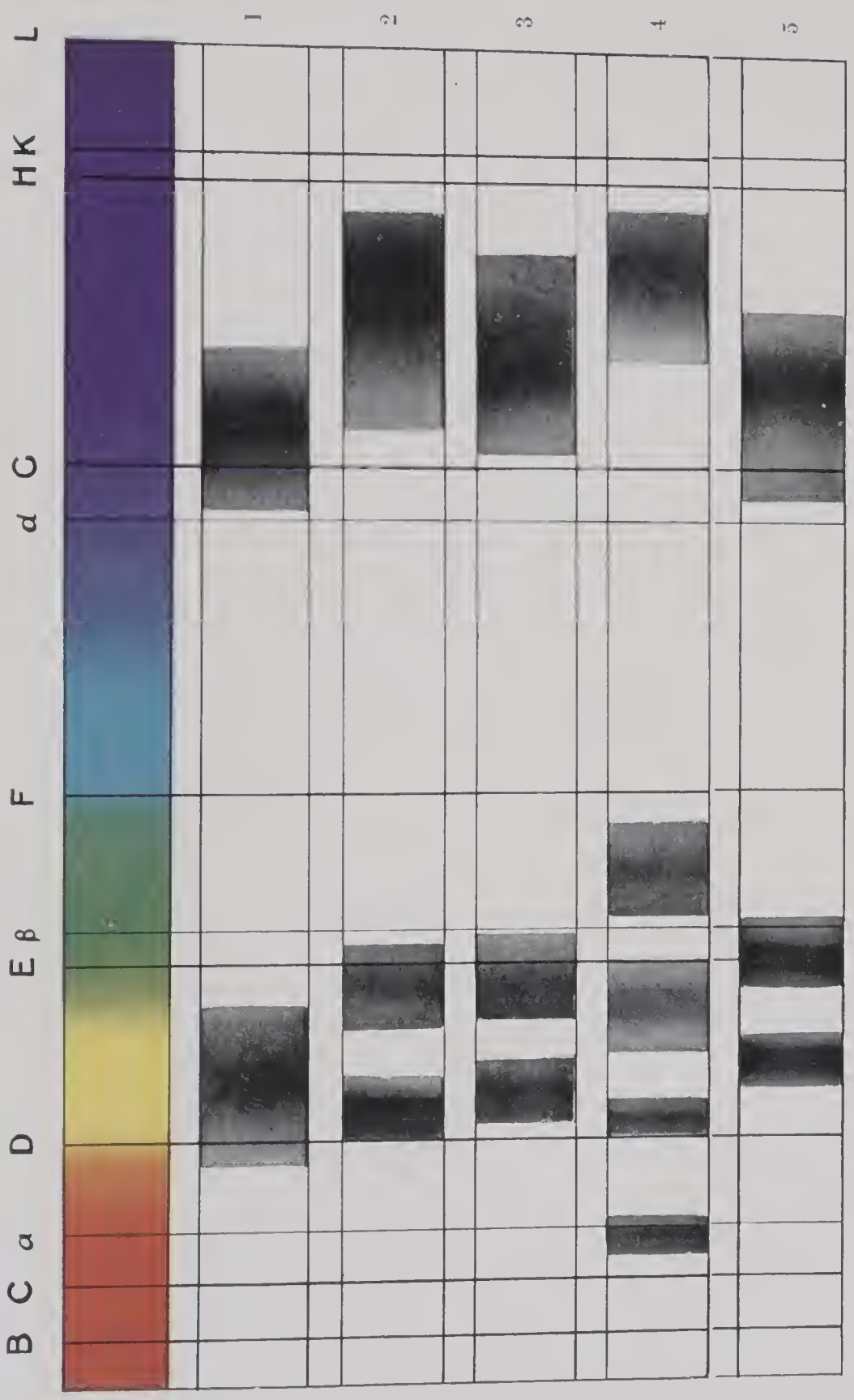


Fig. 184

1. Reduced hæmoglobin. 2. Oxyhæmoglobin. 3. Carboxyhæmoglobin. 4. Methæmoglobin (neutral). 5. Hæmochromogen.

alcohol and water, the spectrum of acid hæmatoporphyrin will be obtained. This consists of two bands, one, narrower and less well defined than the other, lying to the left of the D line, and the other, broader and well marked, to the right of the former between D and E. The spectrum of alkaline hæmatoporphyrin is obtained with more difficulty by neutralising the acid solution, slightly in excess, with a solution of potassium hydrate. It consists of four bands, three of which are more marked than the fourth. The band in the green, between E and F, is the darkest and broadest, and the other three are found to the left of this, two being between D and E and the other between C and D. Both spectra are characteristic and suitable as spectroscopic tests with insoluble blood-stains.

The spectroscopic test is one of the most delicate tests for blood, since it is capable of detecting one-thousandth part of a grain of hæmoglobin. Sometimes a stain may not dissolve readily, due to the conversion of hæmoglobin into hæmatin, but if a few drops of diluted ammonia are added to the solution of normal saline, the removal of the colouring matter will be greatly facilitated. In certain cases it is advisable to filter the solution, especially when it contains particulate matter such as rust or dirt. If, however, the solution obtained is fairly clear, it will be quite suitable for spectroscopic examination without filtration. The technique of the examination, however, will depend upon the amount of blood-stain, and, therefore, the amount of coloured solution at the disposal of the examiner. If the quantity is sufficient to yield about $\frac{1}{2}$ ounce of coloured solution, then the spectroscopic examination may be quite easily made with an ordinary hand spectroscope and a flat glass cell. If, however, the amount of solution is extremely small, the micro-spectroscopic method should be employed. A microspectroscope, such as the Abbé pattern, which fits into the microscope tube in place of the eyepiece, is the most convenient instrument for these delicate spectroscopic tests. A comparison spectrum and a scale marked in wave-lengths are also incorporated in this apparatus. Fig. 185 shows a very simple but useful instrument which we have had designed for our use by Messrs W. Watson & Sons Ltd., London. It has proved most efficient for the routine spectroscopic examination of hæmochromogen crystals. Its chief advantage is that no fine adjustments are necessary. All that is required is to remove the eyepiece of the microscope and insert the instrument into the tube.

Fig. 185

A simple form of micro-spectroscope.

When using the ordinary type of microspectroscope, some of the stain extract is added to a glass cell which is placed in a holder, attached to the microspectroscope, so situated that the source of light falls upon the cell before entering the slit of the instrument. The microspectroscope is then finely adjusted. When making a microspectroscopic examination, and using artificial light, the D line can be readily located by placing a platinum loop, carrying a salt of sodium, in a flame situated near the apparatus.

If the stain is very recent the colouring matter will be in the form of oxyhæmoglobin, the spectrum of which will be visible. If a loopful or two of ammonium sulphide is added, the oxyhæmoglobin will be deoxidised and the band of reduced hæmoglobin will become apparent. If, however, the solution is stirred freely, the fluid will become reoxidised when the spectrum of oxyhæmoglobin will reappear. If more ammonium sulphide is added, together with some sodium hydroxide, or other alkaline solution, the spectrum of hæmochromogen will become apparent. When the fluid under test responds in the manner indicated, all substances, with the exception of blood, can be safely excluded.

To produce the spectrum of hæmochromogen, a drop or two of the stain extract may be treated with a strong reducing agent in the presence of an alkali. An excellent reducing agent for this purpose can be prepared by shaking up 2 grammes of sodium hydrosulphite with 5 millilitres of a 10 per cent. solution of potassium hydroxide in a rubber-corked test-tube and then adding 1 millilitre of alcohol. This reagent should be freshly prepared for each occasion, and when a drop or two is added to an extract of blood, a cherry-red colour quickly develops. Alternatively, the reduction may be effected by the use of ammonium sulphide with the addition of an alkali.

When a stain, some weeks old, has been kept dry in a room in which there are products of combustion of coal or gas, methæmoglobin will probably be present. This derivative of hæmoglobin can be converted into reduced hæmoglobin and hæmochromogen in the same way as oxyhæmoglobin.

In carbon monoxide poisoning, the gas forms a stable compound with the hæmoglobin, carboxyhæmoglobin, the spectrum of which closely resembles that of oxyhæmoglobin, although in the former the two bands are nearer the violet end of the spectrum. Differentiation is readily made by the addition of a few drops of ammonium sulphide when oxyhæmoglobin is reduced and only one band is visible. This treatment does not affect the spectrum of carboxyhæmoglobin.

Sulphuretted hydrogen forms a compound with hæmoglobin, sulphmethæmoglobin, the spectrum of which resembles methæmoglobin (neutral). On the addition of ammonium sulphide to methæmoglobin, methæmoglobin (alkaline) is produced, the absorption band in the red portion of the spectrum disappears and differentiation is thus established.

In poisoning by hydrocyanic acid and cyanides, hydrocyanic

acid links up with methaemoglobin to form cyanmethaemoglobin. This is regarded as a post-mortem manifestation.

In poisoning by hydrofluoric acid or sodium fluoride, the spectrum found in the blood will consist of a band between C and D, close to the D line, and a second and broader band between E and F, close to the F line.

The serological or precipitin test.[3]

Since tests for blood permit of its identification as such, the next important step is to determine whether the blood under examination is that of a human being or whether it has been derived from another source. For this purpose the precipitin test must be employed. The rationale of the test depends on the fact that, by injecting an animal, usually a rabbit, with defibrinated blood of an unrelated animal, an antiserum is produced in the blood of the rabbit, which will precipitate the serum of the animal whose serum has been injected, or that of an animal closely related to it—the group reaction. Normal rabbit serum does not possess this power. The action of precipitation is manifest to the naked eye by the formation of a flocculent deposit preceded by an increasing development of a haze or cloudiness, and this constitutes the practical basis of the test. In medico-legal practice, great care must be exercised in the selection of potent and correctly prepared antisera, and accurate technique must be adopted if reliable results are to be obtained and fallacies avoided. The test is not one for blood as such, but is a specific protein test. When the presence of blood has been established by other tests, the source of the blood, human or animal, can be determined from the specific nature of the protein which it contains, by the precipitin test. The test, therefore, is one for the differentiation of protein, whether in blood serum, in other albuminous fluids, or in tissues, and its application is therefore not restricted to the testing of blood serum. When brought into solution, the protein of the tissues gives the same general response to the test as the protein of the blood of the species concerned. It is, therefore, incorrect to refer to the precipitin test in relation to the detection of the origin of the protein element in blood serum only, since the reaction demonstrates the presence of albuminous substances obtained from any part of a human body, or from a body belonging to any member of the mammalian group. In the Ruxton case, portions of tissue recovered from the drains of the house belonging to Dr Ruxton, together with a piece of debris adherent to a carpet in that house, were examined by this test, following microscopy, and were found to be of human origin.

Technique of the test.

Examination of all antisera is essential before use, in order to eliminate the presence either of haze or opalescence. This is best carried out by adding two drops of antiserum to a $\frac{1}{2}$ millilitre

of normal saline. If the resultant mixture remains clear, the antiserum so tested may be regarded as safe for use. With potent antiserum, a distinct cloudiness should result, within twenty minutes, at laboratory temperature, after it has been added to a 1 in 20,000 dilution of related serum. To apply this test to a blood-stain, it is necessary that the blood serum should be obtained by dissolving the stain in normal saline. Should the solution thus obtained not be perfectly clear, it must be subjected to filtration and also, if necessary, to centrifugalisation. Before carrying out the test, it is necessary to obtain some approximate estimation of the degree of dilution of the stain extract, since the higher the dilution giving a positive result, the more specific becomes the reaction, especially in testing group reactions with the blood serum of closely related animals. For medico-legal purposes, the dilutions commonly used are : 1 in 100, 1 in 1,000, 1 in 5,000, 1 in 10,000, and 1 in 20,000. Usually, a drop of blood when it falls upon filter-paper will occupy an area corresponding to the size of a sixpenny-piece, and having regard to this very approximate standard, an attempt should be made to remove a portion of blood-stain corresponding to one drop of blood. The excised portion of stain should then be steeped in 6 millilitres of normal saline. Since each millilitre of saline contains about sixteen drops and the excised portion about one drop, the resultant dilution will be in the region of 1 in 100. It now becomes a simple matter to adjust the additional dilutions.

It is highly important to test the reaction of the dilutions of blood serum before the addition of the antiserum, since an acid or alkaline reaction will vitiate the test. Unless already neutral, the solutions must be neutralised. For the correction of alkalinity, tartaric acid may be used, and for the correction of acidity, 0·1 per cent. solution of sodium hydroxide.

The following are required for the test :—(1) A supply of absolutely clean, small, tapered or other test-tubes ; (2) a suitable stand in which to place them in series ; (3) normal saline ; and (4) anti-human and other sera.

In carrying out the test numerous controls must be used. A series of tubes bearing distinguishing letters must be arranged in a definite order. For example, let it be assumed that a blood-stain from a jacket of an accused man is to be the subject of examination. Tube No. 1 will contain the extract from the stain in a dilution approximating 1 in 100 ; Tubes Nos. 2, 3, 4, and 5, solutions of the extract in approximate strengths of 1 in 1,000, 1 in 5,000, 1 in 10,000, and 1 in 20,000, respectively. To each of these is added anti-human serum in the proportion of 0·1 millilitre to every 0·9 millilitre of the extract. Tube No. 6 holds an extract from the bloodless part of the jacket, and to it is added the proper quantity of anti-human serum. Tube No. 7 contains normal saline solution to which anti-human serum has been added. Tube No. 8 is filled with the extract from the blood-stain, 1 in 1,000, to which normal rabbit serum has been added in the proportion already suggested. Tube No. 9 contains a solution of human blood diluted

to 1 in 1,000, to which anti-human serum has been added. Tubes Nos. 10, 11, and 12 contain 1 in 1,000 dilutions of other different bloods of known origin to which anti-human serum has been added. It will be noted that the contents of Tubes Nos. 6 to 12 act as controls, with which the reactions in Tubes Nos. 1 to 5 can be carefully compared. If blood serum has merely to be identified as human, as opposed to that of the lower animals, the matter is neither difficult nor lengthy, but if the blood is not of human origin and an opinion is necessary as to its source, it then becomes necessary to test the blood serum in turn with different antisera, until a positive result is obtained and the blood is identified.

A positive reaction occurs when an antiserum is added to an extract of related blood serum. The antiserum sinks to the bottom of the tube, and at the points of contact a faint haze is soon perceptible in the stronger dilutions, but may take longer to develop in weaker dilutions. The haze gradually increases and first becomes more dense at the base of the tube. When the reaction is complete, which for medico-legal purposes should occur at the end of twenty minutes at room temperature, the fluid column is clouded in the lower two-thirds of the tube as the result of the formation of precipitum.

Four essential precautions are necessary in performing the test, namely, the solution of the suspected stain, together with the various antisera employed, must be perfectly clear, the tubes used must be absolutely clean, there must be uniformity in the amounts used in the tubes, and the reaction of the fluid under test must be neutral.

In the illustration (see p. 343) some of the results of the test are shown. Set No. 1, tubes 1 to 5, shows dilutions of human blood serum, 1 in 100, 1 in 1,000, 1 in 5,000, 1 in 10,000, and 1 in 20,000, respectively. Note positive results following addition of anti-human serum. Set No. 2 shows negative reactions to the test after sheep's blood serum has been treated with anti-pig serum. Set No. 3 shows positive reactions with solutions of ox blood treated with anti-ox serum. No. 4, lower capillary tube, shows a positive reaction with a very small amount of 1 in 20,000 human blood serum and anti-human serum. In upper tube, normal saline and anti-human serum are shown.

In order to demonstrate the finer degrees of turbidity, the small tubes normally used should be placed in a rack against a proper viewing box with interior illumination. Nickolls and Thomas [4] have designed an improved precipitation viewing apparatus which they claim is capable of detecting the faintest trace of opalescence or flocculation, and which differentiates easily between a simple opalescence and commencing flocculation. The apparatus, which is a somewhat complicated electrical one, ensures that no light is visible in any tube unless there is a positive reaction, when the Tyndall effect operates and the light beam is scattered in all directions by particles in suspension which appear through the viewing slit as a glowing ring.

Capillary tube method.

When only small quantities of material are available, recourse may be had to this method. The stain extract is drawn into capillary tubes by capillary action, and thereafter a drop of the requisite antiserum is similarly drawn up into each. The usual controls are utilised and the test carried out in the ordinary manner, with the exception that the containers are capillary tubes substituted for the small tubes usually employed. The ends of the capillary tubes are sealed with plasticine by pressing them into a cake of this material. In reading the results, the examiner must adjust the position of the tube in relation to light and background to obtain a clear view of the reaction.

Other medico-legal tests based upon the action of precipitins.

Since precipitins are produced for almost any soluble animal or vegetable protein, the fact has been utilised not only for the identification of the source of blood serum, but for other medico-legal determinations, for example, the identification of the source of seminal fluid, bone fragments, and portions of flesh.

There is no available test for the differentiation of male from female blood.

BLOOD GROUPING

The medico-legal application of blood grouping.

Blood grouping may be of considerable value forensically in helping to establish the guilt or innocence of accused persons or in disproving parenthood in certain cases of disputed paternity. The blood of all persons falls within one of four main groups classified as Group O, Group A, Group B, and Group AB. It must be admitted that, from the medico-legal point of view, the grouping test has many limitations at the present time, but the advances which are being made both rapidly and progressively by many research workers are steadily rendering it more valuable. One of the most important of these limitations lies in the fact that approximately 89 per cent. of all bloods belong to Groups O and A, while only about 11 per cent. belong to Groups B and AB.[5] The variation of blood-group with race shows a characteristic fall in the frequencies of groups O and A, and a rise in group B, as progressive observations were taken south and east across Europe. In view of the large percentage of bloods which belong to the first and second groups, O and A, the tendency of the test may more frequently suggest innocence than guilt, since the blood composing a stain found on a suspect's clothing and the blood of the murderer may belong to the same group. The true value of the test becomes apparent when the blood-stains on the clothing of the accused belong to the same group as the blood of the victim, but to a different group from that of the blood of the accused, since the possible defence might be that the stains upon the clothing of the accused person were caused by his or her own

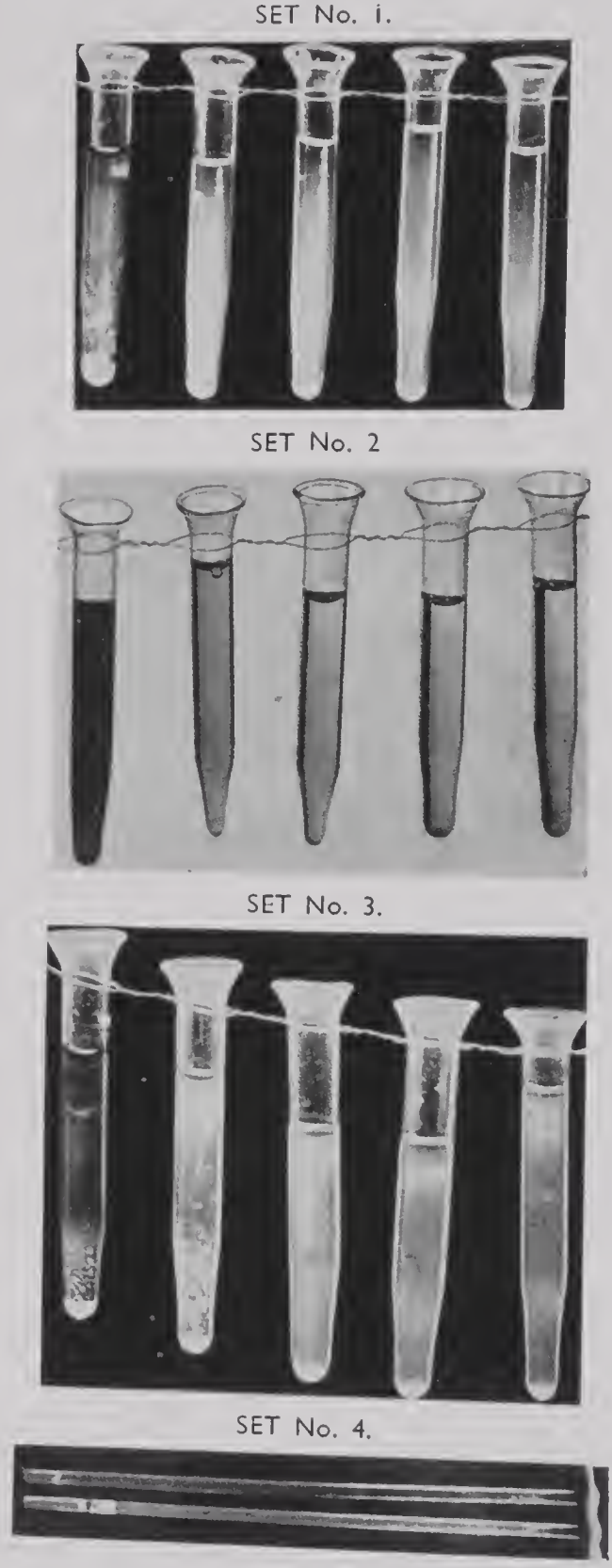


FIG. 186
Precipitin reactions.

(See page 341.)

blood. The blood-grouping reaction, therefore, may narrow down the individuals from whom a given blood-stain may have been derived. Its use for the determination of paternity is of value on account of the Mendelian inheritance of the individuality of the blood by the offspring of a given mating. Although the true father can never be definitely identified by the test, it can frequently be asserted that a certain man could not be the father of the child in question. A man who fails in repeated blood-grouping tests can definitely, and without doubt, be eliminated as the father. The test is also of importance when a child is claimed by two sets of parents.

The phenomenon of hæmo-agglutination in human blood occurs whenever homologous agglutinin and agglutinogen are in contact. It is, therefore, a biological axiom that homologous agglutinin and agglutinogen cannot exist together in the same blood. The four groups were established and classified by the disposition of two agglutinogens, termed A and B, present in the cells, and two agglutinins, *a* and *b*, in the serum, thus :—

BLOOD GROUP CLASSIFICATION

Group.	Serum.	Blood Corpuscles.
O.	Agglutinates cells of the three other groups. Contains agglutinins, *a* and *b*.	Inagglutinable. Contain no agglutinogen.
A.	Agglutinates cells of Groups B and AB. Contains agglutinin *b* (anti-B).	Agglutinated by sera of Groups O and B. Contain agglutinogen A.
B.	Agglutinates cells of Groups A and AB. Contains agglutinin *a* (anti-A).	Agglutinated by sera of Groups O and A. Contain agglutinogen B.
AB.	No agglutinative effect. Contains no agglutinin.	Agglutinated by sera of Groups O, A, and B. Contain agglutinogens A and B.

Agglutinin *a* produces agglutination only of corpuscles which contain agglutinogen A, and agglutinin *b*, of corpuscles which contain agglutinogen B. Thus, with the use of Group A and Group B sera it is possible to classify any blood into its proper group.

Taylor and his co-workers [6] examined 190,000 blood specimens from members of the Royal Air Force drawn from all over the United Kingdom, and among the abnormalities found were fifteen cases (three of group O and twelve of group A) in which there was

a deficiency, or complete absence, of anti-B agglutinin; in only one case (group O) was anti-A agglutinin completely absent. Cold agglutinins were found in the blood of sixty-one persons.

Lattes has stated that the fact of belonging to a definite blood group is a fixed character of every human being and can be altered neither by the lapse of time nor by intercurrent disease. This view is supported generally.

Technique of blood grouping.

Fresh blood.

The two test sera for blood grouping, namely, serum from Group A blood and serum from Group B blood, may either be purchased in prepared form or obtained from the blood of persons of known group and titre. A serum of good titre will cause a macroscopic agglutination when one drop of 1 in 10 dilution of serum is mixed with one drop of a sensitive cell suspension on an open slide at room temperature. When quantities of serum are being stored, a large amount should not be placed in one ampoule but in a number of small 1-millilitre or 2-millilitre ampoules sealed with flame. These should be kept in a refrigerator, when the sera will retain their properties for a long period.

To prepare blood-cell suspensions, a few drops of blood are added to a tube containing several millilitres of normal saline which contains a small quantity of 3 per cent. sodium citrate solution. One drop of blood added to 1 millilitre of normal saline will give approximately a 5 per cent. corpuscle suspension. The suspension is washed once by centrifugalisation, and the supernatant fluid removed. The cell sediment is then added to normal saline to form a 2 to 5 per cent. suspension. Group A and Group B cells are used as test corpuscles.

The group of any given blood can be determined by testing the cells of the blood against the sera of Groups A and B, and by testing the serum of the blood against the cells of these two groups. It is advisable to employ both methods in each examination.

The known serum and cells of both these groups, together with a suspension of the cells of the blood under test, are therefore required. A drop of Group A serum is placed on the left of a slide and a drop of Group B serum on the right. To each is added a drop of the cell suspension under test. The slide is then gently rocked to ensure a proper intermixing of the cells with the serum. The rocking is continued for a few minutes and the slides are then examined microscopically. Snyder recommends that after the reading has been made at the end of a quarter of an hour, it may be advisable to place the slide in an incubator at 37° C. for an additional half-hour to an hour to obviate any chance of missing a weak agglutination due to low titre of the blood.

Small tubes may be used instead of the slide. One drop of unknown cell suspension, normal saline, and test serum are added to each.

When unknown sera are being tested against known cells, the same technique may be adopted.

In cases involving culpa, a sample of the blood of the victim should always be obtained at the post-mortem examination.

Blood-stains.

Before applying blood-grouping tests to stains, it is necessary to establish first that the stain is composed of human blood. The stained garments or other articles must be submitted for examination with expedition. Lattes considers that a large number of practical applications are possible despite various influences to which blood-stains may have been exposed, although, with few exceptions, no evidential value can be attached to any negative results, since the blood may have undergone alterations which prevent an iso-agglutination reaction.

Two methods may be utilised and both should be employed :—

Detection of agglutinins.
Detection of agglutinogens.

Detection of agglutinins.

A small quantity of the dried blood is scraped from the stain, or a portion of the stained material, about $\frac{1}{2}$ square centimetre, is removed. The blood is dissolved in normal saline, 5 cubic millimetres for each milligramme of blood. At a minimum, 15 cubic millimetres are required for the test. The tube in which the dilution is being made should be corked, left at laboratory temperature for an hour, then placed in a refrigerator overnight. The solution may now be tested with A, B, and O cell suspensions. One per cent. and 20 per cent. suspensions are recommended. The results of the tests should be examined many times during several hours, if necessary, until decisive readings are possible.[7]

Detection of agglutinogens.

Agglutinogens cannot be detected by direct agglutination, since the blood cells lose this power on drying, but their presence may be shown by their ability to absorb agglutinins *a* and *b* and their power to inhibit the action of the sera containing these agglutinins. If the blood in the stain contains agglutinogens A and B it will absorb both *a* and *b* agglutinins from the test sera, and when these are subsequently tested against known test corpuscles, the absorption which has taken place will become apparent. Corresponding absorption will result if only agglutinogen A or B is present in the stain. The portion of stained material should be mixed with Group O

serum, about 10 cubic millimetres of extract being required for the test. The mixture, in a corked tube, should be left for an hour at laboratory temperature and then placed in a refrigerator overnight. Two sets of serum and saline dilutions, 1-2, 1-4, 1-8, etc., are prepared on a slide. To each of the sets, a 1 per cent. suspension of A and B cells is added and the results are recorded, after an hour in a moist chamber. When the stain is on fabric a control, using the unstained cloth, should be utilised.[8]

Detection of sub-groups.

Although certain of the blood groups have been divided into sub-groups, the demonstration of these on other than fresh blood is in our opinion fraught with technical difficulties of such a character that their presence cannot yet be safely utilised in the routine examination of blood-stains for forensic purposes.

There are two sub-groups within each of the Groups A and AB. These are classified in Group A as sub-groups A_1 and A_2, and in Group AB as sub-groups A_1B and A_2B. It has been estimated that sub-group A_1 is present in 80 per cent. of these groups and A_2 in 20 per cent. A_1 reacts more strongly than A_2 and the nature of the difference between these sub-groups is probably quantitative, namely, that agglutinogen A is present in the respective blood cells in greater or lesser amount.

Presence of factors in blood other than A or B.

When certain immune sera prepared from rabbits, by injection of human blood, are absorbed with certain human bloods, they still retain agglutinins which are capable of acting on all bloods of the four groups. Two of the agglutinogens revealed are termed M and N, and these are distributed in the four groups. The absence of both M and N from any one group does not occur, but they may be found together or separately. Depending upon these agglutinogens in the red blood cells, bloods can be divided into three groups, namely, M, MN and N, which have no connection with the groups of the A-B-O system. Thus it is possible to distinguish individual samples of blood from one and the same group and to classify blood into twelve groups, namely, ABMN, ABM, ABN, AMN, AM, AN, BMN, BM, BN, OMN, OM and ON. M and N factors are inherited as Mendelian dominants, are present at birth and are important in paternity tests. The agglutinins for M and N are not found in human sera and the agglutinating test sera are prepared by injecting the appropriate type of human red blood cells into rabbits and treating the resultant sera by absorption methods.[9] Other individual blood differences have also been recorded, but require further investigation and elaboration before they can be employed in practice.[10]

The red cells of about 85 per cent. of human beings, irrespective of their A-B-O group, contain an agglutinogen called the Rh factor. This discovery was made by Landsteiner and Wiener [11] who found

that by injecting the red-cells of rhesus monkeys into guinea-pigs and rabbits they obtained a serum capable of agglutinating certain human bloods. Persons whose blood cells were thus agglutinated are termed Rhesus positive, or Rh positive, and those failing to react in this way, Rh negative. The distribution of the Rh factor is the same in both sexes, and this factor is present at birth and is inherited as a Mendelian dominant character. The practical outcome of this finding is that Rh negative subjects are liable to form Rh agglutinins if Rh agglutinogen is introduced into the circulation either by transfusion of Rh positive blood or, in pregnant women, by an Rh positive fœtus, the Rh agglutinogen having been inherited by the child from the father. In such cases, the presence of anti-Rh agglutinins, developed as a result of a transfusion, will bring about incompatibility when a subsequent transfusion of Rh positive blood is given, and an Rh negative pregnant woman may show a serious hæmolytic reaction following a first blood transfusion with Rh positive blood (see p. 410).

Blood grouping in cases of disputed paternity.

Blood grouping can only exclude paternity; it cannot prove paternity. It is sufficient to allude only to Bernstein's theory, since this is now generally accepted. He assumes three allelomorphic genes, A, B, and R, one of which is present in each member of a given pair of chromosomes. Genes A and B are dominant over R. Since each somatic cell has two of these genes, one derived from each parent, there are six possible genotypes.

Wiener aptly summed up the position of the medico-legal application of blood grouping, in relation to questions of heredity (see table, p. 349), when he expressed the views that for reasons, namely, certain exceptions found, there may be slight objections to placing exclusions of paternity or of maternity based on the second law of heredity on the same level as those made in accordance with the first law. Thus, while non-conformity to the first law may be considered absolute proof of non-paternity, non-conformity to the second law should perhaps only be considered strong evidence that paternity is most unlikely. In the former case, the man charged with paternity should be declared innocent regardless of any other evidence. In the latter case, at least, it may be stated that it is highly improbable that the accused man is the true father. He points out that five-sixths of the possible exclusions are made by the first law and that only one-sixth can be attributed to the second law. The technique for the determination of the blood groups has already been described, and one of the several methods may be utilised in paternity cases.

Landsteiner and Levine have shown that the agglutinogens M and N cannot appear in the blood of a child unless they are present in the blood of one or both parents. A type M parent cannot have a type N child and a type N parent cannot have a type M child (see p. 349).

DERIVATION OF OFFSPRING

(After Landsteiner)

Groups of Parents.	Groups of Children.	Exclusion Cases.	Note.
			First Law.
O × O.	O.	A, B, AB.	A and B agglutinogens cannot appear in the offspring unless present in the blood of one or both parents. This is common to the theories of von Dungern and Hirszfeld, and of Bernstein.
O × A.	O, A.	B, AB.	
O × B.	O, B.	A, AB.	
A × A.	O, A.	B, AB.	
A × B.	O, A, B, AB.		
B × B.	O, B.	A, AB.	
			Second Law.
O × AB.	A, B.	O, AB.	A parent whose blood belongs to Group O cannot have a child whose blood contains AB agglutinogens, and a parent whose blood is Group AB cannot have a Group O child. This constitutes Bernstein's further theory.
A × AB.	A, B, AB.	O.	
B × AB.	A, B, AB.	O.	
AB × AB.	A, B, AB.	O.	

INHERITANCE OF MN TYPES

(Theory of Landsteiner and Levine)

Parents.	Possible Children.
M × M.	M.
M × MN.	M, MN.
M × N.	MN.
MN × MN.	M, N, MN.
MN × N.	MN, N.
N × N.	N.

Wiener, Sonn, and Belkin [12] state that the application of the Rh tests in cases of disputed parentage is now justified. In one of the cases described a putative father was excluded by Rh tests, despite the fact that A-B-O grouping, sub-grouping, and MN tests were inconclusive.

Grouping sera are obtained from persons in whom iso-immunisation has taken place either as a result of transfusions or pregnancies. Sera obtained as a result of transfusions will become increasingly rare.

It is now known that a more complete classification of the Rh groups can be made than into Rh+ and Rh—. Wiener [13] has described Rh sub-types. It is generally accepted that there are six common Rh antigens in the red cells of Europeans and each of these antigens can produce its equivalent antibody. These antigens are called C, *c*, D, *d*, E, and *e*. The classification into Rh+ and Rh— depends on the presence or absence of the antigen D. The genes responsible for these antigens are arranged on the somatic chromosomes, which are in pairs. A chromosome can carry C or *c* but not both and the same applies to the other antigens. An understanding of these Rh sub-groups is of great importance in connection with blood-grouping in cases of disputed paternity.

In the instance of innocent persons, the A-B-O grouping system is capable of demonstrating the non-paternity of such persons in one sixth of all cases. If the MN system also is used, innocence can be demonstrated in one third of the cases. If in addition to A-B-O grouping and MN typing the Rh sub-groups are determined it is possible to demonstrate that one half of such men are innocent It will, therefore, be readily understood that when questions of paternity arise it is important to perform MN typing and Rh grouping in addition to A-B-O grouping.[14]

Group specific substances in the organs and body fluids.

Research has shown that group specific substances are present in the cells of practically every organ of the body and in almost every body fluid. The test for detecting these agglutinogens is performed as with blood and blood-stains. The following secretions conform to group type : urine, saliva, milk, nasal secretion, vaginal secretion, seminal fluid, and others (see p. 443). Witebsky was the first to demonstrate the presence of Rh antibodies in breast milk. The titre is usually low.

Agglutinogens in saliva.

Schiff and Sasaki have shown that persons can be divided into two groups depending upon the presence or absence of group specific substances in their saliva. In one group, these substances are present in definite quantity and in the other, they are either entirely absent or present only in negligible amount. Thus persons are either of secretory or non-secretory type depending upon whether the agglutinogens found in the blood cells are secreted in the saliva. Wiener and Belkin [15] state that secretors and non-secretors are sharply defined in infants. The secretion character is inherited as a simple Mendelian dominant by means of a pair of allelic genes. Two individuals who are non-secretors cannot beget

children who are secretors. The agglutinogens, when present in the saliva, correspond with those found in the blood of the individual. In several cases, it has been stated that dried saliva on a cigarette end has been typed successfully.

References

1. Brit. Med. Jour., Vol. I, 650, 1926, Glaister.
2. Ann. Fac. Med., Univ. S. Paulo, Vol. XIV, 405, 1938, Ferreira.
3. "Blood Immunity and Relationship," Nuttal, 1904. "Recent Advances in Forensic Medicine," Smith and Glaister, Churchill, London, 1938. J. Hyg., Vol. III, 259, 1903, Graham-Smith and Sanger. Trans. Med.-Leg. Soc., Vol. I, 21, 1926-27, Glaister. Police Jour., 62, Jan. 1928, Glaister.
4. Brit. Jour. Exp. Path., Vol. XVIII, 55, 1937, Nickolls and Thomas.
5. Lancet, Vol II, 94, 1946, Taylor and co-workers. Ibid., Vol. II, 675, 1919, Hirzfeld and Hirzfeld.
6. Ibid. Vol. II, 94, 1946, Taylor and co-workers.
7. "Medico-Legal Blood Group Determination," Harley. Heinemann, London, 1943.
8. Ibid.
9. "Individuality of Blood," Lattes. London, 1932. "Blood Grouping in Relation to Legal and Clinical Medicine," Snyder, Baltimore, 1929. "Blood Groups and Blood Transfusion," Wiener, New York, 1934. "Die Technik der Blutgruppenuntersuchung," Schiff, Berlin, 1932. "Recent Advances in Forensic Medicine," Smith and Glaister, Churchill, London, 1938.
10. Ibid.
11. Jour. Exper. Med., 74, 309, 1941, Landsteiner and Wiener.
12. Jour. Exper. Med., 79, 235, 1944, Wiener, Sonn, and Belkin.
13. Amer. Jour. Clin. Path., 12, 302, 1942, Wiener.
14. Medical Research Council Memorandum No. 19. Mollison, Mourant and Race. H.M. Stationery Office, 1948.
15. Jour. Immunol., 47, 467, 1943, Wiener and Belkin.

CHAPTER XI

STATES OF INSENSIBILITY

THE differential diagnosis of the various states of insensibility is a subject of considerable importance to the police surgeon, and in the field of forensic medicine. It is therefore necessary to give some consideration to the various symptoms and signs of the commoner causes of unconsciousness.

From what causes may this condition arise? It may be due to disease, for example, cerebral hæmorrhage, embolism or thrombosis of the cerebral vessels, epilepsy, uræmia, or a transitory syncopal attack. Fracture of the base of the skull and concussion of the brain, as a result of accident, are other common causes. Certain drugs such as opium, belladonna, the barbiturates, alcohol, and others, may also produce unconsciousness.

In order to ascertain the cause, the practitioner will naturally endeavour to obtain the history of the case, but unfortunately it too often happens that there is no further history forthcoming than that the person was found unconscious. When this is so, an opinion can only be arrived at by careful differential diagnosis.

Insensibility due to disease.

We must, therefore, first consider the characteristics of those states of insensibility which result from disease, and are more or less sudden in their onset.

Cerebral hæmorrhage.

The points to be noted are :—

Cerebral hæmorrhage generally occurs in persons over the age of forty, but it may arise in much younger persons. The middle cerebral artery is the most frequently involved.

It is found more commonly in the male than in the female.

The symptoms usually begin abruptly, vomiting sometimes supervenes, and unconsciousness, more or less complete, frequently ensues. Convulsions are rare, though muscular twitchings are common.

The face is usually flushed, although occasionally it may be pale, and respirations are laboured and stertorous. The pupillary and tendon reflexes are often absent. Cheyne-Stokes breathing, and an increase in pulse-rate, become evident before death.

When recovery takes place, consciousness gradually returns and is frequently accompanied by a condition of restlessness or delirium. Hemiplegia may then become very obvious.

In cases of pontine hæmorrhage, vomiting and convulsions are common at the onset and there is rapid loss of consciousness. The pupils are markedly contracted and the temperature may rise to 108° F. prior to a fatal issue.

Embolism and thrombosis of the cerebral vessels.

When the lesion is extensive, close similarity exists between cerebral hæmorrhage and these two conditions, and it is frequently difficult to differentiate between them. The points to be noted are :—

When a main vessel is occluded and a large area is affected, convulsions may occur at the onset, unconsciousness is rapid and is followed by signs of hemiplegia. If a large vessel becomes thrombosed, an apoplectic seizure will probably result, although, as a rule, loss of consciousness is much less sudden than in the case of cerebral hæmorrhage. Occasionally there may be a condition of stupor and delirium.

A rough cardiac murmur, usually mitral, may be found on examination, indicating the effects of endocarditis.

In old persons, there will most probably be evidence of degeneration of the arteries.

Epilepsy.

Insensibility, lasting for a variable period, succeeds the convulsive stage of epilepsy. Should the patient be seen before the conclusion of the convulsive seizure, or if information is obtainable from an intelligent eye-witness, the examiner will receive help in his diagnosis. The points to be noted are :—

It occurs at all ages, and is expressive of different causative conditions.

Immediately after the convulsive stage, the face and lips are more or less livid, some froth may be present at the mouth, and the tongue may have been bitten, or scars upon it may be found.

The pupils are dilated during the convulsive stage and for some time thereafter.

The element of time will often clarify the diagnosis, since the person gradually returns to consciousness.

Uræmia.

In its major form the points to be noted are :—

Cardiac enlargement and the presence of abnormal urinary constituents.

Drowsiness or coma with stertorous breathing, with or without convulsions.
Oedema.
In certain cases violent delirium may replace convulsions.

Hysterical coma.

The manifestations of hysteria in women are most varied, and sometimes occur from trivial causes. The characters of an attack are :—

The subject falls to the ground and the body is thrown into convulsive movements of an inco-ordinated character. Foam often appears at the mouth. The reflexes are retained and the tongue is not bitten.

After a variable time, which is usually shortened by the lack of sympathetic treatment, recovery takes place.

Transient syncope or faint.

This condition is common, especially in crowded public places, and its appearances are so familiar and so evanescent that they need not be discussed. Syncope, however, in elderly persons is a much more serious matter, and is characterised, in addition to partial unconsciousness, by an irregular and almost imperceptible pulse. These signs should not be regarded lightly, and prompt measures must be taken to stimulate the heart and, by postural treatment, to restore the cerebral circulation.

Diabetic coma.

In considering coma which is associated with diabetes, it is necessary to distinguish between coma due to hyperglycæmia, when the blood-sugar exceeds 200 milligrams per cent., and insulin coma which results from a condition of hypoglycæmia, in which the blood-sugar is below 70 milligrams per cent. In the differentiation of these two conditions, the points emphasised by Graham [1] cover practically all that need be mentioned.

Hyperglycæmic coma.
Skin is usually flushed.
Odour of acetone is perceived in the breath.
Respirations are deep.
Urine contains sugar and aceto-acetic acid.
Blood-sugar is over 200 milligrams per cent.

Hypoglycæmic coma.
Skin is usually white.
Odour of acetone is not perceived.
Respirations are shallow.
Urine is usually sugar-free, but not necessarily so.
Blood-sugar is under 70 milligrams per cent.

It should also be noted that in insulin coma there may be profuse perspiration, tremors, confusion, and that in some cases the mental attitude may suggest a condition of drunkenness (see p. 630). The symptoms usually develop within a few hours following an insulin injection. The taking of one or two lumps of sugar, which a diabetic patient, under insulin treatment, should always have in his possession, will quickly restore the blood-sugar content and thus prevent the onset of symptoms.

Insensibility arising from head injuries.

Fracture of the skull.

The history of the case is important, but, unfortunately, is often unobtainable. Evidence of the presence of fracture may be found by the following manifestations :—

The presence of scalp injury with depression of an area of the vault of the skull.

Hæmorrhage into the conjunctivæ and eyelids, or bleeding from the nose when the fracture is situated in the anterior fossa. Hæmorrhage, or the escape of cerebrospinal fluid, from one or both ears may be present when the fracture is located in the middle fossa.

The condition of the pupils varies in different cases.

Concussion of the brain.

When concussion is present without further complication, the degree of unconsciousness will vary both in degree and duration (see p. 295). The reflexes may either be diminished or absent. The face is usually pale and the pupils are equal, dilated, and sluggish. If laceration of the brain with accompanying hæmorrhage is present, the coma will be profound, due to compression. In cases of compression, the face is usually flushed, the breathing stertorous, and the reflexes vary on each side. The pupil on the injured side is at first contracted but later dilates, and the other pupil subsequently reacts in a similar manner. The pulse is slow.

The examiner must remember that injury may produce laceration of the brain with hæmorrhage, although there may be no obvious external injury of the head.

Special warning is given to the young practitioner against dealing lightly or carelessly with cases in which there is a definite history of head injury whether the person is under the influence of alcohol or not. If such cases are brought to hospital, it is advisable to retain them under observation for at least twenty-four hours, lest there should be a gradually developing hæmorrhage. We know of several instances in which following dismissal from hospital, after what appeared to be a comparatively trivial head injury, the patient was found in an unconscious condition, and died. Special care is necessary when the injured person is under the influence of alcohol.

Insensibility due to poisoning.

Opium.

The signs and symptoms are :—

The patient may be either somnolent and capable of being roused, or deeply comatose and unable to be roused, depending upon the stage of the case.

The pulse is either full and bounding, or slow and weak.

The face is either flushed or pale, and the lips livid.

The pupils are markedly contracted and immobile.

The temperature, normal at first, becomes sub-normal in the later stages.

The breath may or may not emit the odour of opium, depending upon the preparation which has been taken.

In the later stages the face is pale, clammy, and bedewed with perspiration (see p. 647).

Belladonna.

This form of poisoning is much more rarely seen than that of opium. The characteristic signs and symptoms are :—

The patient is more or less unconscious.

The pupils are widely dilated and immobile.

The face is flushed, and the eyes often have a startled appearance.

There is rambling, muttering, or more excited delirium which in some cases renders the patient almost maniacal.

Speech is thick and often inarticulate, on account of the parched condition of the mucous membrane of the mouth.

The temperature continues normal until the later stages, when it frequently becomes elevated (see p. 650).

Barbiturates.

Many cases of fatal poisoning, suicidal and accidental, occur from overdosage of barbiturates. Large doses cause depression of the central nervous system and the signs and symptoms are :—

The patient may suffer from giddiness, headache, nausea, vomiting, ataxia, weakness, or slurred speech and mental confusion in the earlier stages.

Respiration is shallow.

The pupils frequently show alternate contraction and dilatation.

The blood pressure and temperature fall.

The patient finally lapses into coma, but may recover after a variable interval up to a few days.

In fatal cases death occurs from pneumonia, or, on account of suppression of urine, from uræmia.

In some cases the urine may contain both albumin and sugar, or it may assume a red colour due to hæmaturia or hæmatoporphyrinuria, especially in phenobarbitone poisoning (see p. 642).

Alcohol.

Alcohol, taken in excess, is probably one of the commonest causes of insensibility in police practice.

Since the importance of this subject demands extended consideration, it has been treated in a separate section (see p. 624).

Inebriety in relation to the subject of criminal responsibility is discussed on p. 490.

The essential object in striving to arrive at a correct diagnosis of the cause of insensibility is to facilitate the prompt initiation of appropriate treatment. In most of the cases, admission to hospital whenever possible is the best and safe course, since measures for careful observation combined with adequate facilities for treatment are then available.

Reference

1. Med. Pract. (Sym. No. 1), 1934, Graham.

CHAPTER XII

MEDICO-LEGAL ASPECTS OF SEXUAL FUNCTIONS AND CRIMINAL ABORTION

THE following subjects which relate to the sexual functions are treated in this chapter :—

Impotence and sterility in the male.
Impotence and sterility in the female.
Nullity of marriage on the ground of impotence.
Pregnancy and criminal abortion.

Impotence and sterility in the male.

The question of impotence in the male arises frequently in nullity of marriage cases, and less frequently in disputed paternity cases, and in cases of rape. Impotence is the inability to have sexual intercourse, whereas sterility is the inability to impregnate.

Below the age of puberty, the male is presumed to be sexually impotent. Puberty is generally held to be attained at the age of fourteen years, and by the term "puberty" ought to be meant the attainment of virility, not merely the power of coitus, for the latter power often commences earlier than puberty or virility, and continues for some time after procreative power has ceased. Fairfield [1] reports a case in which a girl aged thirteen was reputed to have been pregnant by a boy of the same age. Absolute certainty was lacking, but the boy himself had no doubt. She points out that the boy's feat was, according to English law, impossible, since it is an "irrebuttable presumption" that a boy under fourteen cannot procreate a child (see p. 424). Although the law fixes the age of attainment of virility, it places no limit upon the period of age above puberty when a male ceases to be sexually potent. Cases of virile power in elderly men are well known and authenticated. This point was raised in the famous Banbury peerage case, where the putative father was eighty years of age at the date of the birth of the claimant, but the judge ruled that there was no legal limit to the age when procreative power ceased.

That procreative power in old men is possible is demonstrated by the fact that spermatozoa have been found in a man of ninety-six.

Certain conditions of the central nervous system, such as hemiplegia, paraplegia, locomotor ataxia, disseminated sclerosis, syringomyelia, and fracture of vertebræ with cord injury may cause

impotence, but this is not always so. In locomotor ataxia, although the procreative power becomes weakened and, latterly, is destroyed, there are well-authenticated cases in which, in the earlier stages at least, procreative power has continued. Impotence may also be produced by excessive masturbation, and ejaculatio preeox, the abuse of alcohol, opium, and tobacco, endocrine dysfunction, and by psychical causes.

The physical eauses of impotenee are probably the most important from the medico-legal aspeet and certainly offer the safest basis upon which to found an opinion.

Absence of the penis constitutes absolute impotenee, since there is no organ for intromission, and in eases of partial amputation, performance of the sexual act may be rendered impossible. Certain malformations of the male external genitals may prevent intereourse, and these include sueh conditions as intersexuality, hypospadias and epispadias. In the last-named condition, the urethral orifice is situated on the upper surface of the penis, which is frequently curved upwards. In hypospadias the external meatus of the urethra is situated at some point upon the under surfaee of the penis or perineum. In both hypospadias, which is fairly common, and epispadias, a more rare condition, even when sexual intereourse may be possible, the seminal ejaculate may not reach the vagina on account of the abnormal position of the urethral orifice. Advanced disease of the penis is another cause of impotence.

Intersexuality is a condition of imperfeet sexual differentiation, or sex formation, and the intersex is an individual in whom male and female features eoexist in various proportions. Cawadias [2] states that the term "hermaphroditism" should be abolished, or if used, to serve only as a poetical synonym of intersexuality whieh is the true scientifie medieal term. In intersexuality the endoerine system is the one most deeply involved. The two elasses of intersex are the male or androgynoid and the female or gynandroid, whieh are distinguished, aeeording to the predominant sex, as male or female. He considers that the term "pseudo-hermaphrodite" is inadequate and should also be discarded. Persons possessing both testis and ovary are either genetie males or genetie females in whom intersexualisation has progressed far. The intersex is either a male possessing certain female sexual features, or a female with eertain masculine sexual features. Bergman [3] holds the view that pseudo-hermaphroditism is characterised by lack of clear-cut differentiation of the external genitalia, while the internal genitalia are iso-sexual. This is due to a hyperplasia of the adrenal cortex in utero, and is an instance of the adreno-genital syndrome. True hermaphroditism is a developmental defect in embryonic life and is probably not related to the disturbances of the adrenal cortex. The external genitalia may be of both sexes and the internal genitalia consist of both ovaries and testes or ovotestes.

The decision of sex, in the case of the intersex, beeomes important medieo-legally in eonneetion not only with questions of impotenee but in those associated with sexual offenees, marriage,

and succession. The question of personal identity in connection with sex has already been mentioned (see p. 88).

Morrison[4] describes the case of a person in whom there was a vagina, from the upper part of which protruded a large penis-like clitoris. Scrotum and testes were not evident, but labia were present. The enlarged clitoris did not have an urethral opening, but there was such an opening at the junction of the base of the organ and a groove which represented the vaginal orifice. On operation, an ovarian formation and testicular tissues were found in the left side of the

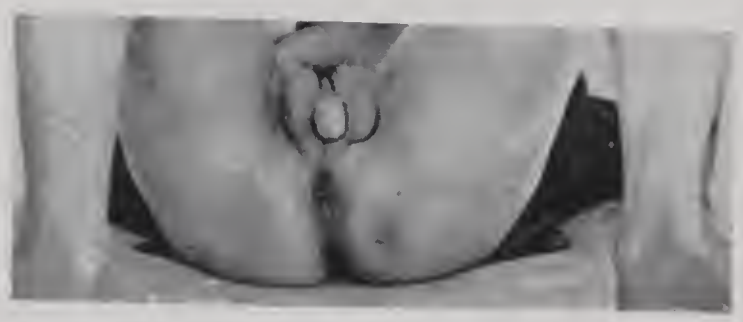


Fig. 187

A case of bifid scrotum.

pelvis. The subject had not experienced menstruation, sexual excitement, or desire.

Raynaud, Marill, and Xiehuna[5] report the case of a person aged eighteen, who had been brought up as a boy. There were feminine appearances—breasts, a large clitoris or small penis, and the left half of the scrotum contained a small testicle. Every few weeks there was a slightly sanguineous urethral discharge. There were also erections of the small penis or large clitoris and ejaculation of seminal fluid which contained active spermatozoa. Laparotomy disclosed a left seminal vesicle, a small uterus with a fallopian tube, and an ovary. There was also a rudimentary vagina. Sections of the testis showed fairly normal seminiferous tubules with spermatogonia and a few sperms.

McFadzean[6] describes a case of an adult male who showed feminisation due to carcinoma of the adrenal cortex.

O'Farrell[7] publishes an interesting series of cases of intersexuality. Five instances occurred in one family which consisted of three males and three females.

Relative impotence is sometimes present, and may be due, among other causes, to neurasthenia, frigidity, or sexual perversion. Sterility, or inability to impregnate, in the male is commonly due to a variety of causes which include hypospadias, absence or atrophy of the testes, pituitary dysfunction, hypothyroidism, and the effects of gonorrhœal infection. Following the removal of both testicles, procreative power is progressively lost.

Impotence and sterility in the female.

In the consideration of this aspect of the subject from the medico-

legal point of view, it is essential that the difference between the terms impotence and sterility should be understood. In the female, as in the male, impotence means the inability to have sexual intercourse. The term sterility, with regard to the female, means the incapacity to conceive. The legal definition of impotence may be considered to be such physical and irremediable conformation of the sexual organs as prevents the act of coition.

The causes of impotence in the female may either be of a permanent or temporary character.

The absence of a vagina, or one which is rudimentary in character, is often found in cases of intersexuality of which the varieties are many.

Hodgson[8] reports a case of congenital absence of the vagina in a woman of thirty-two, who, apart from the fact that she had not menstruated, was unaware of her condition until her marriage, when coitus was found impossible. Surgical operation originated an artificial vagina, but showed that there was no trace of uterus or of any tissue representing it.

Patel[9] describes a young native woman of twenty-two, who, at her second confinement, gave birth to a living child, through the rectum. The vagina, 1½ inches long, ended blindly. The cervix, which opened directly into the rectum, could be seen through the anal canal. The child died immediately following its birth. The first child must have been delivered also by the rectum.

Among the temporary causes of impotence are excessive constriction of the vagina, hyperæsthesia of the vagina, imperforate hymen, prolapse of the uterus or bladder, and vulvar or vaginal tumours.

Turning to the question of sterility, the possibility of pregnancy must primarily depend upon the function of ovulation. Leaving on one side cases of precocious menstruation in young children, there are abundant records to show that the average ages between which girls in this country attain puberty are fourteen and sixteen years. The age of onset of menstruation is very variable, and such factors as race, climate, heredity, general health, environment, diet, and hygiene play a part. While it is the rule that a woman usually becomes pregnant only after the establishment of menstruation, many cases have been recorded in which impregnation has occurred where the usual signs of menstruation have never appeared. At the other end of female reproductive life, the time when impregnation becomes impossible through cessation of the function of ovulation is equally difficult to determine, and, although the age range may be given as between forty and sixty, an opinion on such matters must be based solely upon the circumstances of each specific case (see p. 376). As it is possible for a female to conceive before the external signs of menstruation have appeared, so also must conception be reckoned as possible in a woman within a limited time after these manifestations have disappeared. We are acquainted with the case of a woman, aged forty-seven, who had ceased to menstruate for over a year and who became pregnant. Two cases

have been reported, in one of which there was cessation of menstruation at the age of twenty-three, and in the other the menstrual flow continued until the age of seventy-five. Generally speaking, females who have not yet menstruated, and those who have reached the menopause, are sterile. The only question on which menstruation may have a bearing in civil cases is that which may arise with regard to women who have ceased to menstruate at or about the usual age, and the possibility of their bearing further children, in relation to the provisions of a will. With regard to the age of fertility, in a case before the court it was held that a presumption that a woman of fifty-three was past child-bearing was a rebuttable presumption.[10] In some instances medical examination may not disclose any cause for sterility, and it has been suggested that, in a very small proportion of these, the reason may conceivably be that although the woman is menstruating normally, the menstruation may be of an anovulatory type. Another reason, in the absence of disclosure of any cause in the female, may be found by the appraisal of seminal specimens taken from the male partner.

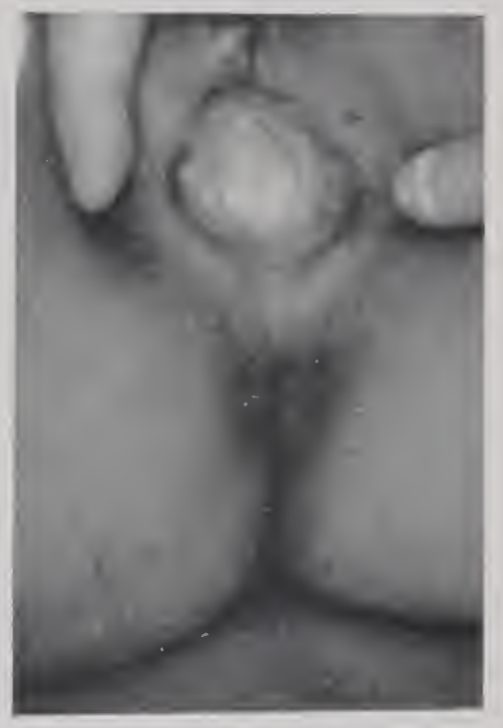


FIG. 1

Imperforate hymen. Note bulging of hymen due to retained menses

Other causes of sterility include removal of the ovaries, certain uterine displacements and diseases, ovarian disease, and lesions affecting the tubes. Congenital abnormality may also produce a condition of sterility.

Anderson[11] describes a very unusual case of a Mussulman woman, aged sixteen, who was sterile. On making a vaginal examination, it was found that unless the anterior wall was followed, the rectum was attained. Thus the rectum opened entirely within the vulva. The cause of the sterility probably resulted from coitus taking place in the rectum instead of the vagina.

Nullity of marriage on the ground of impotence.

Every man is presumed capable of begetting, and every woman of bearing, children. The only proof allowed, in order to rebut this presumption, is that of facts establishing that there is a permanent de facto incapacity.

Where there exists in one of the parties to a marriage such physical and irremediable defect or deformity as will prevent the

marriage being consummated, the marriage is null, and the other party may apply to the competent court to have such nullity declared. If, after evidence, the court is satisfied that the wife or husband, as the case may be, at the time of marriage was and still is unable to consummate, it will annul the marriage. The judgment of the court is termed a Declarator of Nullity of Marriage. Divorce is the judgment of a competent court, after evidence, that a marriage which has been duly consummated is dissolved. An action of declarator will lie against a husband, if he, by reason of impotence, arising from imperfect conformation of congenital origin, or from malformation of the external genitals of an irremediable kind, is prevented from consummating the marriage; and against a wife, if she is of such congenital conformation as prevents her from permitting the consummation. Actions of declarator of nullity of marriage by wives against husbands, on the grounds of neurasthenia, psychical causes, and sexual perversion, have also been heard in the courts. It has been held that an impotent spouse has title and interest to sue for declarator of nullity.[12]

Legally, marriage is a contract between two persons of opposite sex, which presupposes on the part of each the lawful use of the body, or in other words, capability of the fulfilment of the act of physical union by coitus, and any cause however originating on the part of either of them which creates a barrier to coitus would, according to civil law, enable declarator of nullity of marriage. From the legal point of view, apart from statutory enactment (see p. 365), if it is satisfactorily proved that repeated endeavours of a potent husband who has tried all means short of force, have been uniformly unsuccessful, it is for the court, in the absence of any alleged or probable motive for wilful refusal, to draw the inference that the non-consummation was due to some form of incapacity on the part of the wife. From the medico-legal point of view, the primary aspect to be considered is whether there exists a physical deformity or deficiency of the body of the party against whom the action is laid which will prevent, and is likely to continue to prevent, the consummation.

When an allegation is made by a wife or husband that impotence exists, it is the duty of the court to call medical evidence. Two medical examiners are appointed and both parties must submit to such an examination as the medical men think necessary, in order to verify the existence, or otherwise, of the alleged impotence. If the allegation is made by a wife, in order to establish her averment, it is necessary for her to show evidence, by medical examination of her own body, that she is physically capable of having sexual intercourse.

Where the husband sues for declarator of nullity, his wife, should she defend the action, must lead medical evidence of her condition to show whether or not the allegation is true.

The examination of a husband should include the physical conformation of his genitals and microscopical examination of seminal fluid, for living spermatozoa. An opinion regarding virility

must depend upon a man being like, or unlike, other men, for unless there is marked deviation from normal, an opinion implying diminished virility naturally cannot be expressed. When the male external genitals are normal, an examiner is unable to state that the virile and procreative powers do not exist, and the opinion expressed must be to the effect that no good reason exists why these should not be present. In the case of the female, however, assuming the husband is normal, the impediment is likely to be obvious, and will probably consist of such defect of the vaginal portion of the genitals as will establish the inability to permit coition.

The development of any condition, which prevents coitus, after the marriage has been consummated will not afford legal grounds for declarator of nullity.

The principal grounds for a suit of declarator of nullity are :—

There must be an evident and irremediable defect which forms a complete impediment to the consummation of the marriage.

The defect, if it existed prior to marriage, must not have been disclosed to the other party.

The defect must not be due to a condition which has developed after the marriage has been consummated.

The case of Clark v. Clark [13] is both interesting and unusual. The parties were married in 1926, and, according to the evidence of the husband, there was never any consummation of the marriage. By the term "consummation" penetration was presumably meant. In 1930, the wife gave birth to a son of whom the husband was unquestionably the father. The judge held that it was common ground between the parties that the birth of the child did not in itself establish consummation. It was stated in evidence that conception could occur without penetration of the vagina. Fecundation ab extra was an established medical fact previously noted by the courts. The judge found in favour of the husband and he was granted a decree of nullity. This case has been stated as the first of a finding of non-consummation where the husband is admittedly the father of a child of the marriage.

The case of Baxter v. Baxter (1947), 1 All E.R., 387, C.A., is of importance since from it emerged a decision by the House of Lords that the procreation of children was not the fundamental purpose of marriage. Mr Baxter's contention was that his wife's refusal to permit marital intercourse unless he used a contraceptive amounted to wilful refusal on her part to consummate the marriage, although under protest he acceded to her demand. The Lord Chancellor, Viscount Jowitt, said that he could not agree with Mr Justice Hodson and the Court of Appeal that the husband's acquiescence in the conditions imposed by his wife barred him from obtaining a decree. On that view of the matter it was necessary to consider whether the case of Cowen v. Cowen was rightly decided in 1945 (2 All E.R., 197, C.A.). In that case the Court of Appeal held that sexual intercourse meant ordinary and complete intercourse and did not mean partial and imperfect intercourse. They further

declared that the use of contraceptives frustrated one of the principal ends, if not the principle end of marriage. Lord Jowitt said that the institution of marriage generally was not necessarily for the procreation of children, nor did it appear to be a principal end of marriage as understood in Christendom, which, as Lord Penzance said years ago, might be defined as "the voluntary union for life of one man and one woman to the exclusion of all others." His Lordship was unable to believe that Parliament in passing the Matrimonial Causes Act, 1937, intended the Courts to be involved in inquiries as to the merits and effects of various contraceptive precautions. He took the view that in this legislation Parliament used the word "consummate" as that word was understood in common parlance and in the light of social conditions known to exist. There was, in his opinion, no warrant for the decision in Cowen v. Cowen and the present appeal should be dismissed. Lord Wright, Lord Merriman, Lord Simonds, and Lord Normand concurred and the appeal was unanimously dismissed.

(Note.—There is no statutory definition of what consummation means.)

Physical resistance, or persistent refusal, are now recognised by law as the expression of an invincible repugnance not within mental control, or amenable to reason, and if a husband petitions on the ground of wilful refusal and his evidence is accepted, he will succeed in his suit of nullity. The Matrimonial Causes Act made a marriage voidable if it had not been consummated and if the non-consummation was owing to the wilful refusal of the respondent.

Matrimonial Causes Act, 1937, and Divorce (Scotland) Act, 1938.

The first-named Act, which applies to England only, amends the law relating to marriage and divoree, and the chief provisions of medico-legal interest are :—

(1) No petition for divorce shall be presented to the High Court unless at the date of the presentation of the petition three years have passed since the date of the marriage :

Provided that a judge of the High Court may allow a petition to be presented before three years have passed on the ground that the case is one of exceptional hardship suffered by the petitioner or of exceptional depravity on the part of the respondent.

(2) A petition for divorce may be presented to the High Court either by the husband or the wife on the ground that the respondent—

(*a*) has since the celebration of the marriage committed adultery ; or

(*b*) has deserted the petitioner without cause for a period of at least three years immediately preceding the presentation of the petition ; or

(*c*) has since the celebration of the marriage treated the petitioner with cruelty ; or

(*d*) is incurably of unsound mind and has been continuously under care and treatment for a period of at least five years immediately preceding the presentation of the petition ;

and by the wife on the ground that her husband has, since the celebration of the marriage, been guilty of rape, sodomy, or bestiality.

In addition to any other grounds on which a marriage is by law void or voidable, a marriage shall be voidable on the ground—

(*a*) that the marriage has not been consummated owing to the wilful refusal of the respondent to consummate the marriage ; or

(*b*) that either party to the marriage was at the time of the marriage of unsound mind or a mental defective within the meaning of the Mental Deficiency Acts, 1913-27, or subject to recurrent fits of insanity or epilepsy ; or

(*c*) that the respondent was at the time of the marriage suffering from venereal disease in a communicable form ; or

(*d*) that the respondent was at the time of the marriage pregnant to some person other than petitioner :—

Provided that, in the cases specified in paragraphs (*b*), (*c*), and (*d*) of this sub-section, the Court shall not grant a decree unless it is satisfied—

(i) that the petitioner was at the time of the marriage ignorant of the facts alleged ;

(ii) that proceedings were initiated within a year from the date of the marriage ; and

(iii) that marital intercourse with the consent of the petitioner has not taken place since the discovery by the petitioner of the existence of the grounds for a decree.

A rule of court provides that in proceedings for nullity on the ground of impotence or incapacity the petitioner shall apply for the appointment of medical inspectors to examine the parties, and that the registrar shall appoint two medical inspectors and order the parties to attend for examination. A medical inspection is optional for the parties in a case where wilful non-consummation is alleged.

"Incurably of unsound mind" has been interpreted to mean a person who has been of unsound mind for a long time, and who is shown by medical and other evidence to be so unlikely ever to be cured as to be for practical purposes incurable. "Incurable" is not taken to mean "irrecoverable."[14]

The similar act, applicable to Scotland, is the Divorce (Scotland) Act, 1938. The following are the provisions of medico-legal interest :—

Without prejudice to the power of the Court to grant decree of divorce on the ground of adultery, it shall be competent for the

Court to grant decree of divorce on any of the following grounds, that is to say, that the defender—

(*a*) has wilfully and without reasonable cause deserted the pursuer and persisted in such desertion for a period of not less than three years ; or

(*b*) is incurably insane ; or

(*c*) has been guilty of such cruelty towards the pursuer as would justify, according to the law and practice existing at the passing of this Act, the granting of a decree of separation ; or

(*d*) has since the date of the marriage been guilty of sodomy or bestiality.

Provided that, where the ground of action is incurable insanity, the Court shall not be bound to grant a decree of divorce if in the opinion of the Court the pursuer has during the marriage been guilty of such wilful neglect or misconduct as has conduced to the insanity. In an action of divorce on the ground of incurable insanity, the defender shall not be held to be incurably insane, unless it is proved that he is, and has been for a period of five years continuously immediately preceding the raising of the action, under care and treatment as an insane person, and where such care and treatment is proved, the defender shall, unless the contrary is shown to the satisfaction of the Court, be presumed to be incurably insane.

It should be noted that in England the Act goes on to say that a person of unsound mind is deemed to be under care and treatment if retained under an Order under the Lunacy and Mental Treatment Acts, or while receiving voluntary treatment under the 1930 Act, immediately following a period of such detention, provided the patient is not allowed to be absent during any part of this treatment. If petitioning in a Scottish Court, all that need be shown is the continuous operation of the reception order for the previous five years, and the petition cannot be defeated by the fact that the respondent spouse was allowed to be absent during these years for any period on trial, except by section 6 of the Lunacy Acts (Scotland) Amendment Act, 1866, whereby any temporary absence from the mental hospital, if exceeding twenty-eight days, automatically terminates the reception order. It should also be noted that a Scottish petitioner can succeed if the respondent spouse has been detained under care and treatment in England. The Matrimonial Causes Act, 1937, does not give an English husband or wife such a right if the other party to the marriage is under care and treatment in Scotland. In the case of Lang v. Lang[15] the definition of the term " incurable insanity " arose in connection with divorce proceedings. The defendent was detained in a criminal lunatic asylum during His Majesty's Pleasure and the question was whether detention under section 87 of the Lunacy (Scotland) Act, 1857 (see p. 464), was detention . . . " as a lunatic " in terms of the Divorce (Scotland) Act, 1938. It was held that " lunatic " as used in the Divorce (Scotland) Act, 1938, embraced all persons

detained under the Lunacy Acts. The word "lunatic" as used in the Divorce Act, 1938, is not used in a narrow or technical sense, but embraces all persons who are detained under the Lunacy Acts as lunatics whether they be of the ordinary class, or whether they belong to the class of criminal or dangerous lunatics. A recent decision of the Court of Appeal in an English case, Safford v. Safford, has widened the interpretation of the word "detention."[16] Lord Greene, Master of the Rolls, stated that detention had been made the sole test of the continuous "care and treatment" which the petitioner must establish. Care and treatment were the objects of the detention; they ended when the detention ended; when they were no longer required, the detention was terminated by discharge of the patient. Section 55 of the Lunacy Act, 1890, specified three kinds of absence namely, absence on trial; the sending of a private patient to some named place for the benefit of his health; and absence, not for any specified purpose, for not more than forty-eight hours by permission of the medical officer of the hospital or licensed house. All three of these types of absence were treated by the statute as part of the care and treatment provided under the law, not as interruptions thereof. They were, or might be, highly beneficial when the circumstances were favourable and they should not, therefore, be regarded as breaking the continuous period of care and treatment. Detention, in fact, was a matter of status during which it was possible and proper to prescribe methods of care and treatment and, if so, the application of those methods must not be deemed to interrupt the detention. It has been stated that this decision is a common-sense development of a perhaps too meticulously worded statutory experiment.

Some medico-legal aspects of artificial insemination.

Artificial human insemination may be defined as the deposit of semen in the vagina, the cervical canal, or the uterus by instruments to bring about pregnancy unattained or unattainable by sexual intercourse.

The seminal fluid used for this purpose may either have been obtained from the woman's husband or from a donor. Certain recommendations have been made when a donor is used for this procedure,[17, 18] and these include that—

1. the donor must not be a relative of either spouse, he should be potent, should be of age, his age should not exceed forty, and he should have had children of his own.
2. his race and characteristics should resemble as closely as possible those of the husband of the woman to be inseminated, his mental and physical history, together with his personal, familial, and general health must have proved satisfactory, and such examination should include the Wasserman reaction and Rh grouping, and should exclude such diseases as tuberculosis, diabetes, epilepsy, endocrine dysfunction, and psychosis.

3. he must be willing to donate his seminal fluid for the purpose, his wife must agree that he may do so, and both he and his wife must aecept that the donation will result from an act of masturbation.
4. he should be unaware of the destination of the donated seminal fluid and of the result of the insemination.
5. the woman to be inseminated, and her husband, must desire in written form that a donor, preferably unknown, should be used.
6. the physician in eharge of the case should keep the relevant doeuments in his possession, with instructions that, in the event of his death, they should be destroyed unread. He should never undertake the procedure without the knowledge and full consent of both spouses, and a nurse should be present when the insemination is undertaken.
7. for practical purposes, a semen bank might be established.

Possible legal issues arising from artificial insemination.

There is, at the present time, neither law nor obiter dicta in connection with this subject applicable to Britain, thus when either the semen of a donor, or a specimen from a semen bank, is used, a number of important issues may arise for solution. Among many, the following may be mentioned :—

1. What would be the legal position of a mother in relation to the registration of the birth of her ehild born as the result of artificial insemination, or of the woman's husband in similar connection ? It would seem that the proper course would be to state that the child was fatherless. If the mother registers the ehild as that of her husband, or the husband registers it as his own, it would constitute the making of a false deelaration under the Perjury Act.
2. Since the tendency would be to regard the child as illegitimate, what of the possible problem of succession ?
3. Should the marriage be annulled, or dissolved, what of the legal position and custody of the child ?
4. Could the donor be cited as co-respondent in divorce proeeedings ?
5. What of possible incest between offspring from the same seminal donor ? It has been estimated that a fecund donor, submitting two specimens weekly could produce some four hundred children weekly, or some twenty thousand annually, and it has been pointed out that thus a considerable risk of the mating of the children of the same father could arise.
6. What might be the attitude of the General Medical Council toward a practitioner who undertook the procedure of artificial insemination ?

It would appear that the legal aspects of the whole question have not so far come before the courts in Britain, but it seems elear

that if the practice becomes prevalent, some form of legislation will become imperative.

The question of artificial insemination arose recently in the instance of the mother of a child, conceived by this means, who was granted a decree nisi of nullity, in the Divorce Court in London, on the ground of her husband's incapacity to consummate the marriage. The case is believed to be the first of its kind to come before the courts in this country.

Pregnancy in its medico-legal aspects.

The question of pregnancy may come before a medico-legal examiner in various ways and for diverse reasons. The following are the principal :—

As a reason why a woman should be excused attendance as a witness at a trial.

In a case of slander in which it has been said of an unmarried woman, a widow, or a wife living apart from her husband, that she is pregnant.

To declare whether or not a woman, who has raised an action of damages for breach of promise of marriage and seduction, is pregnant, or has been delivered of a child.

To say whether or not a woman is with child, the possible heir to an estate, her husband having but shortly before died, in order that in the disposition of the estate the law of succession may be duly satisfied.

In cases of supposed imposture of pregnancy, to say whether or not the woman has been pregnant, and has been recently delivered of a child.

To form an opinion whether or not a woman, at or about the age of the climacteric, is likely to become pregnant either for the first time, or in continuation of a series of pregnancies, with relation to the question of succession to real or personal estate.

To say whether a woman, who has been sentenced to death, and who pleads pregnancy in bar of execution of sentence, is pregnant.

To declare in cases of alleged criminal abortion whether or not the woman has actually been pregnant.

In cases of alleged concealment of birth or pregnancy and infanticide or child-murder, to determine whether the woman has or has not been pregnant, and, if she has been pregnant, to say whether or not she has been recently delivered of a child.

With relation to pregnancy as an excuse for the attendance of a woman as a witness, it should be clearly understood that pregnancy in itself is not an excuse, except when it is so far advanced that delivery is imminent. At the same time, a valid excuse for non-attendance may be offered, and will probably be accepted, if, owing to any intercurrent condition arising out of the pregnant state, she would suffer risk or prejudice the child by such attendance (see p. 35).

Imposture of pregnancy is rare, but such cases occasionally occur. We have knowledge of two cases.

The Sentence of Death (Expectant Mothers) Act, 1931, makes provision in the instance of a pregnant woman who has been convicted of an offence punishable with death. It enacts that where a woman convicted of such an offence is found to be pregnant, the sentence to be passed on her shall be a sentence of imprisonment for life instead of sentence of death. If the woman alleges that she is pregnant, or where the Court before whom the woman is so convicted thinks fit so to order, the question whether or not the woman is pregnant shall, before sentence be passed on her, be determined by a jury, which will be the jury before whom she was tried for the offence, and the members of the jury need not be resworn. The jury shall determine the question of the pregnancy on such evidence as may be put before them either on the part of the woman or on the part of the Crown, and the jury shall find that the woman is not pregnant unless it is proved affirmatively to their satisfaction that she is pregnant.

Having dealt with the principal occasions upon which the question of pregnancy may arise, the signs and symptoms of pregnancy next fall to be considered.

Diagnosis of pregnancy.

During the first twelve weeks.

Amenorrhœa.

Morning sickness.

Enlargement of uterus felt on bimanual examination. Hegar's sign, due to incomplete filling of the uterus by the ovum, gives rise to the following characteristic which is frequently elicited on careful bimanual examination between the eighth and twelfth weeks of pregnancy. The upper part of the body of the uterus is soft and distended by the ovum, while the lower part is soft and empty and is therefore unduly compressible by the examining fingers. The left hand should be placed on the suprapubic region and two fingers of the right hand inserted into the vagina and placed in front of the cervix.

Slight softening of the cervix.

Slight discoloration of the vagina and cervix.

During the twelfth to twentieth weeks.

Amenorrhœa.

Enlargement of uterus perceived by abdominal palpation.

Ballottement.

When a movable solid body of heavier specific gravity than water is surrounded more or less by fluid in a closed cavity, it falls by gravity to the lowest part of the cavity. If the finger suddenly tilts the solid body by an upward movement, it will momentarily

leave the finger, ascend in the fluid, and then fall back into its original position. In doing so, it will impart to the finger the sensation of a falling body if the finger is retained in position. This constitutes the sign of ballottement which is elicited on bimanual examination when the fingers in the vagina are sharply pressed into the anterior fornix. The recoil is often missed, but the displacement of the fœtus can frequently be detected. The fœtus may, however, occupy a position which is out of reach of the fingers on vaginal examination, and the sign cannot be elicited.

Softening and discoloration of cervix.

Breast changes.

Quickening. About the sixteenth to eighteenth weeks.

During the twentieth week and onwards.

Amenorrhœa.

Enlargement of uterus.

At the end of the twelfth week, in a primipara, the uterus may be felt just above the pubis, at the end of the sixteenth week the fundus will be nearer the umbilicus than the pubis, and at the end of the twenty-second week it reaches the level of the umbilicus. About the thirtieth week the uterus will be found about midway between the umbilicus and the tip of the xiphoid cartilage. The highest point is attained about two weeks before term, when the uterus extends to the tip of the xiphoid cartilage. These estimates of the height of the uterus are very approximate.

Palpation of fœtus.

Auscultation of fœtal heart sounds.

Palpable fœtal movements.

Marked breast changes.

Marked softening of the cervix.

Until the sixteenth week of pregnancy, physical examination may afford presumptive evidence of pregnancy, but absolute proof cannot be obtained by this means. During this early period the use of hormonic tests is of clinical value. The fœtal skeleton will not be apparent by radiological examination until about the eighteenth week.

The average uterus weighs about 2 ounces in the resting phase. Its weight at term is about 2 pounds, due to increase of muscular tissue, when it is about 12 inches long and 9 inches at the maximum transverse diameter.

Cessation of menstruation, and morning sickness by themselves are very unreliable indications of pregnancy, since the menses may disappear temporarily from causes other than pregnancy, and in certain odd cases a hæmorrhagic discharge may recur after conception for a variable period. On the other hand, morning sickness may not be present at all, or at such comparatively rare intervals that no stress can be laid upon it as indicative of pregnancy.

Although colostrum is a sign of pregnancy it must not be forgotten that it can also occur in conditions associated with abnormal ovarian

secretion. The breasts may contain colostrum or a little milk for months or even years after the last pregnancy.

Hormonic diagnosis of pregnancy.

The hormonic diagnosis of pregnancy is of importance and value in the very early stages, since physical and radiological examinations are unreliable. This means of diagnosis may be established as early as the sixth week in more certain measure than any clinical diagnosis.

Aschheim-Zondek test.[19]

The test is based on the detection of the gonadotropic hormones due to the presence of living products of conception, or of live and active chorionic tissue in contact with the maternal circulation. Thus a positive reaction is obtained not only in uterine gestation, but also in all forms of ectopic pregnancies. A positive reaction is also given in cases of hydatidiform mole or chorio-epithelioma, and in a few cases of malignant disease. The test is accurate in 95-97 per cent. of cases and there are more false negative than false positive reactions. In pregnancy the test usually becomes positive within a very short period after the first disappearance of the menses, but this is by no means constant and a negative reaction after two weeks amenorrhœa should not be accepted as definite proof of the absence of a live pregnancy. If clinical signs of pregnancy persist the test should be repeated after an interval of one week.[20] The test is performed upon immature female mice, about three weeks old, by the subcutaneous injection of urine from the suspected case of pregnancy. When the hormones are present in the urine they produce definite and typical changes in the ovaries of the animal injected. The urine taken from the patient is treated before injection, since untreated urine frequently produces toxic effects. To each 25 millilitres of urine, 1 gramme of sulphosalicylic acid is added. The mixture is allowed to stand for half an hour, with occasional shaking, then filtered, after which it is neutralised with sodium bicarbonate, prior to injection. Five mice are used, and each is given six injections of 0·4 millilitre of urine over a period of three days. On the morning of the fifth day, following the first injection, the mice are killed and the ovaries examined. The following changes may be observed :—The corpora lutea are easily recognised, blood spots may be seen, and also enlarged follicles. When, in one or more ovaries of the five animals, a corpus luteum or blood spots or both are seen, a positive diagnosis is established.

If a pregnancy has been diagnosed by this test and subsequent testing proves negative before the end of term, there is a strong suggestion that an abortion has occurred. The test has been used in cases of suspected abortion, since the reaction shows a positive result for a period of about nine days after the products of conception have been evacuated.

Friedman test.

This is a modification of the Aschheim-Zondek test. Ten millilitres of urine are injected intravenously twice, with an eighteen hour interval, into non-gravid mature female rabbits. Forty-eight hours following the last injection, the ovaries are examined. The reaction is positive for pregnancy if hæmorrhagic follicles or corpora lutea are present in the ovaries on macroscopic examination. If the follicles are clear and unruptured, the reaction is negative. Schneider [21] reported a series of one hundred observations in a group of patients in which there were twenty-five pregnant women, twenty-five non-pregnant, and fifty in whom pregnancy was expected. With the exception of two cases, the reaction was in accord with the subsequent findings. Repeated tests in these two cases checked with the clinical course. The test takes a much shorter time than the Aschheim-Zondek test. Robertson,[22] in reviewing 2,368 cases reported by thirteen investigators, found an average error of 1·47 per cent. (see Figs. 189 and 190).

Xenopus or Hogben test.

For this test, 2 to 3 millilitres of prepared pregnancy urine are injected intraperitoneally into six females of Xenopus lævis, the South African claw-toed frog. The animals are kept at room temperature and a positive reaction is indicated, twelve to eighteen hours later, by the extrusion of ova through the cloaca or by the presence of eggs in the oviducts. If the animals are kept at 27° C., this result can be obtained in five to six hours after injection. The test possesses a very high degree of accuracy, and it has been asserted that it can replace the Friedman test but not the Aschheim-Zondek test.

Haines [23] describes a new test performed by the injection of 10 millilitres of the patient's untreated urine into the lymph sac of the male toad of the species Bufo arenarum Hensel. A positive result is shown by the appearance of masses of spermatozoa in the toad's urine after two to four hours.

Signs of pregnancy in the dead body.

From the external appearances of the body alone the chief signs will be those present at the stage of the pregnancy at which the woman died, but no special importance need be attached to these, since by opening the uterus both the existence of pregnancy and the stage which it has reached can be determined.

The ovaries, especially in cases of abortion, should be examined to detect the presence of a corpus luteum (see Fig. 191). The significance of its presence lies in the fact that when the ovum is discharged from the ovary, the follicle becomes a corpus luteum and forms progesterone which is the typical substance with progestin activity. It prepares the subject for pregnancy, and if pregnancy occurs, the placenta liberates a luteinising substance which causes the corpus

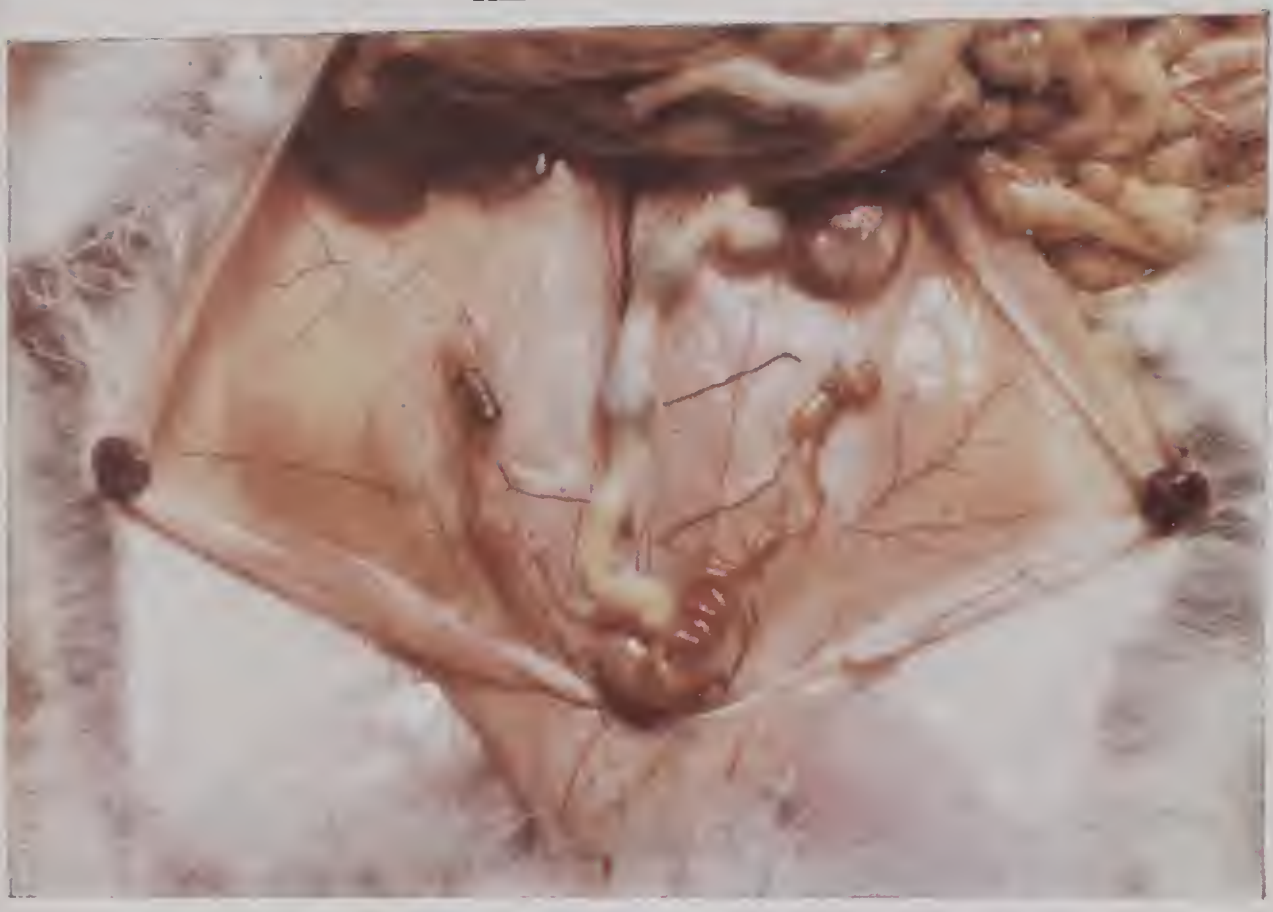


FIG. 189

The Positive Reaction. Showing corpora hæmorrhagica in the ovaries. Note also the congestion of the uterus due to the action of œstrogenic hormone. (Natural colour photograph.)

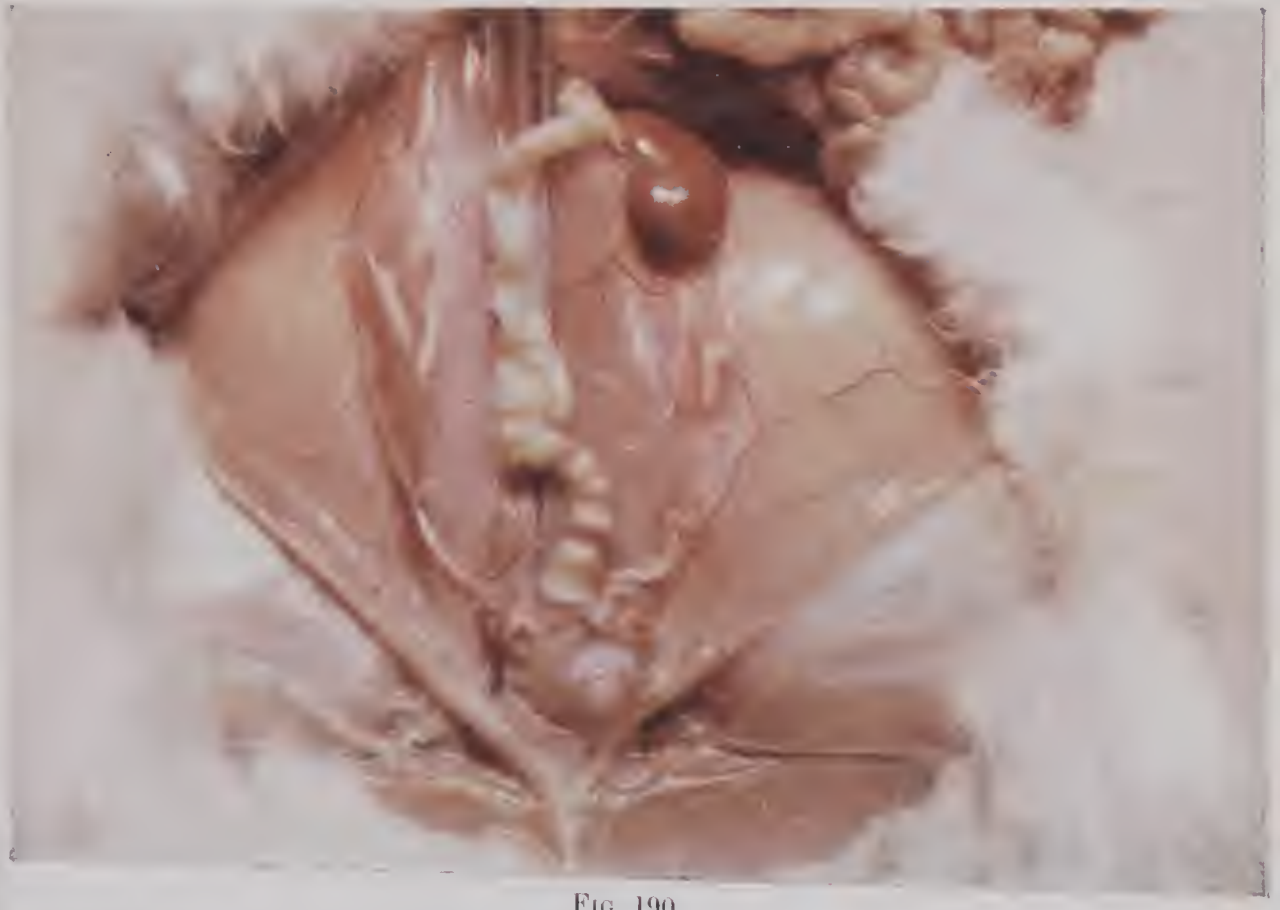


FIG. 190

The Negative Reaction. The ovaries and uterus are normal in appearance. (Natural colour photograph.) (From Evan's Journal, 1938, by courtesy of Evan's Medical Supplies Ltd.)

luteum to persist and to continue to form progesterone. If pregnancy does not occur the corpus luteum atrophies and the hypertrophied uterine mucous membrane is discharged. This is accompanied by loss of blood.[24] If pregnancy occurs, the corpus luteum will increase in size until about the fifth month, when it commences to atrophy. In the absence of pregnancy it will atrophy in about ten days' time and will finally disappear, its site being marked with scar tissue. Cases have occurred in which there has been absence of a corpus luteum, although the subjects were pregnant. Reliance must therefore be placed upon the condition of the uterus and its contents in the determination of pregnancy in medico-legal autopsies.

Pregnancy with reference to female age.

From time to time, young female subjects are submitted for examination to determine the existence of pregnancy, following sexual delicts. Fairfield[25] records some interesting facts with regard to a survey of seventy-four mothers under the age of sixteen years at the age of confinement. Five were aged thirteen, ten were fourteen years old, and the remaining fifty-nine were fifteen years of age. Some of the younger girls had been the victims of incest. There were no maternal deaths, although there were two still-births and three neonatal deaths. Most of the labours were short and easy. Half of the babies born weighed 7 pounds or more, and one, whose mother was thirteen, weighed 8 pounds and 6 ounces. In this instance, the mother was only thirteen hours in labour, and there was no perineal tear. The youngest child in the series was thirteen years and three months at the date of confinement. Only one girl was a certified defective. There was no evidence that maternity had produced excessive nervous or mental strain in these girls, and no case of mental or nervous breakdown had been traced. Our records point in similar directions (see p. 450).

Fig. 191

Corpora lutea. Specimen on top is a corpus luteum of pregnancy. Specimen below is a corpus luteum associated with ovulation.

A case of child-bearing at the precocious age of six and a half years has been recorded.[26] We have notes of a case in which a girl, aged twelve years and two months, was delivered of a child at full time. She must have conceived, therefore, at the age of eleven years and five months. Within recent years, a case of a girl of fourteen who gave birth to twins was reported in Glasgow.

Greater importance, probably, is associated with the age of certain women at the time of their delivery late in life, since important legal issues may emerge. While it is generally true that the fecundity of women lessens the nearer the age approaches the

climacteric period, and that impregnation does not usually occur after forty-five years of age, women have conceived and borne children when they were past fifty (see p. 361).

Gilbertson [27] has recorded the case of a woman whose last child was born when she was fifty years and seven months.

Duration of pregnancy or period of gestation.

The period of gestation is ten lunar months, forty weeks, or two hundred and eighty days; but in legal issues, evidence may be led to show the possible extension of this period. For practical purposes, the assessment of the date of term may be arrived at, with a fair degree of accuracy, by adding forty weeks to the date of the beginning of the last menstrual period. Since ovulation probably takes place about two weeks before the first missed period, the above estimate may be slightly excessive. So that, while it is correct to say that in a large number of cases the period of gestation is two hundred and eighty days, on account of the menstrual cycle of many women being twenty-eight days, it must be considered as possible that the period of gestation may be longer or shorter where the individual menstrual cycle is longer or shorter.

Any data can only be founded upon cases in which, by reason of a single coitus with resulting impregnation, the duration of pregnancy may be reckoned. Even in these cases, however, coitus is not necessarily contemporaneous with conception, since a day or two may elapse between the two occurrences.

Seymour's [28] investigations on the viability of spermatozoa following the introduction of seminal fluid into the cervical canal of five patients, is of interest. The spermatozoa of the donor used in the tests had marked viability. After ninety hours, the sperms had disappeared in two cases, were found to be dead in two other instances, and were alive, but sluggish, in the fifth case. In the last-mentioned case, after one hundred and ten hours, their condition was still the same, and pregnancy resulted. Pregnancy occurred in three out of the five women injected. Experimental work indicates that it is unlikely that spermatozoa retain their fecundity for more than five days after introduction to the female generative tract.[29]

Investigation has shown that fertilisation in the human subject is much more likely to occur during four to five days in each twenty-eight days and that a mating occurring at any other time in the cycle is much less likely to lead to fertilisation. It appears that ovulation varies with the length of the menstrual cycle and ranges from the twelfth to the seventeenth day, and that fertilisation is much more likely to occur at the times in the cycle to which ovulation is restricted.

Sevitt [30] found by biopsy that ovulation can take place on any day of the first fortnight of the menstrual cycle, including the last days of menstruation. This was ascertained by correlating the

menstrual dates and the date of operation with the state of the endometrial biopsy material and the removed ovaries. The finding supports the belief that ovulation can occur on any day of the first half of the cycle. It follows that there is no "safe" period in the first half of the menstrual cycle.

The following cases indicate the difficulty in determining that an abnormal period of gestation is impossible.

In the case of Gaskill v. Gaskill[31] the Court refused to grant divorce on the ground of adultery. The husband, who was a soldier, left on service three hundred and thirty-one days before his wife gave birth to a child. There was no evidence of adultery. Medical evidence was led and showed that it was not impossible for the husband to be the father of the child. Thus a period of gestation amounting to three hundred and thirty-one days has been recognised by a court of law in this country.

The case of Clark v. Clark[32] is one which involves a short gestational period. It came before the President of the Divorce Division in 1939. The petitioner sought divorce on the ground that his wife had given birth to a child after one hundred and seventy-four days' gestation. There was no evidence of misconduct. The child weighed $2\frac{1}{2}$ pounds at birth, which it survived. His lordship stated that it was impossible to ascertain with certainty the date when conception takes place, and it was therefore possible that a child which notionally has had a gestation period of one hundred and ninety-six days, or seven months, has in reality only had one of a hundred and seventy-four days. The child might therefore be comparable with any of the recorded cases whose notional fœtal life had been between a hundred and ninety and two hundred days. This was a rare combination of an extremely premature child with a fixed and not a notional maximum length of fœtal life, and there was no ground whatsoever for saying that the child was illegitimate.

That a period as long as three hundred and forty-nine days, although unusual, was not impossible was decided in the case of Hadlum v. Hadlum[33] while a gestational period of three hundred and forty-six days was admitted in the case of Wood v. Wood.[34] In the latter case, Lord Merriman said that "on the information before us in this case, I absolutely decline to say that we are judicially bound to hold that the period of three hundred and forty-six days (fifteen days longer than the period with which Lord Birkenhead was dealing in 1921 (case of Gaskill v. Gaskill)) is on the wrong side of any line which can possibly be drawn, and that we are judicially bound to hold that the wife had committed adultery." In a recent case in the Divorce Court the judge accepted medical evidence that it was impossible for a normal child to be born after three hundred and forty days' gestation.

It may be taken, therefore, that while the law considers about two hundred and eighty days to be the normal period of gestation, evidence may be led to show reason why, in a given case, the period may be protracted or shortened.

Signs of recent delivery in the living.

This subject is closely connected with concealment of birth and concealment of pregnancy, and medical practitioners are frequently asked to examine women involved in such charges, as to whether they have been recently delivered. The following are the principal signs within eight or ten days of recent delivery :—

Breasts.

These are found to be enlarged, firm, and turgid, and a milky fluid will probably be easily expressed from the nipples. The surface veins are dilated, and there is a more or less dark coloured pigmentation around the nipples. The colour tone of the pigmentation varies ; in blonde women it is usually light in colour, and in brunettes it is dark brown. Montgomery's tubercles are present.

Abdomen.

In multiparæ, the skin of the abdominal wall is frequently flaccid, and may be wrinkled. In primiparæ, the condition of flaccidity may not be so marked, due in large measure to the better tone of the abdominal structures. There is usually evidence of striæ gravidarum, slightly pink in colour, due to the stretching of the skin of the abdomen and resultant formation of scar tissue in the deeper layer of the cutis. As time elapses the striæ become white in colour as the result of diminishing vascularity of the scar tissue, and are termed lineæ albicantes. Some degree of pigmentation, chiefly in the neighbourhood of the umbilicus and median abdominal line, is frequently present. On completion of labour, the fundus lies about an inch below the umbilicus, and a few hours thereafter may be slightly higher. By the tenth day it is on a level with the brim of the pelvis. In from two to three weeks the fundus sinks below the level of the pubis into the pelvic cavity.

Vagina, os, and cervix.

The following conditions are likely to be found :—

There will be a vaginal discharge, and the lochia for the first four or five days will be blood-stained. By the end of the first week it is yellowish or greenish. It then gradually loses all colour, becoming white and turbid until its final disappearance in about ten to fourteen days.

Appearance of bruising or laceration of the external genitals, usually consisting of rupture of the fourchette, swelling of the vulva, and, perhaps, some degree of tearing of the perineum.

The os uteri will be more or less patent, usually to an extent sufficient to enable one or two fingers to be passed into it for a short distance. Its lips are likely to be swollen, and one or both of them may be lacerated.

When these appearances and conditions, or most of them, are found, it may safely be concluded that the woman has been recently delivered of a child.

Signs of previous pregnancy.

The extent and character of the signs found will depend upon whether the woman is primiparous or multiparous. The following signs, or some of them, are likely to be found :—

Lineæ albicantes on the abdomen, the result of hyper-distension of the skin and resultant scar formation in the cutis.

Persistent dark areolæ around the nipples.

Rupture of the fourchette or perineum, or both.

Frequently a healed cervical tear, or tears, in the region of the external os.

Absence of signs of virginity, and a non-rugose condition of the vaginal walls.

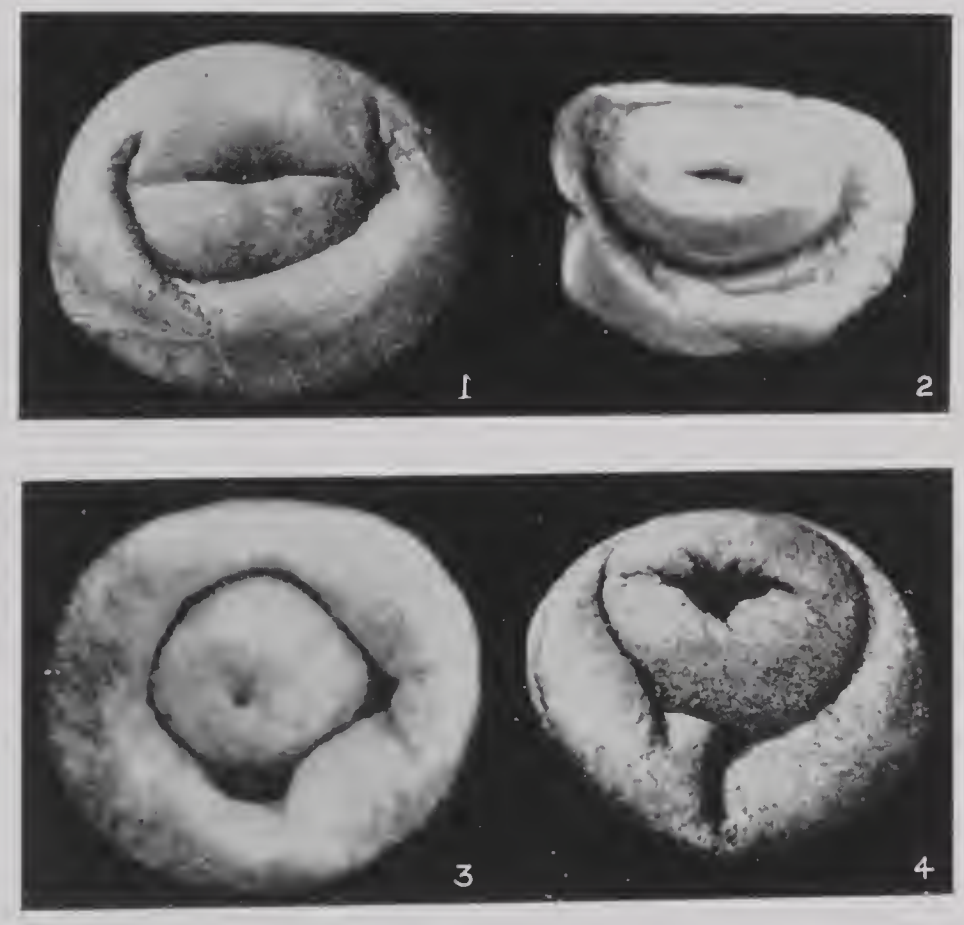


Fig. 192

External os of uterus. (Hoffman)

1. Adolescent virgin. Edges smooth.
2. Adolescent virgin. Edges smooth.
3. In advanced age, but in virginal state. Edges smooth.
4. Multiparous woman. More or less deeply and cicatricially notched. May admit finger-tip.

The examiner, however, must be prepared in exceptional instances to meet with cases where a woman who has borne one or more children may not exhibit on her body any of the above signs, or little evidence of them.

Superfecundation and superfœtation.

Superfecundation is the term applied to the fertilising of two ova, the products of the same ovulation, which are impregnated as the result of two separate acts of coitus. Superfœtation refers to the fertilisation of two ova, each of which has been liberated by different ovulations. The possibility of superfecundation is generally admitted, but the proof of its occurrence is difficult in the human subject since it becomes indistinguishable from a binovular twin pregnancy. A case has been recorded, however, in which a negress has given birth to a white and a black child. Superfœtation is not generally accepted as a possible occurrence, and there is divided opinion on the subject. It has been submitted that the cases cited are all explicable from the well-known facts attending the unequal growth and development of twin conceptions, where the disparity is the result of delayed delivery of the less favoured fœtus. Eden has stated that if it is true that ovulation continues during early pregnancy, there is no obstacle to the fertilisation of a second ovum, and its lodgment in the uterine cavity up to the fourth month, so long as the decidual space persists. Many hold the view that ovulation discontinues after impregnation and during pregnancy. If it is possible for such cases to occur, their advent is very rare, and each would have to be very carefully considered in the light of every circumstance before an expression of opinion as to the definite possibility of superfœtation was expressed.

Criminal abortion.

In neither England nor Scotland does the law recognise any difference between abortion, miscarriage, and premature labour, nor does it take any cognisance of whether the criminal expulsion of the products of conception was produced before or after the period of quickening. Further, the crime may be committed at any stage of pregnancy.

The law of England is set down in the Offences Against the Person Act, 1861, secs. 58 and 59 :—

58. Every woman, being with child, who with intent to procure her own miscarriage, shall unlawfully administer to herself any poison, or other noxious thing, or shall unlawfully use any instrument or other means whatsoever, with like intent, and whosoever, with intent to procure the miscarriage of any woman, whether she be, or be not with child, shall unlawfully administer to her, or cause to be taken by her, any poison or other noxious thing, or shall unlawfully use any instrument or other means whatsoever, with the like intent, shall be guilty of felony.

59. Whosoever shall unlawfully supply or procure any poison or other noxious thing, or any instrument or thing whatsoever,

knowing that the same is intended to be unlawfully used, or employed, with intent to procure the miscarriage of any woman, whether she be with child or not, shall be guilty of a misdemeanour.

A noxious thing is one which will produce the effect mentioned in section 58, and if a substance given produces an abortion this will establish that it is a noxious thing.[35] The quantity of a substance used may decide the question as to whether or not it is a noxious thing.[36]

In both England and Scotland if a woman should die as the result of criminal abortion, the responsible person is chargeable with the crime of murder. In actual practice, the crime charged is manslaughter, in England, and in Scotland, culpable homicide.

By the Infant Life (Preservation) Act, 1929, which is applicable only to England, it is enacted that :—

In the trial of any person for murder or manslaughter of any child, or for infanticide, or for an offence relating to the administration of drugs or the use of instruments to procure abortion, if the jury are of the opinion that the person charged is not guilty of murder, manslaughter, or infanticide or of administration of drugs or the use of instruments to procure abortion, as the case may be, but is shown by evidence to be guilty of child destruction, such person may be so convicted and punished.

It will be observed that, under the Offences Against the Person Act, when a second party attempts to procure the abortion of a woman, the criminality or culpa of the act lies in the intent to procure abortion, and it therefore follows that the person upon whom the intentional procedure has been practised need not have been pregnant. If, however, a woman attempts her own abortion, she is not chargeable unless pregnant. Neither do the provisions of the statute, in terms, make any allowance for the medical practitioner who, with intent, brings about premature labour, with the object of saving the life of the mother from some concurrent condition or disease, from the effects of which she may die. It is quite true that the act must be performed with a distinct felonious intent, for the law in practice recognises that the operation may be required in certain justifiable circumstances. In order to protect himself, the medical practitioner, when he deems such an operation urgently required, should ask and obtain, after consultation, the concurrence of another medical man, preferably an obstetrician, and, if possible, the consent also of the husband, after explaining the situation to him.

The case of R. v. Bourne[37] is of outstanding interest and importance since it deals with the attitude of the law with regard to members of the medical profession and the induction of abortion by them. An eminent obstetric surgeon was charged with unlawfully using an instrument with intent to procure the miscarriage of a girl under the age of fifteen years. The girl had become pregnant as the result of rape committed upon her, in respect of which two men had been convicted while a third had been convicted of attempted rape. The accused freely admitted that he had performed the operation upon the girl in a well-known

London hospital. There had been no effort to conceal the operation. It was submitted by counsel for the defence that the accused had not acted unlawfully since he had operated because the girl's health would have been seriously endangered if the child had been born, and it was impossible to draw a distinction between danger to life and danger to health. The case for the Crown was that the law only made it lawful to use an instrument to procure miscarriage if the operation was performed to save the mother's life. Mr Justice Macnaghten directed the jury that the test which they must apply was whether the prosecution had proved to their satisfaction, beyond reasonable doubt, that the defendant's act was not done in good faith for the purpose only of preserving the life of the girl, and that in a case where a doctor was of opinion that a child could not be delivered without the death of the mother resulting, he was entitled and indeed it was his duty, to perform an operation as soon as possible with a view to saving the mother's life. If a pregnancy was likely to make the mother a physical or mental wreck, a jury was entitled to take the view that a doctor who operated in those circumstances, and led by that belief, was operating for the purpose of preserving the life of the mother. The defendant was acquitted. The decision in this case may be held to have widened the interpretation of what was previously regarded as justifiable abortion. This trial has been described as "less of a criminal trial than a co-operative effort by judge, jury, counsel, and witnesses to create law out of strong but ill-defined feeling."

The Report by the Inter-Departmental Committee on Abortion, 1939,[38] makes it clear that, in the opinion of the Committee, the law should be amended to make it unmistakably obvious that a medical practitioner is acting legally, when in good faith he procures the abortion of a pregnant woman in circumstances which satisfy him that continuance of the pregnancy is likely to endanger her life or seriously to impair her health, and it points out that it was not until 1938, with the verdict in the Bourne Case, that any real guidance was obtained upon this important aspect of the law. The Committee advised consultations before a pregnancy is terminated and viewed with sympathy the proposal that, to a girl or woman pregnant as the result of rape, legal sanction for termination should be extended, but pointed out the practical difficulties in any attempt to arrive at an effective scheme. The same attitude applied to the results of incestuous intercourse.

By the law of Scotland anyone who feloniously causes or procures a pregnant woman to abort is guilty of a very serious crime, whether it is effected by drugs or by the use of instruments or any other means. An unsuccessful attempt is also criminal. To induce abortion as a necessary medical operation is not an offence. A woman herself may be guilty, if she is aware of the purpose for which the drug is administered or the instrument used. Administration of drugs or use of instruments to procure abortion is criminal, although abortion does not follow.

It is necessary in the libel against the accused to aver that the woman was pregnant and to prove the pregnancy at the trial.

Modes of producing abortion illegally.

From the terms of sections 58 and 59, Offences Against the Person Act, 1861, it will be apparent that, although the illegal employment of a poison, other noxious thing, or an instrument is condescended upon, "other means whatsoever" will embrace any other mode which might not be comprehended under any of the named causes.

It must not be forgotten that somewhere in the region of 20 per cent. of pregnancies end in natural abortion, and therefore that cases of suspected criminal abortion must be investigated with great thoroughness in order to establish whether the abortion has actually been brought about by criminal means.

It has been estimated that between 110,000 and 150,000 abortions occur annually from all causes in this country and that of these some 50,000 are criminal abortions. Successful cases do not engage the attention of the authorities as a rule.

From the medico-legal point of view, the methods of inducing criminal abortion may be divided into two main classes :—

Employment of drugs.
Employment of instruments.

Drugs.

It is impossible to assert that any given drug in any given dose will produce abortion. Death may occur from poisoning without the onset of abortion.

Among quacks, the employment of drugs is a method of attempting to procure abortion, chiefly because the sale or supply may be effected under circumstances incapable of legal proof, but most of them, short of toxic dosage, have little or no effect on the uterus or fœtus, since the uterus would appear to be sensitive towards them only when it is approaching term. Among the ignorant, many methods are employed, such as the free use of cathartic medicines, savin, pennyroyal, aloes, tansy, and other drugs. Certain other drugs are employed by those who are more enlightened, such as cantharides, diachylon, or lead plaster, made up as pills, ergot, the free use of quinine, extract of cotton-wood, and several others. The practitioner who has fallen into devious ways, and who knows the comparative uselessness of drugs for this purpose, usually resorts to mechanical interference by the use of instruments.

Under disguised names, such as "Female Remedies for Obstructions," etc., a considerable traffic in abortifacient drugs exists. By the Pharmacy and Medicines Act, 1941 (see p. 515), no person may take any part in the publication of any advertisement referring to any article or articles of any description, in terms which are calculated to lead to the use of that article or articles of that description for procuring the miscarriage of women.

All drugs having an abortifacient effect operate in one or other of the three following ways, namely : by acting on the body generally as a poison ; by acting locally and indirectly upon the uterus through the gastro-intestinal or genito-urinary tract ; by acting locally and directly upon the muscular structure of the uterus.

Little need be said about the vast number of drugs which have been used with the object of inducing abortion, but some consideration must be given to a limited number which apparently exert a definite action upon the uterus. Of these, pituitary extract, ergot, quinine, and lead are worthy of special mention.

Pituitary extract.

An extract of the posterior lobe of the pituitary gland exercises an oxytocic action, and stimulates the uterine muscle which is extremely sensitive to this particular substance. Pituitrin has a specific effect on uterine muscle, causing definite and strong contractions, especially near term.

Ergot.

This is a true ecbolic which acts on the musculature of the uterus on account of the contained substances, ergotoxine, ergotinine, ergotamine, ergometrine and ergotaminine, in addition to histamine and tyramine. Experiments have shown that the alkaloid, ergotoxine produces uterine contractions in about fifteen to thirty minutes. It is the most important factor in ergot poisoning, and causes contraction of the arterioles. Ergometrine, however, is the substance which is chiefly responsible for the action of ergot on the uterus. When given by the mouth it produces a prolonged contraction of the uterus within about five minutes. The uterus is particularly sensitive immediately following the birth of its contents.[39] When liquid extract of ergot is taken orally, a very marked contraction of the uterus follows after a latent period of a few minutes due to the water soluble alkaloid ergometrine (see p. 672).

Quinine.

It is now generally accepted that quinine definitely increases uterine contractions of a rhythmic character, raises the tone of the muscle, and that it may induce uterine contractions (see p. 669).

Lead.

Diachylon, or lead plaster, which contains oleate of lead, is sometimes employed as an abortifacient. This substance is easily rolled into pills which are coated with substances of different colour. Dilling found that lead causes tonic contraction of the uterus. It is believed to have a toxic action on the trophoblastic epithelium of the ovum. For these reasons lead must be regarded as a definite abortifacient drug. Symptoms of lead poisoning have frequently shown themselves in persons who have taken diachylon for the purpose of abortion, and in some of these cases there have been fatal terminations.

In one of our cases, the pregnant woman had been given pills and had been instructed to take three of them daily until abortion resulted. The woman aborted (see p. 560).

Ergot and lead plaster are included in the Poisons List to which special restrictions apply (see p. 503).

Instruments.

Instruments may be used in the first instance or be resorted to when drugs have failed to induce abortion.

Instrumental interference, usually attempted after the second or third missed period, is brought about by the employment of a wide variety of instruments, which includes such implements as knitting or crochet needles, wooden or metal skewers, catheters, sounds, curettes, syringes, and the bark of slippery elm, among many others. Abortionists may rupture the membranes and then request the patient to report to her own doctor. A woman may induce abortion on herself, but usually this is done by a second person. Sometimes the woman may try to persuade her doctor to pass a sound on the pretext that she is suffering from a uterine displacement.

For many years the bark of slippery elm, Ulmus fulva, has been employed, either in the form of a tent or as a long stem which is passed into the cervix, since it has the property of exuding mucilaginous material and of absorbing moisture, thus causing dilatation of the cervix by the resultant increase in bulk of the portion inserted. The bark is obtained from a tree which grows in Central and North America. It occurs in flat pieces of varying length and width and is about $\frac{1}{8}$ inch in thickness. The pieces are cut to the desired length and breadth prior to insertion.

The chief danger of instrumental interference is from septicæmia following wounding of the parts.

Cook[40] has recorded an interesting analysis of three hundred and fifty cases of abortion which were admitted to hospital. His conclusions were that about 40 per cent. of these had probably been procured, that the commonest cause of procured abortion was probably the insertion of slippery elm bark, and that local interference was usually performed with a certain amount of mechanical skill, since it was rare to find positive evidence of injury. Uterine sepsis, accompanied by pyrexia, was present in 35 per cent. of the cases, and uterine sepsis, without pyrexia, in about a further 10 per cent. of the cases. Of these patients, 20 per cent. were decidedly ill and 3 per cent. died. With one exception, all the deaths were due to sepsis. Definite injury was present in four cases, two of which proved fatal, due to perforation of the uterus and perforation of the vaginal fornix. His analysis of the stage of pregnancy at which abortion took place was as follows :—

Under two months	46 cases.
Between two and three months . .	126 ,,
Between three and four months . .	124 ,,
Between four and five months . . .	44 ,,
Between five and six months . . .	10 ,,

The injection of fluids into the uterus, frequently with a Higginson's syringe, for the purpose of inducing abortion is quite common. Tepid water, soap and water or disinfectants such as carbolic acid or lysol, are often used and each is fraught with danger. Apart from the danger of absorption of poisonous substances, in cases in which these may be employed, there is always the grave risk of sudden death from syncope or from air embolism.

Skilled interference usually leaves no traces. When dilatation of the cervix or perforation of the membranes has been undertaken, abortion usually results in about fifty to sixty hours. During the first three months of pregnancy, skilled interference takes the form of dilatation of the cervix under anæsthesia, and immediate evacuation of the uterus with the index finger and a flushing curette. The preliminary use of a laminaria tent, twelve hours before, is often of considerable assistance as an aid to dilatation of the cervix. During the next three months, the membranes may be ruptured by a sound followed by packing of the cervical canal and vaginal fornices, while during the terminal three months the procedure consists in the introduction of one or more gum elastic bougies into the uterus, between the membranes and the uterine wall.

Duties of medical practitioners in relation to cases of criminal abortion.

The resolutions of the Royal College of Physicians,[41] concerning the duties of medical practitioners in relation to cases of criminal abortion are :—

That a moral obligation rests upon every medical practitioner to respect the confidence of his patient, and that without her consent he is not justified in disclosing information obtained in the course of his professional attendance on her.

That every medical practitioner who is convinced that criminal abortion has been practised on his patient, should urge her, especially when she is likely to die, to make a statement which may be taken as evidence against the person who has performed the operation, provided always that her chances of recovery are not thereby prejudiced.

That in the event of her refusal to make such a statement, he is under no legal obligation to take further action, but he should continue to attend the patient to the best of his ability.

That before taking any action which may lead to legal proceedings, a medical practitioner will be wise to obtain the best medical and legal advice available, both to ensure that the patient's statement may have value as legal evidence, and to safeguard his own interests, since in the present state of the law there is no certainty that he will be protected against subsequent litigation.

That if the patient should die, he should refuse to give a certificate of the cause of death, and should communicate with the Coroner. (Note.—In Scotland, the Procurator-Fiscal of the area is the official to whom the communication should be made.)

The College has been advised that the medical practitioner is under no legal obligation either to urge the patient to make a statement, or, if she refuses to do so, to take any further action (see p. 387).

(Note.—With regard to dying declarations and dying depositions, see p. 55.)

Evidence of Criminal Abortion.

Upon what must medical evidence of criminal abortion depend ? The following should be taken into full and careful consideration before an opinion is offered :—

The history of the woman.
The examination of her body.
The examination of the aborted material, if available.

It is important to remember that criminal abortion is most usually practised between the second and fourth months of pregnancy, although it may be induced at later periods, and that an attempt to procure abortion may be made upon a woman who is not pregnant but who merely fears that she is because her menses have temporarily ceased.

History of the woman.

In many instances, this may not come within the knowledge of the medical examiner, but when there is a history of the use of drugs or instruments, all the available facts and circumstances should be carefully reviewed.

Examination of the woman's body.

When opportunity offers, in living cases, the examination must be made as soon as possible, and should include a careful scrutiny of all evidence consistent with recent pregnancy together with a vaginal examination. The finger may not be able to detect small breaches of continuity, but it will probably discover changes in the "feel" of the mucous membrane of the os, and of the patency of the os. Visual examination with a speculum is essential, since not only will the general condition of the genital tract be ascertained but, if injury is present, it will be more readily detected. In some cases of criminal abortion, hæmorrhage may persist due to a retained portion of the membranes or placenta.

A suspicion of criminal abortion should be aroused when the patient shows signs and symptoms of localised sepsis or of general septicæmia. It must not be forgotten, however, that sepsis may follow natural abortion, but in such cases there is usually some preceding clinical history.

In cases of criminal abortion, death may result from shock due to internal injury, hæmorrhage, sepsis, which may assume the form of peritonitis or septicæmia, or from embolism. When poisonous substances have been administered, death may supervene from their effects.

In making a post-mortem examination, the examiner should note carefully the presence or absence of the signs of pregnancy which may be visible upon the exterior of the body. The breasts should be examined and an incision on the under surface of each made to ascertain secretory activity. Having inspected the vaginal tract, dissection of the body should be proceeded with in the ordinary manner and special attention directed to the condition of the pelvic contents. The first thing to note will be the presence or absence of inflammation in this cavity, and, if present, its incidence and extent. Before removing any of the parts from the cavity, careful search must be made for the presence or absence of any penetrating wound of the peritoneum, and if present, the position and character with relation to the uterus, and the tissues penetrated must be noted. Special attention must be directed to the posterior fornix of the vagina, in the region of the pouch of Douglas, since this is a common site of injury in criminal abortion. Thereafter the pelvic viscera are removed en masse (see p. 41). The parts are then examined, carefully and systematically, in a good light. Portions of tissue, including tissue from the placental site, for microscopic examination, should not be excised until the general examination has been completed.

Fig. 193

Fœtus lying in unruptured membranes.

The urinary tract is carefully inspected for signs of inflammation, such as might be produced by cantharides or turpentine, and the vaginal tract, for evidence of injury.

The length, breadth, and thickness of the uterus, the length of the uterine cavity, the circumference of the external os, and the length of the cervix should be recorded (see p. 701). The uterus and the cervix must be examined in detail to establish or eliminate the presence of injury. When small lacerations of the cervix are detected, inquiry should be made regarding possible remedial surgical treatment in hospital prior to death, since such marks are

sometimes due to tenaculum injuries so sustained. The ovaries are examined for the presence of a corpus luteum (see p. 376). In septic cases, any source of infection from organs other than those of the pelvis, especially the appendix, must be eliminated.

Where there are no signs of local injury, the condition of the gastro-intestinal tract must be examined very carefully for evidence of irritant poisoning, and when such evidence is found, the whole tract and its contents, together with other organs of the body, should be placed in clean jars for the purpose of subsequent analysis.

Examination of the aborted material.

Any available material passed by the vagina should be carefully examined, since if it can be proved that it is composed of products of conception, the fact of pregnancy will at once be established. If a fœtus has been retained, an estimation of the stage of its development should be made (see p. 80). In the dead body, portions of material found within the uterus should be the subject of detailed naked-eye and microscopic examination. It is always advisable to remove a piece of tissue from what is thought to have been the placental site, so that the presence of chorionic villi may be identified microscopically (see Figs. 194 and 195).

Has the abortion been produced criminally?

It must be kept prominently in mind that abortion may be produced accidentally, or from disease. The commoner diseases are chronic nephritis, diabetes, acute fevers, especially of the exanthematous type, degenerative changes in the villi of the chorion or in the placenta, together with accidental hæmorrhage between the uterine wall and the placenta. Maternal syphilis causes most serious harm from the third month onwards, and in the later weeks of pregnancy may result in miscarriage and still-birth. Diseases of the fœtus are also a cause of abortion. Violent emotion in certain types of women is occasionally provocative.

Abortion may also be produced in some women by apparently trifling accidents, and a miscalculated step from a height, a marked and sudden jar to the body, or an accidental knock against the abdomen, may prove a precipitating cause. In other women, even serious abdominal injury may fail to precipitate the expulsion of the fœtus, despite the fact that pregnancy may be approaching full-term.

The question of criminal abortion arises when there is evidence of injury to the vaginal wall, the cervix, or to the uterus.

Cases arise from time to time in which, from attendant circumstances, medical practitioners may be compromised, but in which the abortion has been brought about by the woman herself, either by the administration of drugs or by instrumental interference.

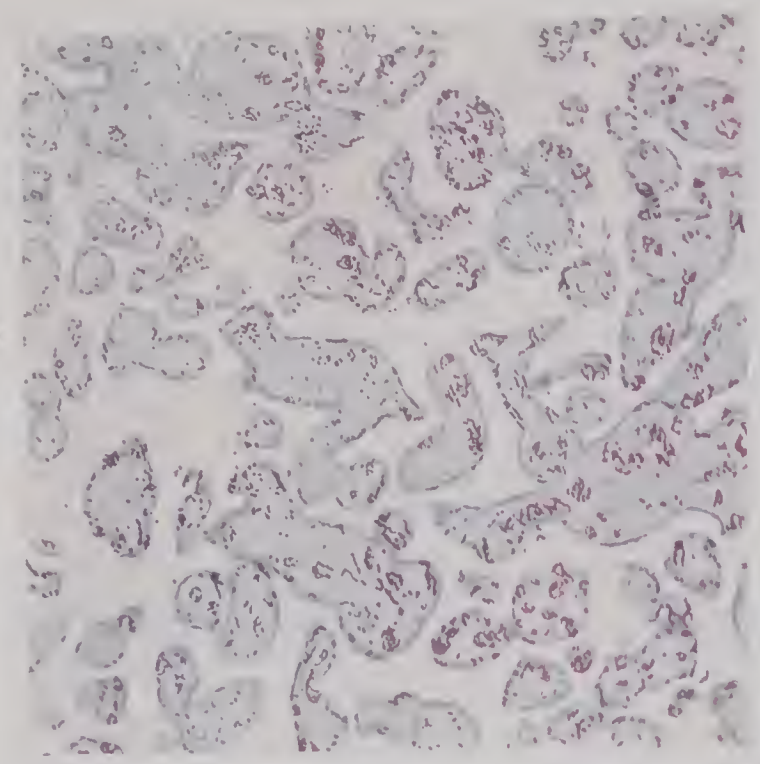


Fig. 194

Chorionic villi at earlier stage of development and vascularisation. (Section by courtesy of Dr D. McKay Hart.) ×80.

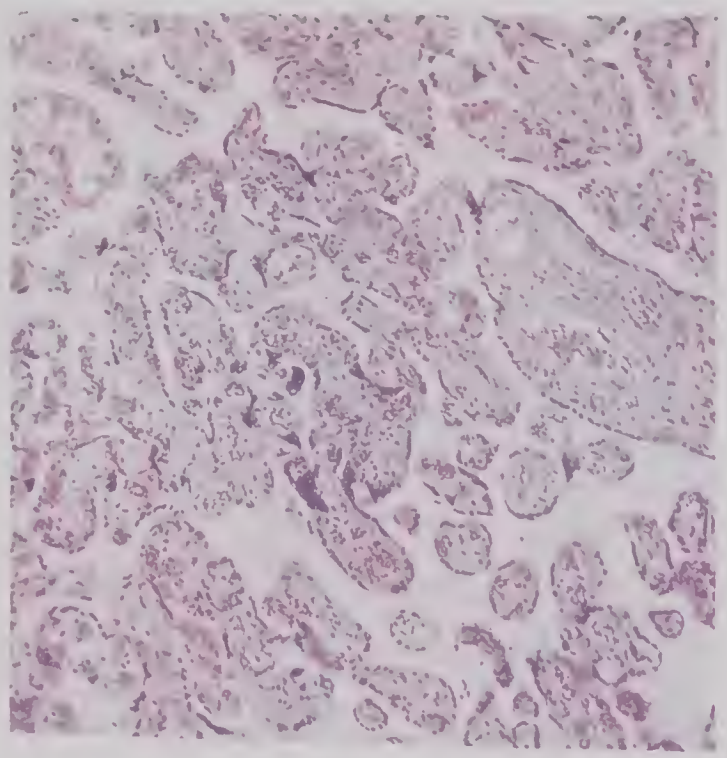


Fig. 195

Chorionic villi at later stage of development and vascularisation. (Section by courtesy of Dr D. McKay Hart.) ×90.

A medical practitioner may be called in after the process of abortion has been initiated by the woman herself, or by another, to save the life of the woman, and should she die without confession, and a post-mortem examination of her body should follow, it is not difficult to understand how he might be compromised in the eyes of the law, and how serious consequences to him might result. In any doubtful case he should call in a fellow-practitioner in order to safeguard himself. It is also advisable to make arrangements for a neighbour of the patient, or preferably a nurse, to be called in so that she may be able to corroborate the facts concerning the abortion, prior to admission of the patient to hospital.

ILLUSTRATIVE CASES

The following illustrative cases include many of the points which have been discussed :—

General peritonitis.

On the right side of the anterior fornix was a lacerated wound, $\frac{1}{2}$ inch by $\frac{1}{4}$ inch. It passed upwards and backwards for 2 inches, and penetrated the peritoneal cavity. The track admitted a No. 10 metal catheter. Two pints of pus were present in the abdomen. Around the entrance of track were five additional superficial wounds varying in length from $\frac{1}{2}$ to $\frac{1}{16}$ inch. These involved the mucosa and showed parallelism. There were also two other superficial wounds.

General peritonitis and septicæmia.

Two lacerated wounds perforated the posterior wall of the uterus and entered the pelvic cavity (see Fig. 196).

Slippery elm bark retained in vagina.

A woman, when examined, was found to have a piece of slippery elm bark in her vagina. It measured 4 inches by $\frac{1}{2}$ inch by $\frac{1}{8}$ inch and was retained in position by a plug of cotton-wool about the size of a small walnut. One of its ends had been cut into an arrowhead shape. The other end had a small pledget of cotton-wool wrapped around it. The woman, who was four months pregnant, did not abort. The slippery elm had not been self-inserted. The woman had given birth to six children.

Localised peritonitis involving uterus and pouch of Douglas.

The following injuries were present :—

A lacerated wound, immediately to right of midline of anterior wall of vagina, 8 millimetres in length and in antero-posterior direction.

A similar wound 9 millimetres long, to right of, and just below, the first.

A similar wound, 7 millimetres long, on right wall of vagina, to right and slightly below the last-mentioned wound.

All three wounds were situated peripherally at points 3 centimetres from the external os, and pus exuded from the openings on slight pressure. The first wound extended upwards in the posterior wall of bladder and utero-vesical tissue to the utero-vesical fold of peritoneum. The second wound extended upwards and to the right, passing more obliquely and to a slightly higher level than the first, to terminate in cellular tissue infiltrated with pus. The third wound terminated in septic cellular tissue at the root of the right broad ligament. In the middle of the posterior lip of cervix was a small septic opening 3 millimetres in diameter, and extending upwards in the posterior wall of cervix for a distance of 5 centimetres. A few millimetres to the right of this was a punctate wound $1\frac{1}{2}$ millimetres in diameter which penetrated tissue for 3 millimetres.

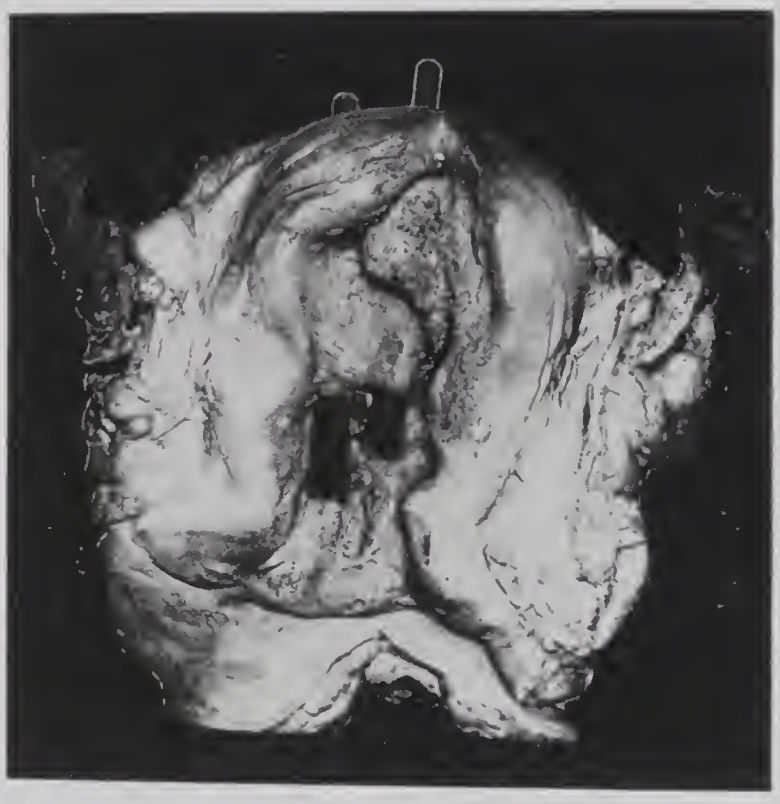


Fig. 196

Two lacerated perforations of posterior wall of uterus.

Sudden death following use of syringe.

A woman was charged with inserting a syringe into the private parts of a young woman and injecting a solution of soap and water, in consequence of which she died. The woman was convicted. The victim, who was between four and five months pregnant, died suddenly from cardiac inhibition during the injection. About 11 ounces of pultaceous, white, cloudy-looking material, containing many sago-like grains and with a scented odour, were found lying between the amniotic sac and the wall of the uterus. The cervical

canal and both its external and internal os were dilated. The external and internal os each measured ¼ inch in diameter. The cervical canal contained some slightly blood-stained, glairy mucus. On the inner surface of the lateral wall of the cervical canal, running upwards for ½ inch from the external os, was a surface laceration which communicated externally with a small lacerated wound on the outer surface of the os. Microscopic examination showed that the laceration had been inflicted during life, and its general appearances were consistent with recent injury and could readily have been caused by the forcible introduction of a foreign body. The extraneous substance, in the uterine cavity, was examined chemically and found to consist of soap. A cake of soap, similar to that used, was also analysed and the results compared with those obtained from analysis of the material found within the uterus. Both were similar in odour and chemical composition. The general development of the fœtus and placenta present in uterus indicated a fourth to fifth month pregnancy.

Gordon [42] describes the case of an eighteen-year-old girl, five months pregnant, who was found lying on her back on the floor, dead, with a basin of soapy water nearby. Post-mortem examination showed air bubbles in the cerebral veins and in the external jugular veins. Frothy blood was present in the internal jugular veins, the right ventricle, right auricle, and in the pulmonary artery. A moderate amount of blood-stained fluid was in the uterine cavity. The placenta was detached from the uterine wall along its lower edge. The fœtal membranes were unruptured.

Brown [43] writes of fatal air embolism after insufflation of the vagina owing to vaginal discharge, eight weeks prior to term. The substance used was "Pieragol" powder, a compound powder containing 1 per cent. silver picrate in purified kaolin. Within a minute of the start of insufflation, the woman, aged twenty-five, and a primipara, complained of a feeling of being "blown up," acute dyspnœa supervened, she had a fit, recovered slightly, then died. Post-mortem examination showed dilatation of the right side of the heart. The right ventricle contained very little blood, frothed into large bubbles. The right uterine plexus contained frothy blood. The right ovarian vein contained particles of yellow powder. The edge of the placenta was stripped for a depth of about 1 to 1½ inches from the right side of the uterus. The membranes on this side were completely free. Blood sinuses on the right side of the uterus, in the vicinity of the stripped part of the placenta and membranes, contained frothy blood, and these drained directly from the uterine plexus already mentioned. Insufflation of the vagina with the cervix partly dilated had allowed the entrance of air into the blood sinuses of the uterus, after the ballooning effect of the insufflation had stripped the membranes and a portion of the placenta from their site.

Dodds and Mayeur [11] report a case of perforation of the posterior vaginal wall and rectum by the hard nozzle of a Higginson's syringe. The patient was twenty-four and was pregnant. Following suture,

she made a complete recovery and was subsequently delivered successfully by Cæsarean section.

Slippery elm bark in urethra.

Farncombe [45] describes a case of attempted abortion by self-introduction of slippery elm into the urethra instead of the vagina, in error. There was a small amount of granulation tissue surrounding the urinary meatus. Radiological examination showed the presence of a cylindrical foreign body lying in the region of the urethra and neck of bladder. The woman admitted having introduced a piece of slippery elm into the womb, as she thought, during the third month of pregnancy. The patient, who was at full time when she came under observation, was thirty-nine and the mother of six children, went into labour unexpectedly before operation for removal of the foreign body. The labour was normal, and she was delivered of a healthy, full-time child which weighed 7 pounds. Considerable damage resulted to the urinary tract. Suprapubic cystotomy was performed and the foreign body, projecting from the urethra into the bladder, was removed. It consisted of a core of slippery elm, about 10 inches long and $\frac{1}{6}$ inch in diameter. The piece, which was doubled on itself, had a deposit of phosphates upon it which formed a conical mass, $4\frac{1}{2}$ inches by $2\frac{1}{2}$ inches. The woman made a good recovery.

References

1. Lancet, Vol. II, 61, 1940, Fairfield.
2. "Hermaphroditos," Cawadias. Heinemann, London, 1943.
3. Jour. Pediatrics, 31, 142, 1947, Bergman.
4. Brit. Jour. Surg., Vol. XXII, 619, 1934-35, Morrison.
5. Presse méd., Vol. XLVII, 459, 1939, Raynaud, Marill, and Xicluna.
6. Lancet, Vol. II, 940, 1946, McFadzean.
7. Jour. Amer. Med. Ass., Vol. CIV, 1968, 1935, O'Farrell.
8. Brit. Med. Jour., Vol. I, 822, 1933, Hodgson.
9. Ibid., Vol. I, 1159, 1937, Patel.
10. G.'s Trustees v. G. S. C., 837, 1936.
11. Brit. Med. Jour., Vol. II, 211, 1935, Anderson.
12. F. v. F., Scots Law Times, Part XVII, 16th June 1945.
13. Clark v. Clark, Lancet, Vol. I, 89, 1943.
14. Med.-Leg. Rev., Vol. X, Part IV, 202, 1942, Latey and Kitchin.
15. Lang v. Lang (1945), Scots Law Times, Parts XXXIV and XXXV, 310.
16. Lancet, Vol. I, 480, 578, 768, 1944, Safford v. Safford.
17. "Artificial Human Insemination." Report held under the auspices of the Public Morality Council, 1947. Heinemann, London.
18. Med. Leg. Rev., Vol. XII, Part III, 138, 1944, Forbes.
19. "Recent Advances in Sex and Reproductive Physiology," Churchill, 1934, Robson.

20. Brit. Med. Jour., Vol. I, 254, 1943.
21. Surg. Gynec. Obstet., Vol. LII, 56, 1931, Schneider.
22. Trans. Edin. Obstet. Soc., 124, 1929-30, Robertson.
23. Lancet, Vol. II, 923, 1948, Haines.
24. "Pharmacology," 56, Gaddum. Oxford Med. Press, 1940.
25. Lancet, Vol. II, 61, 1940, Fairfield.
26. Med.-Leg. Rev., Vol. IV, Part I, 16, 1936, M'Cann.
27. Brit. Med. Jour., Vol. I, 378, 1917, Gilbertson.
28. Jour. Amer. Med. Ass., Vol. CVI, 1728, 1936, Seymour.
29. "Sterility and Impaired Fertility," Roberts, Sharman, Walker, Wiesner, and Barton. Hamish Hamilton, London, 1948.
30. Lancet, Vol. II, 448, 1946, Sevitt.
31. Ibid., Vol. II, 357, 1921, Gaskill v. Gaskill.
32. Med. Annual, 288, 1940, Clark v. Clark.
33. Hadlum v. Hadlum [1948], 2 All E.R., 412, 425, C.A.
34. Wood v. Wood [1947], C.L.Y., 807.
35. R. v. Hollis (1873), 28, L.T., 455.
36. R. v. Hennah (1877), 13, Cox C.C., 547.
37. R. v. Bourne (1938), Police Jour., Vol. XI, No. 4, 398.
38. H.M. Stationery Office, London, 1939, Report by Inter-Departmental Committee on Abortion.
39. "Pharmacology," Gaddum. Oxford Med. Press, 1940.
40. Brit. Med. Jour., Vol. I, 1045, 1938, Cook.
41. Ibid., Vol. I, 207, 1916, Resolutions of Royal College of Physicians.
42. Clin. Proc., 4, 135, 1945, Gordon.
43. Lancet, Vol. I, 616, 1943, Brown.
44. Brit. Med. Jour., Vol. I, 921, 1939, Dodds and Mayeur.
45. Lancet, Vol. II, 825, 1935, Farncombe.

CHAPTER XIII

INFANTICIDE OR CHILD MURDER

THE killing of a newly-born child is, in England, known as infanticide, and the crime may be committed either by acts of omission or by acts of commission.

Infanticide by an act of omission means neglect to do what is necessary for the continuance of the life of a newly-born child, for example, neglecting to tie the umbilical cord after severance, since by omitting to do this the infant may bleed to death, neglecting to clothe it in a reasonable way so as to protect it from cold, or failing to remove such obstacles as would prevent it from breathing.

Infanticide by commission is the performance of any positive act which prevents a newly-born child from living or which destroys its life.

Law in England.

Infanticide has been the subject of statutory enactments, the most recent being the Infanticide Act of 1938, by which, under certain circumstances, the killing of a newly-born child by the mother is the crime of manslaughter. This Act provides that :—

1. Where a woman by any wilful act or omission causes the death of her child, being a child under the age of twelve months, but at the time of the act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child, or by reason of the effect of lactation consequent upon the birth of the child, then, notwithstanding that the circumstances are such that but for the Act the offence would have amounted to murder, she shall be guilty of felony, to wit, of infanticide, and may for such offence be dealt with and punished as if she had been guilty of the offence of manslaughter of the child.

2. At the trial of a woman for the murder of her child, the child being under the age of twelve months, if the jury are of opinion that she by any wilful act or omission caused its death, but that she was in the condition already described, then the jury may return a verdict of infanticide.

3. Nothing in the Act shall affect the power of the jury upon an indictment of murder of a child to return a verdict of manslaughter, or a verdict of guilty but insane, or a verdict of concealment of birth, in pursuance of Section 60 of the Offences against the Person Act, 1861, except that for the purposes of the proviso to that section

a child shall be deemed to have recently been born if it had been born within twelve months before its death.

In every charge of infanticide the legal presumption is that the child is born dead, and the onus of proving that the child was born alive rests on the Crown.

As an alternative charge to infanticide, the accused may be charged, under Section 60 of the Offences against the Person Act, 1861, with concealment of birth. This section provides that if a woman is delivered of a child, every person who has by any secret disposition of the dead body of the child, whether such child died before, at, or after its birth, endeavoured to conceal the birth, shall be guilty of a misdemeanour, subject to the proviso that if any person is tried for the murder of any child and is acquitted, the jury may find, if it so appears on evidence, that the child had been recently born, and that the accused did, by some secret disposition of the dead body of the child, endeavour to conceal the birth, in which case the Court may pass such sentence as if the accused had been convicted upon an indictment for the concealment of the birth.

With the advent of the Infant Life (Preservation) Act in 1929, the law was amended with regard to the destruction of children at or before birth. It provides that :—

Any person who, with intent to destroy the life of a child capable of being born alive, by any wilful act causes a child to die before it has an existence independent of its mother, shall be guilty of felony, to wit, of child destruction, but not if it is proved that the act which caused the death of the child was done in good faith for the purpose only of preserving the life of the mother. For the purposes of the Act, evidence that a woman had at any material time been pregnant for a period of twenty-eight weeks or more shall be prima-facie proof that she was at that time pregnant of a child capable of being born alive.

The Act, which applies to England only, also provides that in the trial of any person for murder or manslaughter of any child, or for infanticide, if the jury are of the opinion that the person charged is not guilty of any of these offences, but if shown by evidence to be guilty of child destruction, he may be so convicted and punished.

Law in Scotland.

In Scotland, the crime is known as child murder and at common law there is no degree of difference between the murder of a child and the murder of an adult. During the seventeenth century, however, the crime became one of such frequent occurrence and the difficulty of proving it so great, that an Act was passed in 1690 which provided that if certain indicia were proved, the jury were entitled to presume that the crime of child murder had been committed. Owing to its severe nature, this enactment was repealed by the Concealment of Pregnancy Act, 1809, which reduced the crime to one of culpable homicide.

The Act of 1809 provides that if any woman conceals her being with child during the whole period of her pregnancy and does not call for and make use of help or assistance at the birth, and the child is found dead or amissing, she may be convicted and imprisoned.

Under this Act the onus is on the Crown to prove (1) that the woman was pregnant and that she concealed this fact during the whole period of pregnancy, (2) that she failed to call for or make use of help at the birth, and (3) that the child has been found dead or amissing.

It is a conclusive common law presumption that a woman who is pregnant and who does not reveal her condition or call for assistance at child-birth is recklessly indifferent to the life of the child.

It is advisable at this stage to give the legal definitions of certain terms. At common law, to constitute "live-birth" the child must have been fully extruded from the parts of the mother, and have achieved an independent existence, but it is not necessary that the child should have breathed, or that the cord should have been cut, but the child must have given some active evidence of life. The issue as to whether a child can have a separate existence before the umbilical cord is severed arose in a case of infanticide at Cambridge Assizes.[1] The ruling was that the entire child must actually have been born into the world in a living state, and the fact that it had breathed was not conclusive proof of separation, because it might have breathed during the act of separation. The fact that the child had breathed, although not conclusive, went some way toward the proof of independent existence. Before a child can be considered alive, it must have had an independent circulation and no longer derived its power of living by or through any connection with the mother. The fact that the child was still connected to the mother by the cord did not prevent the killing from being murder. The jury had to decide whether or not the child had shown a separate existence, if not, the mother must be acquitted of the charge of infanticide.

It will thus be seen that the legal definition of live-birth is divergent from the medical definition, as in the latter, evidence of respiratory action of the child whether initiated partly within or wholly without the maternal parts, is indicative of live-birth. Frequently, on account of the difficulty in determining by physical appearances of the body whether respiratory function was initiated while a portion of the child was within the maternal parts, or after complete extrusion, the alternative charge to infanticide or child murder, namely, a charge of concealment of birth, in England, or of pregnancy, in Scotland, is returned as the verdict of the jury.

When may a child be declared still-born?

The terms "still-born" and "still-birth" shall apply to any child which has issued forth from the mother after the twenty-eighth week of pregnancy, and did not at any time, after being completely expelled from its mother, breathe or show any other signs of life (see pp. 154 and 156).

This question has arisen in a court of law in connection with a charge against a practitioner of alleged falsification of a birth certificate. During the evidence, a number of witnesses stated that the child was born alive about half an hour after the doctor had left the house of the mother, who was then in labour, that they heard it cry, or make a whining noise, and that it had died between three and six o'clock next morning. The accused gave a certificate stating that the woman had been delivered of a still-born child. He stated in his defence that the child was a six months' child, and that he inferred from this fact that it could not be born alive. The jury gave the accused the benefit of the doubt, but stated that they believed he was guilty of gross carelessness in giving such a certificate. It must be clearly understood that a child, irrespective of its intra-uterine development, which manifests signs of independent existence, as, for example, attempts at respiration, the act of crying, or the continuous action of the heart even for some minutes after birth, must be considered as born alive.

The following medico-legal questions arise in cases of infanticide or child murder :—

Has the woman charged been recently delivered ?
Is the body of the child found that of a viable child ?
Is the body that of the child of the person accused ?
Was the child born alive ?
If the child was born alive, what caused its death ?
How long has the child been dead ?

Is the body of the child found that of a viable child ?

The answer to the first question has already been discussed (see pp. 370 and 379). With regard to the second question, it should be explained that unless the child born has reached a stage of development which is consistent with the possibility of a living birth, a charge of infanticide or child murder will not usually be preferred against the mother. In law, a fœtus which has not attained the completion of the seventh month of intra-uterine life is held to be incapable of maintaining a separate existence, and is therefore non-viable. Viability means, therefore, the capability of a fœtus to maintain a separate existence after birth by virtue of a certain degree of development. The presumption of the law, concerning the body of a child found dead and which shows evidence of less than seven months' intra-uterine age, is that the child has failed to live by reason of its immaturity. On the other hand, should there be evidence that the immature fœtus has lived after birth, and that it has been deprived of life by the act of the mother, a charge of infanticide or child murder would lie.

Is the body that of the child of the person accused ?

Proof on this point is most usually established from the evidence of persons with whom the suspected woman has been more or less

intimately associated in work or in social life. A careful examination by the police of the wrappings which envelop the body of a child may throw some light on the identity of the mother.

Was the child born alive ?

The answer to this question opens up a wide field for consideration.

A monster is generally considered to be incapable of living a separate existence, but this will depend upon the character of the monstrosity. Siamese twins have lived for many years. Acephalous, anencephalous, hemicephalous, ectocardiac, and other teratological subjects usually die quickly after birth, although an anencephalous monster has survived birth for sixty-one hours.

The body of a child may show evidence of maceration, due to surface changes which occur within the uterus after the death of the child, and prior to its birth. In certain cases, however, one has to decide whether such appearances were produced before or after birth, since many bodies which are examined show some evidence of decomposition.

Evidence of immaturity should be carefully noted. Infants of less than seven months of intra-uterine development are as a rule incapable of maintaining a separate existence (see p. 400). Hoffmann, Greenhill and Lundeen [2] have reported the birth and survival of a premature infant which weighed 735 grammes at birth. By the eighth day the infant had lost 140 grammes in weight. Whether or not any of the foregoing conditions are present, it must still be determined, by detailed examination of the body, whether there is evidence of live-birth or still-birth, since this question will arise in every case which is the subject of medico-legal inquiry.

The post-mortem examination in all cases of infanticide or child murder must include a detailed investigation of :—

The condition of the lungs.
The changes in the umbilicus.
The changes in the digestive tract.
The changes in the circulatory system.

Condition of the lungs.

Some have considered the position of the convex surface of the diaphragm in relation to the ribs as of diagnostic value. The height of this structure relative to the ribs is greater after a child has breathed than before, but the height of the diaphragm in any case will depend chiefly upon the degree of lung inflation, which in itself is a variable condition in any series of cases of newly-born children.

A detailed examination of the lungs will afford valid proof of the condition of the child with respect to respiration. Prior to independent life, the lungs are functionless, and it is only when the living child is born that they become organs of function. Before

birth the lungs receive only a limited supply of blood necessary for their vitality and growth, but following birth the pulmonary circulation in the body of the child becomes established. In order that the process of aeration may be accomplished, respiration becomes operative. The establishment of these two vital functions, which occurs at the birth of a living child, produces physical changes in the lungs, with respect to volume, colour, and weight.

Changes in volume of the lungs.

Before birth the lungs do not fill the thoracic cavity, and the left lung does not even partially overlap the pericardium. In texture they resemble the consistence of liver tissue, are uniformly coloured, the lobes being indistinctly marked and the sharp edges lying in close contact. After the complete establishment of respiration they more or less fill the thoracic cavity, the degree of completeness depending upon the thoroughness of the respiratory action. When complete aeration of the lungs has been in operation, the left lung more or less covers the pericardium. In the apex of the right lung, the appearances of inflation are usually most marked, since the air entry to this part of the lung meets with the least obstruction. Between the condition of non-inflation and complete inflation there are intermediate stages of incomplete inflation and, to determine the extent of the air entry, further examination is necessary.

Colour of the lungs.

Prior to respiration, the lungs are of a uniform dark, bluish-red colour, but at the margins, where the tissue is thinner and therefore more translucent, they may appear lighter in colour. Unrespired fœtal lungs do not present patches of different colour, but after respiration they become marbled in appearance ; a dark bluish-red background with many red irregular patches may be seen, or the background may be of reddish colour, and the circumscribed patchy areas of dark bluish-red tint. The edges assume a more rounded contour. The marbling in appearance and colour is characteristic of natural inflation, and cannot be simulated by any artificial method. Fully respired lungs feel crepitant to the touch, whereas unrespired lungs feel solid and liver-like in consistence.

Hydrostatic test.

This test depends upon the ratio of the specific gravity of the lungs to that of water, it being a well-established fact that a lung which contains air is specifically lighter than water, and will therefore float. Flotation of the lungs, however, may occur for a reason other than that of contained atmospheric air, and, on the other hand, the lungs may sink, although the child may have breathed following birth, as the result of atelectasis or disease.

Before discussing these points, however, the practical procedure

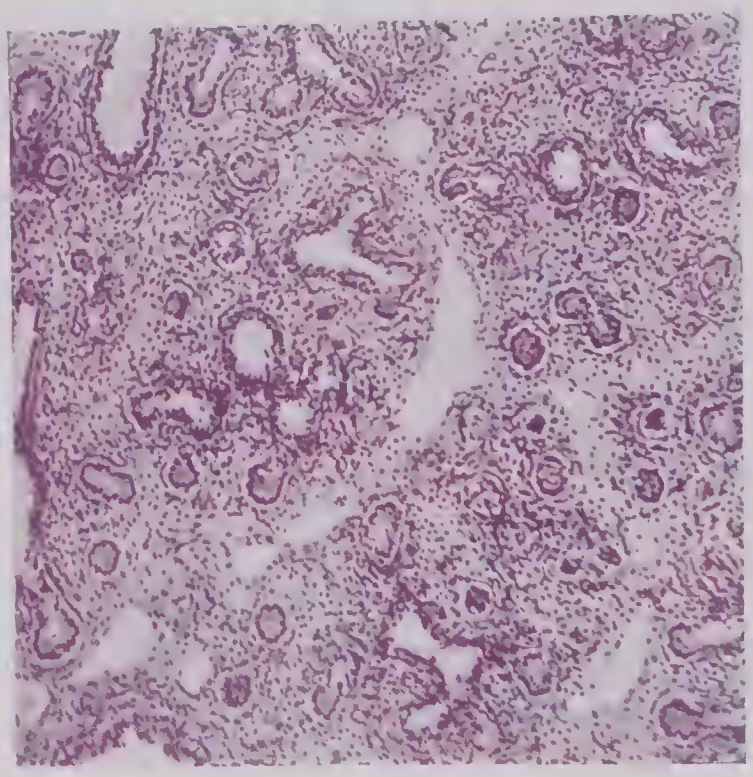


FIG. 197
Unrespired fœtal lung. ×85.

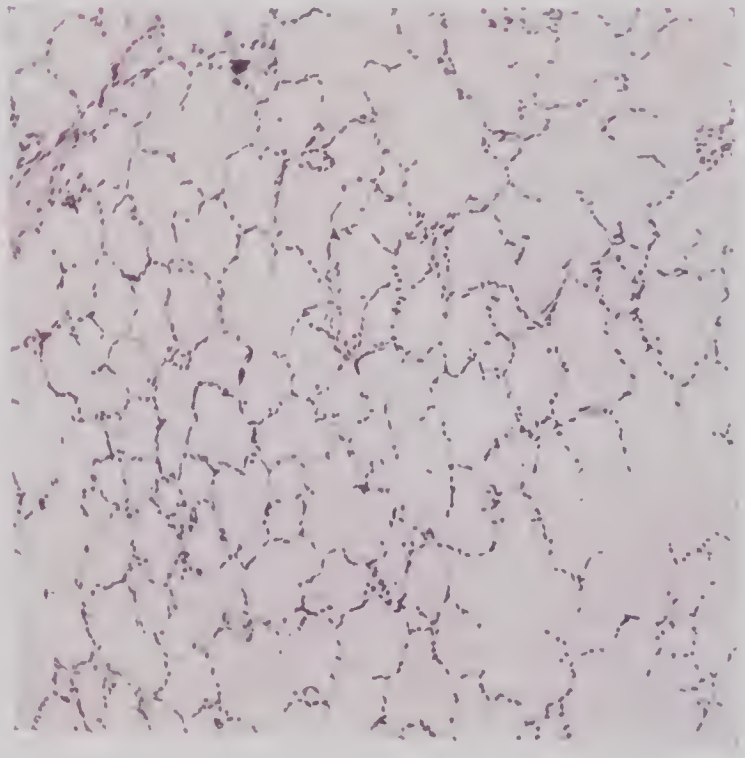


FIG. 198
Respired fœtal lung. ×85.

of the hydrostatic test will first be described. Both lungs are placed in a suitable vessel sufficiently filled with water, and their behaviour with regard to buoyancy is observed. One or other or both may float, partially float, or sink. Should the lungs float, each lung is separated into its respective lobes, and the flotation of each is tested. If all float, the next step consists in cutting up each lobe into a number of pieces and again testing their flotation. If these pieces float, each should be compressed. A number of the pieces are wrapped in a portion of cloth, placed on the floor, and strong pressure is applied with the foot, using the weight of the body for the purpose. This process is repeated until all the pieces of each lung have been so compressed. They are then placed once again in water and their behaviour in regard to flotation is carefully observed. The number and situation of the pieces which float should be noted. The examiner is now in a position to pronounce an opinion as to whether the respiration has been completely or partially established. Should one or both lungs sink when placed in water, it is still necessary to test the lobes and the portions of the divided lobes, by the flotation test, in order to eliminate partial respiration.

In cases of imperfect inflation, our experience is that the upper part of the right lung is likely to float, when the major portion of the left and the bases of both lungs sink.

It is necessary to explain why the procedure above mentioned should be employed in all cases. Following respiration, the air in the lungs consists of tidal and residual air. Tidal air may be expressed comparatively easily from the lungs, but residual air can only be excluded by destruction of the lung tissue. In the process of decomposition, putrefactive gases may develop in the lungs and cause flotation when these organs are placed in water. By the use of pressure in the manner described, putrefactive gases are dispelled, and, accordingly, as a factor in flotation, can be eliminated. If after this procedure the lungs float, it is a definite indication that they contain atmospheric air and that the process of respiration has been in operation. If both lungs and all the portions of both lungs sink, the tissue is airless, although for reasons to be described, in some instances the child may have been born alive (see p. 406). Although these tests establish whether or not air has penetrated the lungs, they cannot establish the fact that the air entered the lungs as the result of breathing after complete birth of the child, since air may have entered during the process of birth. "Vagitus uterinus" is the term applied to the crying of a child while its head is still within the uterus.

Clouston[3] gives details of one of his cases. The patient was in labour with her third child and had been so for ten and a half hours. It was a brow presentation and the os was almost fully dilated. The head was firmly engaged and as he was withdrawing his hand, after the vaginal examination, the child began to cry. It was the normal crying of a newly-born child and was heard by the mother, the nurse, and himself. A woman, in a cottage situated directly below the bedroom, also heard the crying. The loud crying persisted

at frequent intervals for at least one minute. Four hours later the child was delivered by forceps. It was alive and normal. Vagitus uterinus is thought to be due to air present in the uterus and may result from suction of air along the examining hand during relaxation of the uterus. Air may also be sucked into the uterus during a uterine relaxation. It has been suggested that once there is air within the uterus it may suddenly be forced past a fold of membrane or the vagina by a contraction of the uterus and, as a result, there may be a sound which simulates a fœtal cry. This seems an unsatisfactory explanation having regard to the description of the crying heard in several cases.

Objection to the hydrostatic or flotation test has been taken on the grounds that a lung may float from causes other than respiration, as for example, from artificial inflation or putrefaction, and that a lung may sink, although respiration has been imperfectly established, as the result of disease or atelectasis.

These objections must receive some detailed consideration :—

Artificial inflation.

If, following birth, a child shows respiratory difficulty, the common practice is to resort to artificial respiration, and thus it is quite likely that the lungs will receive some air in this way. A point of practical significance, however, lies in the fact that it is most unlikely that an untrained person could accomplish this, and, therefore, the presence of air in the lungs from this cause could only be attributed to resuscitative treatment given by a skilled person whose presence at the confinement would negative any criminal intent.

Artificially inflated lungs do not exhibit the mottled or marbled coloration of naturally inflated lungs, only a portion being likely to be expanded by the process, and on cutting them, the exposed surfaces will present a dry appearance, whereas from the cut surfaces of naturally inflated lungs, blood-stained, frothy fluid will escape when pressure is applied. This is due to the fact that when respiration is established the pulmonary circulation becomes operative. Without the normal function of breathing, there will be no more blood in the lungs than that which existed during the fœtal condition, in which state the lungs still remain, except for the presence of air which has been introduced by artificial means. Apart from these points of differentiation, there is no hard-and-fast method of distinguishing between lungs which have been partially inflated by respiration and those which have been partially distended by artificial means. It is, however, recognised that inflation of fœtal lungs by artificial means is difficult to accomplish even by a skilled person.

Putrefaction.

The gaseous products of decomposition in the lungs may assist in causing them to float, but there must be substantial evidence

of putrefaction generally, together with a non-inflated state of the lungs, before flotation can be attributed solely to this cause. Fœtal lungs are resistant to the process of putrefaction and, therefore, marked evidence of putrefaction of the body is usually present in cases where these gaseous products prove an important factor in the flotation of the lungs. When the lungs and the body generally are far advanced in decomposition, it becomes impossible for the examiner to determine whether flotation, if it takes place, is due to the presence of atmospheric air, or of putrefactive gases, or of a combination of both factors. He should, therefore, frankly admit his inability to affirm that the child had breathed. In cases where the degree of putrefaction is not far advanced, pressure on the portions of lung tissue, as already described, will expel these extraneous gases without total destruction of the tissue, and their resultant behaviour in water will indicate the presence or absence of contained atmospheric air.

Sinking of lungs after respiration.

Turning to this question, it must be stated that occasionally a child may have breathed and yet the lungs may show a negative response to the test of flotation. Two interesting cases in this connection have been reported :—

1. A child, who was weakly, lived for about five hours after its birth. The post-mortem examination showed that the child was at full term, and was fairly well developed. The lungs were in a condition of complete atelectasis. On using the hydrostatic test, the lungs when tried together, separately, or in portions, sank, and presented the appearances and results of those of a still-born child. The doctor who made the dissection stated at the inquest that had he not heard the evidence of five different persons on oath that the child had lived five hours, he should have been compelled to swear that the child had been still-born and had never breathed. He offered the explanation that the child had lived the number of hours it had by whatever aeration the blood received through the mucous membrane of the trachea and larger bronchi.[4]

2. A child, which was born at seven months, lived for about seven hours and was heard to make moaning sounds. Post-mortem examination showed that the lungs were collapsed, and that no developed air cells could be seen. The lungs as a whole sank in water, and although they were cut into small pieces, every piece sank to the bottom of the vessel.[5]

It should be noted that in these cases the infants were weakly. In certain cases, breathing may be of such a shallow nature that the air in the vesicles is absorbed by the blood without appreciable expansion of the walls of the vesicles. Experimental investigation has shown that air which has entered the lungs may be entirely absorbed after respiration has ceased by the blood circulating through them.

Congenital pneumonia cannot be considered as an entity, since

the pathological findings in such cases are due to pulmonary congestion, œdema, and hæmorrhages. Kaldor [6] states that in 75 per cent. of these cases, examined by him, aspirated material was present in the lung tissue, and he assumed that aspiration was responsible for the inflammation and, together with the atelectasis, might be the cause of death. He stresses the advisability of a microscopical examination of the lungs of still-born and newly-born children. Atelectasis, variable in its distribution, may be general or affect only the bases, apices, or margins.

Pulmonary collapse, resulting from atelectasis, and pulmonary consolidation, due to acute œdema, may cause respired lungs to sink in water. If disease is present to this extent it will provide a sound reason for death quite apart from a homicidal act.

In the examination of infants, it is prudent to excise a series of small pieces of lung-tissue from different parts of both lungs for microscopical examination. By this means the condition of the alveoli in relation to contraction or expansion, together with the degree of dilatation of the vessels in their walls, can be ascertained, and the existence of any pathological lesions may be detected (see Figs. 197 and 198).

Shapiro [7] states that there is difficulty in correlating the microscopical appearances of the lungs in cases of still-birth and post-natal death with the results of the hydrostatic test in these cases. He expresses the opinion that the alveolar pattern is the result of a normal developmental process occurring in utero, possibly accentuated by intra-uterine fœtal respiratory movements, but independent of extra-uterine respiration. The gland-like appearances of the lung shown in standard text-books as an example of unrespired lung is probably seen only in relatively early embryos in whom the problem of respiration is unlikely to arise. At, or near term, the alveolar pattern in the human fœtus has developed to such a degree in utero that it cannot readily be distinguished from the pattern seen in the newly-born infant who has breathed for a short time in an extra-uterine environment. The view that the alveolar pattern is already fairly well developed at full term makes for a more intelligible appreciation of the structural appearances seen in the immediately post-natal lung as well as of the physiology of respiration with the first extra-uterine breath. The views outlined for the development of the human lung in utero are completely corroborated by experimental studies in pig embryos. Microscopical examination of the lungs may assist in determining fœtal age.

Stomach-bowel test.

When respiration has been established, there is a probability that air will be swallowed, and so may pass into the bowel. The test is based upon this assumption, and is confirmatory for the establishment of respiration in a newly-born child. Since the object of the test is to detect the presence of air in the gastro-intestinal tract, the stomach should be ligatured with a double ligature at

both the cardiac and pyloric ends prior to removal. The intestine is similarly ligatured at different points. The various parts are tested by flotation, and if a positive result is shown they are punctured under water, when the contained air may be seen to escape to the surface. The important fallacy of the test lies in the presence of gases of putrefaction. When the test is negative, it cannot be inferred that breathing has not occurred.

Miron Hajkis[8] has carried out a number of radiographic investigations with regard to this subject, and has stated that he was unable to demonstrate air in the stomach or intestine of still-born children. He made experiments on still-born infants, immediately after birth, to discover whether air could be made to enter the bowel by mouth to mouth insufflation, and found that even by gross exaggeration of the clinical method he could only inflate the stomach. He is of the opinion that air in the duodenum is strong evidence of live-birth. Even after twenty-four to forty-eight hours of insufflation, radiography did not disclose the presence of air in the duodenum. He was unable to inflate the lungs artificially in these still-born children.

Assuming that the child has survived its birth, but that death has occurred at a later date, attention must be directed to conditions which will provide some indication of the period it has lived.

Changes in the umbilicus.

The changes which occur in the stump of the umbilical cord are of value in determining this question. The first sign of separation

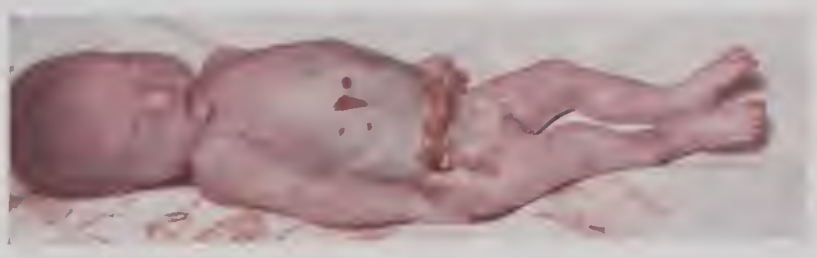


FIG. 199

Note ruptured, but untied, umbilical cord. Commencing putrefaction of abdominal wall is seen, also lividity of face and upper part of chest.

of the stump from the abdominal wall may be apparent in from thirty-six to forty-eight hours after birth, and consists of an inflammatory line at their junction. The line of separation becomes deeper daily until separation is completed. Probably in the largest number of cases the separation is accomplished on the sixth day, in the next largest on the fifth, while in graded smaller proportions of cases, on the seventh, eighth, and ninth days, respectively.

Changes in the digestive tract.

The important sign to be looked for in the stomach and intestines is the presence or absence of food, and if present, its character.

The presence or absence of meconium in the large intestine should also be noted. The presence of food, such as partly digested milk, would point to the fact that the child had survived its birth, although its absence would not point to the opposite conclusion. With regard to meconium in the bowel, its absence does not necessarily indicate that the child has survived its birth, since, for example, in breech presentations, which, in the absence of skilled assistance, are perilous to the life of the child, the meconium may be voided, partly as the result of abdominal compression, and partly from asphyxial manifestations induced by pressure on the cord.

Changes in the circulatory system.

The changes in the heart and circulatory system should receive attention, although not much practical benefit may accrue from the examination because of the comparative variations in time when the fœtal structures close. The foramen ovale, ductus arteriosus, ductus venosus, and umbilical vessels are all pervious in a newly-born child, but they become closed at variable periods following birth. The foramen ovale, for example, does not become permanently closed till the second or third month. The ductus arteriosus and ductus venosus, however, begin to shrink within three or four days. The umbilical vessels are the first to become obliterated. They begin to contract some hours after birth, and are completely impervious within a few weeks.

Was the child born alive?

The following conditions, when found, would warrant an examiner in affirming that a child had been born alive :—

When the child is sufficiently mature to be able to live an independent existence.

When the lungs more or less completely fill the thoracic cavity.

When the colour of the lungs is marbled, or mottled.

When the lungs, or portions of them, after being duly tested, float in water.

When a blood-stained, frothy fluid exudes from the cut surfaces of the lungs on the application of pressure, and when fine bubbles of air are seen to escape when pressure is made on the cut surfaces of the lungs submerged in water.

When microscopical examination shows expansion of the alveoli, and patency of the vessels.

A child has survived its birth for some appreciable time when there is evidence of :—

Changes in the umbilical cord.

Presence of food in the stomach or intestinal tract.

If the child was born alive, what caused its death?

A child may be born alive in a weakly condition and may only survive its birth for a few minutes or hours. This may result from

various causes, such as congenital debility, difficult parturition causing injuries of the brain or skull, and imperfect establishment of respiration.

The study of the causes of neonatal deaths by Cruickshank,[9] based on eight hundred autopsies, demonstrated that in almost 68 per cent. of these cases death resulted from asphyxia neonatorum. He defined the neonatal period as the first month of life. In many cases of prematurity, the cause of death is often the incomplete relief of atelectasis with secondary failure of respiration. Luff[10] considers that it is the circulatory system which is most frequently involved in the death of the fœtus, probably due to compression of the placenta during labour forcing overmuch blood into the fœtal circulation.

Moncrieff[11] classifies the causes of respiratory failure in the newborn as follows :—

Central.

Immaturity of respiratory centre.

Damage to respiratory centre, increased intracranial pressure, œdema, and hæmorrhage.

Administration of narcotics to mother.

Effects of oxygen lack or carbon dioxide excess.

Circulatory disturbances in utero, and pressure on cord affecting respiratory centre.

Peripheral.

Premature or obstructed inspiration.

Delayed expansion of lung.

Muscular feebleness.

Circulatory failure.

Cerebral hæmorrhage of the newly-born is a relatively common occurrence, and infants who die within a few days of birth quite frequently show lesions of the brain attributable to trauma sustained during delivery. The commonest site of intracranial injury with hæmorrhage is the triangular area at the junction of the falx and tentorium. Fractures of the skull are rare in normal labours, or even in difficult labours, unless the aid of forceps has been required. Mace[12] records the case of a woman of twenty-five, a multipara, who awoke in the middle of the night without pains. During the act of micturition the fœtal head appeared at the vulva and was soon born. One of the parietal bones was fractured, although there had been no interference, the cord was unruptured, and the child did not fall to the ground.

In the case of an Rh positive fœtus, and a mother whose blood lacks Rh agglutinogen, anti-Rh agglutinins are formed in her plasma, due to the passage of the Rh agglutinogen from the fœtus across the placenta, and these pass back across the placenta into

the fœtal circulation. This results in the destruction of some of the fœtal blood cells by hæmolysis, which is the chief feature of the group of conditions known as erythroblastosis fœtalis, a frequent cause of still-born or moribund children (see p. 347).

In the examination of a body of a newly-born child, it is necessary to consider not only the cause of death but the manner in which death has been produced. Where, for example, the cause of death is asphyxia, it might have arisen from accident attending the birth or from a criminal act.

A child may die at the moment of delivery from suffocation in the clothing of the mother, by bedclothes, or by discharges which accompany delivery, or from being projected from the maternal parts into a vessel containing water, such as a water-closet. The last-named event may be caused by the fact that, just before birth, the pressure of the head on the rectum and bladder frequently creates a strong desire on the part of the mother for evacuation,

Fig. 200

Note that umbilical cord encircles the neck and is attached to placenta, beside right hand.

which, if acted upon, may, by the concurrence of a strong labour pain, cause the child to be projected into the water-closet, and its death by drowning. We have examined two cases of this kind. In one of these, a young woman, in labour with her first child, delivered her child into a chamber-pot which contained a quantity of urine. We found the child, a small one, head downwards in the position described. In the other, a married woman, in labour with her third child, went to a water-closet on a tenement stair-landing, and while there was seized with violent pains, and delivered her child into the closet-pan. She shouted for help, and the child was removed and resuscitated.

In an isolated case, when a mark is found upon the child's neck, it may become necessary to decide between strangulation by natural causes and by applied violence. Coiling of the umbilical cord round the neck of the child is by no means uncommon, and it is not difficult, therefore, to conceive a case in which a woman might be suspected of the murder of her child, when in point of fact, it has been strangled by the cord during birth. We have examined at least two cases in which this was the apparent cause. The mark produced by the umbilical cord on the neck is never well shown, and the death is not so much due to the constriction exercised as

to asphyxia induced by interference with the placental circulation through the cord (see Fig. 200).

Is the child viable or non-viable?

As has already been mentioned, a fœtus under the age of seven months will not live under ordinary circumstances. According to law, a fœtus which has not attained the completion of the seventh month of intra-uterine life is held to be incapable of maintaining a separate existence, and is therefore non-viable. In the examination of the body of a fœtus, the determination of its stage of development is of great importance in cases of suspected infanticide or child murder (see p. 80). The stage of development of the child can be ascertained by a careful examination of the ossific centres of certain bones, the calcaneus, the talus, the cuboid, the femur, and the tibia. The centres, in the two last-named bones, are situated at the lower end of the former, and at the upper end of the latter. To examine the lower end of the femur, the leg should be flexed against the thigh, and an incision made across, and into the knee-joint, when the end of the femur is pushed forward through the wound. Thin slices of cartilage are then cut, in transverse plane, until the centre is exposed. The head of the tibia is next pushed forward through the wound, and examined in a similar manner. To expose the bones of the foot, an incision should be made through the interspace between the third and fourth toes and carried backwards through the sole of the foot and heel. The centres in the os calcis, astragalus, and cuboid can then readily be examined.

The following is the order of appearance of these ossific centres :—

Calcaneus . . .	At fifth to sixth month of intra-uterine development.
Talus . . .	About seventh month of intra-uterine development.
Lower end of femur .	About eighth month of intra-uterine development.
Cuboid . . .	Usually at full term.
Upper end of tibia .	At full term, or shortly after full term.

The centre of ossification in the lower end of the femur at birth usually measures from $\frac{1}{5}$ to $\frac{1}{4}$ inch in diameter.

In odd instances, even in an apparently mature child, the femoral ossific centres may not be visible. We have noted this in two cases.

It has been stated that the centre in the upper end of the tibia is present in about 80 per cent. of full-term infants.

The absence of an ossific centre in the upper end of the tibia and in the cuboid bone does not necessarily imply immaturity, but their presence indicates maturity.

While it is advisable that the height and weight of the fœtus should be taken, it is better to rely upon the presence or absence of the centres of ossification as evidence of maturity, since small children, as well as large, may be born mature.

Death due to criminal violence.

The bodies of illegitimate children are often concealed in strange places, and sometimes may so remain for long periods before discovery. We have examined bodies found in boxes, in trunks, and in other receptacles, the mothers being at the time of delivery in domestic service, and the births unknown to their employers. In many instances, the poorer classes dispose of the body of a dead newly-born infant in the most casual fashion to avoid the expense of burial. The body may be thrown on to a waste-heap, into a receptacle for refuse, or even into a river or canal. It should be appreciated that the character of the place in which the body

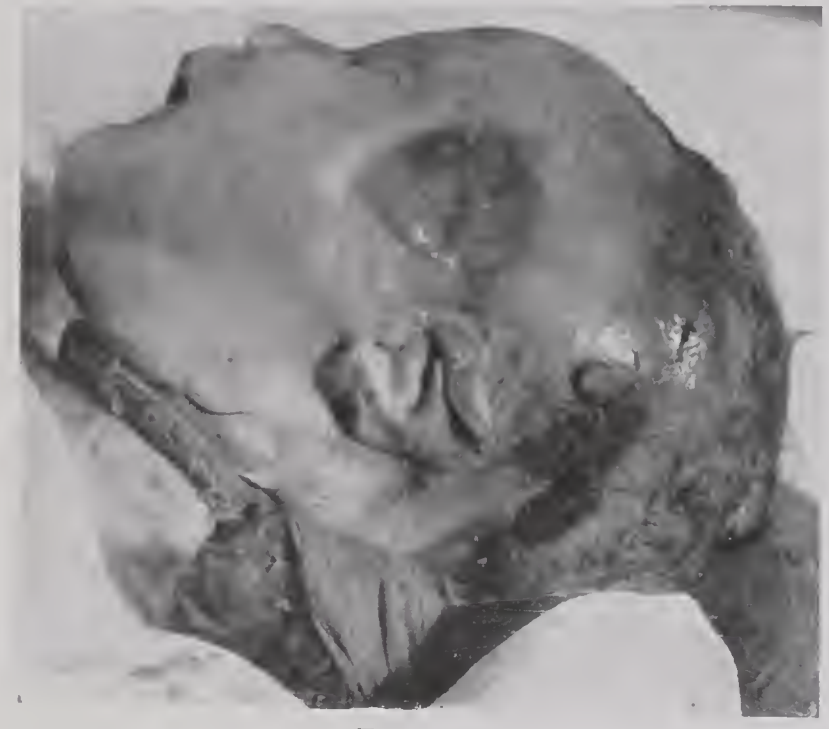


FIG. 201

Body of newly-born child with silk stocking tied tightly round neck. Note signs of putrefaction.

may be found does not provide definite indication either that the child has been born alive or that it has met with a violent death. The examiner should therefore approach the case with an entirely open mind.

Frequently the bodies of newly-born children are found bearing marks of violence. In such cases, careful deliberation is often necessary to differentiate between accident and homicide.

In some instances there may be evidence of extensive injury, the result of brutal violence. In one of our cases the infant had been thrown from a window on the third floor of a building. It is obvious, however, that any method may be employed, as in cases of murder in which adults are the victims. In cases of infanticide or child murder there is a high incidence of asphyxial deaths, which result from suffocation, strangulation, throttling, drowning, or by the

impaction of foreign bodies which have been deliberately inserted into the month or air-passages.

In some cases it is impossible to determine the cause of asphyxia, although this form of death is clearly demonstrated by the internal appearances, or whether it has been produced accidentally or homicidally. When a ligature is found tightly encircling the neck and internal signs of asphyxia are present, it is not difficult to arrive at an opinion (see Fig. 201). In such cases the nature of the ligature should be noted, the ligature preserved, and the mark upon the neck carefully dissected for evidence of extravasation. The appearance of the lungs, froth in the air-passages, and blood-

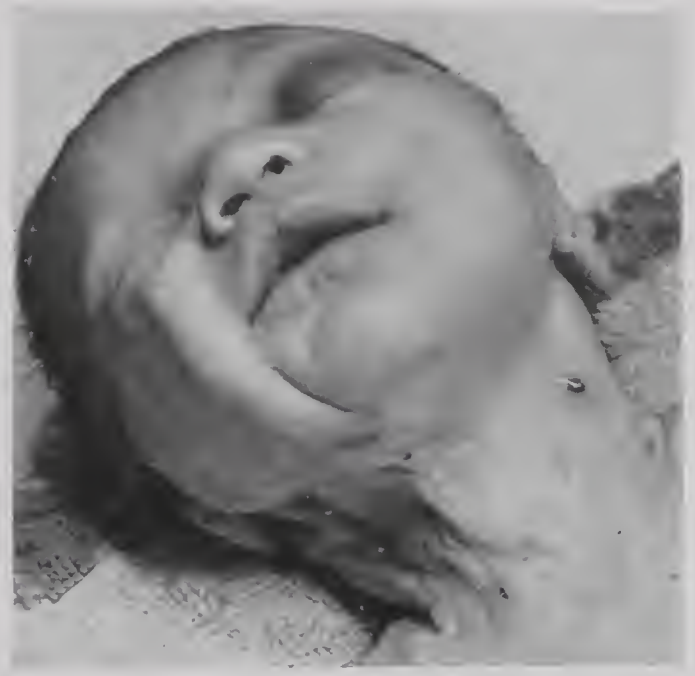


Fig. 202

A case of infanticide showing contusion of left eye, associated with fracture of skull, and abrasions on neck due to compression.

stained, frothy exudation on lung-section would point to drowning. The presence of foreign bodies in the mouth or air-passages when accompanied by internal signs of asphyxia points to homicidal suffocation.

In one case, a woman was convicted for having suffocated her newly-born child by forcing pieces of paper into its month, presumably with the object of stifling its cries. In another case, where the body of a male child, contained in a box loaded with pieces of iron, was found in a river, post-mortem examination showed that it had been strangled.

Often children are throttled, and in such cases the plea of attempting self-delivery may be advanced (see Fig. 202).

Precipitate labour is sometimes a defence in a case of infanticide or child murder, where fracture of the skull is present. The value of the relevancy of this plea may be established by the medical

evidence obtained by an examination of both mother and child. The questions to be considered in the examination of the former are :—

Whether the woman is a primipara or multipara.
Whether the pelvic parts are roomy.
Whether or not there is laceration of the perineum.

And in the examination of the latter :—

Whether or not the umbilical cord is ruptured.
Whether or not the placenta and fœtus are still united by the cord.
The character of the cranial lesions.
The presence or absence of a caput succedaneum.
The size and development of the fœtus relative to the pelvis of the mother.
The condition of ossification of the fœtal cranium.
The presence or absence of other marks of violence upon the body.

Careful consideration and review of all the circumstances will enable the examiner to test the plea offered, by the logic of the facts.

In precipitate labour, a child may be projected to the ground from the mother's body, while she is in the erect position, and thus sustain fatal injury to the skull. To test this possibility, experiments have been made. The dead bodies of twenty-five newly-born children were dropped, head downwards, from a height of 30 inches from the ground, the average height of the female genitals from the ground in the standing posture. Cranial fractures were produced in twenty-four of the bodies, and of these, twenty-two sustained fractures of either one or both temporal bones.

FIG. 203

The appearances of the ossific centre in the lower end of the femur at different stages of development.

Another cause of death may arise from the deliberate omission to ligature the umbilical cord after cutting. Such a case provides a typical instance of infanticide or child murder by omission. The appearance of severance of the cord with a sharp instrument, the absence of ligature, and the comparatively bloodless condition of the body, are indications which would point in this direction.

It rarely happens that a child's death is caused exclusively by neglect and exposure after its birth, but such cases are not unknown. In one case a young farm servant gave birth to a living male child in a cowshed and left it lying on the floor, in consequence of which the child died from pneumonia. She was found guilty.

Newly-born children may be killed by poisoning. Such cases are rare, since a woman who desires to rid herself of newly-born offspring usually adopts the quickest and readiest means of preventing it from crying and thus announcing its birth.

When marks of violence are present, the effects of violence being the cause of death, and there is evidence of complete respiration, is there justification for expressing an opinion that the child was live-born, in the legal sense, at the time the injuries were inflicted?

If the violence on the body is so distributed, or is of such a character, that full delivery of the child is indicated, there would be adequate justification for stating that the child was live-born in the legal sense. If the child had survived its birth long enough to have been suckled, the presence of milk in the stomach would place the matter beyond dispute.

It would appear that by the law of Scotland, the destruction of a child during the parturient process does not constitute the crime of homicide. With the advent of the Infant Life (Preservation) Act, the law, as applicable to England, was amended with regard to the destruction of children before they have attained an independent existence (see p. 398).

How long has the child been dead?

The answer depends upon the post-mortem changes present and the factors attendant on their production (see p. 127).

PROCEDURE AT AUTOPSY OF NEWLY-BORN CHILD

External examination.

The examination of the body of a newly-born child is made in the same manner as the examination of the body of an adult, but the following points, after the wrappings of the body have been noted for identification purposes, demand special attention:—

The general development of the body with respect to maturity or immaturity.

The appearance of the skin in relation to the absence or presence of vernix caseosa, indicative of whether the body has been washed or not.

The state of the natural orifices of the body, and in respect of the mouth and upper passages, the presence or absence of foreign bodies.

The presence or absence of marks of violence.

The condition of the cord: if the cord has been severed, the character of severance: length of the cord: if ligatured or not, and if tied, the character of the ligature.

Whether or not the placenta is still attached to the body by the cord.

The position and character of the caput succedaneum.

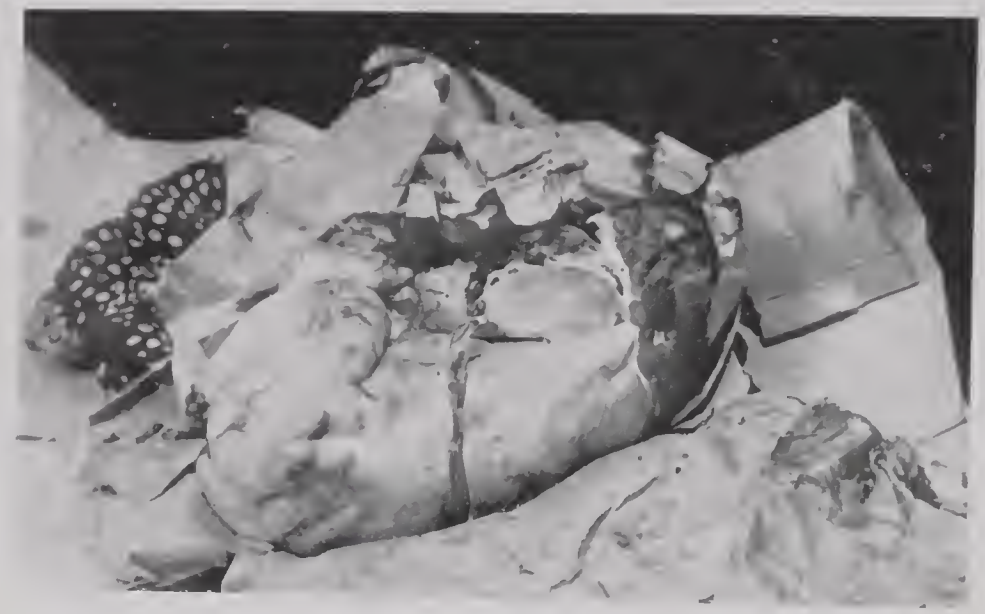


FIG. 204

Body of newly-born child, the victim of infanticide, shown in wrappings as found. Early formation of adipocere affected the tissues of the neck. The umbilical cord can be seen encircling the trunk.

Internal examination.

Head.

Examine as in adult cases, except that the brain may be exposed by cutting through the membranous connection of the skull bones. Should the brain substance be unduly soft, the skull may be divided with strong scissors and the brain cut across on a level with the bone incision, when it can be removed with the calvarium.

Neck.

Examine carefully for marks of constriction.

Attention should be directed to the possibility of there being foreign bodies in the air passages.

Chest cavity.

Examine organs in natural position. Note the volume of the lungs and the space occupied by them: the position of the highest

convex part of the diaphragm ; the colour of the lungs, and their consistence by palpation ; the presence or absence of petechiæ or Tardieu's spots. Remove lungs and apply the hydrostatic test.

Abdominal cavity.

Avoid the umbilical site when making the primary incision through the skin and underlying tissues composing the abdominal wall. Note condition and appearance of organs in natural position ; the condition of the umbilical vessels : the presence or absence of air and food in the stomach and intestines ; the presence or absence of meconium in the large intestine, and if present, the amount and distribution.

Examination of spine.

Any injury or congenital defect, such as spina bifida, should be noted.

Examination of ossific centres in bones of foot, in lower end of femur, and in head of tibia.

The following tabulated criteria will assist in the assessment of the development of a newly-born child.

DIFFERENTIATION OF NEWLY-BORN INFANTS AS TO AGE AT BIRTH

	Seven Months.	Eight Months.	Nine Months (Calendar).
Length . .	13 to 15 in.	14 to 17 in.	18 to 24 in.
Weight . .	3 to 4 lbs.	4 to 5 lbs.	$6\frac{1}{2}$ to 7 lbs. and upwards.*
Ossific centres	Calcaneus. Talus.	Calcaneus. Talus. Centre in lower end of femur appears.	Calcaneus. Talus. Lower end of femur—size : $\frac{1}{5}$ to $\frac{1}{4}$ in. in diam. Cuboid—usually at full term. Head of tibia—frequently at full term.

* Infants at birth have occasionally been found to weigh as much as 12 lbs. and 15 lbs., and on the other hand, but still more rarely, to be much less than the average weight at birth and still survive. Hubbard [13] records a case in which the infant weighed only 1 lb. at birth, and another observer of the same infant states that at the age of six weeks it weighed $32\frac{3}{4}$ ozs., having been partly breast fed and partly bottle fed. Shackleton [14] also records a case of an infant who weighed 17 ozs. at birth, but when over a year old weighed $22\frac{1}{2}$ lbs.

ILLUSTRATIVE CASES

Application of violence.

The body was that of a mature child. Ossific centres were present in bones of foot, in lower end of femur, and in head of tibia. The

placenta was attached to the cord which had not been severed from body. A moderate-sized caput was present over right parietal bone. The undernoted injuries were present :—

Extensive bruising over anterior half of scalp, entire left side of scalp, left side of forehead, and left eyelid. These areas contained a considerable quantity of extravasated blood.

Two linear abrasions and three small punctured incised wounds were present on front of neck (see Fig. 202).

Skull and brain.

There were extensive extradural and subdural hæmorrhages, and the vault of skull on left side of frontal bone showed a stellate fracture, a limb of which involved roof of left orbit where it terminated in a curved fracture. There was also a stellate fracture of left parietal bone which divided the bone into four portions with resultant depression. The right parietal bone was also the seat of a stellate fracture which divided the bone into three pieces and caused depression. There was a small laceration of right occipital lobe of cerebrum.

Neck.

Dissection of the tissues showed four small areas of bruising over sides and on the floor of the mouth.

Lungs.

These showed complete expansion and several punctate hæmorrhages, or Tardieu spots, were present over the pleuræ. On section, copious, frothy, blood-stained exudate was expressed.

Heart.

Some Tardieu spots were present on the surface and the right ventricle was slightly dilated.

Stomach.

This organ was almost completely filled with liquid and clotted blood, together with some mucus. The blood had resulted from an injury present on right side of pharynx.

Opinion.

The body was that of a mature child who had respired freely. Death had resulted from the effects of asphyxia due to pressure applied to the neck, and multiple fractures of the skull with resultant cerebral hæmorrhage. From the character of the cerebral hæmorrhage, the head injuries had been produced during life, and from the microscopical examination of tissue removed from the various sites of injury, these had also been produced ante-mortem. The body of the child was found in a suitcase together with blood-stained clothing and bed linen.

Attempt to dispose of body by burning.

The child weighed $5\frac{3}{4}$ pounds, and its length was $20\frac{1}{2}$ inches. The umbilical cord, attached to the body, measured 10 inches and appeared to have been evenly severed, but was not ligatured. The finger and toe nails were well developed. Putrefactive changes were present, and the right side and back of head, face, neck, right arm and hand, back and sides of body, and entire legs and feet showed effects of burning. The scanty hair upon the scalp was singed and soot deposit was present in most of the areas affected by burning. On the right side of the skull was a gap, due to injury of the parietal and occipital bones, through which liquid brain tissue had escaped. The edges of bone, over posterior part of gap, showed charring and powdering. In the left lumbar region, the tissues were split for a distance of 5 inches and bowel protruded. The tissues over both groins and over right side of abdomen were similarly affected. In the last-named situation, the aperture communicated with the interior of abdominal cavity. The burns on body varied from third to sixth degree. Vesicles were absent. Microscopical examination of tissue from the areas of burning showed that these lesions had been produced after death, and similar examination of tissue from the edges of the apertures gave the same indication. The lungs did not show any evidence of establishment of respiration. The circumstances made it clear that an attempt had been made to dispose of the dead body of this illegitimate child by placing it in an open fireplace.

An unique case.

The case of R. v. Lloyd and Hampson, Liverpool Assizes, June 1944, is exceptional. The accused were charged with the attempted suffocation of a newly-born male child. The evidence was that a full-term child, weighing $8\frac{1}{4}$ pounds, had been buried in a garden, in the bank of an air raid shelter, and that three hours later it was dug up alive. The after-birth was first unearthed, and when further digging was in progress a short sigh was heard. The remainder of the earth was then removed manually and the child was found. It lay on its right side, was naked, and the body was covered with wet soil. Seven inches of umbilical cord, which had been ruptured, but had not been tied, remained attached to the abdominal wall. The child showed signs of life and following clearance of the air passages, a warm bath, and treatment for shock, recovery gradually ensued. There were a few superficial scratches and a small bruise on the body. The infant had been buried 18 inches to 2 feet below the soil which was wet due to falling rain.

References

1. Brit. Med. Jour., Vol. II, 652, 726, 1940.
2. Jour. Amer. Med. Ass., Vol. CX, 283, 1938, Hoffmann, Greenhill and Lundeen.

3. Brit. Med. Jour., Vol. I, 200, 1933, Clouston.
4. Ibid., Vol. II, 1567, 1900, Dilworth.
5. Ibid., Vol. I, 146, 1901, Randolph.
6. Amer. Jour. Obstet. and Gyn., Vol. XXV, 113, 1933, Kaldor.
7. Clinical Proceedings, Vol. VI, No. 4, 149, 1947, Shapiro.
8. Lancet, Vol. II, 134, 1934, Miron Hajkis.
9. Jour. Amer. Med. Ass., Vol. XCVI, 495, 1931, Cruickshank.
10. Amer. Jour. Obstet. and Gyn., Vol. XXXII, 286, 1936, Luff.
11. Lancet, Vol. I, 531, 1935, Moncrieff.
12. L'Obstét., Vol. VI, 54, 1901, Mace.
13. Brit. Med. Jour., Vol. II, 878, 1928, Hubbard.
14. Ibid., Vol. II, 1076, 1928, Shackleton.

CHAPTER XIV

RAPE AND CARNAL KNOWLEDGE AND OTHER SEXUAL CRIMES

RAPE, in Scotland, is the carnal knowledge of a woman forcibly and against her will, or, whether by force or not, of a female under twelve years, or of an idiot. In England, under the Offences Against the Person Act, 1861, carnal knowledge of a woman forcibly and against her will is rape. By the Criminal Law Amendment Act, carnal knowledge, whether by force or not, of a female under thirteen, or of an idiot, is a felony. Although in Scotland an idiot is deemed incapable of giving consent, in England the state of idiocy must be such as to render the woman incapable of expressing assent or dissent. Accordingly, in England there must be some evidence that the act was without consent. Consent produced by mere animal instinct would, however, be sufficient to prevent the act from constituting rape, but it would still be a misdemeanour. Under the Criminal Law Amendment Act, 1885, applicable to both England and Scotland, carnal knowledge of a woman obtained by personating her husband is deemed to be rape.

To constitute the crime, in the case of females above twelve years in Scotland, and thirteen years in England, three things are necessary, namely, the use of force to overcome the woman's will to resist; resistance to the utmost by the woman; and penetration, but not necessarily emission.

The necessary force is the overcoming of such resistance, and it is immaterial what means are used to this end. Actual violence is unnecessary so long as the woman's will to resist is overpowered, for example, drugging her into a state of insensibility, coercing her into submission by fear or intimidation, or, as already mentioned, by personating her husband.

In the case of an adult woman in possession of all her faculties, it is essential for the commission of the crime that the woman has resisted to her utmost. It would not amount to rape if the female, after half-hearted resistance, gave consent. The resistance must be maintained to the last, and until the female is overcome by unconsciousness, complete exhaustion, brute force, or fear of death. The crime may be committed against any female, whatever her character, except when the relationship of husband and wife obtains. The use of force on the part of the husband does not constitute the crime of rape. A husband may, however, be guilty as an accessory to the rape of his wife by aiding another person in its perpetration. Where the assaulted woman is physically incapable of resistance,

force does not require to be proved, and to a lesser degree this is the case when the victim suffers from weakness of mind, not amounting to idiocy. Further it is unnecessary to prove force in the case of females under thirteen years in England, or of females under twelve years in Scotland, or of idiots. Such are deemed incapable of consent, and the mere fact of penetration is enough.

By the law of England, administering to a female any stupefying or overpowering drug, matter, or thing, is a felony if done with intent to commit any indictable offence. When a woman is found asleep, or insensible from intoxication or other cause, she is incapable of such consent as excludes criminality.

By the law of Scotland, taking sexual advantage of a woman while she is asleep is an indictable offence termed Clandestine Injury to Women. Further, sexual connection with a woman found in a state of insensibility from intoxication does not constitute the crime of rape.

The case of H.M. Advocate v. Grainger[1] is one of interest on the latter point. Two men were indicted on a charge that they did ravish a woman while she was in a state of insensibility or unconsciousness from the effects of intoxicating liquor. Their counsel maintained that the indictment was irrelevant in respect that it did not disclose the crime of rape according to the law of Scotland. The presiding judge sustained this objection.

In stating the objection counsel argued that, as rape (save in the exceptional cases of pupils and idiots) is the carnal knowledge of a woman forcibly and against her will, the crime could not be committed unless the woman is in a condition, physically and mentally, to exercise her will-power and offer resistance, and as the libel set forth that the woman was in a condition when she was incapacitated by reason of intoxication from offering any resistance or from exercising her will-power in the way of giving or refusing consent, the offence charged did not amount to rape.

In giving his decision the judge stated that it was not alleged that the accused supplied the liquor with which the woman became intoxicated. Had this allegation been made, the charge of rape might have been sustained, as it has been decided that it is rape to have connection with a woman whose resistance has been overcome by drugging her. It might be suggested, he continued, that the present case fell to be assimilated to that of a female idiot; but that did not seem to be a true analogy. The idiot had, in law and in fact, no will; in the present case the woman assaulted had a will, the activity of which had been but temporarily suspended by her intoxication. The true analogy seemed to him to be the case of the woman who was taken advantage of while asleep, and such an offence was not rape. Just as a sleeping woman was temporarily in a state of unconsciousness wherein she was incapable of exercising her will-power, so here it seemed that the woman was in the same temporary condition of unconsciousness by reason of intoxication. What was said to have been done by the accused, although not rape,

was a criminal offence, the crime of inflicting clandestine injury on a woman, and must be indicted as such, and not as rape.

In the case of H.M. Advocate v. Logan,[2] the Lord Justice-Clerk in charging the jury directed that if the drink were administered by the accused with the object of overcoming the resistance of the victim, the crime might be rape ; but that if the drink had been taken by the victim of her own free will and not been given with a view to overcoming her resistance, the crime, so far as it was possible to define it, came within the category of indecent assault.

In Scotland, there is no age fixed by law before which a male person is held incapable of committing the crime of rape. Probably the earliest age at which conviction has followed was that of a boy aged thirteen years and ten months. By the law of England, a boy under fourteen is presumed incapable of committing this crime. This is a presumption which is not rebuttable by evidence of precocious development (see p. 358). It would seem that a male under fourteen can be convicted of attempt to commit rape or of an indecent assault.

Penetration means the actual penetration of the private parts of the female by the male organ, although there is no emission of semen. As to the degree of penetration necessary to constitute the offence, it has been ruled that the slightest penetration of the penis within the vulva is sufficient, even to such a slight degree that the usual signs of virginity are not interfered with.

Anyone having carnal knowledge, or attempting to have carnal knowledge of any woman or girl who is a mental defective, and who is under institutional care or guardianship, under circumstances which do not amount to rape, but knowing at the time of the commission of the offence that the woman or girl was under such care or guardianship, commits a crime and offence under Section 46 of the Mental Deficiency and Lunacy (Scotland) Act, 1913 (see p. 471).

Unlawful carnal knowledge and indecent practices affecting girls.

The Criminal Law Amendment Acts, 1855-1928, deal with unlawful carnal knowledge of girls above the age of twelve and under the age of sixteen, and also cover indecent practices with girls within the same range of ages, together with acts of gross indecency between male persons. The crime of unlawful carnal knowledge is not the crime of rape, since the act is committed with the consent of a girl who, being under sixteen, cannot give valid consent, whereas carnal knowledge of a female of any age without her consent and against her will is rape.

As a girl under sixteen cannot give valid consent, any male person who has intercourse, or attempts to have intercourse, with a girl above the age of twelve and under the age of sixteen, commits a criminal offence, namely, having unlawful carnal knowledge, or attempting to have unlawful carnal knowledge. Consent, after the age of sixteen, eliminates the legal offence, but below the age of

consent, even solicitation on the part of the girl would not avoid the criminality of the sexual act committed by the male.

The following is a brief résumé of the provisions of the Act of 1885 :—

Section 4 enacts that any person who unlawfully and carnally knows any girl under the age of thirteen years shall be guilty of felony, and any person who attempts the foregoing shall be guilty of a misdemeanour.

Section 5, that any person who unlawfully and carnally knows or attempts to have unlawful carnal knowledge of any girl being of or above thirteen and under sixteen years of age, or of any female idiot or imbecile woman or girl, shall be guilty of a misdemeanour, provided that it shall be a sufficient defence to the first part of the section if it shall be made to appear to the court or jury that the prisoner had reasonable cause to believe that the girl was of or above the age of sixteen years.

By the Age of Marriage Act, 1929, which makes marriages under sixteen void, the following further proviso was added to the last-mentioned section, namely, that in any proceedings against a person charged under Section 5 of the Act of 1885, or charged with indecent assault, it shall be a sufficient defence to prove that, at the time when the offence is alleged to have been committed, he had reasonable cause to believe that the girl in respect of whom it is alleged to have been committed was his wife.

The Criminal Law Amendment Act, 1922, amended the Act of 1885 with respect to offences against persons under the age of sixteen.

Section 1 provides that it shall be no defence to a charge or indictment for an indecent assault on a child or young person under the age of sixteen to prove that he or she consented to the act of indecency.

Section 2, that reasonable cause to believe that a girl was of or above the age of sixteen shall not be a defence to a charge under Section 5 of the principal Act, except in the case of a man of twenty-three years of age or under where he had reasonable cause to believe that the girl was over the age of sixteen. This defence shall be valid only on the first occasion a person is charged with an offence under this section.

Section 4, that in the application of the Act to Scotland, the following provision shall be substituted for Section 1 of the Act :—

> Any person who uses towards a girl of or above the age of twelve years and under the age of sixteen years any lewd, indecent, or libidinous practice or behaviour which, if used towards a girl under the age of twelve years would have constituted an offence at common law, shall, whether the girl consented to such practice or behaviour or not, be guilty of an offence against this Act.

A further Act to amend the foregoing law with respect to offences against persons under sixteen years of age was passed in 1928, but has no medico-legal importance.

Arising out of the Act of 1922, the comments made at the hearing of the appeals in the cases of R. v. Keech [3] and R. v. Chapman [4] are not without interest. In the first case, leave to appeal was granted by the judge, who desired the direction of the Court of Criminal Appeal as to whether a judge ought never to pass more than a nominal sentence in cases where there is a conviction for indecent assault, but where the defence of belief in the age of the girl is an answer to the charge of carnal knowledge. When the appeal was heard, Mr Justice Avory pointed out that this would be encouraging the "permanence" of what he designated as "this grotesque statute," namely, the Criminal Law Amendment Act, 1922. On the same point the Lord Chief Justice remarked :—"This statute in this part of it is sufficiently bad, but it would be indescribably worse if it were to lead to the view that, since it had been passed, nobody can properly be punished for an indecent assault if he is under the age of twenty-three." The appeal was dismissed and the sentence confirmed.

In the second case, the question under consideration before a full bench of five judges was whether a person who has passed his twenty-third birthday, but has not attained the age of twenty-four when he commits the offence, is entitled to avail himself of the provision in Section 2 of the Act. It was generally admitted that the wording in that section was loose, but it was argued, on behalf of the Crown, that the words used were "words of art" and must be construed in their technical sense. The Court applied the rule that "where an equivocal word or ambiguous sentence leaves a reasonable doubt of its meaning which the canons of interpretation fail to solve, the benefit of the doubt shall be given to the subject and against the legislature which has failed to explain itself." The appeal was allowed, with the result that the defence in Section 2 is valid for the use of a prisoner up to midnight of the night before his twenty-fourth birthday.

As we have already seen, the law, under certain circumstances, permits a prisoner charged with having carnal knowledge of a girl between the ages of thirteen and sixteen, to show that he had reasonable cause to believe that she was over the age of sixteen years. A defence cannot be founded upon impressions formed from the appearance of the girl, for, if such a defence could be entertained, it would nullify the statute altogether, because the accused would only have to say that while the girl's age might be fourteen, he believed, and had grounds for believing from her appearance, that she was over sixteen. It must not be mere supposition on the part of the accused. It must be that he formed the opinion upon information, or other intelligible and reasonable grounds of belief. Credible information obtained perhaps from the girl herself, or from other sources, by a man twenty-three years of age or younger, is alone a valid defence. It is a matter of constant observation in daily life, and it is well known by medical men, that the evidences of physical maturity in girls between the ages of thirteen and sixteen are as variable as is their stature or their weight, and that there

are many girls of such ages who might well be mistaken for a more advanced age than that of sixteen.

Signs of physical resistance in cases of rape.

From what has been said respecting the legal interpretation of the kind of force and amount of penetration necessary to constitute rape, it will be obvious that from the medico-legal point of view the physical signs will vary in different cases, or may even be absent, although the crime has been committed.

It is easy to conceive the possibility of legal commission of the crime without any physical evidence whatever being found on the body of the female to justify a medical examiner in doing more than reporting the negative facts. Since the slightest degree of penetration, without emission, constitutes the crime, no physical signs of defloration may be produced; indeed, there may be no local evidence whatever of the juxtaposition of the male and female parts. Therefore, the medical examiner is not justified in affirming, because no such physical evidence is forthcoming, that rape has not been committed. If evidence exists, the range of physical signs will vary according to the capacity for physical resistance. In the case of young children, females who have been drugged, or those who are in a state of unconsciousness from any other cause, the evidential results of resistance will probably be absent, while the local signs of accomplishment of the act are likely to be well marked, but even then the signs will depend upon whether or not the female has been accustomed to sexual intercourse. In cases of rape, we must therefore distinguish between the general signs of physical resistance and the local evidence. Assuming that a healthy, vigorous young woman alleges that she has been ravished, the first thing the medical examiner must look for is evidence of the signs. If the woman has resisted to her uttermost, she will probably bear marks upon her body, face, neck, and limbs of her violent resistance, and probably, also, she will have inflicted injuries upon the body of her assailant. In such a case, therefore, the examiner should look for evidence of the signs of violence upon her body, such as wounds, bruises, or scratches. In the absence of these, he should exercise at first a healthy scepticism as to the truth of her statements, unless she avers that she had fainted, been overcome by fear, or had been drugged, and her sexual parts bear evidence of interference. At the same time, our experience has led us to the belief that this scepticism may be carried too far. There are, unquestionably, girls who become panic-stricken when an attack of this kind is made upon them, and are rendered incapable of offering serious resistance, with the consequence that their bodies do not bear evidence of injuries such as might be expected from a severe struggle, while locally there may be all the expected signs of the accomplished act of penetration. In such a case, the acts and demeanour of the girl immediately after the alleged commission of the crime should be subjected to very critical investigation, as these may provide valuable evidence, corroborative or otherwise, regarding the alleged ravishing. In the

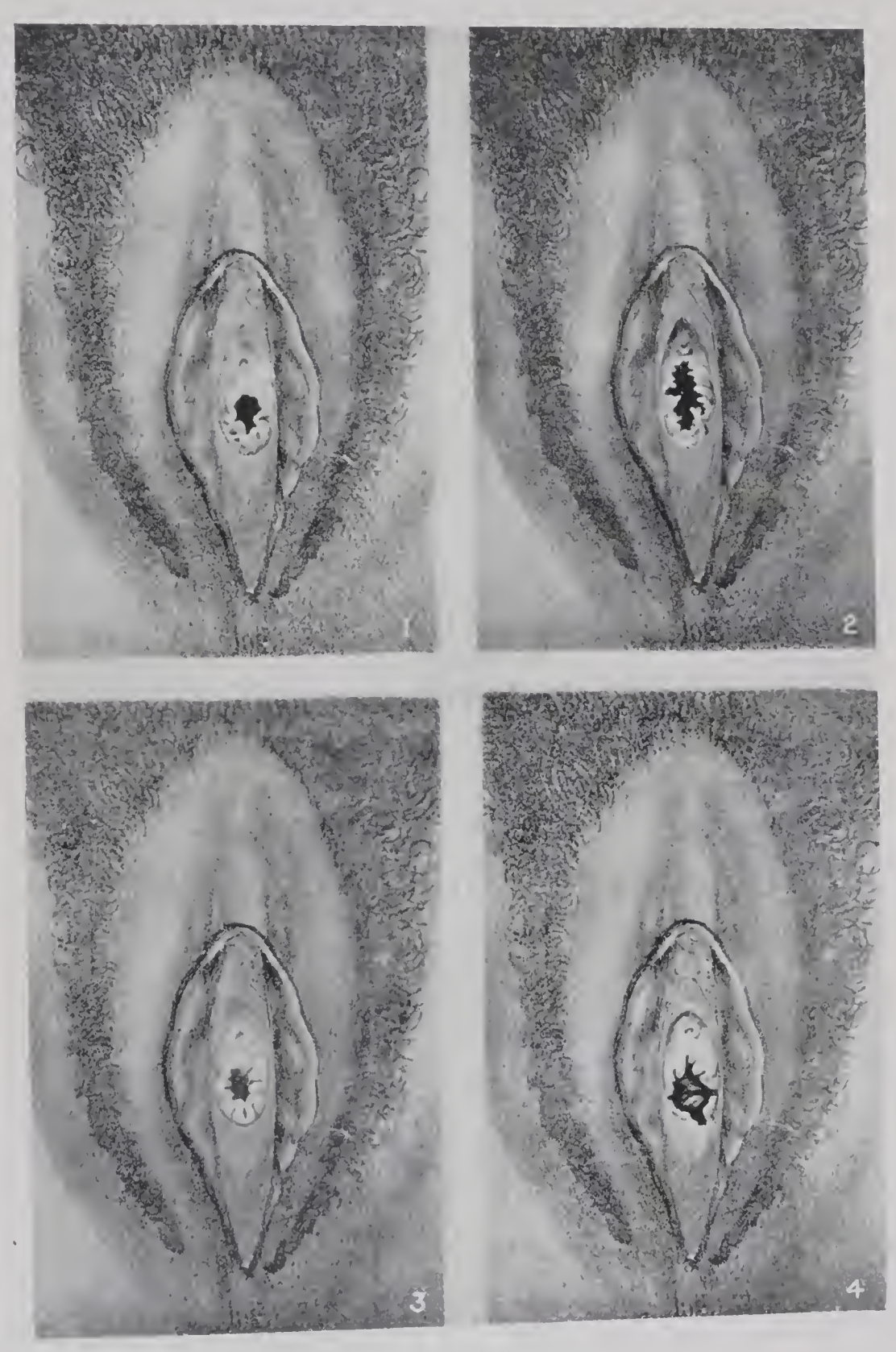


FIG. 205

Types of Hymen. (Photographs of prepared models.)

1. Intact—Annular, with natural notches.
2. Intact—Fimbriated.
3. Ruptured, following coitus.
4. Ruptured extensively, following delivery.

case of children, the examiner must not expect evidence of physical resistance, since children, as a rule, are incapable of exercising sufficient resistance to cause the production of injury.

Before considering the appearances of the sexual parts associated with recent loss of virginity, it is advisable to discuss the physical signs of virginity.

Signs of virginity.

The following are the principal signs :—

An intact hymen.
A normal condition of the fourchette and posterior commissure.
A narrow vagina with rugose walls.

These signs, taken together, may be regarded as evidence of virginity, but taken singly they cannot be so reckoned.

The hymen, which varies greatly in shape and consistence, is found as a normal structure in most virgin females, except in certain cases in which from varied causes it has been more or less destroyed by factors other than sexual intercourse, principally masturbational acts, the insertion of foreign bodies, disease, self-scratching due to irritation of the parts from lack of cleanliness, gynæcological examination, operation, or accident. In cases of habitual masturbation, which is quite a prevalent habit in females, some hypertrophy of the labia and clitoris may be present. Mason [5] reports the case of a girl, aged seventeen, who was a confirmed clitoris masturbator from the age of eight, in whom the erected clitoris measured 5 centimetres in length and $1\frac{3}{4}$ centimetres at the base, and showed a similar measurement of the glans (see Fig. 218). The form of the intact hymen is variable in character and may show merely as a slight annular ring fringing the opening of the vagina, it may be tight and rigid or, very rarely, may exist in such pronounced form as to constitute an imperforate curtain which entirely shuts off the vagina from the external genitals (see Fig. 188). Between these two extremes there are varying forms. The following types of intact hymen may be met with—

Fig. 206
Intact annular hymen with natural notches.

The infantile hymen with a small, slit-like opening near the centre ;
The annular hymen ;

The annular hymen with natural notches which may give a lobulated appearance ;
The fimbriated, or irregularly-notched hymen ;
The hymen showing a septum and unequal openings, or with two openings, one serrated, and the other lobed ;
The hymen showing a posterior rudimentary septum ;
The hymen showing an irregular, puckered opening and undistended ;
The crescentic hymen.

From a want of knowledge of these forms, mistakes may be made by examiners, since natural notches and fimbriations might be mistaken for rupture in sexual cases (see p. 428). While recent rupture of the hymen would signify the forcible introduction of an instrument of some kind, and while loss of the hymen does not necessarily indicate loss of virginity, its persistence does not unequivocally point to the existence of virginity. In odd cases, the hymen has remained unruptured after coition and during resultant pregnancy, and has remained intact until ruptured by the birth of the child, or until incised to permit the passage of the child. In these rare cases, the hymen has been of annular and distensile type which has permitted the entry of the male organ without rupture. It should has permitted the entry of the male organ without rupture. It should not be forgotten that pregnancy can occur even when penetration is impossible, since only the entry of spermatozoa is essential to achieve this.

In the virgin female the vagina is narrow and its walls are slightly rugose. The absence of these characteristics does not, however, necessarily indicate habituation to sexual intercourse, since masturbation, or repeated mechanical dilatation, may affect the virgin condition.

From a review of the foregoing signs, it will be apparent that they must be found conjointly to afford satisfactory evidence of the virgin state.

Local evidence of rape.

We turn now to consider the signs in the sexual parts of a virgin female which when found would support the examiner in concluding that rape had been committed.

These are :—

Recent rupture of the hymen.
Presence of blood, fresh or dried, about the vulva.
Marks of bruising, abrasions, or inflammation of the parts.
Presence of semen in the vagina.

Additional signs such as discomfort in walking or frequency of micturition may be present in certain cases, especially in young girls.

When there has been forcible attempted penetration or complete penetration of the vagina, evidence of rupture of the hymen will be present, as a rule, but both the character and extent of the injury will vary in different cases depending upon the nature of the hymen,

the disproportion between the male and female parts, the extent of the penetration, and the amount of force used. The site of rupture presents a tear, or a series of stellate tears, in the membrane marked in recent cases by a blood-stained or inflamed line or lines.

Rupture of the hymen is almost invariably accompanied by some degree of bleeding. The severed edges do not unite, but become rounded off in the process of healing which, in the case of slight tears, occurs in from two to three days ; more extensive tears take a longer period to heal, usually about seven to ten days or even slightly longer, depending upon circumstances. It is not possible to date an injury of the hymen after it has completely healed. In women who are habituated to intercourse, and in those who have borne children, the remains of the hymen constitute what are known as

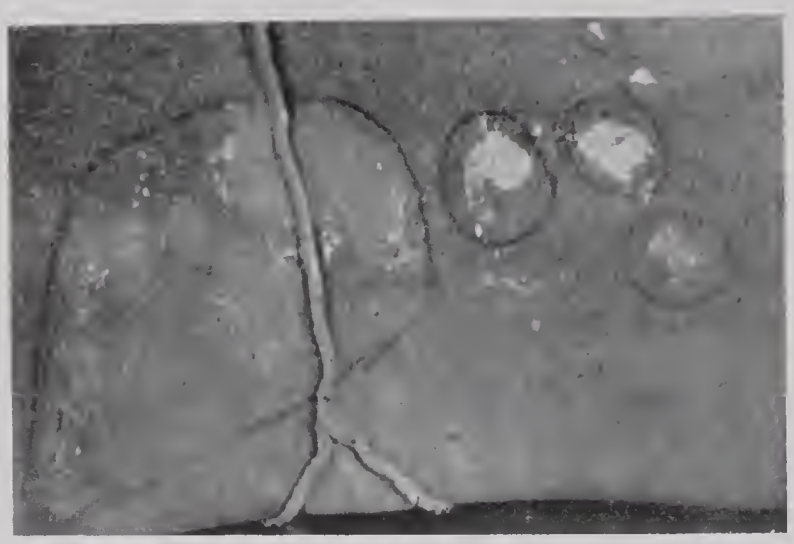


Fig. 207

Seminal stains, close to crutch of undergarment, photographed in filtered ultra-violet light.

carunculæ myrtiformes which are situated around and close to the vaginal orifice and present the appearance of different-sized but small, round, fleshy projections or tags.

Attention should be paid to the presence of blood about the vulva, thighs, and pubic area of the body, and on the clothing. Whether blood is present or not, and, if present, the amount, will depend upon the extent of the injury and the vascularity of the hymen. It sometimes happens that, from the unusual quantity of effused blood, the examiner may be led to suspect that the assailant has also received injury to his genitals. In three cases which we examined, the quantity of blood found on the girls' underclothing, and at the place where the crime was committed, was greater than would reasonably have been expected from an injury to the hymen. On examination of the suspected males who had been apprehended, a recent rupture of the frænum of the penis was found in each case. Apart from such injury to the male, coitus may cause considerable bleeding where, in the hymen, a small vessel has been incompletely

torn. We have not seen this in rape, but we have seen it in the case of the first coitus of marriage. The examiner should be on the alert, however, that he is not deceived by a false charge of rape, and that the presence of blood is not merely menstrual. There will be corroborative evidence of rape should bruising or abrasions of the genitals be found, since either of these conditions is indicative of violence.

Accompanying the foregoing signs, evidence of their cause is afforded when semen is found in the vagina. In all cases of alleged rape, smears from a vaginal swab should be examined microscopically for spermatozoa. The swab should be moistened with normal

Fig. 208

Filtered ultra-violet lamp of Hanovia pattern.

saline, or 0·3 per cent. solution of glacial acetic acid in distilled water. Several microscope slides are then lightly and evenly smeared with it. The smears should be thin. To some of the slides a drop or two of the diluting fluid mentioned are added. Cover-slips are applied and the preparations examined microscopically. The remaining slides should be dried and a staining process employed (see p. 438). The pubic hair and the hair around the external genitals, together with any suspicious staining on the skin or clothing, should also be examined for evidence of seminal fluid. The absence of semen, however, must not be interpreted as an indication that rape has not been committed since an emission is not a requisite of the crime.

The use of filtered ultra-violet light will prove helpful in the detection of seminal staining, whether upon hair, the body surface,

or clothing.[6] The ultra-violet lamps of Hanovia pattern have proved most useful in this respect (see Fig. 208).

In some cases, the victim of rape or unlawful carnal knowledge may become infected with gonorrhœa or syphilis. Owing to the existence of a belief among persons of the lower classes that coitus with a young virgin will cure them of gonorrhœa, children are sometimes infected. In one of our cases two young children were affected with gonorrhœa, the result of sexual contacts with a male lodger who was suffering from the disease. Many years ago, in Glasgow, a woman was tried and convicted on a charge of lewd, indecent, and libidinous practices and behaviour towards a boy, aged seven years, from which he contracted gonorrhœa.

A far more common cause of purulent discharge, however, is vulvo-vaginitis. The differentiation between a simple vulvo-vaginal inflammation and gonorrhœal infection should be determined by bacteriological examination (see p. 443).

Our case records show a wide variance in the extent and character of local injury in sexual cases. In one instance, a child of seven years, violence had been extreme, and the perineum was so torn that the injury communicated with the rectum. In the case of a girl aged sixteen, of muscular build, who alleged that forcible and complete intercourse had been performed upon her by five men on ten occasions within four hours, the hymen had not been recently torn, and there was only a small amount of dried blood on the labia. There was no evidence of local injury or of inflammation. In a series of thirty-six cases involving young females, there was evidence of a ruptured hymen, but only in sixteen of these was the rupture recent. The ages of the girls varied from thirteen to seventeen years. In eleven of these instances there was definite evidence of sexual trafficking. The findings confirm the view that sexual offences with girls under sixteen are more common than the number reported to the authorities would indicate.

ILLUSTRATIVE CASES

A post-mortem examination of the body of a girl, aged seven, made by us, showed that the cause of death was asphyxia due to compression of the air-passages. There were some bruising and abrasions of both legs.

Examination of the genital region showed that the private parts, fork of the legs, region of anus and lower parts of buttocks, were more or less covered with a thin layer of blood mixed with fæces. Detailed examination showed that the tissues between the vagina and rectum had been completely torn for a distance of fully an inch. In our opinion the injuries described in these parts had been produced either by the fingers of an assailant or by the forcible intromission of a male organ.

A man was indicted and tried at the High Court at Glasgow on a charge of rape and murder. The post-mortem examination of the woman's body showed the following features :—(1) vertical lacerated

wound 1½ inches long from bridge of nose upwards in the middle line, extending down to bone; (2) similar wound on eyebrow, ½ inch long, and close to right of (1); (3) similar wound about 1 inch to left of (1), ½ inch long; (4) oblique lacerated wound, ½ inch above and outward from (3), 1 inch long and reaching bone; (5) severe bruising of right upper eyelid and neighbouring parts, and a lesser degree of bruising of left upper eyelid; (6) incised wound on front of nose extending into right nostril and separating right side of nose from underlying bone which was exposed; (7) perforating lacerated wound of lower lip, V-shaped, opening into mouth; (8) bruising of upper lip and extensive bruising of both sides of neck with abrasion of skin; and (9) severe and extensive bruising of upper and inner sides of both thighs, and a recent rupture of hymen with extensive blood-staining around vaginal orifice.

Examination of the scene of the crime showed a patch of blood-stained turf with a sharp stone projecting slightly from it.

Blood-stains were found upon nearly all the garments of the accused, including the trousers, and on these, seminal stains were found on the inside of the front opening. Blood-stains were also found on all the garments of the deceased. The piece of turf showed a blood-stained area 24 inches by 19 inches. On the pubic hair of the dead woman, complete spermatozoa were found.

Hairs found on the trousers of the accused had been pulled out by the roots and corresponded, in microscopical characters, to the pubic hairs of the deceased. At the trial a plea of not guilty was tendered, but a special defence was lodged that if the accused committed the crime, he was at the time either insane, or in such a condition mentally, as not to be responsible for his actions. After lengthy evidence for the Crown, including medical evidence as to the sanity of the accused, the defence called medical evidence as to his mental abnormality, which indicated in the opinion of these medical witnesses that accused suffered from sadism. The jury, however, found the accused guilty of rape and culpable homicide, and he was sentenced to fifteen years' penal servitude.

The next case is illustrative of a false charge of sexual assault. We are indebted to Dr A. G. Mearns for the particulars.

Dr Mearns was called to see a girl, aged eleven, who, her mother alleged, had been indecently assaulted by a boy aged fourteen. The girl who was lying quietly in bed, was well grown for her years, and physical examination revealed a normal appearance of the vulva. The hymen was normal and intact. The girl's story, in which she persisted, was as follows :—

At seven the previous evening, during the temporary absence of her parents, the boy from the next house called her into his house to look at some comic papers She went in and found him alone in the living room. He produced the papers and then pushed her into a deep clothes cupboard, forcibly pulled down her knickers and, having exposed himself, attempted to introduce his penis into her private parts. She was actually touched by the boy's genitals, and she said that "it hurt." Repeated questioning failed to shake her.

The boy was interviewed in his father's presence. He was a rachitic specimen though, physically at least, somewhat precociously developed sexually. He denied in the strongest terms that he was guilty of the alleged act, and made an important statement, namely, that the girl had entered his house as the siren of a neighbouring mill blew seven. His father, who was employed in the mill, arrived home about two minutes later. The father confirmed this, saying that he had left very punctually that evening as he had to attend a masonic lodge meeting. He arrived home at four minutes after seven at the latest. The mother said that she had returned home very shortly after her daughter had re-entered the house, which she thought was about five to ten minutes after seven. The girl's clothing was not disarranged but she was flushed. The clothing, at the time of the alleged assault, consisted of a woollen vest, cotton closed knickers suspended at the waist by elastic, a cotton petticoat, and a serge pleated skirt which fitted closely at the waist like a kilt. The knickers were new, and being rather long, had been hitched up and fastened to the undervest by two safety pins. Bearing in mind the shortness of the time occupied by the alleged act, the following points regarding the clothing are important:—

It was impossible to reach the waistband of the knickers with the hands unless a tuck-like panel was loosened in the side of the skirt. The girl made no mention of this when questioned originally.

If the knickers had been torn down, as alleged, the safety-pins would have been ripped out, leaving tears in both knickers and undervest. Neither garment showed such damage. The minute pin-holes were alone visible. The boy could not have known about the pins, and in any case he could not have reached them.

As regards time, the girl would require to have been exposed, assaulted, and her clothing re-arranged, and have returned to her own home in three minutes. Tested in the surgery it took one and a half minutes to undo her garments.

The child later confessed, amid tears, that the entire story was a fabrication, and that the boy never had, at any time, behaved improperly in her presence. It was later found that the girl had been punished for raising her skirts in the playground at the school which she attended, and for making improper gestures to boys who were passing.

Another case is rather unusual. A man was charged with rape. His defence was that intercourse had taken place with consent. Examination of the woman did not show any extensive injury of the body. Local examination revealed oozing of blood from the vulva a few hours after the alleged assault. The hymen showed two recent tears, the first, situated on the left side, close to junction of upper and lower quadrants and associated with bruising, the second, on the right side in approximately a corresponding position. The latter tear also showed some bruising. A tear, $\frac{1}{4}$ inch long,

was present close to mid-line of fourchette. Spermatozoa were found in the vaginal swabs. Examination of the accused did not show any evidence of injury affecting the genitals. In flaccid condition, the penis measured $4\frac{1}{4}$ inches from base to tip, circumferential measurement around mid-part was $3\frac{3}{4}$ inches, and similar measurement around coronal part was 4 inches. The size of the penis was thus in considerable excess of the average in European subjects. The defence raised the question of penile dimensions as an explanation of the infliction of injury during the act of coitus with consent. The jury unanimously found the accused not guilty of rape.

Examination of the accused.

As the examination of an accused person may disclose evidence which would tend to connect him with the charge preferred against him, it can only be made with his full consent. Such consent should be obtained in the presence of a third person, after cautioning the accused of the possible results of the proposed examination and informing him of his right to withhold consent (see p. 65). Scratches upon the face or hands of the accused, bites on his body or limbs, or bruises on his legs from kicks, are strong corroboratory evidence of the averment of the complainant that she resisted vigorously. Rupture of the frænum of the penis, in rare cases, may be found, and affords evidence of forcible intromission of the organ into an incommensurately smaller passage. This was found in one case where a man was charged with the rape of a girl, whose underclothing was saturated with blood. The floor of the vestibule where the crime was committed was also heavily stained with blood. The man denied any knowledge of the crime, but the finding of blood in the seams of the front part of his trousers, which showed evidence of recent washing, and on the nails and soles of his boots, heightened the suspicion of his guilt. He pleaded guilty at his trial.

Apart from general or local injury, the penis may show evidence suggestive of recent turgescence, the effects of friction, or of moisture at the meatus due to seminal fluid.

Examination of seminal stains.

It is usually necessary, as corroborative evidence of sexual offences, to examine the clothing of the person assaulted, and that of the person accused, for the presence or absence of seminal stains and blood-stains. The underclothing in such cases is frequently not characterised by its cleanliness, and consequently the examiner may expect to find a variety of stains ranging in colour from red, brown, or yellow to a faint, almost imperceptible, greyish colour. Those of a reddish colour are commonly composed of blood; of a yellowish colour, from vaginal discharge, or urine; of a brownish colour, from fæces; while those of a greyish appearance may be due to semen. To narrow the line of investigation, a preliminary examination of the garments under filtered ultra-violet light should

be made, when stains, such as those composed of urine, leucorrhœal discharge and seminal fluid, will show a bright fluorescence. The fluorescence of seminal stains is of a bluish-white colour, and stains reacting in this manner should first be selected for examination (see Fig. 209). Whenever a photograph is taken by filtered ultra-violet light with the view to producing it in court, an ordinary photograph should also be taken. Courts are inclined to distrust photographs which may not record what is normally seen but show only what is normally not seen. This also applies to infra-red photography (see pp. 78, 267).

Seminal stains, when dry, present a greyish-white appearance, impart a stiffened feeling to the fabric, and show an irregular, map-like contour. Many leucorrhœal stains present similar characters and microscopical examination will have to be employed to determine the difference. The stains when they are presented for examination are usually in a dried condition, and before their constituents can be examined microscopically they must be moistened in order to obtain an extract. Experience has shown, however, that excellent results are obtained by microscopical examination of scrapings, taken from the surface of a stain, following the addition of a drop or two of a 0·3 per cent. solution of glacial acetic acid in distilled water to the scrapings on a slide. In the majority of cases, particularly when the stains are fairly thick and not of recent origin, this method has been found preferable to the soakage method, and is recommended. The cutting edge of a scalpel is a suitable instrument to employ when making the scraping, but care should be taken to avoid the use of an excess of the material and to exclude, so far as possible, fibres from the fabric. The procedure for the soakage method is as follows :—

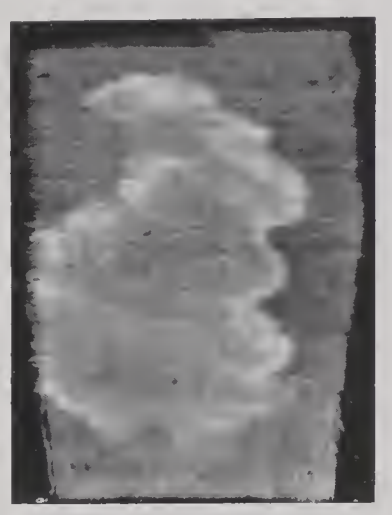


FIG. 209

Photograph of a seminal stain during exposure to filtered ultra-violet light.

Excise a portion from the stains, keeping each in a separate watch-glass.

Add sufficient 0·3 per cent. solution of glacial acetic acid in water (preferably distilled) to moisten the stained material.

Place the specimens under a small bell-jar until there is complete saturation of the fabric.

Prepare a series of six clean glass slides, and with a pair of flat-bladed forceps smear each with the moistened stain.

On three of them place cover-slips. These are now ready for examination. Dry the other three slides, and stain with hæmalum, gentian violet, or methylene blue, with eosin as a counter-stain, or with any other suitable dye.

We strongly recommend the hæmalum and eosin staining method :—

The smears must be thin and spread uniformly on the slide.
Dry by fanning.
Fix with methyl alcohol.
Refix, for five minutes, first with 95 per cent. and then with 70 per cent. ethyl alcohol.
Rinse quickly in water.
Stain for twenty minutes with hæmalum. (A 2 per cent. solution of crystalline hæmatin in 90 per cent. alcohol is added to a 5 per cent. solution of potassium alum.)
Steep in running water for thirty minutes or longer.
Restain with hæmalum for five minutes.
Rinse in water for ten minutes.
Stain, for contrast, with 3 per cent. alcoholic eosin solution for three minutes.
Differentiate in sequence of 70 per cent., 90 per cent., and absolute alcohol
Clear in carbol-xylol.
Mount in canada balsam.
Apply cover-slip.

The preparations are thus examined in the unstained and stained conditions. The $\frac{1}{6}$ inch objective should be employed.

Scott [7] recommends the following improved technique for fixation and staining of spermatozoa.

Fixation.

Put a small drop of specimen seminal fluid on a clean slide and draw it gently with the edge of another slide, making an even smear.

Place slide in stoppered bottle containing equal parts of ether and 95 per cent. alcohol. Let slide stand for three hours, or preferably, overnight. Prevent friction of specimen on prepared surface of slide.

Staining.

Pass through alcohols, 70 per cent., 50 per cent., and distilled water. Stain by Baecchi's method :—

Stain for five minutes in Baecchi's fluid (1 per cent. methylene blue, 1 millilitre ; 1 per cent. acid fuchsin, 1 millilitre ; 1 per cent. hydrochloric acid, 40 millilitres).

Wash in distilled water, blot gently, dehydrate rapidly in absolute alcohol, clear in xylol, and mount in neutral balsam.

Taylor [8] advocates the following method for staining spermatozoa in forensic work. The suspect stain is soaked directly on to a microscope slide in a few drops of acidulated water (28 millilitres distilled water, 1 drop of concentrated hydrochloric acid).

According to stain and the material, soak from five minutes to one and a half hours. It is advisable to tease the material into individual fibres during soakage and to squeeze as much as possible of the extract from the material by means of forceps.

The film is allowed to dry at room temperature.

Staining.

Fix film by passing through Bunsen flame.
Stain in ripened Mayer's hæmalum, two to five minutes.
Wash in tap water.
Blue by exposing to ammonia vapour.
Wash in tap water.
Counter-stain in 0·5 per cent. to 1 per cent. aqueous solution of eosin (yellow) for one to three minutes.
Wash in tap water.
Dry film by gentle heating.
Mount in Canada balsam.

The spermatozoa show the following staining reactions: head cap, pink-blue; head and neck, bright blue; and tail, pink.

The characters of seminal fluid.

The normal quantity of seminal fluid in a single ejaculate is from 2 to 5 millilitres, and on the average about 3 millilitres, although the volume varies widely even in the same person. Volumes of 6 millilitres and over may be regarded as excessive, but a case has been reported of an ejaculate amounting to 13·5 millilitres. The total number of spermatozoa in an ejaculate from a healthy young man is about 400 to 500 millions, but it must not be forgotten that seminal stains may not disclose the presence of spermatozoa if the subject from whom the seminal fluid came suffers from aspermia or oligospermia. Such states may be either temporary or permanent. In these instances, since the finding of spermatozoa is necessary to prove that stains are seminal, there is a considerable chance of guilty persons evading conviction. The fresh ejaculate is of a gelatinous, stieky character, but after it has been exposed to the air for about a quarter to half an hour, it tends to become more liquid, probably due to enzyme action. Semen consists of two unities, seminal fluid and certain formed cellular elements including spermatozoa, and epithelial cells and crystals, composed of choline and lecithin. It is slightly alkaline in reaction.

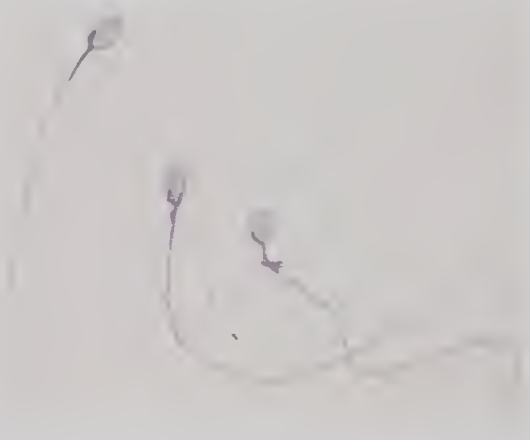


Fig. 210

Spermatozoa. Stained preparation —methylene blue. ×850.

It is not difficult to recognise a spermatozoon when it is seen entire because of its large and obvious head, which is about 4·5 μ in length, and its neck and filamentous tail, which are about 50 μ long. The seminal fluid of young male subjects frequently contains spermatozoa, and we have found them plentifully in stains produced by the ejaculate of boys of thirteen.

Although stains are proved to be of seminal origin when spermatozoa are present, their absence does not prove that the stains have not been produced by human semen. For medico-legal purposes, we hold the view that if spermatozoa, or at least one complete spermatozoon, are not found in the stain, we cannot positively affirm that such stains are seminal, and that the apparent absence of these bodies does not permit of an opinion that the stain has not been produced by human seminal fluid. In the latter instance, an examiner should merely state that, as the result of his examination, he was unable to detect the presence of spermatozoa. The discovery of spermatozoa in stains is not always an easy matter, for the obstructive elements are many. The important responsible factors are the character of the fabric, the age of the stain, and the conditions to which it has been exposed before reaching the laboratory for examination. Spermatozoa have been discovered in stains long after their emission. We have found many complete sperms in a stain upon a garment after the elapse of five months following the sexual offence, the garment having been in police custody during that period. The undergarment in question, apart from the seminal staining, was clean and relatively new. On the other hand, we have frequently examined seminal stains which, when moist and fresh, were found to contain spermatozoa, but which did not reveal them after they had become dried from exposure to the air of a warm room, even after comparatively

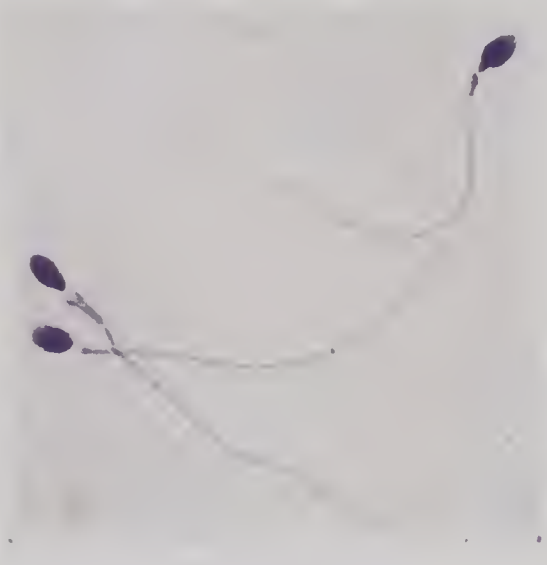


FIG. 211

Spermatozoa. Stained preparation—methylene blue and Biebrich scarlet. ×850.

FIG. 212

Florence test. Microscopical appearance of crystals (acicular type). ×75.

short intervals of time. Desiccation would seem, therefore, in some cases, to lead quickly to disintegration of spermatozoa. We have been able, however, in unequivocal stains kept in stoppered bottles, to detect spermatozoa after the lapse of six months. Our view is that it is not so much the continuous desiccation which militates against their discovery on fabrics, as the occurrence of decomposition in the early stage before the stains become dried, due to their admixture with urine or other discharges which undergo rapid decomposition. Readily identifiable spermatozoa may be found in the vagina for a considerable period after death. The writer has had no difficulty in detecting complete and well-preserved spermatozoa in a vaginal swab after some eighty-five hours, the estimated interval between death and the taking of the swab. A number of circumstances play an important part in the preservation or early destruction of sperms within the vagina of the cadaver. (For their survival period within the vagina of the living subject see p. 377.)

Florence test.

This is merely a preliminary chemical test, and is not regarded for medico-legal purposes as a positive test for seminal fluid. The reagent employed consists of a solution of 1·65 grammes of potassium iodide and 2·54 grammes of iodine in 30 millilitres of distilled water. The reaction is shown by the formation of dark brown crystals, rhombic or needle-shaped, when the reagent is mixed with an aqueous extract of seminal fluid. The test is employed as follows:—The seminal extract is prepared by warming the stains in a little water on a water-bath, acidifying with dilute hydrochloric acid, cooling and adding some dry ammonia sulphate. The extract so treated is filtered, the reagent is added, and the mixed fluid examined microscopically. In most instances the reaction is obtained simply by adding a drop of the reagent to a wet film of seminal solution, placing a cover-slip on the slide and examining microscopically. The reaction is due to choline. The crystals are composed of periodide of choline, which may also be produced by tissue extracts. Forbes,[9] on our suggestion, submitted this test to critical survey, and arrived at some important practical conclusions. He states that if the result is negative, the spermatic fluid may not have reacted due to a very low choline content, the stain may have been over-diluted, or it may not have been composed of seminal fluid. When positive, one need not necessarily find spermatozoa since it may be a case of aspermia. In his opinion, a fallacy might be due to tissue extracts in the stain.

Barberio's test.

Another preliminary spermatic reaction is produced within a few minutes when a saturated aqueous or alcoholic solution of picric acid is mixed with fresh, dried, or dissolved semen. Certain crystals

are formed which, when examined microscopically, appear as slender yellow-tinted rhomboid needles with obtuse angles, sometimes crossed longitudinally by a line of refraction, or as ovoid crystals when the angles are rounded off. These crystals are composed of spermine picrate. The extract on which the test is applied should not be too concentrated. Like the Florence test, this reaction is not a positive one for seminal fluid, and the results are not so uniform as in the former test.

Since both iodine and picric acid stain spermatozoa, either of these tests can be applied simultaneously with the microscopical examination for the detection of spermatozoa.

Fig. 213

Barberio's test. Microscopical appearance of crystals. ×75.

The attention of the examiner must not be confined solely to seminal stains. It is important, especially in cases where there has been recent rupture of the hymen, that blood-stains also should be examined for spermatozoa, since they may be present in the blood which flows from the breach in the hymen.

Leucorrhœal stains are characterised by an abundance of squamous epithelial cells, and if the discharge has been muco-purulent, by pus cells also, the compound nuclei of which can be rendered apparent by treatment with weak potassium hydrate.

In conclusion, it should be stressed that the presence of at least one spermatozoon must be established to identify positively the stain as semen, and that no one should undertake this examination without adequate experience.

Precipitin or serological test with seminal fluid.

The spermato-precipitins are of value in the identification of seminal fluid in certain cases, admittedly few in number, for example, in bestiality cases, where it might be desirable to differentiate between the seminal fluid of man as opposed to animal, by having a corroborative serological test to substantiate an opinion based on the microscopical differences in character of the spermatozoa. To apply the test, an immune serum must be prepared by injecting a rabbit with human seminal fluid or the seminal fluid of a particular animal, in the same manner as for the production of anti-blood

serum. When the anti-serum is obtained, the serum precipitates must be removed from it in order to produce the immune serum. This is done by mixing it with an equal part of 1 in 200 dilution of human serum or animal group serum. It is necessary to leave the mixture at room temperature for an hour, then in a refrigerator overnight, and thereafter it should be thoroughly centrifuged. As a rule, this removes all precipitum from human serum or from the serum of the particular group of animals, and leaves a serum which is specific for human seminal fluid or that of the particular animal group. The supernatant fluid is used for the test which is carried out in the manner already described (see p. 339). With a properly prepared serum, 1 millilitre of seminal fluid extract to which 0·1 millilitre of immune serum has been added will give a reaction in one hour at room temperature.

Determination of the group of seminal fluid.

Serum from Group A and serum from Group B are mixed with saline to form dilutions of 1 : 2, 1 : 4, 1 : 8, 1 : 16, etc. 0·1 millilitre of each dilution of each serum is added to the same amount of seminal fluid in a small test-tube. The tubes are allowed to stand for two hours at laboratory temperature when they are centrifuged, the supernatant fluid tested with cells of Group A and Group B, respectively, and the effects of the previous absorption carefully noted. In this manner the group of the seminal fluid is determined. By this means it is therefore possible in certain cases to show that the group of seminal fluid found on stained articles differs from that of the seminal fluid of a suspect who may be charged with a sexual offence.

Examination for venereal disease.

The examination for the presence of venereal infection in an accused or assaulted person must include both clinical and laboratory procedures and should be conducted only by those specialised in the appropriate fields. The venereal diseases are gonorrhœa, syphilis, and soft sore.

Gonorrhœa.

The causative organism, Neisseria gonorrhœae, is usually abundant in acute gonorrhœal discharges from males, but in chronic infections, as well as in the female, it may be scanty, and less in evidence than concomitant coliform or diphtheroid bacteria. Smear, culture, and serological methods are available for its demonstration.

Smear method.

In the male, both urethral and prostatic secretions should be examined. The urethral meatus should first be thoroughly cleansed, both with saline and spirit, by means of gauze swabs. A bead of secretion, obtained by introducing a sterile platinum loop into the

meatus, should then be transferred to a slide and spread into a thin film. Prostatic fluid should be obtained by finger massage as follows. The subject, having first urinated in order to expel adventitious bacteria, should separate his legs and bend forward till the palms touch the ground. The middle finger of the operator's gloved right hand, lubricated with vaseline, should then be inserted into the anal canal. When the prostate is felt, it should be massaged with a hooking motion of the examining finger until prostatic secretion exudes from the urethra and may be collected upon a slide. Both urethral and prostatic material should be fixed by gentle heat and stained by Gram's method, of which the following modification will be found convenient :—

Gentian violet (2 per cent.), 2 minutes.
Lugol's iodine, 1 minute.
Wash in water.
Acetone, 20 seconds.
Wash in water.
Ziehl's fuchsin (1 in 200), 30 seconds.
Wash in water. Dry off above flame.

The integrity of the staining may be controlled by making at the same time a film of a known Gram-positive organism, such as Staphylococcus aureus.

When the subject is female, material should be obtained from both urethra and cervix. Preliminary cleansing should be as in the male, but more thorough, particular care being required in swabbing the vulva. For the successful acquisition of specimens a gynæcological table should whenever possible be employed to ensure proper position of the patient, while a suitable expanding speculum and a good light are essentials. The cervix is not affected under the age of puberty, and in such cases it is sufficient to collect discharge from the vulvar margins and vaginal introitus.

The presence of bean-shaped Gram-negative diplococci occurring intracellularly in multiples of two may be taken as diagnostic. It should, however, be noted that in very early infections, and also in chronic stages, the gonococci are often extracellular. In all cases care must be taken to discriminate the gonococcus from other wholly or partially Gram-negative diplococci, notably the Diplococcus magnus, which resembles it, but is usually larger. In this connection the routine use of the same magnification for all smears is desirable.

In all cases of vulvo-vaginitis in children, especially where medico-legal issues are involved, it is imperative that the bacteriologist be sure of his ground before returning a diagnosis of gonorrhœa.

Culture method.

As a corroborative procedure, or whenever the smear method leaves doubt, culture of the discharge should be attempted, but this is, even in skilled hands, far from uniformly successful. New

techniques, employing enrichment media, have given promising results and are now being developed.

In males, according to Jadassohn, cultures should always be made from prostatic secretion. The organism can be cultured also from fresh seminal fluid. If the cultures are consistently negative, gonorrhœal infection may be eliminated.

Serological method.

The complement-fixation test is now receiving a wider recognition in diagnosis, and is frequently of value especially in older infections and, as in the female, when smears have proved equivocal. In expert hands, using a suitable antigen, the test has yielded more than 90 per cent. of positive results in late and chronic cases. The test is in general similar to the Wassermann procedure.

Syphilis.

Dark-field method.

The diagnosis of syphilis in the primary stage is established by demonstrating Treponema pallidum alive in exudate from the chancre prior to the therapeutic administration of arsenic or the local application of an antiseptic. The sore should first be cleansed by rubbing with plain gauze soaked in saline, and then dried. The serum which exudes should immediately be examined by the dark-field technique, or else be collected in a capillary tube, which is then sealed and sent to a laboratory with expedition. In the absence of serum after the preliminary cleansing, it is permissible to scarify the lesion gently, but the collection of blood in the capillary tube should be avoided, as corpuscles interfere with the subsequent examination.

Material obtained by like method from the fauces or exanthem in acute secondary syphilis may also reveal the presence of the treponema.

Serological method.

When syphilis is suspected, 10 millilitres of blood should be withdrawn from the basilic vein and submitted to a laboratory for the Wassermann test. A positive reaction will not have reached full development until six to eight weeks following the date of infection. For medico-legal purposes, it is important to remember that in the absence of clinical signs a positive serological finding does not necessarily denote the presence of active syphilis; nor does it necessarily affirm infectivity. Congenital lues and Wassermann "fastness," a not uncommon occurrence in the treated and non-infective subject, should be kept prominently in the mind of the examiner, particularly in the approach to cases of alleged criminal abortion in which the defence may raise the question of syphilis as the determining factor. In cases of long standing the importance of Wassermann-testing of the cerebrospinal fluid must not be overlooked.

The Kahn and Meinicke tests (see Fig. 214) are of value in confirming Wassermann findings. A quantitative modification of the Meinicke test which returns the titre of circulating antibody is now under investigation.

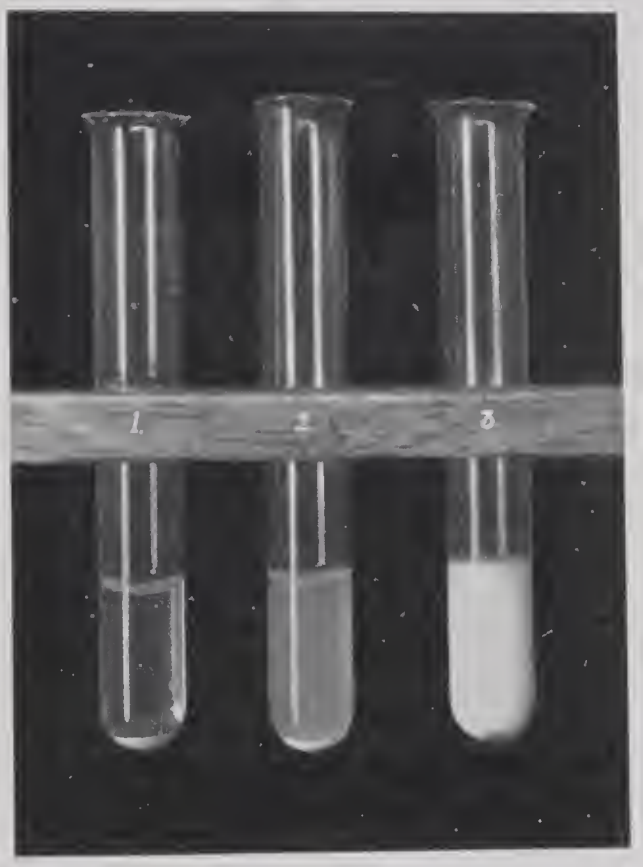


FIG. 214

Meinicke test.

1, Positive reaction. 2, Doubtful reaction. 3, Negative reaction.

Can a healthy adult female be raped by one man?

There is no doubt that rape can be perpetrated in the instance of a woman who has become exhausted by the resistance which she has offered, or when, on account of fear or injury, she has lost consciousness. One should refrain, however, from expressing an opinion on this important point until careful consideration has been given to all the facts of the particular case. There have been many undoubted cases in which a woman has been raped by an unaided man despite the fact that so long as a woman retains complete possession of her senses it does not require great physical strength to deny sexual entry by apt disposition of her limbs. When the act has been accomplished, one is almost forced to the conclusion that she must have become physically exhausted and incapable of further resistance, or that from fear or injury there had been temporary loss of consciousness, or that there was marked disproportion in strength between the woman and her assailant, or that she finally became acquiescent to the sexual act. On the other hand, it must not be forgotten that an assailant might have overcome

her resistance quite readily by an appropriate disposition of her clothing.

When marked disproportion of age, size, and apparent strength in favour of the female are present, a charge of rape is often unsuccessful, because consent is likely to have been given. In most cases of this kind, however, such questions will probably not be asked of a medical witness, because the judge and jury are able to form their own opinions. The duty of the medical witness is to decide, from the appearances of the body of the female, whether carnal knowledge of the woman has been effected forcibly, and the court will form its own opinions on the other points.

It is obvious that rape might be perpetrated quite easily when there is more than one assailant.

Is it possible for a woman to be violated during ordinary sleep without her knowledge?

The answer to this question is that it is highly improbable in the case of a virgin. We should say that it is impossible, because of the pain attendant upon a first coitus. In the case of women who are accustomed to sexual intercourse, it is unlikely, although not impossible. Some Scottish cases are of interest in this connection.

A man was convicted on the charge of having carnal knowledge of a woman when asleep; and another man, in a second case, was also convicted on the same charge. By the law of Scotland this offence is not rape but clandestine injury to a woman (see p. 423). In our experience we have only known one attempt of this kind. The prisoner tried to personate the husband of a young woman and entered her bed, but the woman, awaking when he tried to have coitus, raised the alarm, whereupon the man fled. On examination we found her to be six months pregnant but not to have sustained any injury.

Carnal knowledge during unconsciousness.

Cases of violation of females during a state of unconsciousness occasioned by fits, faints, hysteria, and anæsthetics have occurred. That the act may be accomplished under such circumstances is obvious. It is unfortunately true that medical men and dentists, by reason of their professional work, are liable occasionally to have baseless charges of this kind preferred against them by women following the administration of an anæsthetic. We have been informed by the police of a division in which a large hospital is located that such charges are by no means uncommon, and that, having investigated several of these, they have found them without foundation.

Procedure in examination of females in cases of alleged rape.

In proceeding to the examination of persons in connection with a charge of rape or unlawful carnal knowledge, there are certain points which should prove useful to the inexperienced.

When such a charge is laid with the police, the practice is that a doctor is requested to examine the complainant, who, by laying a charge, is presumed to be willing to afford all the evidence, even to the examination of her person. Expressed consent to make the examination must, nevertheless, be obtained (see p. 65). Another female should always be present during the examination. In cases of unlawful carnal knowledge and other sexual offences with girls under the age of sixteen, the consent of the girl is insufficient, since she is held incapable of giving or withholding consent until she has attained the age of sixteen. The consent of her parent or guardian is, therefore, first required. The medical examiner should not ask the female any questions concerning the details of the alleged offence, except as to the time and place of the assault.

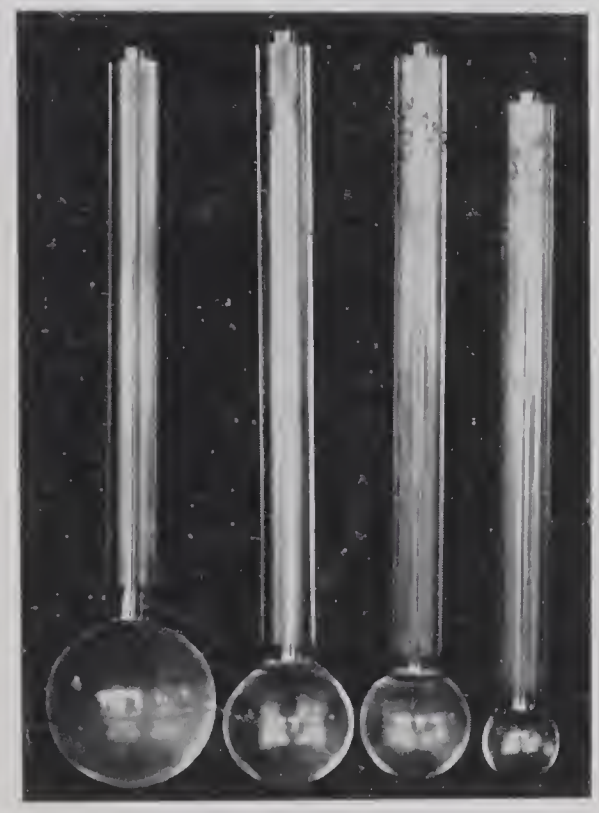


Fig. 215

Graduated, solid glass globes for deployment of hymeneal edges during examination.

When making a local examination of the genitals, modesty must not be allowed to obstruct a detailed investigation. Either the lithotomy or knee-chest position may be utilised for the purpose, adequate illumination must be available, and the subject under examination should be treated with consideration.

In cases which present an annular notched hymen or a hymen which shows an irregular purse-like opening, it may be, and frequently is, a difficult matter to differentiate between the natural notches and the results of old and small laeerations. Practice has shown that this matter is greatly simplified by the use of a glass rod with a small, spherical head. This is gently passed through the hymeneal aperture and then eased forwards, when the edges of the hymen become slightly everted. By slowly rotating the sphere, around which the edges are plaeed, natural notches are readily differentiated from tears, recent or old. This method does not cause either pain or injury, and its use by us over a long period has amply demonstrated its value. These rods should be composed of Pyrex glass, and we recommend that a set, with spherical heads of different sizes, should be at hand (see Fig. 215). The rod should be at body temperature before insertion.

The following examination should be made in such cases :—

The general physical appearance and demeanour of the female.

The presence or absence of marks of violence upon her body ; their character, and position, when present.

The presence or absence of marks upon the clothing, when the assault is alleged to have taken place in the open.

The condition of the genitals, with respect to : (1) the presence or absence of blood ; (2) the signs of bruising, or other injury ; (3) the condition of the hymen with regard to old or recent injury, together with the character, extent, and situation of the injury ; (4) whether, from the character of the vagina, the female has been habituated to coitus.

Some vaginal secretion for microscopical examination should be obtained as already described (see p. 432).

If a muco-purulent or purulent discharge is present, a specimen should be taken for bacteriological examination.

The examination of stains on garments for spermatozoa should be made subsequently.

INDECENT AND LEWD PRACTICES

The provisions of the Criminal Law Amendment Act, 1885, in relation to these offences, have already been described (see p. 424). Such practices have assumed a varied form and have covered a wide range of indecent conduct towards girls and boys in the many cases which we have investigated. Among the most prevalent, are handling of the private parts, including the introduction of the finger into the vagina, masturbatory acts performed upon the girl, inducing her to handle the male organ, mutual masturbatory acts, intercrural connection, and lingual contact with the private parts. When the subjects of such offences are under the age of puberty, girls of twelve years and boys fourteen years, the crimes are generally under common law, but where the girls are above the age of twelve and under the age of sixteen the charge is brought under the Criminal Law Amendment Act, 1922, section 4.

The age of the girl, if under sixteen, must be set out in the indictment and must be proved at the trial. In the case of gross indecency between males, whether in public or in private, puberty, for obvious reasons, is not made the limit of a charge. Further, in the case of boys, consent cannot legitimise the offence, and where a boy old enough to consent, has consented, instead of the act not constituting an offence as in the case of females over the age of sixteen, the act becomes a crime in both parties. In cases of indecent assault between females, if the assaulted person is over sixteen, the defence of consent could be employed, since an adult and consenting woman may behave indecently, in private, but this is not permitted by law between males. Conversely, it is an offence for a female to commit an indecent assault upon a boy.

In the case of R. v. Hare,[10] a woman was convicted of indecent assault on a boy of twelve. On appeal it was argued that the offence of indecent assault on a male referred to in Section 62, Offences Against the Person Act, 1861 (see p. 452), could not be committed by a woman, since offences in that section were limited to sodomitical offences. Mr Justice Avory, in dismissing the appeal, pointed out that "unnatural offences" did not govern the words "any indecent assault upon a male person" and said there was no ground for limiting these words in the manner suggested.

In all cases of indecent practices, it is held to be an aggravation of the offence when the offender is in a relation of trust to the person offended, as for instance, a teacher or a nurse. It is also an aggravation if venereal disease is communicated, or if previous convictions for indecent, lewd, and libidinous practices have been recorded against an accused person.

The examination of the subjects resolves itself into an examination of the private parts of the offended girls or boys, and we have not infrequently found that such practices have been made by one offending male against a series of girls or boys of tender age. In some cases, injury to the hymen may be found due to the insertion of the finger of the offender. The only evidence of any value as to whether the male organ has been used and emission of semen has taken place, is obtained by an examination of the underclothing, bedding, or other material. On a number of occasions, we have had to examine the male offender where there was a charge of communicating venereal disease to the offended girl or girls. Reviewing the records of sixty-seven cases of indecent practices with girls, we find that seventy-three men were charged, but that one hundred and forty girls were molested. In thirty-one cases, more than one girl was assaulted by the accused, and in several instances the accused was responsible for indecent practices with no fewer than four, five, six, and seven young girls. Drink played an important rôle in the cases of four accused men. One of the men charged was an epileptic. Venereal disease was communicated to the young victims in three cases. Many of the offenders were married and three were the fathers of children. The ages of the male accused varied from sixteen to sixty-four, while the age range of those molested was four to fifteen years, the commonest ages being between seven and twelve.

With regard to cases of unlawful carnal knowledge, eighty-seven cases showed that the most common age incidence of the young females was between eleven and fifteen. Venereal disease was communicated in four cases, and in fourteen cases pregnancy resulted between the age of thirteen and sixteen.

In one of our cases, the accused was linked up with the offence, which took place in an outside lavatory, by a comparison between the stains on his overcoat and the substance covering the walls of the lavatory. Analysis showed that the powder composing these stains was similar in composition to scrapings taken from the walls. This similarity was based on the fact that both contained barium, zinc, and sulphide.

UNNATURAL SEXUAL CRIMES

Allen [11] states "that the law of the land discourages any public manifestation of sex, no matter how normal it may be." Any transgression of this tends to be regarded as an act of indecency and may be punished accordingly. Apart from "natural" manifestation, there are six so-called "unnatural offences" which are punishable. These are :—

1. Incest.
2. Sodomy.
3. Bestiality.
4. Masturbation, if publicly performed.
5. Indecent exposure.
6. Tribadism, if publicly performed.

Incest.

The crime of incest is statutory in Scotland (the Act of 1567), while it is an offence against the canon law, and also against statute law in England (Punishment of Incest Act, 1908). Incest is the crime of carnal connection between persons held to be within the forbidden degrees of relationship. There must have been actual sexual connection to constitute the crime. In Scotland, an attempt to commit incest is a relevant charge both at common law and by statute. The parties must know that they were related within the prohibited degrees, but if coitus is established the onus of disproving presumed relationship is thrown on the persons accused. The Criminal Procedure (Scotland) Act, 1938, has enacted that carnal knowledge between a man and a woman, whose marriage to each other is authorised by the Marriage (Prohibited Degrees of Relationship) Acts, 1907-31, or would be authorised on the death of any person, is not incest. Medical evidence in such cases resolves itself into affirmation of non-virginity of the female, or the existence of pregnancy. We have examined many girls for evidence of this crime, and in all of them there were definite signs of trafficking in the sexual parts, and in one, pregnancy resulted, while in others seminal stains on the undergarments were found.

In one of the many cases in which we have made examinations, the incestuous intercourse was between brother and sister, both under the age of majority. The girl at the time of our examination was seven months pregnant. In many other instances, the intercourse was between father and daughter.

In one case, two sisters were involved in incestuous intercourse with their father. The elder, aged sixteen and ten months, explained that the acts of intercourse had occurred twice weekly over a period of eight years ; the younger, aged fourteen, stated that her father had had connection with her on at least ten occasions, over a period of two years.

In reviewing thirty-two further cases of incest, we found that the father of the victim was involved in nineteen instances, the

step-father in nine, an uncle in one, and a brother in three. The ages of the females ranged from six to twenty-three years, the most frequent age incidence being fifteen years. Pregnancy resulted in four cases, in one of which the girl was twelve and a half years old. In two cases, where the sexual relationship took place between brother and sister, the age of the girls was fifteen and that of the brothers seventeen and eighteen, respectively. Another case involving three adults, was most unusual, since the woman, already married and with one child, committed incest on many occasions not only with her adult brother but with her aged father.

Sodomy.

Sodomy is the unnatural anal intercourse between two male persons. The passive agent, if consenting and above the age of sixteen, is equally guilty with the active participant. Lord Dawson [12] in a debate on the Matrimonial Causes Act, 1937 (see p. 365), stated that homosexuality was regarded as a pathological condition. In the future it might be regarded as a deficiency disease. It had one foot in the realm of disease and was not wholly in the realm of crime. Few people were 100 per cent. female or 100 per cent. male. Some were predominantly one or the other, and some near the border line; these went wrong or right according to the circumstances of their lives. For practical purposes homosexuals were potential or active. By the Offences Against the Person Act, 1861, the punishment is limited to imprisonment. In England, this crime may be prosecuted under the Offences Against the Person Act, 1861, Sections 61 and 62, and under Section 11 of the Criminal Law Amendment Act, 1885.

The Criminal Law Amendment Act, 1885, Section 11, enacts:—

That any male person who, in public or private, commits, or is a party to the commission of, or procures or attempts to procure the commission by any male person of, any act of gross indecency with another male person, shall be guilty of a misdemeanour.

In Scotland, the crime is prosecuted at common law or under the above section of the Criminal Law Amendment Act. It should be clearly understood, however, that prosecution under Section 11 of the Criminal Law Amendment Act, 1885, in such cases, is as an alternative charge to that of sodomy where the facts which can be proved fall short of what is necessary to sustain a charge of sodomy, or a definite attempt to commit sodomy, but where the act committed is one of gross indecency between males. This section has therefore widened the scope of the law, with respect to the commission of indecent offences by two or more male persons, but does not of itself necessarily deal with the crime of sodomy. It includes such offences as penile friction on the gluteal folds, handling of the male parts, mutual masturbation, or intercrural connection.

Under the Offences Against the Person Act, 1861, it would appear that, in relation to the crime of sodomy, the sex of the passive agent would be immaterial since the term used is "man-

kind," the mass of human beings.[13] Proof of penetration, but not of emission is necessary to constitute the offence. In R. v. Wiseman,[14] a man was indicted for having committed this offence with a woman, and in R. v. Jellyman[15] for committing the offence on his wife. Pederasty is the form of sodomy in which the passive agent is a boy. This unnatural crime is, perhaps, more common than is supposed, but charges frequently break down at, or before, trial for want of adequate evidence.

The disproportion between the penis and the anal orifice, together with the absence or use of violence, and the use or otherwise of a lubricant, in the course of penetration, are important factors in determining whether injury to the anus will result.

ILLUSTRATIVE CASES

Two young men assaulted a boy, and while one held him the other committed the act. On examination, we found four recent linear lacerations of the anal mucous membrane with evidence of bleeding.

A man was charged with repeated acts of sodomy with a boy aged eight over a period of seven months. Examination of the anal parts showed irritation around the entrance, and a healing fissure on the mucous membrane, together with an infundibuliform shape of the rectal orifice. The boy suffered from incontinence of fæces.

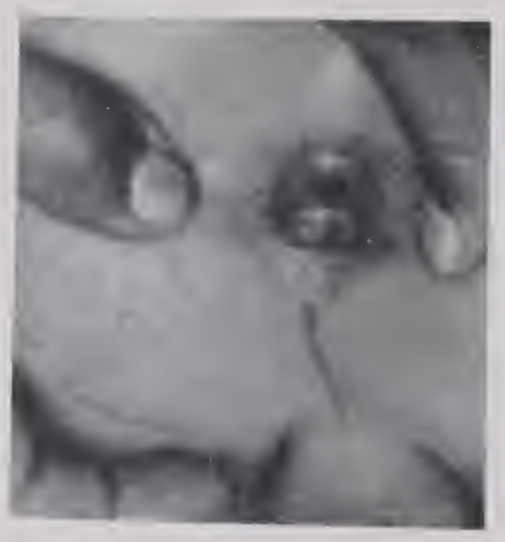


Fig. 216

Case of sodomy. Passive agent was a boy of ten. Some dilatation of anus, together with bruising and inflammation, were present

In a case of sodomy, involving two adults, smears from the glans penis and urinary meatus of one of the men and from the rectum of the other showed numerous spermatozoa. Stains from the jacket, raincoat, and trousers of the former man, and from the overcoat and trousers of the latter, also disclosed the presence of many spermatozoa.

In a case of attempted sodomy by a man with a boy, examination under filtered ultra-violet light of the pyjamas worn by the boy showed a fluorescence of certain oleaginous stains which was identical to the fluorescence given by the contents of a jar of vaseline said, by the boy, to have been used by the accused.

The lesions which ought to be looked for, in the passive agent, by the examiner are :—

Recent lacerations, bruising, or inflammation of the anal mucous membrane.

Dilatation of the anus, absence of puckering of the anal orifice, and diminished sphincter-grip, in varying degree, in habitual cases.

An infundibuliform shape of the anus in confirmed passive sodomists.

The presence of old lacerations, as indicated by scarring, and of external piles.

Incriminating stains are important.

Without condescending upon details, this unnatural crime may be committed in ways which leave no lesions behind them, and consequently, in many cases, the crime either goes undetected, or fails to be proved through lack of evidence.

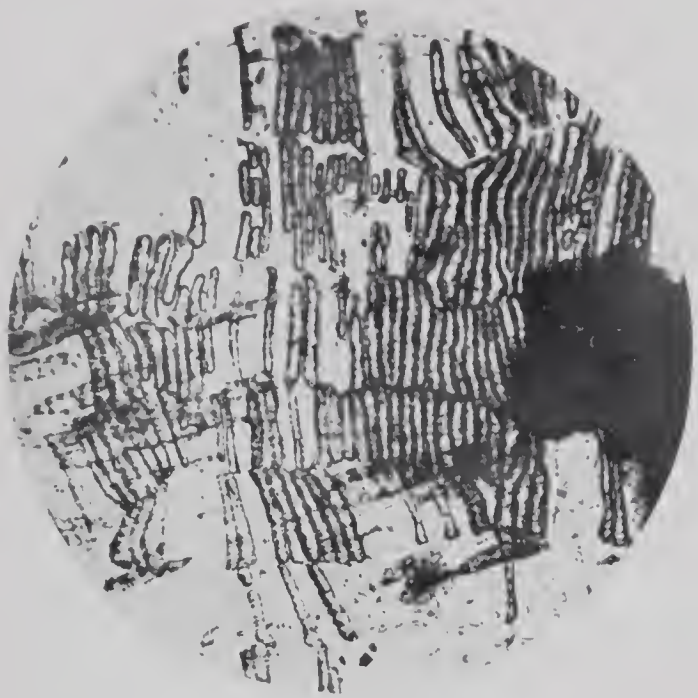


Fig. 217

Striated muscle fibre in a fæcal stain associated with sodomy.

Sodomy most frequently results from the condition of male sexual inversion or homosexuality. A review of sixteen relatively recent cases of sodomy shows that the age of the active agent varied from twenty-five to sixty-four years, whereas that of the passive agent was from six to forty-three years. In three instances, the active partner had been previously convicted of sodomy, and in six others, with the offence of gross indecency with another male. A case (reported to us by Dr Mearns) is of special interest, since it shows precocity of the active agent. He was a boy of fourteen, who suffered from urethral gonorrhœa and had experienced sodomitic relations with several men whom he had importuned for the purpose of gratifying his perverted sexual urge.

So far as boys are concerned, charges at common law are usually preferred when the boy is below the age of puberty. Such cases can still be brought at common law when the youth is above that age but usually such charges are brought under section 11 of the Criminal Law Amendment Act, 1885.

With regard to gross indecency between males, points which emerge from our records are :—

The ages of the accused varied from twenty-five to forty-five :

The assaulted males varied in age from six to twenty-six ;

One of the accused had four previous convictions for sodomy and indecent practices, one had five convictions for lewd practices and was found to be insane, another had a conviction for sodomy and gross indecency with a male, and several had previous convictions for gross indecency.

Taylor's [16] monograph on homosexual offences and their relation to psychotherapy contains some very interesting information, and allusions to it are freely made. He states that in 1946, ninety-six persons were received into Brixton Prison charged with homosexual offences. Of these, thirty-nine were charged with indecent assault on boys, twenty-four with importuning, seventeen with gross indecency, and sixteen with the crime of sodomy. Of the ninety-six cases, fifty-seven involved offences against boys, including all the indecent assaults. He classified all the cases into four groups :—

1. those with heterosexual tendencies in whom the homosexual offence was in the nature of a substitution for the normal heterosexual act and who were classified as "pseudo-homosexuals" ;
2. "bisexuals," individuals in whom strong heterosexual, as well as homosexual, tendencies were obvious ;
3. prostitutes, individuals who would have fallen into the "pseudo-homosexual" group, but were characterised by the fact that the homosexual acts were carried out for gain : and
4. "true inverts," which numbered only thirteen in the total of ninety-six cases. Of the "true inverts," eight were charged with importuning, three for gross indecency, and two for sodomy. Of the thirteen, only one was interested in boys, but twelve were attracted to men. Five were always passive, two were always active, three were either active or passive, and in three the act was in the form of mutual masturbation.

Androgynous physique was present in six cases. Of the two active men, one was quite effeminate in appearance. Of the five preferring the passive role, three were of feminine, and two were of masculine build. Perversions were much more evident in those preferring the passive role. Fellatio was admitted by four men. Of the true inverts, thirteen cases in all, seven had had treatment with improvement, three refused treatment, and only one of the remainder was considered likely to benefit by treatment.

Bestiality.

Bestiality is the term applied to sexual intercourse between a person of male sex and an animal, whether by the anus or by the vagina. Under the Offences Against the Person Act, 1861, applicable to England, it constitutes a felony. In Scotland, bestiality is an offence at common law. Of this crime little need be said. Any evidence of a medical character must either be supplementary to that of eye-witnesses, or purely circumstantial. When in the latter

form, it usually consists in finding upon the person of the accused evidences of contact with the animal, as, for example, hairs which correspond with those of the animal in question, or the presence of human seminal fluid in, or around, the parts of the animal.

ILLUSTRATIVE CASES

A man was tried for bestiality with a mare. A pair of trousers which the accused was wearing at the time of apprehension, and hair from the hinder parts of the mare, were examined. Stains consisting of blood were present in the fork of the trousers, and also a hair, similar to the hairs from the mare, was found. Certain other whitish stains on the trousers were examined and some were found to contain squamous epithelium. When fluid from the neighbourhood of the genitals of the mare was examined, immediately following the offence, spermatozoa were found. The accused pleaded guilty, and, having been previously convicted of a similar crime, was sentenced to a long period of imprisonment.

At the Circuit Court, Glasgow, a man was tried for bestiality with a cow.

At the High Court, Dumfries, a man was convicted of bestiality with domestic fowls.

A man was apprehended near Renfrew after having been seen attempting to have unnatural intercourse with a duck. On examination as to his mental condition, he was found to be of unsound mind and unfit to plead.

At Fort William, a man was charged with committing this offence with a female goat, and was convicted (see Fig. 31, p. 33).

OTHER SEXUAL PERVERSIONS

Sadism.

The term is applied to the association of sexual desire and the inflicting of cruelty or violence. The condition is frequently associated with " lust " murder (see Fig. 171, p. 316).

Masochism.

This is the opposite of sadism, since the condition is the association of sexual desire with the desire for submission to cruelty and violence or mutilation.

Fetichism.

Fetichism is sexual gratification produced by contact with, or the sight of, an object that normally does not exercise a sexual influence on the mind. An infinite variety of sexual references may thus be developed among different individuals.

Indecent exposure or exhibitionism.

These terms apply to the acts of men whose sexual desire consists principally of the exhibition of the genitals with or without the performance of masturbatory acts in the presence of women or young girls. Occasionally women may expose themselves in public.

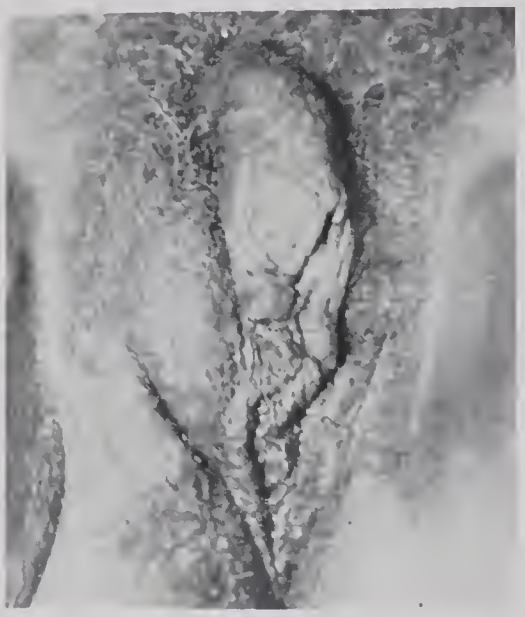


FIG. 218

Abnormal enlargement of clitoris in a woman long habituated to lesbianism.

Female sexual inversion, tribadism, or lesbianism.

These terms are applied to sexual relationships between females (see Fig. 218).

Male sexual inversion or homosexuality.

These terms are applied to sexual relationships between males (see p. 452).

Transvestism or eonism.

Transvestism is the name given to the perversion in which males find sexual pleasure in wearing female apparel. This condition is sometimes found in females who dress themselves in male attire.

Necrophilia.

The term is applied to the act of defiling the dead by sexual intercourse.

References

1. H.M. Advocate v. Grainger [1932], Scots Law Times, 28.
2. H.M. Advocate v. Logan [1937], Scots Law Times, 104.
3. R. v. Keech, 21 Blackman Crim. App. Rpts., 125.
4. R. v. Chapman (1932), Police Jour., Vol. V, 1, 146.
5. Amer. Jour. Obstet. and Gyn., Vol. XXV, 144, 1933, Mason.
6. "Recent Advances in Forensic Medicine," Churchill, Smith and Glaister. 2nd edit., 1938.
7. Bull. Instit. Med. Lab. Technology, Vol. XII, No. 6, 1946, Scott.
8. Bull. Path. and Bacteriological Lab. Assistants Assoc., Vol. VII, No. 4, 33, 1941, Taylor.
9. Police Jour., Vol. XIII, 2, 162, 1940, Forbes.
10. R. v. Hare (1934), Police Jour., Vol. VII, 3, 263.
11. "The Sexual Perversions and Abnormalities," Allen, 1940, Oxford Univ. Press.
12. Med.-Leg. Rev., Vol. VIII, 2, 121, 1940, Lord Dawson.
13. R. v. Jacobs (1817), Russ. and R., 231.
14. R. v. Wiseman (1718), Footes Rep., 91.
15. R. v. Jellyman (1838), 8 C. and P., 604.
16. Brit. Med. Jour., Oct. 4, 525, 1947, Taylor.

CHAPTER XV

LUNACY IN ITS MEDICO-LEGAL ASPECTS

ALTHOUGH the statutory enactments dealing with lunacy are mainly administrative in character, it is necessary for his intelligent and legal conduct that the practitioner of medicine should be familiar at least with those provisions by which his relations to the person of unsound mind are regulated. His relationships with the public will doubtless bring him sooner or later into contact with cases of idiocy, of inebriety, or forms of lunacy or mental unsoundness, and he may be required: (1) to certify mental unsoundness; (2) to pronounce an opinion as to the mental capacity of a prisoner to instruct counsel for his defence, or to plead to a given charge; (3) to pronounce an opinion regarding testamentary capacity; or (4) to certify in other contingencies, such as in the appointment of a curator to a person who, owing to mental unsoundness, is unable to manage his own estate.

PROCEDURE IN ENGLAND RESPECTING LUNATICS

Procedure for detention of an insane person in an asylum.

This is to be found in the **Lunacy Acts of 1890-1911.** The procedure depends upon:—

Whether the person is living with relations.

Whether the person is under proper care or control, is being cruelly treated or neglected by relatives or those in charge of him, or is wandering at large.

Procedure when the person is living with relations.

There are three ways in which his detention may be effected:—

By Judicial Order on Petition.

By an Urgency Order.

By an Inquisition in Lunacy.

By Judicial Order on Petition.

This is the method most commonly adopted, and consists in presenting to the Judicial Authority the following documents:—

I. A petition by the wife or husband, or parent, or other relative of the person named. If by any other person than a near relative,

where nearer relatives are living, the reason why it is presented by the petitioner and not by the nearer relative must be stated. The petitioner must be at least twenty-one years of age. The petition must contain the following particulars :—

Full name ; age ; occupation ; postal address of petitioner.
Full name ; occupation ; address of insane person.
Relationship of petitioner to insane person ; or, if not related, the reason for his signing the petition.
A declaration that petitioner is not related to, or connected with, either of the persons signing the certificates of lunacy, in the relationships of husband, father, father-in-law, son, son-in-law, brother, brother-in-law, partner, or assistant ; or, in the case of a female petitioner, in the opposite relationships.
An undertaking by the petitioner to visit the insane person, or to have someone appointed for the purpose, once every six months while in hospital.

The Judicial Authority is a County Court Judge, a Stipendiary Magistrate, or one of the Justices of the Peace appointed annually for the purpose by the Justices of the County concerned.

II. A statement of particulars with reference to the history and condition of the insane person. If the insane person has already been received into an asylum under an Urgency Order, the date on which the order was presented must be given.

III. Two medical certificates on separate forms.

In each certificate the name, designation, and address of the person must be given ; a declaration that the signator is a registered medical practitioner ; the date upon which his examination was made, and the address at which it was made ; a statement of facts indicative of insanity observed by the signator at the examination ; similar facts observed by others, whose full description must be such that their identity may easily be established ; a statement of the bodily health of the insane person, with reference to fitness for removal ; a declaration that the signator has first read Section 317 of the Lunacy Act, 1890, before granting the certificate ; and the signature, the date of signature, and the address of the signator.

IV. The Reception Order, which is signed by the Judicial Authority, who, before signing, must :—

Be satisfied with the evidence contained in the medical certificates ; or,
Appoint a date within seven days of the presentation of the petition for its consideration ; at which diet he may, however, adjourn consideration until some subsequent date, within fourteen days of the date of the presentation of the petition ; or, he may
Visit the alleged lunatic.

When the Reception Order is signed it becomes the Order for the detention of the patient in the place named in the Order, and

permits the necessary steps to be taken for the removal of the patient.

In the event of the Judicial Authority being dissatisfied with the evidence of insanity and dismissing the petition, and another petition being presented as to the same allegedly insane person, the person presenting such other petition must :—

In making out the new petition, state that a previous petition had been dismissed, and give the reasons for its dismissal ;

Procure from the Lunacy Commisioners a copy of the statement of the reasons for dismissing the previous petition, and present this with the new petition.

By an Urgency Order.

This method is adopted where, either for the welfare of the alleged lunatic, or for the public safety, it is expedient to place the insane person under care and treatment without delay. The Urgency Order must be signed by a near relative ; it must be accompanied by a statement of particulars and one medical certificate. The Order may be made before or after the medical certificate, or before or after a petition for a Reception Order is presented. If made before, the fact must be stated in the petition ; if after the petition has been presented, a copy must be sent forthwith by the petitioner to the Judicial Authority. An Urgency Order remains in force for seven days, or if a petition for a Reception Order is pending, then until the petition is finally disposed of. The person signing the Urgency Order must personally have seen the allegedly insane person within two days before signing.

By an Inquisition.

This method is used when difficulty is anticipated in dealing with the property of the insane person. The original procedure by this method was slow, cumbrous, and expensive. By the Lunacy Act of 1890, the procedure is easier, more rapid, and less expensive. All the necessary powers for granting to any named person full control of the property of any lunatic or person of weak mind, either from disease or age, are vested in a Judge of Lunacy.

When the person is not under proper care or control, or is cruelly treated or neglected or is wandering at large.

By Summary Reception Order (by virtue of the Lunacy Act 1890, as amended by the National Health Service Act, 1946). If a duly authorised officer of the local health authority—

(*a*) has reasonable ground for believing that a person in the area of the authority is a person of unsound mind and a proper person to be sent to a mental hospital ; and

(*b*) is satisfied there are no relatives or friends who intend and are able to take proceedings by petition for a Reception Order under the foregoing provisions of this Act ;

he shall, within three days, give notice thereof to a justice having jurisdiction in the place where the said person is.

A justice, upon receiving such a notice, shall by order require the officer giving the notice to bring the said person before him or some other justice having such jurisdiction, at such time, within three days of the receipt of the notice, and at such place as may be specified in the order.

A duly authorised officer of the local health authority or any constable, who has reasonable ground for believing that any person wandering at large in the area of the authority is a person of unsound mind, shall immediately apprehend and take the said person, or cause him to be apprehended and taken, before a justice.

Any justice, upon the information upon oath of any person that a person wandering at large within the limits of his jurisdiction is of unsound mind, may by order require any constable, or duly authorised officer of the local health authority for the area where the person is, to apprehend him and bring him before the justice making the order, or any justice having jurisdiction where the said person is.

The justice shall then call in a medical practitioner, and shall make such examinations and inquiries as he thinks advisable and if he is satisfied that the alleged lunatic is a proper person to be detained and the medical practitioner signs a certificate with regard to the lunatic, then the justice may by order direct the lunatic to be received and detained in the institution for lunatics named in the order.

Having considered the procedure in connection with the detention of an insane person, the method for his discharge will next be described. A patient detained in any institution for persons of unsound mind, or under care as a single patient, shall, if he is detained under a reception order made on petition, be discharged on a direction in writing by the person on whose petition the order was made or, in the event of this not being possible, by the person who made the last payment on account of the patient, or by the appropriate relative. If there is no person qualified to direct the discharge of a patient, the Board of Control may order his discharge.

Voluntary patients and detention without certification.

Persons may voluntarily desire their own detention in an institution to protect themselves against the risk of an attack of mental illness which they feel, or may think, is impending. In virtue of the Mental Treatment Act, 1930, they may do so by filling in a special form of application which is sent to the superintendent of the mental hospital or home to which they desire admission. No further formalities are necessary. The patient may be received into the charge of a person approved by the Minister of Health. Should the age of the patient be under sixteen, the application must be made by a parent or guardian, and be supported by a recommendation by the family doctor or by a doctor approved for

the purpose by the Minister of Health or by the local health authority.

Such a patient may be discharged from an institution upon giving three days' notice in writing of his desire to do so, but if under sixteen, the intimation must be made by a parent or guardian. Should the patient's condition become such that he is incapable of expressing his willingness or unwillingness to remain in the institution, he must be discharged within twenty-eight days provided he does not resume his capacity for expressing his willingness to remain or has been certified insane. If the Minister of Health is of opinion that a voluntary patient is unfit to remain on this understanding, he may order the person in charge either to discharge the patient or to take steps to deal with him either as a person of unsound mind or as a person who is likely to benefit by temporary treatment. There is also provision in the Act for persons suffering from mental ailments which are likely to improve with temporary treatment. In these cases, although the patient is incapable of expressing willingness or unwillingness to receive treatment, he may be detained, upon a written application signed by a relative to the person in charge of the hospital or by a magistrate's order, in an institution, hospital, or home. Certification is not necessary, but the application must be accompanied by medical recommendations from two doctors who have examined the patient, of whom one shall be a medical practitioner approved for the purpose by the Minister of Health while the other may be the patient's doctor. The medical examination may be made jointly or separately, and if the latter, the examinations must be made within five days of each other and such recommendations will only remain in force for a period of fourteen days following the last examination.

The period of detention shall not continue longer than six months, except when an early recovery seems reasonably probable, when the period may be extended by three months at a time, so that the total period does not exceed one year. Should the patient become mentally capable of expressing his willingness or unwillingness to continue treatment, he cannot be detained, against his wish, for a period longer than twenty-eight days from the date of the return of that capacity, unless again he loses that capacity.

Mental Deficiency Acts, 1913 and 1927, applicable to England (see p. 472).

PROCEDURE IN SCOTLAND RESPECTING LUNATICS

The statutory enactments dealing with lunacy in Scotland are the **Lunacy (Scotland) Act, 1857,** the **Lunacy (Scotland) Act, 1862,** and the **Lunacy (Scotland) Act, 1866,** and it is necessary that the practitioner should be acquainted with certain of the provisions of these Acts.

The Lunacy (Scotland) Act, 1857.

By section 35 it is provided that every medical person, that is, every person registered as a practitioner in medicine or surgery, under the Medical Act, 1858, who signs any certificate under or for the purposes of the Act shall specify therein the facts upon which he has formed his opinion that the person to whom such certificate relates is an insane person, an idiot, or a person of unsound mind, and shall distinguish in such certificate facts observed by himself from facts communicated to him by others. The section further provides that no person shall be received into any asylum or house under any certificate which purports to be founded only upon facts communicated by others. Any person granting a certificate without having seen and carefully examined the person to whom it relates or granting a false certificate renders himself liable to severe penalties (section 38).

Section 71 prohibits unqualified persons from practising under the Act and makes it incompetent for any medical person who has any pecuniary or patrimonial interest or concern with or in any asylum or house, or any co-partnership or participation of profits with any superintendent of any asylum or house, or whose father, brother, or son is such superintendent, to practise or to be employed, or to grant certificates under the Act, but does not prevent any medical person so practising, being employed, or granting certificates with reference to any asylum or house where no such interest or relationship exists. The section further provides that nothing therein contained shall prevent the medical officer of a district asylum from granting certificates with reference to any lunatics of the district to which the asylum belongs.

Section 87 makes provision for procedure in cases in which insanity stands in bar of trial, and section 88 for cases of lunatics acquitted of a criminal charge on the ground of insanity.

Section 92 empowers the Sheriff to authorise the liberation of a lunatic on production to him by a relation or others of a certificate of two medical persons, approved of by him, certifying the recovery of the lunatic, or bearing that he may without risk of injury to the public or himself be set at large. The section further authorises the General Board of Control in like manner to order the liberation of any lunatic on being satisfied, by the certificate of two medical persons whom they think fit to consult, of the recovery or sanity of the lunatic. It will be noted that under this section the Board's powers are limited to the case where the lunatic has entirely recovered. This restriction was removed by section 55 (5) of the Mental Deficiency and Lunacy (Scotland) Act, 1913, and the Board have now the same authority as the Sheriff.

The Lunacy (Scotland) Act, 1862.

By section 14 the Sheriff of any county in Scotland may grant an Order for the reception into and detention in any asylum, or house, of any lunatic, if such lunatic be resident or be found within

the county, or if the asylum or house mentioned in such Order be situate within such county ; but no Order shall be granted unless upon a petition subscribed by the party applying for the same, accompanied by a statement of particulars in the form of Schedule (C) annexed to the Act of 1857 (see p. 466), and setting forth the degree of relationship or other capacity in which the petitioner stands to such lunatic, and also accompanied by certificates in the form of Schedule (D) annexed to that Act (see p. 467), bearing date within fourteen clear days next preceding the date of petition, under the hands of two medical persons having no immediate or pecuniary interest in the asylum in which the lunatic is to be placed. The Order of the Sheriff shall be in the form of Schedule (E) annexed to the Act of 1857 (see p. 468). No superintendent of any such public, private, or district asylum or house shall receive or detain any person without such an Order dated within fourteen clear days prior to the reception of such lunatic except in the case of a lunatic whose case is duly certified by one medically qualified person to be one of emergency, in which case the lunatic may be detained for a period not exceeding three days without an Order. By Section 4 of the Act of 1866 it is provided that it shall not be lawful for the medical superintendent, ordinary medical attendant, or assistant medical officer of any asylum, to grant a certificate of insanity for the reception of any lunatic, not a pauper lunatic, into such asylum, except a certificate of emergency under section 14 of the Act of 1862. Such a certificate cannot however be granted by the superintendent of a private asylum.

Section 15 provides that where any lunatic has been apprehended, charged with assault or other offence inferring danger to the lieges, or where any lunatic has been found in a state threatening danger to the lieges, or in a state offensive to public decency, the Sheriff of the county in which such lunatic has been apprehended or found, may upon application by the Procurator-Fiscal, authorised officer, under section 112, Lunacy (Scotland) Act, 1857, or other person, accompanied by a certificate from a medical person, bearing that the lunatic is in such state, forthwith commit him to some place of safe custody. The Sheriff shall then direct notice to be given in some local newspaper, of such commitment, and of his intention to inquire into the condition of such lunatic on a stated date, and at the same time direct notice of the application to be given to an authorised officer of the area of a local health authority within which the lunatic has been apprehended or found, and such further notice as he shall think fit. If the authorised officer does not within twenty-four hours undertake, to the satisfaction of the Sheriff, to make due arrangements for the safe custody of such lunatic, the Sheriff shall proceed to take evidence of the condition of such lunatic, and upon being satisfied that he is a lunatic, and in a state threatening danger to the lieges, or offensive to public decency, commit him to an asylum. An Order authorising the superintendent of the asylum to which the lunatic is to be committed to receive the lunatic, and authorising the transmission of the lunatic to such

asylum, shall be granted by the Sheriff in respect of every such commitment; and such lunatic shall be detained in such asylum until cured, or until caution is found for his safe custody, in which last case the Sheriff may, upon application to that effect and on being satisfied as to such caution, and the safety and propriety of such custody, authorise the delivery of the lunatic to the person so finding security.

Section 16 provides that on the application of the person at whose instance a lunatic is detained in an asylum, the Board may authorise his removal or liberation on probation without an Order of the Sheriff. Section 17 enacts that the superintendent of any asylum or house shall give intimation of the recovery of a lunatic to the person at whose instance such lunatic is detained, or in the absence of such person to the nearest known relative. If after fourteen days from the despatch of such intimation the person to whom the same was transmitted does not take steps for the liberation of such lunatic, the superintendent shall intimate the facts to the Board, who may direct inquiry into the circumstances, and if satisfied that the lunatic has recovered, or may safely be liberated without risk or injury to the public or himself, may order his discharge forthwith. To fulfil this section, it does not appear that complete recovery of the person is requisite, since all that is insisted upon is that the patient has so far recovered that he may safely be liberated without risk or injury to the public or himself.

The schedule which is in use under the Act of 1857 for taking the necessary steps for the detention of a patient in an asylum consists of the following parts :—

I. The petition to the Sheriff or his Substitute by the near relative of the alleged insane person for authorisation to convey such person to a given asylum, the date of which petition must be within fourteen clear days following the dates of the medical certificates.

II. The statement of particulars regarding the patient, which must be signed by the petitioner. The form used is that contained in Schedule (C) of the Lunacy Act, 1857, and is as follows :—

Form of Statement to be lodged with a Petition to the Sheriff for the Reception of a Lunatic.

Christian name and surname of patient at length.
Sex and age.
Married, single, or widowed.
Condition of life, and previous occupation (if any).
Religious persuasion (so far as known).
Previous place of abode.
Place where found and examined.
Length of time insane.
Whether first attack.
Age (if known) on first attack.
When and where previously under examination and treatment.
Duration of existing attack.

Supposed cause.
Whether subject to epilepsy.
Whether suicidal.
Whether dangerous to others.
Christian name and surname and place of abode of nearest known relative of the patient, and degree of relationship (if known), and whether any member of his family known to be or to have been insane.
Special circumstances (if any) preventing the insertion of any of the above particulars.

I certify, that to the best of my knowledge the above particulars are correctly stated.

Dated this day of One thousand nine hundred and . [Signature of person applying.]

In the printed Form of Petition in general use the word "lunatic" has been changed to the word "patient." It is most important that the definition of the expression "lunatic," given in section 1 of the Act of 1862, should be kept clearly in the mind of the practitioner. It reads as follows: "The word 'lunatic' shall mean and include every person certified by two medical persons to be a lunatic, an insane person, an idiot, or a person of unsound mind." These terms are not defined but mean nothing more than a person of unsound mind. In view of the definition of "idiot" in the Mental Deficiency and Lunacy (Scotland) Act, 1913, that term in the Act of 1862 appears to have been superseded (see p. 467).

III. Medical Certificate (No. 1), and Medical Certificate (No. 2).

The form of Medical Certificate given in Schedule (D) of the Lunacy Act of 1857, is as follows :—

I, the undersigned [*set forth the qualification entitling the person certifying to grant the certificate*], and being in actual practice as a [*physician, surgeon, or otherwise as the case may be*] do hereby certify on soul and conscience that I have this day at [*insert the street and number of the house (if any) or other like particulars*] in the County of separately from any other medical practitioner visited and personally examined A. B. [*insert designation and residence*], and that the said A. B. is a person of unsound mind, or an idiot, and a proper person to be detained under care and treatment, and that I have formed this opinion upon the following grounds, viz. :—

Facts indicating insanity observed by myself (*state the facts*).
Other facts (*if any*) indicating insanity communicated to me by others (*state the information and from whom*).

(Signed) [*Name and medical designation and place of abode.*]
Dated this day of One thousand nine hundred and .

Reception Order.

IV. The Order of the Sheriff, for transmission and reception of the lunatic, given in the form of Schedule (E) annexed to the Act of 1857 sets forth :—

His name and judicial office.

The fact that a petition and medical certificates, which state that the alleged lunatic is of unsound mind, and that he ought to be detained in an asylum, have been submitted to him.

Order for detention of the person named.

Date ; and signature of Sheriff.

This Order is addressed to the superintendent of the asylum, named in the Order, in which the person is to be detained under care and treatment.

Emergency certificate.

The lunatic, in cases where there are risks to his own safety, or to that of the public, may in virtue of section 14 of the Act of 1862 be committed upon one medical certificate. This section states : " provided that the superintendent of any Public, Private, or District Asylum may receive and detain therein, for any period not exceeding three days, and without any Order of the Sheriff any person as a lunatic, whose case is duly certified to be one of emergency by one medical person qualified as aforesaid." The certificate in this case is termed the Certificate of Emergency, and is accompanied by an application by a relative.

The Certificate of Emergency is not a scheduled certificate, but its terms are as follows :—

I, the undersigned [*set forth full name of certifier*], being [*set forth the qualifications of the certifier*] hereby certify on soul and conscience that I have this day at [*set forth particulars as to place of examination*] in the County of , seen and personally examined [*designate the patient and his residence*] and that the said person is of unsound mind and a proper patient to be placed in a mental hospital, and is in a sufficiently good state of bodily health, at this date, to be removed to the mental hospital at .

And I further certify that the case of the said person is one of emergency.

[Signature.]

Dated this day of One thousand nine hundred and .

As it is usually anticipated that a petition to the Sheriff for a Reception Order will be presented during the period the Certificate of Emergency is in force, it is customary, although not obligatory, for the doctor who signs the Certificate of Emergency also to complete the Medical Certificate given in Schedule (D) of the Lunacy Act of 1857 (see p. 467).

The Certificate of Emergency should be accompanied by the following additional document, which should be filled up by the applicant :—

I hereby request the superintendent of the [*name of mental hospital to be inserted*] to receive therein [*name of patient*] to whom the foregoing Certificate of Emergency refers.

Relationship or other capacity in which applicant stands to patient. .....

Signature and Address.....

.....

Date.....

In the case of a lunatic wandering at large, the Procurator-Fiscal initiates the procedure for his detention by instituting an inquiry in lunacy before the Sheriff, and the lunatic is committed upon the certificates of two medical practitioners who have been named by the Sheriff on the petition of the Fiscal.

Detention of involuntary patients in other than mental hospitals.

By virtue of the Lunacy (Scotland) Act, 1857, provision is made for the reception and detention of certain patients. This class of patient is composed of persons whose mental illness is unconfirmed and in whose instance it is considered expedient, with a view to recovery, that they should be temporarily detained. To secure this end such patients may be admitted on one medical certificate to a nursing home or private dwelling specially licensed for the purpose and the period of detention is limited to six months.

Voluntary patients.

Section 15 of the Act of 1866 makes provision for allowing persons to enter asylums voluntarily. It provides, that it shall be lawful for the superintendent of any asylum, with the previous assent in writing of the Board, which assent shall not be given without written application by the patient, to entertain and keep in such asylum, as a boarder, any person who is desirous of submitting himself to treatment, but whose mental condition is not such as to render it legal to grant certificates of insanity in his case. The only formality required is that the patient shall sign two letters, one addressed to the Board and the other to the medical superintendent of the institution to which he desires admission, stating that he wishes to place himself under care and treatment as a voluntary boarder. No such boarder shall be detained for more than three days after having given notice of his intention or desire to leave such asylum.

PROCEDURE IN SCOTLAND—MENTAL DEFICIENCY

Mental Deficiency and Lunacy (Scotland) Act, 1913.

The following provisions have a medico-legal bearing.

Section 1 defines defectives as—

Idiots ; that is to say, persons so deeply defective in mind from birth or from an early age as to be unable to guard themselves against common physical dangers.

Imbeciles ; persons in whom there exists from birth or from an early age mental defectiveness not amounting to idioey, yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so.

Feeble-minded persons or morons ; persons in whom there exists from birth or from an early age mental defectiveness not amounting to imbecility, yet so pronounced that they require eare, supervision, and control for their own protection or for the protection of others, or, in the case of children, that they by reason of such defectiveness appear to be permanently incapable of receiving proper benefit from instruction in ordinary schools.

Moral imbeciles ; persons who from an early age display some permanent mental defect coupled with strong vicious or criminal propensities on which punishment has had little or no deterrent effect.

Section 3 describes the circumstances under which defectives are dealt with. Sub-section 1 enacts that a person who is a defective shall be subject to be dealt with, if in addition to being a defective, he is a person :—

who is found neglected, abandoned, or without visible means of support, or cruelly treated ; or

who is found guilty of any offence punishable in the case of an adult with imprisonment, or who is ordered or found liable to be ordered to be sent to an industrial school ; or

who is undergoing a sentence of imprisonment (except under civil process) or is undergoing detention in a place of detention by order of court, or in a reformatory or industrial school or in an inebriate reformatory or in any asylum or other lawful place of detention for lunatics or a state mental hospital ; or

who is an habitual drunkard within the meaning of the Inebriates Acts ; or

in whose case notice has been given by the Education Authority ; or

who, being a woman and unmarried, is in receipt of national relief at any time during her pregnancy or at the time of birth of her child ; or

who, during any consecutive period of six months in the year immediately preceding the commencement of proceedings under this Act, has been in receipt of assistance through the National Assistance Act, 1948, in accommodation provided under Part III, Local Authorities Services, on three or more than three occasions.

In the case of defectives under the age of sixteen, who are capable of education, the local authority concerned is the Education Authority, and in the case of defectives incapable of education or of defectives over sixteen years of age, the local authority is the local health authority within the meaning of the National Health Service (Scotland) Act, 1947.

Section 6 provides that :—

A judicial order may be obtained upon an application to the Sheriff by petition of any relative or person contributing towards the support of the alleged defective, or by the local authority concerned, or by the Procurator-Fiscal in cases reported to him ;

The petition should be accompanied by two medical certificates, signed not more than one month previous to presentation of petition, one to be signed by a practitioner approved for the purpose by the Board, and the other, whenever practicable, by the usual medical attendant of the defective, if any, and by a statutory declaration signed by or on behalf of the petitioner and by at least one other person, who may be one of the medical certifiers, stating that (*a*) the person to whom the petition relates is a defective within the meaning of the Act, and the class of defectives to which he belongs, (*b*) that that person is subject to be dealt with under the Act and the circumstances which render him so subject, and (*c*) whether or not a petition under this section or a petition for an Order under the Lunacy Acts has previously been presented relating to that person, and if so, the date thereof and the result of the proceedings thereon.

Section 46 deals with the protection of defectives from acts of sexual immorality or procuration.

Any person who unlawfully and carnally knows, or attempts to have unlawful carnal knowledge of, any woman or girl who is a defective under the Act, under care or treatment in an institution or certified house, or placed out on licence therefrom, or under guardianship under the Act, under circumstances which do not amount to rape, but which prove that the offender knew at the time of the commission of the offence that the woman or girl was under such care or treatment, or so placed out or under guardianship, shall be guilty of a crime or offence.

If on the trial of an indictment for rape the jury are satisfied that the accused is guilty of an offence, as stated in the above paragraph, but are not satisfied that he is guilty of rape, the jury may acquit him of rape and find him guilty of that offence.

Section 56 extends the same protection from acts of sexual immorality to women and girls who are lunatics duly certified under the Lunacy Acts as to women and girls who are defectives under this Act.

Section 10 of the Criminal Law Amendment Act, 1885, shall apply in the case of a woman or girl who is a defective in the same manner as it applies in the case of a girl who is under the age of sixteen years (see p. 425).

In 1946, at a sitting of the Glasgow High Court of Justiciary, William Martin was charged with the murder of a man by shooting and with the attempted murder of another man. The accused was not insane and was certified as fit to plead. He was found not guilty of attempted murder, but guilty of culpable homicide. The prisoner was found defective within the meaning of the Mental Deficiency and Lunacy (Scotland) Act, 1913. In his summing-up at the trial, Lord Mackay expressed the view that the 1913 Act applied only to crimes of punishment by penal servitude, and that, if the jury found Martin guilty of murder it could not be applied. If it was applicable in such cases, he said, we would have the dangerous situation of our institutions crowded with feeble-minded persons convicted of murder. The charge was deserted simpliciter by Lord Mackay at a continued diet in the High Court in Edinburgh. It was stated by the Lord Advocate that an instruction had been issued to the Procurator-Fiscal, Glasgow, to take certain steps under the Mental Deficiency Act. These steps had been taken and a warrant for Martin's detention under the Act was placed before his Lordship. In making an Order to desert the diet, Lord Mackay said that the jury, in order to found the subsequent proceedings, had to make a finding which was, in his opinion, not a verdict of guilty, or a verdict of any sort, except a verdict of fact. That finding, he said, should not anywhere be treated as a finding of guilt against Martin, even of culpable homicide. Martin was committed to an institution for mental defectives under a judicial order granted at Glasgow Sheriff Court. It is believed that never before have the provisions of the Mental Deficiency and Lunacy Act been involved in a murder trial.

PROCEDURE IN ENGLAND—MENTAL DEFICIENCY

The law concerning mental defectives in England is governed by the **Mental Deficiency Act, 1913.** This act was similar in its provisions to the Scottish Act, but was amended by the **Mental Deficiency Act, 1927.** This latter Act provides that the mental defectiveness need not have existed from birth or from an early age, but that the condition of arrested or incomplete development of mind may have arisen during the first eighteen years of life, whether from inherent causes or induced by disease or injury. So far as England is concerned, mental deficiency means a condition of arrested or incomplete development of mind existing before the age of eighteen years, whether arising from inherent causes or induced by disease or injury.

Mental tests of intelligence.

These special tests are based on the work of Binet and Simon which has been modified by Terman [1] and others. A number of authorities hold the view that the results of intelligence tests should never be used alone in assessing the degree of mental defectiveness. Henderson and Gillespie [2] express the view that the capacities and resources of a person can be assessed only by all-round observation, by careful history-taking, and by an examiner of wide experience of morbid as well as normal reactions.

MEDICAL CERTIFICATES OF LUNACY

From what has preceded, it will be obvious that the medical certificate plays a most important part in a petition for the detention of a person of unsound mind. Since certification forms the pivot upon which the whole procedure for the detention of an insane person turns, it is imperative that all members of the medical profession should not only make themselves conversant with the signs and symptoms of insanity and other allied mental conditions, but that they should exercise the duty of certification in the most conscientious and painstaking manner. It is therefore necessary to describe the legal essentials associated with this duty.

What does the law demand of the medical practitioner who signs a certificate of lunacy?

In England:

He must not be related to the petitioner, for example (where petitioner is a man), as father, father-in-law, son, son-in-law, brother, or brother-in-law.

He must not be a partner or assistant of the other signator.

He must not be the person under whose charge the alleged lunatic is to be placed or the usual medical attendant of an insane person in a "single-patient" house, if he has signed the medical certificate upon which the Reception Order or Urgency Order is based.

He must not be a person interested in the payments on account of the patient, or in the profits of the institution in which the alleged lunatic is to be placed.

The examination upon which the certificate is founded must be made within a period of seven clear days before the presentation of the petition, or within two clear days, where an Urgency Order is used.

It must be made separately from any other practitioner.

The terms of the certificate must be definite and clearly expressed, and it must contain sufficient evidence of the insanity of the person named in it to be convincing to any average intelligent individual.

One of the certificates should be given by the usual medical attendant of the alleged insane person, or, if not, the reason should be stated.

Before granting the certificate, the signator must have read over Section 317 of the Lunacy Act of 1890. ["Any person who makes a wilful misstatement of any material fact in any medical or other certificate, or in any statement or report of bodily or mental condition under this Act, shall be guilty of a misdemeanour."]

The foregoing extract from this section is printed at the end of the Form of Medical Certificate.

In England the two blank certificates are printed on separate schedules.

In Scotland:

He must not be related to the superintendent of the asylum or house into which a patient is to be placed, as son, brother, or father.

He must not have any pecuniary or patrimonial interest or concern with or in the asylum or house into which a patient certified by him is to be placed, or have any co-partnership or participation of profits with the superintendent of any such asylum or house.

He must examine a patient before granting a medical certificate of his lunacy within fourteen days preceding the date of petition to the Sheriff, and he must be in actual practice.

His certificate must show that, on the date and at the place named, he personally and separately, from any other medical practitioner, examined the person named in the certificate.

The certificate must specify the facts on which an opinion of insanity has been formed, (*a*) as ascertained by himself from examination of the lunatic, and (*b*) as ascertained from other persons named and designated.

The certificate must be attested "on soul and conscience." In Scotland both blank certificates are printed on the same schedule.

What is meant in law by an examination made "separately from any other practitioner"?

Obviously, the intention of the legislature is that the statements made by the certifiers, since they have the force of statements made upon oath, should have all the cumulative force of concurrent testimony by each examiner separately noting facts which are indicative of unsound mind in the person examined. The absence of, or the semblance of the absence of, a separate examination by the medical certifiers would, therefore, vitiate each certificate. It is, however, quite a common practice in such cases, for a medical practitioner to call in a specialist and for both to meet at the house

of the patient, when the previous history and present condition of the patient are fully explained to the specialist before he makes his examination.

It might be held illegal for both doctors to sign certificates under such circumstances, even although the examination of the patient by one has been immediately followed by an examination made by the other. It is, therefore, advisable for the consultant to examine the patient first and the usual medical attendant to do so later on the same day or on the following day. Such procedure will conform to both the spirit and the letter of the law.

The medical certificate of lunacy is a legal document, and in it must be embodied the identity of the signator and also of other persons. Technical error arising from want of identity will invalidate the certificate. For example, if the signator omitted to give the necessary identity of the persons who have provided him with the "facts observed by others," the certificate would be null and void, since upon the facts so given he has partly established his opinion of the insanity of the person named in the certificate.

Probably there is no more common fault in such certificates than imperfectly or improperly expressed data upon which the opinion of insanity has been determined, or from which it has been deduced. The certifier should always remember that it is insufficient that he alone is convinced of the insanity of the person, since the facts which he has observed of the insanity must satisfy the mind of the Judicial Authority and convince him that the patient, whom he has never seen, is insane. It is not enough to say that the person is suffering from delusions which the certifier may know are delusions: he must state that he has inquired into the truth or reality of the patient's statements and has proved them to be unfounded.

No statements which fall within the sphere of verification should be used as evidence of insanity unless and until they have been proved to be non-existent, and this should be stated in the certificate. Language which is capable of conveying to different minds other reasonable meanings than that intended should not be used in the certificate. Such words as "incoherent," "irrational," "morose," "wild," or such phrases as "curious in manner," or, "behaves absurdly," should be avoided, since there are no standards by which the average mind can grasp the precise meaning intended to be conveyed.

The certifier should, therefore, state facts which by themselves exemplify the condition, and should cite instances of incoherency, irrationality, moroseness, wildness, absurdity of behaviour, or singularity of manner. This enables any other person to infer from the stated facts the qualities of mind, devious from normal, which the patient exhibits. In the certificate, the names of persons should be used instead of pronouns. Plain, short sentences are preferable to long and complicated ones, and permit the facts to emerge more easily. The certifier should satisfy himself that he has acted in good faith, has shown reasonable skill and care, and that the opinion expressed can be judged an honest and careful one.

Protection of practitioner against wrongful certification.

In England.

Protection to medical practitioners against actions arising out of certification of lunacy was recognised by the **Lunacy Act, 1890.** It enacted that :—

" A person who before the passing of this Act has signed or carried out or done any act with a view to sign or carry out an order purporting to be a Reception Order, or a certificate that a person is of unsound mind, and a person who, after the passing of this Act, presents a petition for any such order, or signs or carries out or does any act with a view to sign or carry out an order purporting to be a Reception Order, or any report or certificate purporting to be a report or certificate under this Act, or does anything in pursuance of this Act, shall not be liable to any civil or criminal proceedings, whether on the ground of want of jurisdiction or on any other ground, if such person has acted in good faith and with reasonable care.

" If any proceedings are taken against any person for signing or carrying out, or doing any act with a view to sign or carry out any such order, report, or certificate, or presenting any such petition as in the preceding sub-section mentioned, or doing anything in pursuance of this Act, such proceedings may, upon summary application to the High Court or a Judge thereof, be stayed upon such terms as to costs and otherwise as the Court or Judge may think fit, if the Court or Judge is satisfied that there is no reasonable ground for alleging want of good faith or reasonable care."

The Mental Treatment Act, 1930.

This Act affords increased protection to members of the medical profession against claims alleging negligent certification in lunacy proceedings. No civil or criminal action can be brought against a doctor, in this respect, without leave of the High Court, which leave is not granted unless the Court is satisfied that there is a substantial ground for alleging bad faith or lack of reasonable care. The onus of proof thus now rests upon the plaintiff.

In Scotland.

Similar provision to the terms expressed in the first sub-section of the Lunacy Act of 1890, as applicable to England, is provided by **section 73 of the Mental Deficiency and Lunacy (Scotland) Act, 1913.**

While, however, such enactments as the foregoing will not prevent the raising of actions, the practitioner who has conformed to the requirements of the law may rest assured that the law will protect him.

TESTAMENTARY CAPACITY

The duty of giving evidence as to the testamentary capacity of a testator usually falls upon medical witnesses, although it is not confined to them.

In the case of a disputed will in which the ground of action is the incapacity of the testator to make such will, doubtless the evidence of the medical attendants of the testator would, as a general rule, prove of the greatest value. It is therefore advisable for practitioners, in order to maintain an attitude free from the semblance of bias or partiality, to avoid being witnesses to documents of a testamentary nature.

In probate suits, the ultimate burden of proving testamentary incapacity rests on the party contesting the will.

For a will to be valid, from the medico-legal point of view, the testator must be in possession of a "sound and disposing mind." This state of mind, however, may be quite consistent with certain departures from sanity, and it may be inconsistent with an absence of insanity in the ordinary sense, where, for example, by reason of degenerative changes in the brain of an old person, the mind has become weakened and facile. If the legal definitions of what constitutes a sound and disposing mind are reviewed, it will be found that probably one of the best definitions is that given by Lord Cockburn in the case of Banks v. Goodfellow.[3] He stated that if the human instincts and affections, or the moral sense, became perverted by disease, if insane suspicion or aversion took the place of rational affection, if reason and judgment were lost and the mind became a prey to insane delusions, calculated to interfere with and destroy its functions, and to lead to a testamentary disposition due only to their baneful influence, then the will was bad. If, however, the testator had an insane delusion, but it did not affect the general faculties of the mind and had no effect on the will, the mere fact that he had it did not take away his testamentary capacity.

The law admits that there may be sufficient intelligence remaining, although reduced from an average standard, for the legal exercise of the disposition of an estate. This is well exemplified in many legal decisions in cases in which the testators were the acknowledged subjects of delusions, sometimes of a most definite character. Further the reasonable nature of the document is an important element. It is therefore apparent that the law allows considerable latitude in the interpretation of the mental state with reference to testamentary capacity.

A lucid interval is not necessarily a complete restoration to mental vigour previously enjoyed nor is it merely the cessation or suppression of the symptoms of insanity. The burden of proof of a lucid interval rests on the party asserting its existence.

The testator should have a sound and disposing mind either at the time of giving instructions for the preparation of the will or at the actual moment of its execution. The law does not demand that

this state of mind should exist on both these occasions, but in the case of a holograph will it must exist at the time when the will is made and signed by the testator. The testator must fully comprehend :—

that he is disposing of his property to the person or persons named ;
the extent of his possessions of which he is disposing ;
the nature and effect of his act in its bearings on the claims of others, which should have had his consideration.

It is also necessary that :—

the instructions for the preparation of the will have been given, and its subsequent execution made voluntarily and without any undue influence by any other person.

Thus the question which the medical practitioner has to answer is, whether the testator is, or was, in a mental condition, by reason of natural incapacity, old age, illness, or insanity, such as to render him incapable of understanding that he is, or was, disposing of his property to a certain person or persons, or of knowing the extent of his estate, and of understanding the nature and effect of his act with relation to the natural claims of those excluded from the will. The only means available to the practitioner for answering the question are afforded by the behaviour and statements of the testator, either in connection with the act of testamentary disposition, or with reference to the acts of his daily life and his contacts with those around him. Even with evidence of mental impairment in one direction, it still remains to be considered whether the degree and character of the impairment prejudices the ability of the testator to dispose of his estate in a proper fashion. The testamentary capacity of an aphasic person can only be measured by the character and extent of the brain lesion and its effects upon the receptivity of the mind.

The Inheritance (Family Provisions) Act, 1938, which is applicable only to England, authorises husbands, wives, unmarried daughters, infant sons, or other incapacitated children of the deceased to apply to the courts to alter the will where it is found not to have made " reasonable provision for their maintenance." Such applications must not raise the issue of the soundness of the testator's mind. Despite this judge-made ruling, the absence of a " sound and disposing mind " can still be relied upon as a ground for upsetting the will altogether. Thus in England, as in Scotland, it is not possible for a testator completely to disinherit those standing in close family relationship.[4]

Management of property of person mentally incapacitated.

When a person, by reason of mental incapacity, is precluded from managing his affairs efficiently, a legal application to the court may be made by relatives or friends of that person for the appointment of someone to administer his affairs. Frequently such mentally affected persons are not confined to an asylum.

Procedure in England.

By the Lunacy Act, 1908, a Judge in Lunacy may appoint a Receiver. The application must be supported by two medical certificates which embody the reason for the view expressed that the person concerned is incompetent to manage his affairs, or of giving direction for their management, on account of mental infirmity whether from disease or age.

Procedure in Scotland.

The application for the appointment of a Judicial Factor or Curator Bonis is made either by petition to the Court of Session, or, where the yearly value of the estate does not exceed £100, by application to the Sheriff. The person concerned must be mentally unfit to manage his affairs, but it is not necessary that he be absolutely insane. Prima facie evidence of incapacity is the production of two medical certificates signed by two practitioners, "on soul and conscience," to the effect that the person is incapable of managing his affairs and stating specifically the cause and its duration, copies of which are appended to the principal petition or application. One of the certificates at least should be given by one unconnected with the asylum should the person be detained. The certificates should include the date and place of interviewing such person, and should be dated within the period of one month prior to the presentation of the application. The petitioner usually makes a nomination for a Curator Bonis and the court may accept the nominee and appoint him as Curator. The petition or application contains the name of the person suggested for appointment as Judicial Factor or Curator Bonis, but if the nomination is objected to on the ground of adverse interest or for other reason, and such objection is upheld, the court may appoint some other suitable party. The effect of the appointment is to put the Curator in such person's place and to supersede the latter in dealing with the estate.

CRIMINAL RESPONSIBILITY

Before discussing the medico-legal aspects of criminal responsibility in relation to mental states, it is perhaps expedient to review the law in relation to criminal offences committed by insane persons.

Law relating to criminal offences and insanity.

England.

The statutory enactments connected with insanity as it may emerge in the case of persons charged with crime or offences, and which are of medico-legal interest, comprise :—

Criminal Lunatics Act, 1880.
Trial of Lunatics Act, 1883.
Criminal Lunatics Act, 1884.
Criminal Appeal Act, 1907.

Criminal Lunatics Act, 1800.

The principal provision of this Act is contained in section 2 which provides that if any person indicted for any offence is insane, and is found upon arraignment to be so by a jury lawfully impanelled for that purpose, so that he cannot be tried upon such indictment, or if upon the trial of any person so indicted he shall appear to the jury to be insane, it shall be lawful for the Court before whom he is brought to direct such finding to be recorded, and thereupon to order him to be kept in strict custody until His Majesty's pleasure is known. If any person charged with any offence is brought before any Court to be discharged for want of prosecution, and he appears to be insane, it shall be lawful for the Court to order a jury to be impanelled to try the sanity of such person ; and if the jury so impanelled find that he is insane, the Court may order him to be kept in strict custody until His Majesty's pleasure is known.

Trial of Lunatics Act, 1883.

Section 2 of this Act provides that where in any indictment or information any act or omission is charged against any person as an offence, and it is given in evidence on his trial that he was insane, so as not to be responsible according to law for his actions at the time the act was done or omission made, and it appears to the jury that he did the act or made the omission charged, but was insane at the time when he did or made the same, the jury shall return a special verdict to the effect that the accused was guilty of the act or omission charged against him, but was insane at the time when he did the act or made the omission. Where such a special verdict is found, the Court shall order the accused to be kept in custody as a Broadmoor patient in such place and in such manner as the Court shall direct till His Majesty's pleasure is known, and it shall be lawful for His Majesty, from time to time, to give such order for the safe custody of such person during pleasure in such place and in such manner as to His Majesty may seem fit.

Criminal Lunatics Act, 1884.

This Act consolidated and amended the law relating to Broadmoor patients. Its principal provisions are to be found in sections 2. 3, and 4.

Section 2 provides that where it appears to any two members of the visiting committee of a prison that a prisoner in such prison, not being under sentence of death, is insane, they shall call to their assistance two legally qualified medical practitioners, and such members and practitioners shall examine the prisoner and inquire as to his insanity, and after such examination and inquiry may certify in writing that he is insane.

In the case of a prisoner under sentence of death, if it appears to a Secretary of State, either by means of a certificate signed by

two members of the visiting committee of the prison in which such prisoner is confined, or by any other means, that there is reason to believe such prisoner is insane, the Secretary of State shall appoint two or more legally qualified medical practitioners, who shall forthwith examine the prisoner and inquire as to his insanity, and after such examination and inquiry such practitioners shall make a report in writing to the Secretary of State, and they, or the majority of them, may certify in writing that he is insane.

By section 3 it is provided that where it is certified by two legally qualified medical practitioners that a person being a Broadmoor patient (not being a person with respect to whom a special verdict has been returned that he was guilty of the act or omission charged against him, but was insane at the time when he committed the act or made the omission) is sane, a Secretary of State, if satisfied that it is proper so to do, may by warrant direct such person to be remitted to prison to be dealt with according to law.

Section 4 enacts that the superintendent of any asylum or other place in which any Broadmoor patient is detained shall make a report not less than once a year to a Secretary of State giving such particulars as such Secretary may require of the condition and circumstances of every Broadmoor patient in such asylum or place, and such Secretary shall at least, once in every three years, take into consideration the condition, history, and circumstances of such Broadmoor patient, and determine whether he ought to be discharged or otherwise dealt with, and further that where a Broadmoor patient is conditionally discharged, a report of his condition shall be made to a Secretary of State by such superintendent at such times and containing such particulars as may be required by the warrant of discharge. (The term " Broadmoor patient " is employed in the Criminal Justice Act, 1948, to describe persons of unsound mind who have been ordered to be kept in State Mental Hospitals in strict custody until His Majesty's pleasure be known.)

Procedure at trial.

The issue to be tried is the state of the prisoner's mind at the date of arraignment, and evidence as to this must be led. The jury may find that the prisoner is insane, and when this is so, the judge will order him to be confined during His Majesty's pleasure.

The jury may, on the other hand, find that the prisoner is sane, and in this event the prisoner must plead to the indictment.

Should a prisoner who is mentally unfit to plead be placed upon trial in error, and should his unfitness to plead become apparent in the course of the trial, the judge may discharge the jury and order him to be detained during His Majesty's pleasure.

The question of the prisoner's sanity may also arise from the evidence given at a trial. In such a case the issue is not whether the prisoner is sane or insane at the time of the trial, but whether he was sane at the time when the crime charged against him was committed.

By the Trial of Lunatics Act, 1883, it is enacted that, in such cases, when it is found by the jury that the prisoner was insane when the crime was committed, the jury will return a verdict of guilty but insane. The judge will then order the prisoner to be detained during His Majesty's pleasure. The term, "to be kept in strict custody until His Majesty's pleasure shall be known," usually means detention for life, consequently insanity as a defence is rarely put forward in other than charges of murder.

Criminal Appeal Act, 1907.

This Act by section 5 provides that if on any appeal it appears to the Court of Criminal Appeal that, although the appellant was guilty of the act or omission charged against him, he was insane at the time the act was done or the omission made so as not to be responsible according to law for his actions, the court may quash the sentence passed at the trial and order appellant to be kept in custody as a Broadmoor patient under the Trial of Lunatics Act. 1883, in the same manner as if a special verdict had been found by the jury under that Act.

In the case of R. v. Dashwood,[5] the question of the defence of insanity was raised on appeal. The Court of Appeal pointed out that it had no power to order a new trial by jury and that it would require some very remarkable circumstances to justify the Court in turning itself into a tribunal of fact to decide such an important matter as insanity when that matter had been deliberately withheld from the consideration of the jury by the person who was asking them to embark upon the inquiry. After his conviction the accused changed his mind and seemed to have been only too willing to rely on the defence of insanity. To grant the application before them would be to open the door to every person charged with any serious crime before a jury to instruct his counsel to run such defence as he thought right at the trial, and if that defence failed, to come to the Appeal Court and ask that his state of mind should be inquired into, and to invite the Court to say that the proper verdict would be that he was insane. In their view, the Act which defined the powers of the Court precluded them from doing such a thing.

Scotland.

The statutory enactments, of medico-legal interest, are the **Lunacy Act of 1857 and the Criminal and Dangerous Lunatics (Scotland) Amendment Act, 1871.**

Lunacy Act, 1857.

The relevant sections of this Act are sections 87 and 88.

Section 87 provides that where any person charged under any indictment or criminal libel with the commission of any crime shall be found insane, so that he cannot be tried upon such indictment, or if upon the trial of any person so indicted he shall appear to the jury charged with such indictment or criminal libel to be insane, the

Court before whom he is brought shall direct a finding to that effect to be recorded, and thereupon the Court shall order such person to be kept in strict custody until His Majesty's pleasure is known.

Section 88 provides that in all cases where it is given in evidence upon the trial of any person charged under any indictment or criminal libel with committing any crime or offence that he was insane at the time of committing such crime or offence, and he is acquitted, the jury shall be required to find specially whether he was insane at the time of committing the crime or offence, and to declare whether he was acquitted by them on account of such insanity. If they shall so find and declare, the Court before whom such trial takes place shall order such person to be kept in strict custody, in such place and in such manner as to the Court shall seem fit, until His Majesty's pleasure shall be known.

Procedure at trial.

A plea of insanity in bar of trial must be stated at the pleading diet held in the Sheriff Court of the district in which the prisoner is confined. If present insanity is established before trial, the judge, without a jury, after hearing medical evidence, will find that the accused cannot be tried, and will order him to be confined during His Majesty's pleasure.

If the jury after trial consider that the accused was insane at the time of the commission of the offence charged against him, they will acquit him on the ground of insanity. If at any stage of the trial they arrive at the conclusion that the accused is insane, a finding to that effect is recorded. When the acquittal is on the ground of insanity, the accused is ordered to be detained in custody during His Majesty's pleasure.

It will be noted, therefore, that in Scotland a plea of insanity may be raised at two separate stages in a criminal charge, firstly, in bar of trial, and secondly, in bar of sentence. Insanity in bar of trial is pleaded where the accused is precluded, by insanity, from instructing his defence, whereas the plea of insanity in bar of sentence is put forward when the accused is proved insane at the time of the commission of the crime.

The Criminal Justice (Scotland) Act, 1949, empowers courts to adjourn the hearing of a case for the purpose of enabling inquiries to be made into the mental condition of a person charged with a criminal offence.

Criminal and Dangerous Lunatics (Scotland) Amendment Act, 1871.

This Act deals principally with the detention and disposal of state mental patients and provides by section 2 that where a person charged under indictment is ordered by the Court to be kept in custody during His Majesty's pleasure, any order which His Majesty shall be pleased to issue in relation to the custody of such person

may be renewed and varied from time to time. The section further provides that it shall be lawful for His Majesty, by a Secretary of State, to authorise on terms, the liberation from custody in prison or elsewhere, of any person who has been ordered to be kept in custody, and that if any of the conditions of such liberation are broken, any principal Secretary of State may direct such person to be taken into custody again and revert in all respects to the same position as he was in at the time of liberation. (The term "state mental patient" is employed in the Criminal Justice (Scotland) Act, 1949, to describe persons of unsound mind who have been ordered to be kept in State Mental Hospitals in strict custody till His Majesty's pleasure be known.)

The legal aspects of criminal responsibility.

The law presumes that every person is sane and accountable for his actions until the contrary is proved. The burden of proving otherwise rests upon the person setting up the defence of insanity. There are many people tainted with insanity who are, nevertheless, influenced by the same motives as ordinary persons and to whom the fear of punishment has a sufficiently strong deterrent effect. For such individuals the defence of insanity would be unsuccessful.

The present law on the defence of insanity is based upon the answers given by fourteen judges, to certain questions put to them on abstract issues by the House of Lords in 1843. The principal decisions which were then taken are embodied in what is called the M'Naghten Rule, because it was drawn up following the trial of a man named M'Naghten. He was tried for the murder of Mr Drummond, whom he shot in the belief that he was Sir Robert Peel, for whom he had lain in wait. His acquittal followed on the ground of insanity. Medical evidence was led to the effect that a person of otherwise sound mind might be affected with morbid delusions and that the prisoner was in that condition. The evidence also indicated that a person labouring under a morbid delusion might have a moral perception of right and wrong, but that in the case of the prisoner it was a delusion which carried him beyond the power of his own control, leaving him no such perception, and that he was not capable of exercising any control over acts which had a connection with his delusion. The answers which the jury gave to the questions put to them were to the effect that at the time the act was committed the prisoner did not have the use of his understanding so as to know that he was doing a wrong and wicked act, and that the prisoner was not sensible at the time he committed the act that he was violating both the laws of God and man.

Put briefly, the M'Naghten Rule is that, to establish a defence on the ground of insanity, it must be clearly proved that at the time of committing the act the accused was labouring under such a defect of reason from disease of the mind that he did not know the nature and quality of his act, or if he did, that he was not aware that he was doing wrong.

Legislation has not yet disturbed the criteria of insanity laid down by the judges in the M'Naghten case. It is clearly seen, therefore, that there can be no conviction for crime in this country in the absence of a criminal mind or mens rea. Those falling within the M'Naghten Rule must be held to be without a mens rea. Nevertheless, an act may be a crime although the mind of the person who commits it is affected by disease, if such disease does not in fact produce upon his mind one or other of the effects above mentioned. With respect to the legal interpretation, in this connection, of the term "wrong," the criterion is not legal right and wrong, but moral right and wrong. It must be a knowledge which is not merely general and abstract, but a knowledge which must exist with regard to the particular act under consideration.

The legal aspects of criminal responsibility have been clearly put as follows :—

"In a strictly legal sense there is no insane criminal. Concede insanity, and the homicidal act is not criminal. The act of the insane, which in the sane would be criminal, lacks every element of crime. . . . A sane man, who has committed crime, may thus become insane, either before or after conviction for the crime. He may be rightfully called an insane criminal. If the insanity develops before the trial, the law would suspend his trial while the insanity continued. If the insanity came after conviction, he should be treated as an insane man, not as a criminal. The medical mind frequently views the question of insanity as one of responsibility only. To the legal mind, insanity conceded, responsibility ends. . . . If a man is clearly insane, he is not a criminal, even though 'he has within him a tendency sufficiently strong to cause him to commit offences against both person and property.' He needs care, and the State has the right, and it is its duty, to protect him from the consequences of his own mental condition. . . . It is a question of fact, usually for the jury—Is the man, or was the man, insane at the time the act was committed ? If the jury says by its verdict he was insane, then the Court sends him to an asylum for the treatment of the insane." [6]

Sir James Stephens' comment upon the answers of the English judges in the M'Naghten case is that the questions were put in a very general form, and that they could hardly have been meant to be exhaustive, because if they were so meant, the implication is that the effect of insanity upon emotions and will is left out of consideration, and is not to be taken into account in deciding whether an act done by an insane person did or did not amount to an offence, which, if it were put in the form of a proposition, would be contrary to experience. The interpretation of the law, as laid down in the answer quoted, differs, however, in the rulings of different judges. Some adhere to its literal terms, and hold that the medical evidence must be confined to a description of the state of mind of the accused at the time the act was committed, which, in many cases, may be absolutely impossible, although an opinion might be hazarded on the point from a consideration of his mental condition at the time

of the examination. Other judges leave the jury to say from the evidence led whether the accused was prevented by any disease affecting his mind from controlling his own conduct.

This certainly enlarges the purview, and it permits of medical evidence being led regarding the mental condition of the accused at the time of examination, and, based upon that, as to whether any disease then present was likely to have existed at the time of the commission of the act, thus preventing him from controlling his conduct. Other judges put the question to the jury, whether the accused knew the nature and quality of the act, and whether he was of sound mind. This is still a more liberal interpretation of the law, since, by implication, a person may be insane who at the time he committed the act knew the nature and quality of the act, and, consequently, any relevant medical evidence would be admitted which had relation to the mental condition of the accused. It will be obvious that the interpretation which different judges may place on the law will affect the range and character of the medical evidence which is permissible.

Assuming, however, that the witness is permitted to offer such relevant evidence as will throw light upon the mental condition of the accused at the time of the examination, and, by implication, of his mental state at the time of the commission of the crime; what kind of evidence would justify the witness in offering an opinion of the insanity and irresponsibility of an accused person? Generally, the existence of delusions, especially delusions bearing upon the particular crime which has been committed, would be strong evidence, because their existence betokens dissociated cerebral action. A delusion may cause abnormality either of the whole mental outlook or only of certain actions.

A man may be the subject of a delusion which, although it affects his actions, does not offer any causal connection with the crime he has committed.

It must not be forgotten, however, that the intellect may have been so abnormal at the time when the crime was committed that, in the ordinary process of reasoning on the part of the examiner, the connection between the delusion and the crime cannot be traced. If delusions are found to exist in a person who has committed a criminal act, it would be the duty of the examiner to discover, if possible, how far the processes of ideation and volition are affected thereby, and to demonstrate the path by which he has arrived at the conclusion that the person is sane or insane.

A delusion is a persistent and incorrigible belief that things are real which exist only in the imagination of the patient and which no rational person can conceive that the patient, when sane, could have believed. It is usually associated with the personality of the individual, and consists of a belief in some beneficial or prejudicial influence. When the delusion is believed to be prejudicial to the affected individual, acts of violence either towards himself or others frequently result. A delusion differs from an hallucination in that the latter is expressive of a disordered sense,

and is a perception by one or other of the senses without external causation, for example, a person labouring under an hallucination may state that he hears voices speaking to him, when in point of fact no one is speaking.

The medical witness must rely solely upon his examination of the accused to arrive at an opinion as to sanity or insanity, and the evidence obtained will depend upon the range and method of that examination.

The examiner must take into account such factors as the mental history of the progenitors, the physical condition of the accused, together with his personal mental condition, in relation to the character of the crime committed. Frequently, the crime is purposeless or motiveless in character, is committed upon persons usually held dear by the culprit, or upon those who are strangers to him, and its perpetration is accompanied by complete unconsciousness on the part of the perpetrator. The mental history of the accused, together with his present condition, must be exhaustively inquired into, so that from the complete examination, the examiner may be able, in giving evidence, to place all the facts before the Court in such a way that they may be appreciated by laymen.

For the descriptions of the various types of insanity and states of aberration of the mind, the reader is referred to treatises which deal with the clinical aspects of this subject. Neither from a statutory nor a legal point of view is a definite diagnosis of the specific form of insanity required, although a record of all the facts indicative of insanity is essential.

Uncontrollable or irresistible impulse.

In 1923, Lord Justice Atkin's Committee, which was appointed to review the criteria of insanity as incorporated in the M'Naghten Rule, reported that they reaffirmed these criteria, but made a recommendation that it should be recognised that a person charged criminally with an offence is irresponsible for his act when the act is committed under an impulse which the person was, by mental disease, deprived of any power to resist. This is the criterion of uncontrollable or irresistible impulse. The only attempt to add this to the M'Naghten Rule was made in the House of Lords in 1924, by Lord Justice Darling in moving the second reading of the Criminal Responsibility (Trials) Bill which he had introduced. This was opposed so strongly that the Bill was withdrawn. It has been suggested, from time to time, that allowance, which reduced full criminal responsibility, should be made for persons who suffer from an abnormal mental condition which renders them liable to commit crimes under the influence of an irresistible impulse. It has been felt, however, that if such a defence was recognised, the resulting exemption would be more than a potential risk, that violent tempered persons would make less effort to control their tempers and then avail themselves of such an excuse. The trend in the direction of the inclusion of this condition as a defence has shown

itself in England rather than in Scotland, and the following divergent opinions which have been expressed in two criminal cases are worthy of note.

In the course of his charge to the jury in the trial of Ronald True, for the crime of murder, at the Central Criminal Court in 1922, Mr Justice McCardie touched upon the important question of uncontrollable impulse as a defence. He said that the case had given him much anxiety because of the conflict of opinion which had existed in the past and existed at the present time as to the law, namely, that even if the prisoner knew the physical nature of the act, and that it was morally wrong and punishable by law, yet was he through mental disease deprived of the power of controlling his actions at the same time? If the answer was "yes," then, in his view of the law, the verdict should be guilty, but insane. He had explained his view, but he wanted to point out to the jury that this particular head of exemption, if he could call it such, was one which should be applied by the jury with great care, since it would never do to diminish unduly the doctrine of responsibility for criminal acts. It would be unwise to allow any man to think that he was saved from the need of controlling his actions because he would be able to say to the jury, "From mental disease, I could not control myself." His lordship next referred to the question of epilepsy. He asked the jury whether the defendant took the life of the girl whilst in an actual epileptic seizure. If so, even then, though he knew the physical nature of the act, and also that it was morally wrong and punishable by law, should the verdict again be guilty, but insane? The jury returned a verdict of guilty of wilful murder. An appeal was heard and dismissed. The prisoner was later certified insane, following an order by the Home Secretary that he be examined as to his mental condition by experts.

In the case of R. v. Burton,[7] a trial for murder, the jury desired instruction on the point whether if the accused knew perfectly well what he was doing, but owing to mental disturbance was totally unable to control his actions, the crime would amount to manslaughter. The judge informed them that uncontrollable impulse had no place in English law. The panel was found guilty but insane.

Two other cases may also be instanced. In that of Sodeman v. R., 1936, before the Judicial Committee of the Privy Council, the defence was that the accused had a mind which could not resist doing meaningless acts. It was suggested that the M'Naghten Rule was no longer to be treated as an exhaustive statement of the law relating to insanity, and that there must be grafted on to it a rule that where a man knew that he was doing wrong, none the less, he might be insane if he was caused to do the act by an irresistible impulse produced by disease. This view was not accepted by the Court.

At the hearing of the appeal of Kopsch,[8] the Lord Chief Justice said that the fantastic theory of uncontrollable impulse was not yet part of the criminal law, and it was to be hoped that the time was very far distant when it would be made so.

Diminished responsibility.

Turning now to Scotland, the established proof of diminished responsibility may reduce a charge of murder to that of culpable homicide. In the case of R. v. Savage,[9] Lord Aness stated that the law had come to recognise those who, while they may not merit the description of being insane, were nevertheless in a condition as to reduce the quality of their act from murder to culpable homicide. In such cases there must be proved aberration or weakness of mind, some form of mental unsoundness, a state of mind bordering on, though not amounting to insanity, a mind so affected that responsibility is diminished from full responsibility to partial responsibility, and the prisoner must be only partially responsible for his action.

The case of R. v. Kirkwood [10] is also worthy of note in this connection. The accused was tried for murder before the Lord Justice Clerk. A special defence was put forward that at the time of the crime charged the accused was insane and not responsible for his actions. At the close of the medical evidence, counsel for the defence tendered a plea of guilty of culpable homicide, and this plea was accepted by the Crown on the ground of diminished responsibility of the accused. In passing sentence, the presiding judge, Lord Aitcheson, said :—"It is quite impossible for me to assess what the precise degree of your responsibility is, but the only sentence I can pronounce upon you that can in any way be commensurate with the crime you have committed and adequate in the public interest, is that you be detained in penal servitude for life." The accused appealed against the sentence, and it was argued for the appellant that it was the duty of the presiding judge to discriminate between what was due to responsibility and what to irresponsibility, and to assess to what extent the accused was responsible for the crime. It appeared that the accused had been subject to epileptic fits for several years and had been treated in hospital. These fits were frequent and sometimes prolonged, and were on occasions accompanied by violence. On one occasion there had been an attempt to commit suicide. A medical witness gave evidence to the effect that the appellant was sane at the time of his examination and also when the crime was committed, but that he was not, at the time of the crime, a fully responsible individual. Other medical witnesses went further, and stated that there were circumstances connected with the actual crime which would lead them to say that the appellant was quite insane in what he did and that he had no idea what he was doing.

In intimating the refusal of the appeal, which was heard before eleven Judges of Justiciary, the Lord Justice General, Lord Normand, stated that the defence of impaired responsibility was somewhat inconsistent with the basic doctrine of Scots criminal law that a man, if sane, is responsible for his acts and, if not sane, is not responsible. It was a modern variance of the doctrine, justified by medical testimony directed to the special facts of some particular case. Mental weakness, short of insanity, was regarded by our law

as an extenuating circumstance having the effect of modifying the character of the crime or justifying a modification of sentence or both. But it might be impossible to assess the degree of responsibility and, further, the diminished responsibility of the accused was not the only relevant circumstance. The interests of society included the reformation of the criminal, the prevention of the repetition of the crime by him or by others, and the protection of other members of the community. Inquiry had satisfied him that Kirkwood's mental and physical condition would be carefully considered, that, if necessary, ameliorative treatment would be given to him, and that his condition would be reviewed from time to time by the authorities who had power to control his treatment and to order his eventual release. There was nothing which would justify the Court in interfering with the sentence.

Amnesia in relation to crime.

Hopwood and Snell[11] have thrown light on this subject as the result of an investigation based on the examination and histories of a hundred male inmates of the State Criminal Asylum at Broadmoor. Their views are that the defence was not as successful as its frequency of presentation would presuppose, that amnesia cannot be diagnosed on the patient's word alone, but must be checked by known facts, since independent evidence is essential, and that real recovery does not occur until some weeks or months after the crime. A history of chronic alcoholism, indication of a psychopathic personality, or neuropathic heredity was frequently elicited. They consider that the commencement and especially the end of the amnesic period are of special importance and are frequently blurred. In cases where there is partial amnesia, the patients were found to have a confused recollection of their acts, and the ideas which were accompanied by marked emotion were remembered, but those less emotional in character were not. They hold the further opinions that statements made soon after the crime, which are indicative of a knowledge of the crime, are not inconsistent with a genuine amnesia due to a subsequent repression. Touching upon the nature of the crime, they add that it is frequently without obvious motive, is unpremeditated, and that the perpetrator makes no effort to escape the consequences. A sudden return of memory almost certainly indicates malingering. Those interested in the subject of amnesia in this relationship are advised to consult the original article.

Inebriety.

The abuse of alcohol by persons may, under certain circumstances, bring them within the category of insanity and irresponsibility. Although chronic alcoholism is one of the more indirect causes of insanity, nevertheless the direct effects of alcohol frequently induce insanity of a temporary nature, for example, delirium tremens. The common law rule is that mere drunkenness cannot be pleaded as an excuse for crime, but a state of mental unsoundness produced by

alcohol can be so pleaded, and if established by proof, would be held to be so. The position, therefore, resolves itself into the question whether or not an accused person who has committed a crime while under the influence of alcohol was sane or insane.

Lord Deas, at the trial of a prisoner who was charged with murder, stated in his address to the jury that "if a man was insane at the time he committed the offence, no matter whether the insanity lasted for ten minutes or for half an hour, merely under drink would not do, but if drink produced insanity for however short a time, and the man did certain things while insane that there was no reason to think he would do while sane, that was quite enough." In former cases, Lord Deas made it perfectly clear that, although drunkenness, not amounting to insanity, was no excuse for crime, it was a factor in the reduction of the quality of a charge of murder to one of culpable homicide. Lord Kingsburgh stated in a charge to the jury: "You are entitled to take into consideration in the question whether the full guilt of murder has been incurred in this case, the important fact that the man was intoxicated at the time, and to a certain extent using violence, quite unconscious as to the extent of it. Although he is responsible for that, he may not necessarily be guilty of murder."

In a trial on a charge of murder, counsel for the accused asked the presiding judge to direct the jury that, if they were of the opinion that the accused, while acting in self-defence, had exceeded what was reasonably necessary as retaliation because of a temporary loss of reason caused by an attack of the deceased while he, the accused, was under the influence of drink, they were bound to acquit him of culpable homicide. The presiding judge directed the jury to the contrary effect, namely, that if, although provoked, the accused exceeded in brutality what the ordinary man should have done, drink could not palliate the brutality of his acts. The accused was convicted of culpable homicide. The court, in an appeal on the ground of misdirection, held that drunkenness, short of proved incapacity to form the intent necessary to commit the crime, could not palliate retaliatory violence in excess of what was reasonably necessary, and refused the appeal. (Reid v. H.M. Advocate, High Court of Justiciary, February, 1947.)

Lord Young declined to let a case of a married woman go to the jury, and discharged the prisoner. She was charged with culpable homicide, having, when in delirium tremens, caused the death of her infant by neglect and starvation.

In the case of R. v. Baines, Mr Justice Day charged the jury that if a prisoner was in such a state of intoxication that he did not know the nature of his act, he was insane in the eye of the law, and that it was immaterial whether the mental derangement consequent upon such intoxication was permanent or temporary.

The position with regard to inebriety and criminal responsibility can be summed up shortly. Drunkenness is ordinarily neither a defence nor excuse for crime unless the intoxication is such as to

prevent the individual from restraining himself from committing the act in question or to take away from him the power of forming any specific intention. The reason why ordinary drunkenness is not an excuse for crime lies in the fact that the offender did wrong in getting drunk. If distinct disease of mind is caused by drinking, as opposed to drunkenness, and obliterates the knowledge between good and evil, the jury may bring in a verdict as in the case of insanity.

A man is presumed to intend the natural consequences of his acts, but this is only a presumption, and it may be rebutted, for example :—

In the case of a sober man, in many ways ;
In the case of a man who is drunk, by showing his mind to have been so affected by liquor he had taken that he was incapable of knowing that what he was doing was dangerous, in other words, that he was likely to inflict serious injury.

It will, therefore, be understood that in cases of delirium tremens, the defence will be one of insanity, but that in certain cases of drunkenness, the condition of drunkenness at the time when the crime was committed, although not an excuse for the crime, may be pleaded as a mitigation and, provided the degree of intoxication is proved to be such that it deprived the accused of the power of forming any specific intention, the charge of murder may be reduced to that of manslaughter in England, or to culpable homicide in Scotland. On this matter there is no difference between the law of either country.

Legislation relating to inebriety.

Inebriates Acts.

The Act of 1879 affirmed that an habitual drunkard had power by his own act to secure detention in a licensed institution for a period not exceeding one year, for the purposes of reformation and cure. This Act was limited in its duration to ten years, but the Act of 1888 made its provisions permanent. The Act of 1898, while it contained no provision for the compulsory detention of non-criminal habitual drinkers, made provision for the detention of criminal inebriates of both sexes for reformative purposes. Criminal inebriates convicted of an offence, punishable with imprisonment or penal servitude, may be detained in a State inebriate reformatory for a term not exceeding three years in addition to, or in substitution for, any other sentence.

Before pronouncing such a sentence, the Court must be satisfied that the offence was committed while under the influence of drink, or that drunkenness was a contributing cause, and that the offender himself admits that he is, or has been found by the jury to be, an habitual drunkard. The class of habitual drunkards, called "repeaters," is composed of those who, within the twelve months

preceding the date of the commission of the offence, have been summarily convicted at least three times of minor offences. On conviction they may be detained in any certified inebriate reformatory for not more than three years. The law, therefore, while it does not transgress any principle affecting the responsibility of those committed, acts on the assumption that such persons are less able to resist the perpetration of offences by reason of their habits, and, therefore, are not mentally sound, although the degree of mental unsoundness falls short of insanity and irresponsibility.

The Inebriates Acts apply to both England and Scotland.

Mutism.

The legal procedure adopted at the trial of an accused person who is mute differs in England and Scotland.

English procedure.

Any question whether the accused is mute of malice or by visitation of God is determined by a jury sworn to try the preliminary issue. If their verdict is "mute by the visitation of God," a second issue must be determined, namely, whether the accused is able to plead and understand the proceedings at the trial. If he is not, he is treated as insane, unless the incapacity is temporary. In the case of R. v. Governor of Stafford Prison,[12] a prisoner who was totally deaf and could neither read nor write was arraigned for a felony. He stood mute, and a jury was impanelled and sworn to ascertain whether he was mute by the visitation of God. They found that he was, and were then sworn again to try whether he was capable of pleading to the indictment. They found that he was incapable of pleading to, and taking his trial upon, the indictment and of understanding and following the proceedings by reason of his inability to communicate with, or be communicated with by, others. Upon this verdict the judge, acting under Section 2 of the Criminal Lunatics Act, 1800, ordered him to be kept in custody until His Majesty's pleasure should be known. It was held that this judgment amounted to a finding that the prisoner was insane within the meaning of the Act, and that the order was properly made.

Scottish procedure.

In 1817, for the first time in Scotland, the question as to whether a person deaf and dumb is an object for trial and punishment, came to be tried. The case concerned a woman, Jean Campbell, alias Bruce, who was indicted at Glasgow for the murder of her child.[13] The question was a new one and the Lords on the Circuit certified the case for the consideration of the High Court. The Court was satisfied that she had the power of communicating her thoughts, and showed intelligence of right and wrong. She was committed for trial and the case was remitted to the judges at the next circuit

at Glasgow, when she pleaded not guilty by communicating by a deaf and dumb show. Her evidence was given by signs and gestures and was in her favour. A verdict of not guilty was returned.

In February 1942, at the High Court in Glasgow, a man was charged with murder, and after six doctors had stated that he was fit to instruct his defence, the presiding judge decided that he was insane within the meaning of the Lunacy Act, 1857. The accused man was deaf, unable to talk properly, could not read, was said to be feeble-minded, but was not certifiably insane. After hearing the medical evidence, the judge decided to interrogate the man in the privacy of his room together with counsel and the prisoner's interpreter. Since the case presented an unusual and difficult problem, concerning the disposal of the panel, the judge certified the case for the consideration of the High Court and adjourned it. The decision to adjourn was based upon the findings that either the panel was of such mental unsoundness as to be considered insane within the meaning of the Lunacy Act, 1857, or that, in any event, his admitted mental defect, coupled with his undoubted physical disabilities, which really cut him off from adequate contact with the outside world, rendered it improper that he should be called upon to plead to a capital charge. Five judges in the High Court decided that the prisoner should stand trial on the charge, and that the question of his sanity should be left to be determined by the jury.

The Solicitor-General (Sir David King Murray, K.C., afterwards Lord Birnam) said that the attitude of the Crown was that either the man was insane within the meaning of the Act, and that therefore the statutory course of detention during His Majesty's pleasure should be followed, or that he was not insane within the meaning of the Act, and that in that case the trial should proceed, whatever difficulties the Court and the jury might be faced with. What was meant by insanity in the statute was not certifiable insanity but such insanity as prevented an accused in a reasonable sense from being capable of standing his trial or giving instructions for his defence. The Lord Justice-General, intimating the decision of the Court, said that, since counsel had taken up the position that the accused ought to go forward to trial by jury, who would be capable of determining the question of his sanity, and who would have more extended evidence than had yet been led, his motion for trial should be allowed, and accordingly no determination regarding the prisoner's insanity should be made at the present stage. The other judges concurred. The accused was tried at a later date and acquitted.

References

1. "Measurements of Intelligence," Harrap, London. Terman.
2. "Textbook of Psychiatry," 449, 5th ed., 1940, Henderson and Gillespie. Oxford University Press, London.
3. L.R., 5, Q.B., 549, 1870, Banks v. Goodfellow.
4. Lancet, Vol. I, 31, 1946.

5. Police Jour., Vol. XVI, No. 1, 14, 1943, R. v. Dashwood.
6. Med.-Leg. Jour. of N.Y., Vol. XVI, 268, 1898.
7. 100, J.P., 306, R. v. Burton.
8. Cr. App. R., Vol. XIX, 50, 1925, Kopsch v. R.
9. J.C., 49, 51, R. v. Savage, 1923.
10. Scots Law Times, Reports, 209, 1939 ; Poliee Jour., Vol. XII, No. 2, 163, 1939, R. v. Kirkwood.
11. Jour. Mental Sc., Vol. LXXIX, 27, 1933, Hopwood and Snell.
12. 2 K.B., 81, 1909, R. v. Governor of Stafford Prison.
13. Hume, Vol. I, 45, R. v. Campbell.

TOXICOLOGY

CHAPTER XVI

LAWS RELATING TO POISONS

TOXICOLOGY has been defined as the science which includes the knowledge of the sources, characters, and properties of poisons, the symptoms which they produce, the nature of the fatal results, and the remedial measures which should be employed to combat their actions or effects. Since poisoning is one of the methods by which murder is committed, it is essential that students and practitioners should be familiar with the outstanding symptoms and signs of poisoning in living persons together with its effects as found in the examination of the dead.

Taylor has described a poison as "a substance which, when taken into the mouth or stomach, or when absorbed into the blood, is capable of affecting seriously the health or of destroying life by its action on the tissues with which it immediately, or after absorption, comes into contact." The law, however, does not require that a poison should be defined further than as a substance which, when administered, is injurious to health or life. The law on the subject, as applicable to England, is contained in Sections 11, 22, 23, and 24 of the Offences Against the Person Act, 1861.

Section 11 runs thus:—"Whosoever shall administer or cause to be administered to, or be taken by, any person, any poison or other destructive thing with intent to commit murder, shall be guilty of a felony."

It will be seen, therefore, that even although the poison administered is not followed by bodily injury, either as the result of prompt and effective treatment or from some other cause, if the intent to commit murder is proved, the person administering will be guilty of felony.

Section 22 covers the administration of substances calculated to enable the commission of a crime, and provides that if any person unlawfully applies or administers to, or causes to be taken by, or attempts to apply or administer to, or attempts to cause to be administered to, or be taken by, any person, any chloroform, laudanum, or other stupefying or overpowering drug, matter, or thing, with intent thereby to enable himself or any other person to commit, or with intent, to assist any other person in committing, any indictable offence, he shall be guilty of felony" (see p. 423).

Section 23 likewise makes it a felony for any person, unlawfully and maliciously, to administer to, or cause to be administered to, or taken by, any other person, any poison or other destructive or

noxious thing, so as thereby to endanger the life of such person, or so as thereby to inflict upon such person any grievous bodily harm.

It will be apparent from this last section that if anyone administers any poison, or other destructive or noxious thing, to the danger of life, or which causes the infliction of grievous bodily harm, he is guilty of a felony. To meet the possible plea that the substance administered was not intended to endanger life or to inflict grievous bodily harm, and that the substance so administered did not accomplish either of these things, Section 24 enacts that if the substance was administered with intent only to injure, aggrieve, or annoy, the person who administered or caused it to be administered shall be guilty of a misdemeanour.

The question as to what constitutes a "noxious thing" has been considered in courts of law, and it has been held that there must not only have been an administration of a noxious substance with intent but that it must have been administered in sufficient quantity to be noxious. A further ruling given is that if a person administers, with intent to produce miscarriage, something which, as administered, is "noxious," he administers a "noxious thing" (see p. 382). In Scotland, to cause death by poisoning is a capital crime at common law. At common law an attempt to poison is also a crime, although authoritative opinion is divided as to whether or not the guilty person is subject to the death penalty. Under statute, however, an attempt to poison is also punishable by death, and that whether the administration of the poison was with intent to murder, disable, or do some grievous bodily harm. For the law respecting the application of corrosives to the surface of the body, see p. 223.

STATUTES REGULATING THE SALE OF POISONS

The Acts of Parliament by which the sale of poisons and the practice of pharmacy in England and Scotland are regulated are :—

Pharmacy and Poisons Act, 1933; Poisons Rules, 1935; Poisons (Colouring) Rules, 1936; and Statutory Rules and Orders made in connection with the Pharmacy and Poisons Act, 1933.

Dangerous Drugs Acts, 1920-32; and Statutory Regulations and Orders made in connection with them.

Therapeutic Substances Act, 1925; and Therapeutic Substances Regulations, 1931-44.

Pharmacy and Medicines Act, 1941.

Pharmacy and Poisons Act, 1933.

The Poisons List is divided into two parts :—

Part I of the List consists of those poisons which are not to be sold by retail except by a person who is an authorised seller of poisons, for example, a registered pharmacist whose business premises are registered with the Pharmaceutical Society of Great Britain.

Part II of the List consists of those poisons which are not to be sold by retail except by a person who is an authorised seller of poisons or whose name is entered in a list, kept by a local authority, of persons who are entitled to sell poisons in this part of the List, or who carries on the business of an authorised seller of poisons at one or more chemist's shops to sell drugs, but not Part I poisons, or at other shops where no pharmacist is employed and which are registered by the Pharmaceutical Society and not by a local authority.

In determining the distribution of poisons as between Part I and Part II of the List, regard was given to the desirability of restricting Part II to substances in common use or likely to come into common use, for purposes other than the treatment of human ailments, and which it is reasonably necessary to include in Part II if the public are to have adequate facilities for obtaining them. The Secretary of State may from time to time, after consultation with, or on the recommendation of, the Poisons Board, by order, amend or vary the List as he thinks proper, and the List as in force is referred to as "The Poisons List." In this Act the expression "poison" means a poison included in the Poisons List.

The division of the Poisons List into Parts I and II, together with the distribution of the various substances contained therein, is of subordinate interest to members of the medical profession. The regulations regarding the sale of poisons contained in Parts I and II, however, demand consideration and are important.

These regulations make it unlawful :—

(*a*) For a person to sell any poison included in Part I of the Poisons List, unless :—

(1) he is an authorised seller of poisons ; and
(2) the sale is effected on premises duly registered under the Act ; and,
(3) if the poison is in the First Schedule, the sale is effected by, or under the direct supervision of, a registered pharmacist.

(*b*) For a person to sell any poison, whether included in Part I or in Part II of the Poisons List, unless the container of the poison is labelled in the prescribed manner :—

(1) with the name of the poison ; and
(2) in the case of a preparation which contains a poison as one of the ingredients, with the prescribed particulars as to the proportion which the poison contained in the preparation bears to the total ingredients ; and
(3) with the word "poison" or other prescribed indication of the character of the article ; and
(4) with the name of the seller of the poison and the address of the premises on which it was sold.

(*c*) For a person to sell any poison in Schedule I of the Poisons List to another person unless that person is :—

(1) certified in writing in the manner prescribed by rules, and by

a person authorised by rules to give a certificate for the purpose ; or

(2) known by the seller or by some registered pharmacist in the employment of the seller at the premises where the sale is effected, to be a person to whom the poison may properly be supplied (see pp. 506 and 507).

The seller of any such poison shall not deliver it until :—

(1) he has made or caused to be made an entry in a book to be kept for that purpose stating, in the form prescribed by rules, the date of the sale, the name and address of the purchaser, and of the person by whom the certificate required was given, the name and quantity of the article sold, and the purposes for which it is stated by the purchaser to be required ; and

(2) the purchaser has affixed his signature to the entry.

The provisions enumerated under (*c*) do not apply to :—

(1) medicine supplied by a duly qualified medical practitioner for the purposes of medical treatment, by a registered dentist for the purposes of dental treatment, or by a registered veterinary surgeon for the purposes of animal treatment ; or

(2) to a medicine which is dispensed by an authorised seller of poisons on duly registered premises ; or

(3) to a poison forming part of the ingredients of a medicine which is supplied by an authorised seller of poisons on duly registered premises, provided that :

(i) the medicine is distinctly labelled with the name and address of the person by whom it is supplied or dispensed ;

(ii) that on the day on which the medicine is supplied or dispensed or on the day after, the following particulars are entered in a book used regularly for the purpose, but which need not be used exclusively for that purpose :

(*a*) the date on which the medicine was supplied or dispensed ;

(*b*) the ingredients of the medicine and the quantity supplied ;

(*c*) the name and address of the authorised seller if the medicine was dispensed by him and the person to whom the prescription was given, together with the date ; and

(*d*) the name of the person to whom it was supplied, if the medicine was not so dispensed.

The latter provisions need not be satisfied in the case of a medicine supplied on, and in accordance with, a prescription given by a duly qualified medical practitioner or registered dentist under, and in

accordance with, the provisions of the National Health Service Act, 1946, or the National Health Service (Scotland) Act, 1947, and in the case of a medicine supplied on a prescription on which the medicine has been supplied by the seller on a previous occasion, it is sufficient if the day on which the medicine is supplied together with the quantity are entered in a book, together with a sufficient reference to an entry in the book duly recording the dispensing of the medicine on the previous occasion.

Except as provided for by rules, nothing in the foregoing shall interfere with :—

(1) the sale of poisons by way of wholesale dealing ; or
(2) the sale of an article to a duly qualified medical practitioner, registered dentist, or registered veterinary surgeon for the purpose of his profession ;
(3) the sale of an article for use in, or in connection with, any hospital, infirmary, dispensary, or health centre ; or
(4) the sale of an article by a person carrying on a business in the course of which poisons are regularly sold either by way of wholesale dealing or for use by the purchasers thereof in their trade or business.

It should be noted that if any medicine sold comes within the Dangerous Drugs Acts (see p. 513) the proper entry must be made in the Dangerous Drugs Register.

Under the **Poisons Rules, 1935,** as amended by the Poisons (Amendment) Rules, made by the Secretary of State as empowered under the Act, certain substances have been relegated to specific Schedules. The First and Fourth of these Schedules, to which special restrictions apply, are of high importance to members of the medical profession who should have a clear knowledge regarding the classes of poisons contained therein, and of the restrictions imposed on their sale.

FIRST SCHEDULE

Substances included in the Poisons List to which special restrictions apply

Alkaloids, the following ; their salts, simple or complex :—

Acetyldihydrocodeinone.

Aconite, alkaloids of, except substances containing less than 0·02 per cent. of the alkaloids of aconite.

Apomorphine, except substances containing less than 0·2 per cent. of apomorphine.

Atropine, except substances containing less than 0·15 per cent. of atropine.

Belladonna, alkaloids of, except substances containing less than 0·15 per cent. of the alkaloids of belladonna calculated as hyoscyamine.

Benzoylmorphine.

Benzylmorphine.
Brucine, except substances containing less than 0·2 per cent. of brucine.
Calabar bean, alkaloids of.
Coca, alkaloids of, except substances containing less than 0·1 per cent. of the alkaloids of coca.
Cocaine, except substances containing less than 0·1 per cent. of cocaine.
Codeine, except substances containing less than 1·5 per cent. of codeine.
Colchicine, except substances containing less than 0·5 per cent. of colchicine.
Coniine, except substances containing less than 0·1 per cent. of coniine.
Cotarnine, except substances containing less than 0·2 per cent. of cotarnine.
Curare, alkaloids of; curare bases.
Desomorphine (dihydrodesoxymorphine).
Diacetylmorphine.
Dihydrocodeinone.
Dihydrohydroxycodeinone.
Dihydromorphine.
Dihydromorphinone.
Ecgonine, its esters, except substances containing less than 0·1 per cent. of ecgonine.
Emetine, except substances containing less than 1 per cent. of emetine.
Ergot, alkaloids of.
Ethylmorphine, except substances containing less than 0·2 per cent. of ethylmorphine.
Gelsemium, alkaloids of, except substances containing less than 0·1 per cent. of the alkaloids of gelsemium.
Homatropine, except substances containing less than 0·15 per cent. of homatropine.
Hyoscine, except substances containing less than 0·15 per cent. of hyoscine.
Hyoscyamine, except substances containing less than 0·15 per cent. of hyoscyamine.
Jaborandi, alkaloids of, except substances containing less than 0·5 per cent. of the alkaloids of jaborandi.
Lobelia, alkaloids of, except substances containing less than 0·5 per cent. of the alkaloids of lobelia.
Morphine, except substances containing less than 0·2 per cent. of morphine calculated as anhydrous morphine.
Nicotine.
Papaverine, except substances containing less than 1 per cent. of papaverine.
Pomegranate, alkaloids of, except substances containing less than 0·5 per cent. of the alkaloids of pomegranate.
Quebracho, alkaloids of.
Sabadilla, alkaloids of, except substances containing less than 1 per cent. of the alkaloids of sabadilla.
Solanaceous alkaloids, not otherwise included in this Schedule, except substances containing less than 0·15 per cent. of solanaceous alkaloids calculated as hyoscyamine.

Stavesacre, alkaloids of, except substances containing less than 0·2 per cent. of the alkaloids of stavesacre.

Strychnine, except substances containing less than 0·2 per cent. of strychnine.

Thebaine, except substances containing less than 1 per cent. of thebaine.

Veratrum, alkaloids of, except substances containing less than 1 per cent. of the alkaloids of veratrum.

Yohimba, alkaloids of.

Allylisopropylacetylurea.

Amidone (dl-2-dimethylamino-4 : 4-diphenyl-heptane-5-one) ; its salts.

Amidopyrine ; its salts.

Amino-alcohols, esterified with benzoic acid, phenylacetic acid, phenylpropionic acid, cinnamic acid or the derivatives of these acids, except in substances containing less than 10 per cent. of esterified amino-alcohols.

Antimonial poisons, except substances containing less than the equivalent of 1 per cent. of antimony trioxide.

Arsenical poisons, except substances containing less than the equivalent of 0·01 per cent. of arsenic trioxide and except dentifrices containing less than 0·5 per cent. of acetarsol.

Barbituric acid ; its salts ; derivatives of barbituric acid ; their salts ; compounds of barbituric acid, its salts, its derivatives, their salts, with any other substance.

Barium, salts of.

Beta-aminopropylbenzene ; its salts ; its N-alkyl derivatives ; their salts ; beta-aminoisopropylbenzene ; its salts ; its N-alkyl derivatives ; their salts (e.g., benzedrine). This substance is exempt in appliances used for inhalation.

Cannabis ; the resin of cannabis ; extracts of cannabis ; tinctures of cannabis ; cannabin tannate.

Cantharidin, except substances containing less than 0·01 per cent. of cantharidin.

Cantharidates, except substances containing less than the equivalent of 0·01 per cent. of cantharidin.

Carbachol.

Digitalis, glycosides and other active principles of, except substances containing less than one unit of activity (as defined in the British Pharmacopoeia) in 2 grammes of the substance.

Dinitrocresols, except agricultural and horticultural insecticides or fungicides ; dinitronaphthols ; dinitrophenols ; dinitrothymols.

Ergot ; extracts of ergot ; tinctures of ergot.

Guanidines, the following : polymethylene diguanidines, diparaanisylphenetyl guanidine.

Hydrocyanic acid, except substances containing less than 0·15 per cent., weight in weight, of hydrocyanic acid (HCN) ; cyanides, except substances containing less than the equivalent of 0·1 per cent., weight in weight, of hydrocyanic acid (HCN) ; double cyanides of mercury and zinc.

Lead, compounds of, with acids from fixed oils.

Mercuric chloride, except substances containing less than 1 per cent. of mercuric chloride ; mercuric iodide, except substances containing less than 2 per cent. of mercuric iodide ; nitrates of mercury, except substances containing less than the equivalent of 3 per cent., weight in weight, of mercury (Hg) ; potassio-mercuric iodides,

except substances containing less than the equivalent of 1 per cent. of mercuric iodide; organic compounds of mercury, except substances containing less than the equivalent of 0·2 per cent., weight in weight, of mercury (Hg).

Metanitrophenol; orthonitrophenol; paranitrophenol.

Metopon (Methyldihydromorphinone); its salts.

6-Morpholino-4 : 4-diphenylheptane-3-one; its salts.

Nux Vomica, except substances containing less than 0·2 per cent. of strychnine.

Opium, except substances containing less than 0·2 per cent. of morphine calculated as anhydrous morphine.

Ouabain.

Oxycinchoninic acid, derivatives of; their salts; their esters.

Para-aminobenzenesulphonamide; its salts; derivatives of para-aminobenzenesulphonamide, having any of the hydrogen atoms of the para-amino group or of the sulphonamide group substituted by another radical: their salts.

Pethidine; its salts.

Phenetidylphenacetin.

Phenylcinchoninic acid: salicylcinchoninic acid; their salts; their esters.

Phenylethylhydantoin; its salts; its acyl derivatives: their salts.

Picrotoxin.

Savin, oil of.

Sodium monofluoracetate.

Strophanthus, glycosides of.

Sulphonal and alkyl sulphonals.

Thallium, salts of.

Tribromethyl alcohol.

Tridione (3 : 5 : 5-trimethyloxazolidine-2 : 4-dione).

Zinc phosphide.

This Schedule, which contains certain poisons from both Part I and Part II of the Poisons List, imposes the following restrictions:—

1. The purchaser must be known to the seller or to one of his qualified assistants to be a person to whom the poison may properly be sold.

2. If this condition cannot be complied with, then the purchaser must show a written certificate signed by a householder, and this has to be endorsed by a police officer, in charge of a police station, unless the householder is known to the chemist to be a responsible person of good character.

3. Following the sale of the poison, this certificate must be retained by the seller.

4. An entry of the sale must be made in a book kept for this purpose together with the date, the name and address of the purchaser, and the person granting the certificate, the name and quantity of the poison sold, and the stated purpose for which it was required. The purchaser must sign the Poisons Register before the poison is delivered.

These restrictions do not apply to the following articles:—

(*a*) Machine-spread plasters;

(*b*) surgical dressings;

(*c*) articles containing barium carbonate and prepared for the destruction of rats and mice ;

(*d*) corn paints in which the only poison is a poison included in the Poisons List under the heading of "Cannabis" (e.g., the resins, extracts, tinctures, and cannabis tannate).

(*e*) articles containing zinc phosphide and prepared for the destruction of rats and mice.

Doctors, dentists, and veterinary surgeons may obtain poisons contained in the First Schedule from any authorised seller by a written order or by personal attendance at the premises of the seller and by signing the Poisons Register. If an order is sent, it must be signed, and contain the name and address of the purchaser together with the reason for the purchase of the poison. In the case of emergency, a medical practitioner, dentist, or veterinary surgeon may obtain poisons contained in the First Schedule if they are required in the course of his profession, by giving an undertaking that he will furnish a written order within twenty-four hours. Failure to furnish the order after the drug has been supplied is an offence.

The following provisions govern the supply of poisons by medical practitioners, dentists, and veterinary surgeons :—

1. It is not necessary for a medical practitioner to keep a record of a medicine supplied unless it is a substance included in the First Schedule.
2. Dentists and veterinary surgeons must keep a record of all medicines which contain a poison and which have been dispensed or supplied by them.
3. The date on which the medicine was supplied, the ingredients, quantity, and the name of the person to whom the medicine was supplied must be entered in a book used for the purpose.
4. All medicines containing poisons supplied by medical practitioners, dentists, and veterinary surgeons must be labelled and bear the name and address of the practitioner.

Strychnine may only be sold or supplied as an ingredient in a medicine except :—

(*a*) by wholesale ;

(*b*) for export ;

(*c*) to doctors, or veterinary surgeons, for use in their practices ; or

(*d*) to a person or institution concerned with scientific education, research, or chemical analysis, for these purposes ;

(*e*) under certain conditions for the purpose of killing moles, and not exceeding 4 ounces at one time ; or

(*f*) to a person authorised by the Secretary of State for the Home Department to purchase strychnine for the purpose of killing seals.

FOURTH SCHEDULE

The Fourth Schedule contains a list of the following substances which, subject to the undernoted restrictions, can only be sold by

retail upon a prescription given by a qualified medical practitioner, registered dentist, registered veterinary surgeon, or registered veterinary practitioner.

Allylisopropylacetylurea.
Amidopyrine ; its salts.
Barbituric acid ; its salts ; derivatives of barbituric acid ; their salts ; compounds of barbituric acid, its salts, its derivatives, their salts, with any other substance.
Dinitrocresols ; except agricultural or horticultural insecticides or fungicides ; dinitronaphthols ; dinitrophenols ; dinitrothymols.
6-Morpholino-4 : 4-diphenylheptane-3-one ; its salts.
Para-aminobenzenesulphonamide ; its salts ; derivatives of para-aminobenzenesulphonamide having any of the hydrogen atoms of the para-amino or of the sulphonamide group substituted by another radical ; their salts, except when contained in ointments and surgical dressings.
Phenylcinchoninic acid ; salicyl-cinchoninic acid ; their salts ; their esters.
Sulphonal ; alkyl sulphonals.
Tridione (3 : 5 : 5-trimethyloxazolidine-2 : 4-dione).

These restrictions provide that the prescription must be in writing and must be signed and dated by a duly qualified practitioner, registered dentist, or registered veterinary surgeon. Provided that where an authorised seller of poisons is reasonably satisfied that a person ordering any such poison and who is a duly qualified medical practitioner and who is by reason of some emergency unable to furnish such a prescription immediately, he may, notwithstanding that no such prescription has been given, if the said person undertakes to furnish him within the twenty-four hours next following with such a prescription, deliver the poison ordered in accordance with the directions of the said person, so, however, that notwithstanding anything in such directions, the supply shall not be repeated unless such a prescription has been given. If any person by whom any such undertaking has been given fails to deliver to the seller a prescription in accordance with the undertaking, or if any person for the purpose of obtaining delivery of any poison under the foregoing proviso, makes a statement which is to his knowledge false he shall be deemed to have contravened the provisions of this Rule. The prescription must specify the address of the person giving it, except in the case of a National Health Service prescription, and the name and address of the person for whose treatment it is given, or, if the prescription is given by a veterinary surgeon, of the person to whom the medicine is to be delivered. If the prescription is given by a dentist, it must bear the words " For dental treatment only," or, if given by a veterinary surgeon, the words " For animal treatment only." The total amount of the medicine to be supplied and the dose to be taken have to be added. The person who dispenses the prescription must comply with the following requirements :—

1. The prescription must not be dispensed more than once unless the prescriber has stated thereon either that it may be dispensed a stated number of times or that it may be dispensed at stated intervals.

2. If the prescription contains a direction that it may be dispensed a stated number of times or at stated intervals, it must not be dispensed otherwise than in accordance with the direction. A prescription which contains a direction that it may be dispensed a stated number of times but no direction as to the intervals at which it may be dispensed shall not be dispensed more often than once in three days, and a prescription which contains a direction that it is to be dispensed at stated intervals but no direction as to the number of times that it may be dispensed shall not be dispensed more often than three times.
3. At the time of dispensing or, where a poison has been delivered (as referred to under the proviso described at the foot p. 508), on the subsequent receipt of the prescription there must be noted on the prescription above the signature of the prescriber the name and address of the seller and the date on which the prescription was dispensed or, as the case may be, the poison was delivered.
4. Except in the case of a National Health Service prescription, or a prescription which may be dispensed again, the prescription must, for a period of two years, be retained on the premises on which it was dispensed in such manner as to be readily available for inspection. When a repeat prescription is dispensed for the last time, it must be retained by the chemist.

Poisons (Colouring) Rules, 1936.

It is unlawful to sell any poison included in the Schedule to these Rules and intended for use in agriculture or horticulture for the destruction of bacteria, fungi, insects, vermin, or as weed-killer unless there has been added to the poison a dye of a distinctive colour and soluble in water. This rule does not apply to :—

(*a*) Lead arsenate paste or lead arsenate powder ; or
(*b*) poisons which are of themselves of a distinctive colour ; or
(*c*) sheep dips which are already of a distinctive colour.

Schedule

Arsenates.
Arsenites.
Copper acetoarsenites.
Halides of arsenic.
Organic compounds of arsenic.
Oxides of arsenic.
Sodium thioarsenates.
Sulphides of arsenic.

Poisons Rules, 1949.

It should be noted that no person shall be entitled by virtue of being a listed seller of Part II poisons to sell any arsenical poison, other than lead arsenates, calcium arsenates and copper acetoarsenites, any mercuric chloride, mercuric iodide or any organic compound of mercury, unless the purchaser thereof is engaged in the trade or business of agriculture or horticulture and requires the poison for the purpose of that trade or business.

THE PROVISIONS OF THE DANGEROUS DRUGS ACTS, 1920-32; AND STATUTORY RULES AND ORDERS MADE IN CONNECTION WITH THEM

The medico-legal relations of the medical practitioner to these Acts and to the Statutory Rules and Orders issued from time to time are of great importance.

The Act of 1920 regulates the Importation, Exportation, Manufacture, Sale, and Use of Opium and other Dangerous Drugs. Part III deals with prevention of the improper use of certain drugs of addiction and controls their manufacture, sale, possession, and distribution except on premises licensed for the purpose. It also regulates the issue, by medical practitioners, of prescriptions containing any of these drugs, and the dispensing of any such prescriptions, and requires persons engaged in their manufacture, sale, or distribution to keep such books and furnish such information in writing or otherwise as may be prescribed. The regulations under this part of the Act provide for authorising any person who lawfully keeps open shop for the retailing of poisons, to manufacture at the shop, in the ordinary course of his retail business, any preparation, admixture, or extract of any drug to which this part of the Act applies, and to carry on at the shop the business of retailing, dispensing, or compounding any such drug, subject to the power of the Secretary of State to withdraw the authorisation in the case of a person who has been convicted of an offence against the Act.

If the Home Secretary suspects that a medical or dental practitioner is supplying or prescribing dangerous drugs illegally, or is administering them to himself unlawfully, he may summon him to appear before a tribunal composed of a representative from the General Medical Council, the British Medical Association, the Royal College of Physicians, and a legal assessor. If the charges are proved, on the recommendation of this tribunal, the Home Secretary may withdraw the practitioner's authority to issue such drugs.

By the Dangerous Drugs (Amendment) Regulations, 1936, the words "Persons keeping open shop for the retailing of poisons" have been substituted by "Persons who are authorised sellers of poisons within the meaning of the Pharmacy and Poisons Act, 1933"; and for the words "at the shop" the words "on the registered premises" have been substituted.

Drugs to which the Dangerous Drugs Acts and Regulations apply.

(1) Raw opium; Coca leaves and Indian hemp, including the resins obtained from Indian hemp and any preparations of which such resins form the base:
(2) Medicinal opium;
(3) Any extract or tincture of Indian hemp, and any preparation made from such extract or tincture;

(4) Morphine and its salts, and diacetylmorphine (commonly known as diamorphine or heroin), and the other esters of morphine and their respective salts ;

(5) Cocaine (including synthetic cocaine) and ecgonine and their respective salts, and the esters of ecgonine and their respective salts ;

(6) Any solution or dilution of morphine or cocaine or their salts in an inert substance, whether liquid or solid, containing any proportion of morphine or cocaine, and any preparation, admixture, extract or other substance (not being such a solution or dilution as aforesaid) containing not less than one-fifth per cent. of morphine or one-tenth per cent. of cocaine or of ecgonine ;

(7) Any preparation, admixture, extract, or other substance containing any proportion of diacetylmorphine or of the other esters of morphine, and any preparation, admixture, extract, or other substance containing any proportion of ecgonine or of the esters of ecgonine ;

(8) Dihydrohydroxycodeinone, dihydrocodeinone, dihydromorphinone, methyldihydromorphinone (commonly known as metopon), acetyldihydrocodeinone, dihydromorphine, dihydrodesoxymorphine (desomorphine), their esters and the salts of any of these substances and of their esters, morphine-N-oxide (commonly known as genomorphine), the morphine-N-oxide derivatives, and any other pentavalent nitrogen morphine derivatives ;

(9) Thebaine and its salts, and benzylmorphine and the other ethers of morphine and their respective salts ;

(10) Pethidine (1-methyl-4-phenylpiperidine-4-carboxylic acid ethyl ester) and amidone (dl-2-dimethyl : 4-4 : diphenyl-heptane-5-one) and their salts.

(11) Any preparation, admixture, extract, or other substance containing any proportion of any of the substances mentioned in paragraph (8) or in paragraph (9) except, in the case of preparations of methylmorphine or ethylmorphine, syrupus codeinæ phosphatis, B.P.C., 1934, and preparations, admixtures or other substances containing not more than 2·5 per cent. of methylmorphine or ethylmorphine (calculated as pure drug) associated with other medicinal substances.

Note.—"Ecgonine" means lævo-ecgonine and includes any derivatives of ecgonine from which it may be recovered industrially, and the percentage in the case of morphine shall be calculated as in respect of anhydrous morphine.

The principal restrictions imposed under the Regulations of 1937 are :—

1. A person duly authorised shall not manufacture, or carry on any process in the manufacture of a drug otherwise than in accordance with the terms and conditions of the authority issued to him and then only on the premises authorised.

2. A person shall not supply or procure, or offer to supply or procure, to or for any person, including himself, or advertise for sale, a drug or preparation unless he is duly authorised

to do so and does so in accordance with the terms and conditions of his authority, and the person supplied is authorised to be in possession of the drug or preparation.

The administration of a drug or preparation by, or under the direct personal supervision and in the presence of, a duly qualified medical practitioner, or by, or under the direct personal supervision and in the presence of, a registered dentist, in the course of dental treatment, shall not be deemed to be the supplying of a drug or preparation.

A person to whom a drug or preparation is lawfully supplied on a prescription lawfully given by a duly qualified medical practitioner, a registered dentist, or a registered veterinary surgeon, or to whom a drug or preparation is lawfully supplied by a duly qualified medical practitioner or a registered veterinary surgeon who dispenses his own medicines, shall be deemed to be a person authorised to be in possession of the drug or preparation so supplied.

Persons who are members of the following classes :—

(*a*) duly qualified medical practitioners ;
(*b*) registered dentists ;
(*c*) registered veterinary surgeons ;
(*d*) pharmacists who are employed or engaged in dispensing medicines at a public hospital or other public institution :
(*e*) persons in charge of a laboratory for purposes of research or instruction and attached to an institution approved for the purpose by the Secretary of State ;
(*f*) persons duly appointed by a local authority as analysts for the purposes of the Food and Drugs (Adulteration) Act, 1928 :

are authorised, so far as may be necessary for the practice or exercise of their respective professions or employments, to be in possession of, and to supply, drugs or preparations, provided that a dentist shall not be authorised to supply drugs or preparations otherwise than by the personal administration to persons receiving treatment from him.

The term " institution " means a university, university college, public hospital, or other like institution.

A prescription for the supply of any such drugs must comply with the following requirements :—

1. It must be in writing or typewriting, must be dated, signed by the medical practitioner, dentist, or veterinary surgeon, with his usual signature, and address, and must specify the name and address of the person for whose use the prescription is given. The prescription must specify the total amount of the drug to be supplied. In prescriptions issued under the National Health Service Acts, on the forms provided, the address of the practitioner need not be included.

2. A prescription shall only be given by a medical practitioner when required for purposes of medical treatment. When given by a dentist, it must be marked "For local dental treatment only," and when by a veterinary surgeon, shall be marked "For animal treatment only."
3. The drugs shall not be supplied more than once on the same prescription, provided that if the prescription so directs, the drugs may be supplied on more than one but not exceeding three occasions, as directed in the prescription, at intervals to be specified therein.
4. The prescription shall be marked with the date on which it is dispensed and shall be retained by the person, firm, or body corporate by whom the prescription is dispensed, and, unless for the purposes of the National Health Service Acts, shall be kept in the premises where it is dispensed and shall be available for inspection.

Every person authorised to supply dangerous drugs or preparations shall comply with the following provisions :—

(*a*) He shall keep a register and enter therein, in the forms set out in Part I and Part II of the Second Schedule in the Regulations, true particulars with respect to every quantity of any drug or preparation obtained by him, and with respect to every quantity of any drug or preparation supplied by him.

A separate register or a separate part of the register shall be used with respect to each class of drug and its preparations.

(*b*) The required entry in ink or indelible pencil must be made on the day on which the drug or preparation is received or on which the transaction with respect to the supply thereof takes place, or, if that is not reasonably practicable, on the following day.

(*c*) So much of this Regulation as requires a person to enter in the register particulars with respect to drugs or preparations supplied to him shall not apply to :—

(1) A duly qualified medical practitioner who enters in a day-book particulars of every drug or preparation supplied by him to any person, together with the name and address of that person and the date of the supply, and enters in a separate book a proper reference to each entry in the day-book which relates to the supply of any drug or preparation ; or

(2) an authorised seller of poisons, within the meaning of the Pharmacy and Poisons Act, 1933, who enters in a separate book a proper reference to each entry in a Pharmacy Act book which relates to the supply of any drug or preparation.

Every register, separate book, day-book, or Pharmacy Act book must be kept on the premises, and be available for inspection.

By the Dangerous Drugs (Exempted Preparations) Amendment Regulations, the sale of many preparations containing small quantities of the dangerous drugs, are exempted from the Regulations, but their sale is controlled by the Pharmacy and Poisons Act, 1933. Such preparations in common use are :—

Pasta Arsenicalis, B.P.C. 1934.
Pil. Ipecac. c. Scilla, B.P.C. 1934.
Pil. Digitalis et Opii Co., B.P.C. 1923.
Pil. Hydrarg. c. Cret. et Opii, B.P.C. 1934.
Pulv. Cretæ Aromat. c. Opio, B.P. 1932.
Pulv. Ipecac. et Opii, B.P. 1932.
Suppos. Plumbi c. Opio, B.P. 1932.
Tabellæ Plumbi c. Opio, B.P.C. 1934.
Elixir Diamorphinæ et Terpini c. Apomorphina, B.P.C. 1934.
Linctus Diamorphinæ Camphoratus, B.P.C. 1923 and 1934.
Linctus Diamorphinæ c. Ipecacuanha, B.P.C. 1934.
Linctus Diamorphinæ et Scillæ, B.P.C. 1923 and 1934.
Linctus Diamorphinæ et Thymi, B.P.C. 1923 and 1934.
Mixtures of Pulv. Ipecac. et Opii, B.P. 1932 with any of the following :—
  Hydrarg. c. Cret., B.P. 1914 and 1932.
  Acetylsalicylic acid.
  Phenacetin.
  Quinine and its salts.
  Sodium bicarbonate.
Cocaine eyedrops—a preparation consisting of an admixture of cocaine in castor oil with mercuric chloride in a proportion of not more than 1 part in 200 of cocaine and not less than 1 part in 3,000 of mercuric chloride.
Methylmorphine and ethylmorphine and their respective salts and any preparation, admixture, or other substance containing any proportion of methylmorphine or ethylmorphine associated with an inert substance, whether solid or liquid ; and preparations and admixtures or other substances containing more than 2·5 per cent. of methylmorphine or ethylmorphine (calculated as pure drug) associated with other medicinal substances.

Provided that the percentage of dangerous drugs contained in the foregoing official preparations is not exceeded, these preparations, on prescription, are unrestricted.

Therapeutic Substances Act, 1925 ; and Regulations.

This Act controls the manufacture, sale, importation, purity, and standards of certain therapeutic substances used in the treatment of disease. These include :—(1) The substances commonly known as vaccines, sera, toxins, antitoxins, and antigens ; (2) the substance commonly known as salvarsan (Dioxy-diamino-arsenobenzol-di-hydrochloride), and analogous substances used for the specific treatment of infective disease ; (3) preparations of the specific antidiabetic principle of the pancreas, known as insulin : (4) preparations of the posterior lobe of the pituitary body intended for use by injection, pituitrin ; (5) surgical ligature or suture ; and (6) penicillin (crude filtrate), penicillin (dried crude filtrate), and

penicillin salts; (7) preparations of human blood; (8) organic substances having the specific biological action of curare on neuro-muscular transmission; (9) streptomycin.

No person shall manufacture for sale any of the foregoing substances unless he holds a licence from the licensing authority or in premises other than those in respect of which a licence is in force. The Regulations do not contain matter of medico-legal importance. The Third Schedule deals with general provisions applicable to arsphenamine and derivatives. The licensing authority in England is the Minister of Health, and in Scotland, the Department of Health.

Pharmacy and Medicines Act, 1941.

This Act amends the Pharmacy and Poisons Act, 1933. The chief points of interest are that :—

1. No person shall take any part in the publication of any advertisement referring to any article in terms calculated to lead to the use of that article for the purpose of the treatment of human beings for any of the following diseases, namely, Bright's disease, cataract, diabetes, epilepsy or fits, glaucoma, locomotor ataxy, paralysis, or tuberculosis.

   This does not apply to an advertisement published by a local authority, or by the governing body of a voluntary hospital, or by any person acting with the sanction of the Secretary of State or the Minister of Health. Sanction has been given, until further notice, to the publication of advertisements referring to insulin but not to any particular brand. It shall be a defence for the person charged to prove that the advertisement was published only so far as was reasonably necessary to bring it to the notice of persons of the following classes :—

   (*a*) Members of Parliament ;
   (*b*) members of a local authority ;
   (*c*) members of the governing body of a voluntary hospital :
   (*d*) registered medical practitioners ;
   (*e*) registered nurses ;
   (*f*) registered pharmacists and authorised sellers of poisons ;
   (*g*) persons undergoing training with a view to becoming registered medical practitioners, registered nurses, or registered pharmacists ;
   (*h*) persons carrying on a business which includes the sale or supply of surgical appliances.

2. No person shall take any part in the publication of any advertisement referring to any article in terms calculated to lead to the use of that article for procuring the miscarriage of women.

   It shall be a defence to prove that the advertisement was published only in a publication of a technical character

intended for circulation amongst persons of classes, namely, (*d*), (*e*), (*f*), (*g*), and (*h*) described above.

3. No person shall—

(*a*) sell by retail any article consisting of or comprising a substance recommended as a medicine ; or

(*b*) supply any such article as a sample for the purpose of inducing persons to buy by retail the substance of which it consists or which it comprises ; unless there is written so as to be clearly legible on the article or label affixed—

(i) the appropriate designation of the substance or of each of the active constituents thereof, or of each ingredient of which it has been compounded ; and

(ii) in a case where the appropriate designation of each of the actual constituents or the ingredients is written, the appropriate quantitative particulars of the constituents or ingredients.

This shall not apply to any article made up and supplied for the use of a particular person being an article prescribed by reference to the needs of that person. "Appropriate designation" means—

(i) If the substance is included in the Poisons List, the name required to be labelled in accordance with Section 18 of the Pharmacy and Poisons Act, 1933.

(ii) If the substance is not included in the Poisons List, the description set out at the head of any of the monographs contained in the edition of the British Pharmacopœia or the British Pharmaceutical Codex which was last published before the date on which the article was sold or supplied.

(iii) If not within (ii), then the accepted scientific name or other name descriptive of the true nature of the substance, constituent, or ingredient.

4. No person shall sell by retail any article consisting of or comprising a substance recommended as a medicine unless he is—

(*a*) a registered medical practitioner or a registered dentist ; or

(*b*) an authorised seller of poisons ; or

(*c*) a person not being in any of the foregoing classes, but being a person who—

(i) has served a regular apprenticeship to a registered pharmacist, or to a body corporate authorised to sell poisons ; and

(ii) is at the date of the passing of the Act carrying on, on his own account, a business which comprises the retail sale of drugs.

CHAPTER XVII

GENERAL ACTION OF POISONS, EVIDENCE AND TREATMENT OF POISONING

General action of poisons.

THE action of poisons may be local, remote, or both. They kill either by destroying the tissues with which they come into contact or produce their effects by being absorbed into the general circulation and thus act on organs remote from their point of absorption, such as the central nervous system or heart. Although corrosive substances cause lesions in the parts with which they are brought into contact, the proximate cause of death may vary, depending upon the substance which has been introduced into the body, since the major factor is either shock or other effects following absorption. With regard to the remote action of poisons following absorption, some of them act on certain organs more than others, for example, opium, morphia, the barbiturates, and alcohol affect the brain, digitalis and oxalic acid involve the heart, strychnine, the spinal cord, and chlorine, gaseous in form, the lungs. Certain other poisons, notably arseniuretted hydrogen, carbon monoxide, chlorate of potash and pyrogallic acid act upon the blood. The poisons which are frequently swallowed and cause death are, probably in order of frequency, phenol and phenol derivatives, corrosive acids and alkalis, prussic acid and cyanides, oxalic acid and oxalates, opium derivatives, barbiturates and aspirin, strychnine and nux vomica, arsenic and mercury salts, phosphorus, atropine and belladonna. With regard to the dosage of potent drugs, these can be divided, in arbitrary manner, into two groups, namely, those in which the lethal dose lies far in excess of the active dose and those in which it lies close to the active dose. Of the drugs which may be relegated to the former group, atropine may be mentioned, while of those belonging to the latter group, strychnine is an example. There is a very definite distinction between the terms "toxic dose" and "fatal dose," since the former merely causes symptoms of poisoning while the latter induces poisoning to such a degree that death results.

Circumstances modifying the action of poisons.

Quantity.

The question of dosage has already been touched upon. The larger the quantity and the greater the severity of the symptoms,

the more rapid usually is the fatal result. It must not be forgotten, however, that the actual excess, in respect of some poisons, may prove a merciful dispensation by inducing both rapid and effective emesis. The question of quantity, which separates the medicinal action of a poisonous drug from its toxic action, has prompted the law to differentiate between a poison and a noxious thing.

Condition of administration.

A poison acts most rapidly when inhaled in a gaseous or vaporous form, or when injected intravenously. Next, in order of rapidity, when injected intramuscularly or subcutaneously, and least rapidly when ingested. Further slowing down of absorption occurs when the substance which is swallowed is only partly soluble. With decreasing rapidity, absorption takes place from the mucous surfaces and the unbroken skin.

Chemical combination.

Some substances when in certain combinations are very poisonous, but when in others are comparatively innocuous. While the component parts of a mixture may have a poisonous action when administered singly, the compound itself may be comparatively inert.

Mechanical combination.

When a powdery poisonous substance is administered with fluids of much lighter specific gravity, the substance is liable to sediment in the vessel, and thus the quantity actually swallowed is less than when the vehicle of administration has been a fluid of specific gravity more nearly approaching that of the powder. It is by adopting the latter principle that the poisoner who uses arsenic administers it in a fluid which by its colour will mask the presence of the poison, and which is heavy and viscid. When a poison is administered by the mouth, the stomach being empty, its action will be much more rapid than when swallowed after a meal.

Habit.

Habit diminishes the effects of certain poisons, since a tolerance toward them is gradually developed. It is a matter of common observation, for example, that continuous indulgence in alcohol and tobacco confers a comparative immunity from danger from poisonous doses. Similarly, the morphine habit may be contracted to such an extent that immense doses may be taken with impunity. From continued use, strychnine and arsenic may be taken in doses which otherwise would be liable to act poisonously.

Condition of bodily health.

A relatively small dose, from which a stronger person would probably recover, may kill a weakly person. In certain diseases, some drugs can be given, with impunity, in doses which, in other

circumstances, would be likely to prove harmful as, for example, morphia in mania or delirium tremens, and in cases of acute pain. In some diseases, on the other hand, certain drugs cannot be administered even in small doses without toxic effects. This is seen in cases of renal disease, when mercury and morphia should only be administered with the greatest caution.

Age.

Adults are more tolerant to certain drugs, such as narcotics, than children and old persons.

Idiosyncrasy.

Among persons, there is a varying degree of susceptibility. From time to time this is seen when such drugs as morphia, cocaine, quinine, iodine, and bromide are administered.

Cumulative action.

Certain poisons tend to accumulate within the body as the result of slow excretion, and therefore the continued administration of relatively small doses may occasion symptoms of poisoning, for example, strychnine, lead, mercury, digitalis, and carbon monoxide.

Evidence of poisoning in the living body.

The evidence of poisoning will depend upon whether the poisoning is acute or chronic.

In acute poisoning the symptoms appear suddenly while the individual is in good health. The person is affected with a group of symptoms of a definite character out of consonance with his previous state of health. This feature of the attacks stands out prominently in all recorded cases of poisoning. At the same time, certain groups of symptoms of disease which simulate poisoning may have a sudden onset, and this possibility must receive due consideration.

In chronic poisoning, the onset of the symptoms is more gradual and insidious, on account of the small quantity of poison which has been administered on each occasion, since the intention of the poisoner may be to kill his victim gradually by persistent administration of small doses in the hope of averting suspicion. In such cases, the possibility of detection lies in the absence of causal relationship between the condition of the patient and the symptoms from which he suffers, in the fitful coming and going of the symptoms, in their appearance, usually after food or liquid has been taken, and in their complete disappearance on the patient's removal from his usual surroundings.

In acute poisoning especially, and in chronic poisoning generally, the symptoms frequently appear soon after food, medicine, or drink has been taken. It must be borne in mind, however, that it is precisely under such circumstances that symptoms of disease, for example gastro-intestinal conditions, may arise.

When more than one person has eaten similar food, and when they are similarly affected, there is strong presumptive evidence of poisoning.

The strongest proof of criminal or suicidal administration of poison, however, is established by the discovery of poison in the food taken, in the vomited matter, or in body excretions, when it can be shown that such could not have occurred from accidental extraneous contamination.

If a poison is found in the urine of a patient suffering from unexpected and anomalous symptoms, there can be no doubt whatever that the poison has been introduced into the body, has passed into the circulation, and has then been excreted.

Duty of a medical practitioner in cases of suspected poisoning.

Should a medical man be called to a case which, from the symptoms, he suspects to be due to chronic poisoning, what points should he attend to, apart from the treatment of the patient ?

These may be summarised broadly as follows :—

The time of the occurrence of the symptoms, and their character, should be carefully noted.

Their relation, in point of time, to the taking of food, drink, or medicine, and the order in which they appear, should be fully investigated.

It should be observed whether the symptoms intermit, or increase steadily in severity.

The previous condition of health of the person affected should be ascertained.

If there has been vomiting, the vomited matter should be inspected and, if possible, a sample should be taken for analysis.

A sample of the suspected peccant food, drink, or medicine, and a sample of urine and fæces should be obtained for analysis. These should be placed in suitable containers, sealed, and labelled.

If food is suspected, inquiry should be made as to whether similar food has been eaten previously with impunity, and whether or not any person other than the one affected has eaten it with impunity, or has been affected more or less simultaneously.

Explanations offered or remarks made, regarding the onset of symptoms, should be noted.

When the symptoms do not conform to ordinary illness, and when, in spite of appropriate treatment, they continue, the environment of the patient and the conduct of those looking after him, should be carefully observed, and the services of a consultant utilised.

Should there be suspicious circumstances, the patient should be removed to hospital or to a nursing home where he can be under close observation and supervision remote from his usual environment.

General lines of treatment in acute poisoning.

These may be summed up in a few sentences, but the mode and extent of their application must be left to the judgment of the practitioner according to the needs of individual cases.

First.

The poison should be removed from the stomach expeditiously.

Second.

Antidotes should be administered.

Third.

The poison should be eliminated from the body by the natural channels.

Fourth.

Symptomatic treatment should be administered and stimulation of the respiratory and circulatory systems should be resorted to immediately indications present themselves.

The best and most expeditious method for the removal of poisons from the stomach is by the use of the stomach tube, which is easily introduced, and, when intelligently used, can do no injury. The tube, which is composed of flexible rubber, should be about $\frac{1}{2}$ inch, or slightly less, in diameter, and about 5 feet in length. It should be marked at a point about 20 inches above the lower end, which should be perforated by more than one opening. The actual termination of the lower end should be rounded off in order to avoid friction when the tube is passed. A filter-funnel should be inserted into the upper end of the tube. The average distance between the lips and the cardiac end of the stomach in the adult subject is approximately 18 inches, and it is for this reason that the tube is marked in the manner described, so that the operator may have indication when the end of the tube has reached the interior of the stomach. In the case of young children, a tube of narrower calibre and of shorter length should be employed. In using the stomach tube, it is advisable that the head of the patient should be placed over the end of the bed. In this position, the mouth and pharynx will be at a lower level than the larynx and trachea, and thus regurgitated fluid, around the tube, will not enter the air-passages. After the tube has been warmed and smeared with a lubricant, such as glycerine or vaseline, it is passed into the stomach by depressing the tongue with the finger, sliding the tube along the finger into the pharynx, directing the tip to the back of the pharynx, and thus into and down the œsophagus. Water, the temperature of which has been first tested against the cheek or lip of the administrator, is then poured in by the funnel. The water may contain a suitable dissolved antidote. While pouring in the fluid, the funnel should be held above the patient's head. As the last portion of

water is being poured in, and the tube and a portion of the funnel are full of water, the tube at its junction with the funnel is pinched between the finger and thumb, and the tube is then lowered below the level of the stomach. Atmospheric pressure acting upon the column of water in the stomach and tube will empty the contents of both. The process may be repeated as often as required. To perform complete gastric lavage, about 2 gallons of water in all should be used. About 1 pint of fluid should be employed for each separate lavage. When the poison which has been swallowed is known, a suitable chemical antidote may be dissolved in the solution used for lavage, and when the process is nearing completion, a quantity of the fluid should be allowed to remain in the stomach.

In cases of strychnine poisoning, it is imperative that before attempting to introduce the tube, the patient should first be anæsthetised, otherwise the attempt will induce spasm and frustrate the effort. When a patient is conscious and a stomach tube is not available, simple emetics, such as mustard and water, or salt and water, should be freely used. If the patient refuses or is unable to swallow, an emetic, such as apomorphine, should be given hypodermically.

The stomach tube should not be employed in poisoning by strong corrosives on account of the danger of perforation of the stomach by the tube, following the effects of the corrosive lesions. In such cases it is preferable, when possible, to counteract their effects by causing the patient to drink neutralising demulcent fluids. In poisoning by certain substances, which by irritant action cause nephritis, it is necessary to assist excretion. Perspiration may be induced by hot baths, warm packs, or by hypodermic injection of pilocarpine.

To dilute the poison which has been absorbed into the blood-stream and aid its elimination from the body, intravenous infusion of normal saline solution or glucose saline will be found most beneficial in certain cases. Blood transfusion will be found useful in poisoning due to toxic substances causing blood changes. When asphyxial manifestations threaten, and in certain stages of gaseous poisoning, oxygen with 7 per cent. carbon dioxide is valuable. To clear the airway, the patient should be placed face downwards to allow fluid to drain from the mouth and upper passages. When there is a tendency for the tongue to fall back, its position should be carefully controlled. When breathing ceases, artificial respiration must be employed without delay. Œdema glottidis may call for intubation. The treatment of symptoms should be applied as indications arise, and such stimulatory measures as are necessary must also be utilised. The detailed lines of treatment in cases of specific poisoning are described later under the appropriate headings.

Antidotes.

The effects of certain poisons can be counteracted by antidotes, which may be divided into three groups, in accordance with their mode of operation, namely: (*a*) physical, (*b*) chemical, and (*c*) physiological.

Physical antidotes.

These are few in number. Some of them have also a chemical action to a limited extent. For example, egg albumen or flour and water are mechanical antidotes in poisoning by corrosive sublimate. At the same time, by the formation of albuminate of mercury, which is practically insoluble in the stomach for the time being, egg albumen acts partly as a chemical antidote. Charcoal may be reckoned as a physical antidote in alkaloidal poisoning, as by strychnine, but it is difficult to estimate how far the antidotal effect is not chemical because of the occluded oxygen. Physical antidotes also exercise a beneficial effect in other cases, since they dilute the poison and thus may limit its effects. Demulcent fluids act in this manner both in corrosive and irritant poisoning.

Chemical antidotes.

These neutralise poisons by forming new compounds which are either insoluble or less active; thus alkalis in acid, and dilute acids in alkali, poisoning may be taken as the simplest type. Magnesium sulphate may be used in poisoning by carbolic acid, and sulphates of the alkalis in poisoning by lead or barium. Freshly precipitated oxide of iron, made by treating tincture of perchloride of iron with excess of ammonia, filtering the precipitate, and adding water, may be used in arsenical poisoning as a chemical antidote since it forms the comparatively insoluble ferric arsenite. Tannin and its preparations produce an insoluble tannate in poisoning by antimony. Potassium permanganate is a powerful antidote in morphine poisoning.

Physiological antidotes.

The mode of action of these is widely divergent. Chloroform may be said to exercise an antagonistic effect in strychnine poisoning by inducing general anæsthesia, thus overcoming the tetanic contractions and giving an opportunity for the elimination of the poison from the stomach and from the body generally. Atropine, within limits, is a physiological antidote to morphia. Physiological antidotes are not reliable in their action.

Intravenous injections of normal saline or glucose saline, also blood transfusions, in certain cases of poisoning, yield very beneficial results.

Evidence of poisoning in the dead.

Evidence from post-mortem appearances.

The post-mortem appearances found will be described in detail when specific poisons are considered. In conducting a post-mortem examination where poisoning is suspected, evidence of disease must be differentiated from lesions due to the effects of a poison. Should any doubt exist in the mind of the examiner, histological examination should be made. Adequate and appropriate material for analysis must be secured, and in this respect the nature and

characters of the lesions present, if any, together with the history of the case should provide the necessary indications. When such indications are not available, all the organs, together with a specimen of blood and urine, should be retained (see p. 45).

Evidence from chemical analysis.

The most important proof of poisoning is the detection of the poison within the body. In some cases, however, on account of embalming, decomposition of the tissues, and the lapse of time between death and the examination, certain poisons, although present within the body at the time of death, may not be discovered on analysis. This is possible in cases of poisoning by hydrocyanic acid, and by certain other poisons of vegetable origin.

Difficulties, too, may occur in cases with respect to the particular poison employed, for example aconitine, which is difficult to isolate and which is also adversely affected by putrefactive changes in the tissues.

In the toxicological section which follows, descriptions of the methods for quantitative analysis have been omitted, apart from a few isolated instances, for example, alcohol, the estimation of which forms part of the evidence in so many cases. Toxicological analysis is a highly specialised branch of science which has fallen appropriately within the sphere of the skilled chemist rather than that of the medico-legist who, however, should be responsible for the interpretation of the results obtained by analysis. It certainly lies well outside the scope of the student and practitioner. The reader who desires an intimate knowledge of these methods should consult standard works on the subject.[1]

Classification of Poisons.

Since there is no sufficiently satisfactory classification for the many and varied poisons which fall to be described for medico-legal purposes, we do not propose to follow closely any of the existing classifications. The grouping employed has been dictated by the character of the action of the poisons.

Reference

1. "Poisons. Their Isolation and Identification," Bamford, 1st and 2nd Editions, 1940 and 1947. "Churchill Laboratory Manual for the Detection of Poisons and Powerful Drugs," Autenrieth, Churchill, 1928.

CHAPTER XVIII

CORROSIVE POISONS

General action of corrosives.

THERE is more or less destruction of the parts with which the corrosive substance comes in contact, due to protoplasmic coagulation, precipitation, or dissolution of protein, and extraction of water. As a rule there is no remote systemic action, with the exception of shock. The signs and symptoms after swallowing a corrosive consist of burning, acute pain in the mouth, pharynx, œsophagus, and stomach, continuous retching and vomiting of shreddy blood-stained material, intense thirst, and probably some involvement of the air-passages. There will be signs of corrosion of the mouth or lips or both. Consciousness is usually retained and death may result from shock, due to the extensive destruction of tissue, suffocation from implication of the larynx, or perforation of the stomach. Cicatricial contraction of the œsophagus is an occasional sequela.

Post-mortem appearances.

These may be expressed generally as signs of corrosion and partial destruction of the parts with which the fluid has been in contact and vary in extent from localised patches to extensive areas, particularly in the stomach.

CORROSIVES—MINERAL ACIDS

Signs and Symptoms.

The symptoms come on immediately. There is pain, which extends from the mouth to stomach, gaseous frothy eructations, and brownish or blackish coloured vomit, sometimes mixed with portions of discoloured mucous membrane. The vomit has a strongly acid reaction, stains clothing and fabrics with which it comes into contact, and causes effervescence with a carbonate. There is intense thirst, the efforts to swallow in appeasing it causing difficulty and pain, and there may be considerable dyspnœa from swelling of the epiglottis and larynx. The mouth and lips are excoriated, their mucous membranes being sodden and discoloured, the colour depending upon the nature of the acid which has been swallowed. Articulation is often indistinct. If the acid has been taken from a spoon, or bottle, the lips may escape injury. The vomiting and retching being more or less continuous, the patient becomes

prostrated, but the mental faculties usually remain clear until the terminal phase of the illness is approached. Death may result from convulsions, suffocation, exhaustion, shock, and from perforation of the stomach. Recovery may follow prompt antidotal measures, but death may supervene subsequently from the effects of stricture of the œsophagus.

General treatment.

The stomach tube should not be employed since there is a possible danger of perforation of the wall of the stomach. Alkalis, such as lime water, calcined magnesia, and milk of magnesia, may be employed, but alkaline carbonates which liberate carbon dioxide should not be used, on account of gaseous distension of the stomach. Later, diluent, demulcent drinks, such as barley-water, milk, or thin gruel, should be given freely. To combat respiratory failure, artificial respiration, especially by Schaefer's method, may be employed and oxygen, with 7 per cent. carbon dioxide, used. When there is œdema of the glottis, tracheotomy may have to be performed. When there is circulatory shock, due to fluid loss and blood concentration, the patient should be given a continuous intravenous infusion of normal saline or saline with glucose. This should be maintained until the feeling of thirst has passed, the mouth is moist and urine is being secreted. To alleviate pain, morphia should be administered with caution and other symptoms should be treated as they arise.

Post-mortem changes.

The lips may be stained a yellow, whitish, or brownish colour. The mucous membrane of the mouth is corroded, the tissues being softened and discoloured, and the œsophagus is similarly affected. There may also be irregular patches of extravasated blood. The gastric mucosa is frequently discoloured, and the colour variation will depend upon the nature of the ingested corrosive. Extravasations of blood are common, and the mucous membrane is corrugated. The stomach may be perforated.

Sulphuric acid or oil of vitriol.

This is commonly used in commerce, in the form of strong and invariably impure vitriol. Poisoning usually occurs by accident, but is sometimes caused suicidally.

Post-mortem appearances.

The mucous membrane of the stomach will show a varying degree of blackish discoloration depending both upon the concentration and the amount of the acid swallowed. From post-mortem diffusion, the corrosive action may occasionally be observed in the peritoneal cavity. The œsophagus will also show areas of dark sloughing, and the larynx is frequently involved. When death is delayed, lesions may also be found in the duodenum.

Fatal period.

The shortest period is about an hour, but as a rule about eight to sixteen hours elapse before death supervenes. Death from shock, however, may be instantaneous, but life may be prolonged for weeks or months. Septic pneumonia is a possible complication.

Fatal dose.

Half a drachm has killed a child about one year old in twenty-four hours. The smallest fatal dose in an adult is probably 1 drachm, but recovery has followed a dose of 4 drachms.

Chemical tests (see p. 723).

Hydrochloric acid, muriatic acid or spirits of salt.

This is used in various trades.

Post-mortem appearances.

The parts of the mucous membrane which have come in contact with the acid show, at first, a dirty-white or ash-grey colour, which later turns brown or black. Perforation of the stomach is not so frequently found as in the instance of sulphuric acid. In addition to the discoloration of the mucous membrane, patches of erosion, of varying depth and size, will also be found.

ILLUSTRATIVE CASES

A woman, aged fifty, swallowed 3 ounces of hydrochloric acid with suicidal intent. Six hours later she was taken to hospital in a collapsed condition. There were no signs of corrosion of the mouth or tongue. She died about eight hours after taking the poison. Post-mortem examination showed marked lesions of the epiglottis; the larynx was red, œdematous, and necrotic. The throat was markedly involved. The mucous membrane of the œsophagus was almost entirely destroyed. The external surface of the stomach was black in colour, there being hæmorrhage into the peritoneal cavity. There was no perforation of stomach. Internally, its whole surface was almost black, this being more marked at the pyloric than the cardiac end. The duodenum at its commencement was involved.[1]

A man, aged twenty-three, was admitted to hospital for œsophageal stricture. Four years previously he had swallowed about 4 ounces of hydrochloric acid. Gastrostomy was performed, but he died ten days later. Post-mortem examination showed a stricture of the œsophagus, $2\frac{1}{2}$ inches above the cardiac end of the stomach. It was of a hard, fibrous, and annular character, and admitted a No. 15 catheter. There was also a similar stricture at the pylorus, which scarcely admitted a fine probe. The stomach was greatly hypertrophied and pouched at its cardiac end. The œsophagus showed only a slight degree of pouching.

Fatal period.

From four to thirty hours.

Fatal dose.

Half an ounce. Recovery has followed 2 ounces.

Chemical tests (see p. 715).

Nitric acid, aqua fortis, or red spirit of nitre.

The strong commercial acid varies in colour from pale yellow to deep orange, depending upon the amount of impurity present, the height of the colour being due to the presence of nitrogen peroxide. On organic matter, due to the formation of picric acid, nitric acid produces yellow stains which are darkened by the addition of an alkali.

Post-mortem appearances.

The affected mucous membranes show yellowish discoloration, and the degree of corrosion is very marked. On account of the fumes which are liberated, death is frequently caused by congestion and œdema of the lungs. Capillary bronchitis is quite a frequent complication.

For the detailed description of poisoning by nitrous fumes and the treatment, see p. 597.

Fatal period.

Death commonly occurs within twenty-four hours, but has supervened within one and a half hours.

Fatal dose.

Of the strong acid, the smallest fatal dose recorded is 2 drachms.

Chemical tests (see p. 718).

CORROSIVES—ALKALIS

Corrosive alkalis are used for a variety of purposes in the processes of manufacture.

Potassium hydroxide or caustic potash.

Cases of poisoning by this substance are comparatively rare. In one of our cases a man swallowed a quantity of strong solution of this corrosive alkali in mistake for ginger-beer, and although he recovered from the acute symptoms, he later suffered from partial œsophageal stricture. While poisoning by caustic potash is unusual, poisonous effects are more common from the use of concentrated solutions of the carbonate, or pearl-ash. The symptoms, generally, are of the corrosive type, but are not as severe as those which follow the swallowing of the mineral acids. Swelling of the tissues with which the alkali comes in contact is likely to be marked and severe,

and the surface of the mouth, tongue, and lips becomes highly reddened and eroded.

The post-mortem appearances are indicative of corrosion.

Treatment.

Dilute acids such as vinegar, lemon juice, or tartaric acid, and demulcent drinks should be administered. Olive oil may be given freely or egg albumen may be employed.

Fatal period.

Death has occurred after a few hours. Not many fatal cases have been recorded.

Fatal dose.

Very uncertain.

Chemical tests (see p. 721).

Sodium hydroxide or caustic soda.

Commonly met with as washing soda, a mixture of the hydrated oxide and carbonate.

The symptoms and lesions are similar to those of caustic potash.

Accidental poisoning by caustic soda is relatively more common than by caustic potash.

Willimott [2] reports the case of a person who swallowed 60 grains of the solid dissolved in half a tumbler of water. Vomiting, pain in the mouth, throat, and stomach, and collapse followed. Death occurred in about twenty-nine hours. The lips and mucosa of the mouth, tongue, and pharynx were swollen. The mucosa of tongue and pharynx was of deep chocolate colour.

Ammonium hydroxide or caustic ammonia.

Symptoms.

In two of our cases, suicide had been attempted by drinking ammonia sold for domestic purposes. In both cases, which recovered, the symptoms of dyspnœa and dysphagia were very marked, and the former were accompanied by some degree of cyanosis. In one of the cases, the mouth and throat showed patchy corrosion, and the tongue and pharynx were congested and œdematous. Following recovery from the initial symptoms, stricture of the lower third of the œsophagus developed, which, however, was overcome by the use of graduated bougies. In neither of the cases was the amount of fluid swallowed ascertained. The strength of the ammonia, which had been taken, was 9·8 per cent. Liquor ammoniæ fortis contains 32·5 per cent. by weight of ammonia. Serious symptoms of poisoning have followed the swallowing of ammonium carbonate. When ammonia is swallowed, there are more or less immediate symptoms of acute irritation involving the mouth, upper air-passages, œsophagus, and stomach.

The urgent symptoms in poisoning by ammonia are due to the ammoniacal fumes which are liberated (see p. 596).

Treatment.

As described for Caustic Potash. For the treatment of respiratory complications, see p. 597.

Post-mortem appearances.

These consist of a raw, inflamed condition of the lips, mouth, tongue, pharynx, and larynx, with patchy erosion of the mucous membrane. The œsophagus is also involved and the lining membrane is usually of whitish-yellow colour and sodden in appearance. Frequently there is irregular desquamation with exposure of underlying tissue. Œdema of the glottis is sometimes present. The gastric mucosa may show a similar picture to that of the œsophagus, although the colour is usually somewhat darker. Patchy submucosal extravasations of blood may be seen. As a rule, there is intense congestion. When a quantity of the fumes has been inhaled, the bronchi are covered with a fibrinous membrane which is easily stripped and leaves a raw surface beneath. The lungs are congested and œdematous, or evidence of broncho-pneumonia may be present (see p. 597).

Fatal period.

Shortest period known is four minutes.

Fatal dose.

A drachm and upwards of strong ammonia.

Chemical tests (see p. 707).

CORROSIVES—ORGANIC ACIDS

Oxalic acid.

This acid is used by shoemakers, bookbinders, brass polishers, straw-hat makers, and in the cleaning of brass domestically. It is sometimes taken with suicidal intention, and has occasionally been mistaken for Epsom salts with fatal results.

It is thought that the biochemical lesion of oxalic acid poisoning is due to the removal of calcium ions from tissue fluid incident to the formation of insoluble calcium oxalate.

Symptoms.

If a large dose of the acid is taken, the following signs and symptoms are commonly found, namely, a burning, acrid taste on swallowing, dysphagia, vomiting, which is severe and continuous, and a burning sensation in the œsophagus and stomach. Occasionally a feeling of suffocation, with lividity of face and hurried respiration, may be experienced. Collapse is rapid. The vomited material at first consists of normally coloured stomach contents mixed with a varying amount of mucus, but as sickness continues, the colour

becomes greenish-black or almost black due to the presence of altered blood. In addition to the foregoing manifestations, there are pain and tenderness over the abdomen. If the case is short in duration, the intestinal tract is not affected, but if life is prolonged, purging and tenesmus may be present. The pulse becomes feeble and irregular, and not infrequently the patient complains of a sensation of numbness in the limbs. Convulsions often precede death. The character and severity of the symptoms depend upon the amount and the concentration of the acid swallowed. When in concentrated solution, the acid acts as a corrosive.

Treatment.

Consists in first washing out the stomach and thereafter administering chalk, or lime in milk, or other demulcent drinks in small concentrated quantities. Saccharated solution of lime, in drachm doses, frequently repeated, is recommended and also intravenous injections of calcium chloride or gluconate (Calcium Sandoz).

Post-mortem appearances.

If the dose is large and the acid is in concentrated form, all the parts which have come into contact with the acid are softened, white, and corroded, or may be stained with blackish or reddish streaks. White corroded areas may be found at the corners of the mouth, on the lips, tongue, and lining of the cheeks. The œsophagus is markedly congested, and in patches, the lining membrane is frequently corroded and partly detached. The stomach contains a dark brown, glairy fluid, which has an acid reaction, and the mucous membrane is corroded and detached in varying degree ; the blood-vessels in the submucous layer may be seen distinctly because of their dark-coloured contents. The stomach also shows intense congestion. Perforation is rare, but occasionally occurs. The upper part of the intestinal tract may also show evidence of a hæmorrhagic necrosis. Since this poison is excreted by the kidneys, they will show marked congestion or cloudy swelling if death has been delayed sufficiently long. In such cases death is frequently due to anuria and uræmia. Concentration of oxalate in the kidneys may produce necrosis of the renal tubules.

Fatal period.

The shortest period recorded is three minutes. Death has taken place in ten minutes, after 1 ounce had been swallowed ; in another case, at the end of thirty minutes, from the same dose ; while a person has survived for five days.

Fatal dose.

A boy of sixteen died in eight hours after a dose of 60 grains. Recovery, however, has followed the swallowing of $\frac{1}{2}$ ounce, and $1\frac{1}{2}$ ounces.

Chemical tests (see p. 719).

Acid potassium oxalate.

Salts of sorrel, or salts of lemon, is an acid salt, used for removing iron stains from clothing, and therefore may be taken accidentally or suicidally. It is almost as poisonous as oxalic acid and gives rise to similar symptoms and signs. In one case where about ½ ounce was taken by mistake for Rochelle salt, the patient became ill within three minutes and suffered from severe pain in the œsophagus and stomach, vomiting, and partial unconsciousness; later, purging, and severe pains in the loins and back. He recovered. The same dose, however, proved fatal in another case. A young woman took by mistake, for Epsom salts, a quantity of salts of sorrel. She died before assistance could be obtained. One case recovered, following prompt treatment, after 1 ounce had been taken.

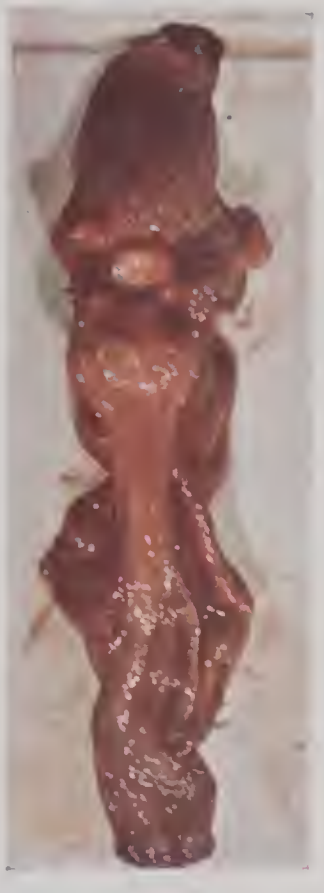


Fig. 219

Tongue, larynx, and œsophagus, in a case of potassium oxalate poisoning.

In a recent case, a woman of thirty-nine, took, in error for a stomach powder, a quantity of salts of lemon which she dissolved in milk. Thereafter she vomited, perspired freely, and complained of burning pain in the epigastrium and œsophagus. The woman died within an hour of taking the poison. Post-mortem examination showed that the palate, and the edges of the tongue and their under surfaces were of white appearance. The pharynx was congested and contained clear tenacious mucus. The œsophageal mucosa was of slate colour and there was patchy exfoliation. The outer surface of the stomach was inflamed and the organ contained a moderate quantity of dark coloured fluid. A brownish-black gelatinous substance covered the gastric mucosa and was composed of mucus and altered blood. The mucosa was inflamed and some corroded and softened patches were visible. The lining of the duodenum showed some inflammation. The amount of the poison consumed was not known.

Chemical tests (see p. 719).

Acetic acid.

In the glacial form, acetic acid acts as a corrosive, but as dilute acid, simply as an irritant. Some cases of poisoning from the glacial form have been recorded.

Symptoms.

The parts with which the acid comes into contact are softened,

and rendered a whitish or pale yellowish colour. Laryngeal complications, because of the volatility of the acid, are commonly found in such cases. Vomiting is usual. The action, locally, is therefore that of a corrosive, and such effects are apparent in the mouth.

The Annual Report of the Chief Medical Officer of the Ministry of Health for the year 1937 refers to cases of acetic acid poisoning. In one case, a man, who in a restaurant took a quantity of vinegar which was labelled "Highly Concentrated Vinegar," died soon afterwards. The contents of the bottle consisted of approximately 60 per cent. acetic acid. If the directions on the label had been followed, the contents should have been diluted to about one part in thirteen, prior to use.

Treatment.

The stomach should be washed out, and an alkali, such as lime-water, chalk, or magnesia administered. Milk or olive oil may be used as demulcents. Special symptoms should be treated as they arise.

Post-mortem appearances.

The mucous membrane of the œsophagus and stomach will be affected in varying degree, and there may be appearances of corrosive action. The upper air-passages will show marked congestion.

Fatal period.

This is variable.

Fatal dose.

One drachm of the glacial acid has killed a child, but an adult has recovered after taking 6 fluid ounces.

Chemical tests (see p. 702).

Carbolic acid, or phenol.

This substance is obtained by the action of nitrous acid on aniline, by the dry distillation of salicylic acid, and by the dry distillation of coal. It is a popular disinfectant, and by reason of its common use, has given rise, suicidally and accidentally, to more deaths than any other poisonous substance.

When pure, the acid consists of long, colourless, prismatic, needle-like crystals, which have a burning, sweetish taste. It is slightly soluble in water (1 in 11), but is freely soluble in glycerine, ether, alcohol, and benzene. Although termed an acid on account of the form of its composition, unlike other acids it does not redden litmus paper. In concentrated form it coagulates albumin. If the pure crystals are exposed for some time to air, they become pinkish-red. For sanitary purposes, however, it is usually sold in a crude form, as a dark-coloured liquid, which, like the purer forms,

possesses a characteristic pungent, penetrating odour, but which consists of a mixture of carbolic acid, cresylic acid, and other derivatives, as ortho-, meta- and para-cresol. The preparations are sold as Jeyes' Disinfecting Fluid, Izal, and as many other compounds. Cresols are the homologues of phenol and are also poisonous. Crude cresol (cresyl oil or cresylic acid) may be used as a disinfectant instead of phenol and is employed in the preparation of numerous products such as cresol soap or saponified cresol and creoline. Lysol consists of 50 per cent. v/v of solution of cresol in a saponaceous solvent, such as saponified linseed oil. Carbolic acid acts as a mild corrosive and anæsthetic upon the skin and mucous membranes when in the crystalline form or in strong solutions. It gives a white, bleached, and puckered appearance to the skin at the point of application, the epidermis is destroyed, and a yellowish-brown, or brown staining results. It sometimes produces gangrene when applied as a dressing to the fingers and toes. Apart from its local action, it also produces a remote action on the central nervous system.

FIG. 220

Corrosion affecting lower lip and chin in a case of lysol poisoning. Bridge of nose also affected by contamination from edge of drinking vessel.

Symptoms.

These are intense burning pain in the mouth, throat, and stomach, vomiting of frothy mucus, which may be neither severe nor continuous, coldness and clamminess of the skin, contraction of the pupils, and lividity of the lips. Stertorous, hurried, or laboured breathing, a small, thready pulse, subnormal temperature, and early onset of insensibility are also prominent manifestations. In many cases, the odour of the acid may be detected in the breath, though not in every case. There may be signs of corrosion on the lips and at the corners of the mouth, the marks being of a pale brown or yellowish colour. The symptoms depend in some measure upon the concentration swallowed. If a strong solution has been taken, death may follow quickly, with or without vomiting, and with rapidly intervening coma and stertorous breathing, but if a dilute solution has been used, the symptoms supervene only after absorption and are not usually so rapid in onset. It should be remembered that, owing to the local anæsthetic action of this acid, symptoms of irritation, such as pain and vomiting, are not so prominent as in poisoning by oxalic acid or the mineral acids.

Death usually results from respiratory or cardiac failure due to paralysis of the respiratory or cardiac centre.

Treatment.

The objects of the treatment are first, to limit the absorption of the poison; secondly, to sustain the patient; and thirdly, to aid the elimination of the poison from the system. Thorough gastric lavage, using a 10 per cent. solution of glycerine in water or plain water, should be carried out without delay and continued until the washings no longer emit an odour. When lavage has been completed, a quantity of medicinal liquid paraffin should be left in the stomach, and since it is not absorbed it is preferable to vegetable or animal oils. Egg albumen, since it precipitates phenol, will delay absorption. Caffeine may be used as a respiratory stimulant, and for continued cardiac action strophanthus has been strongly recommended. Other treatment should be along general lines. Intravenous saline-glucose may prove useful in certain cases. When necessary, rectal feeding should be adopted for some days.

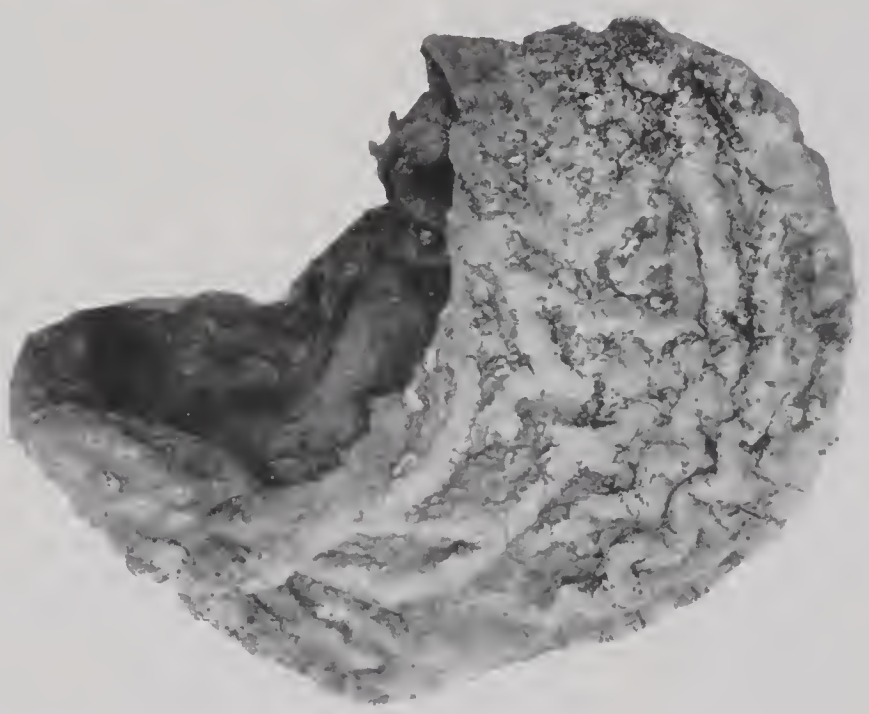


FIG. 221

Gastric mucosa in a case of carbolic acid poisoning.

Post-mortem appearances.

Those parts of the skin surrounding the mouth, and the mucous membrane of lips and mouth, with which the poison has come into contact, show a pale brown or yellowish staining. This, however, will depend upon the strength of the poison used, for example, a 1 per cent. solution produces no effect whatever, a 2 per cent. solution a slight staining, only observable on careful examination, a 4 to 5 per cent. solution, a whitish discoloration, which is likely

to disappear within six hours, whereas a strong solution of the acid or the pure acid causes a white slough. When impure carbolic solutions are swallowed the colour of the stain will be more or less affected. The mucous membranes of the œsophagus and stomach are also more or less similarly affected. The action of carbolic acid upon the gastric mucosa is characteristic. The mucous membrane forms projecting folds, is more or less brownish in colour, and looks leathery. On opening the abdominal cavity, the odour of phenol can usually be detected, owing to the fact that after death some of the acid passes through the walls of the stomach. For this reason, the peritoneal covering of the stomach is injected. A varying portion of the upper part of the small intestine may show evidence of corrosion and injection of the vessels. The odour of the acid is, however, most marked on opening the stomach, and its contents may be found to consist of blood-stained mucus. When a fairly crude cresol preparation has been swallowed the odour may prove very misleading, since it is atypical and may suggest the presence of creosote due to the cresylic acid. We have noted this on a few occasions. In one case, in which this odour was quite aggressive in character, chemical analysis showed the presence of 44·7 grains of cresol in the stomach. The urine, which emitted a similar odour, contained 1·8 grains. The lungs, the abdominal viscera, and brain, are frequently congested. The urine is of greenish-black colour, said to be due to the presence of pyrocatechin and hydroquinone.

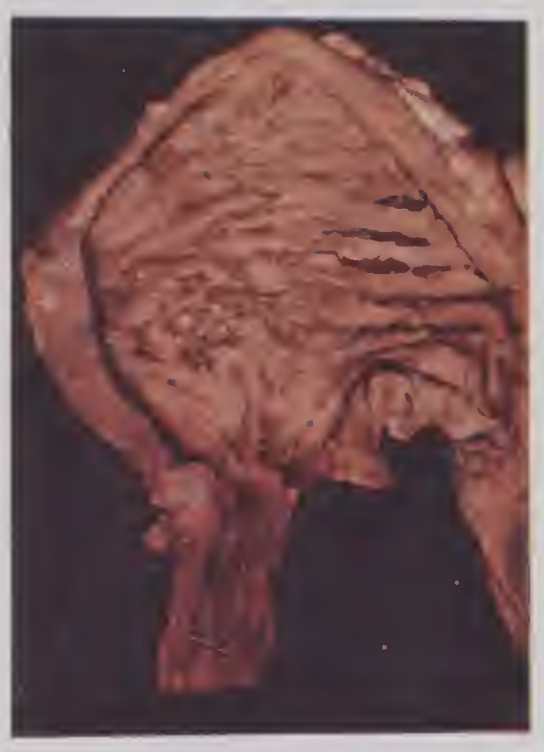


Fig. 222

Gastric mucosa in a case of lysol poisoning.

Death may result from carbolic acid due to its absorption through the unbroken skin, from the effects of inhalation of its fumes, or from rectal and vaginal injections.

Miller[3] reports the case of a youth, aged eighteen, suffering from ringworm on the right shoulder, the scapular region, and over the left arm and trunk, who had a mixture of camphor and carbolic acid rubbed over the affected areas. Before this procedure was completed the youth became faint and giddy. The mixture was diluted and the application continued. The youth then became dyspnœic, staggered to the floor, and was helped to bed, where he died within fifteen minutes after the commencement of the application. Post-mortem examination showed that both

lungs were congested and in a state of acute pulmonary œdema. The mouth was filled with frothy blood.

Fatal period.

Death from carbolic acid usually occurs within three hours after the poison has been taken. It has, however, followed in three minutes, and it has been retarded for sixty hours.

Fatal dose.

One drachm has killed in twelve hours. Recovery, however, has followed large doses; in one case, from 1 ounce of 90 per cent. phenol, in another, from 6 ounces of crude carbolic acid, and in a third, of a child aged two, from ½ ounce of crude acid containing 30 per cent. carbolic acid.

Recovery, after prompt treatment, has followed the swallowing of about a pint of Jeyes' fluid. Creolin and lysol also produce toxic effects. Many cases of fatal poisoning have been recorded.

The toxicity of carbolic acid is about eight times greater than that of lysol.

Chemical tests (see p. 719).

CORROSIVES—CAUSTIC SALTS

There are certain salts of different elements, for example, antimony, copper, zinc, silver, and compounds of chromic acid, which possess caustic properties.

Antimony trichloride.

The trichloride, or "butter of antimony," used for certain trade purposes, and in veterinary practice, has been taken both accidentally and for suicidal purposes. It is a light brown, dark brown, or dark red coloured liquid, and possesses marked caustic properties. The symptoms produced are more markedly those of a corrosive than of an irritant. In the few cases which have been recorded, the prominent signs and symptoms were persistent vomiting, severe burning pain in the mouth, throat, œsophagus, and stomach, and general signs of collapse.

Evidence of corrosion of the parts with which the poison has come into contact, with denudation of the mucous membranes, may be found post-mortem.

The amount which constitutes a fatal dose has not been established with any degree of precision. Taylor records a case in which an army surgeon took, suicidally, from 2 to 3 fluid ounces, and died. A boy of ten years, however, has recovered after taking about ½ ounce.

Chemical tests (see p. 707).

Copper sulphate.

This in the solid form, or in very concentrated solution, exercises a limited corrosive effect, but its action is mainly that of an irritant.

A child aged three swallowed about fifty tablets containing ferrous sulphate and a trace of copper sulphate and as a result died. The maximum amount of copper swallowed was about 2 grains and with vomiting the amount absorbed would be less. The cause of death was acute yellow atrophy of the liver. The explanation for so small a quantity of copper causing death was that in the tablets the copper sulphate was mixed with a relatively large amount of inert material which would prevent evacuation by purgation[4] (see p. 566).

Chemical tests (see p. 714).

Zinc chloride.

This as a solid, or in concentrated solution, acts corrosively, and the signs and symptoms are chiefly those of a corrosive poison. When taken by the mouth, there are frequently no signs of corrosion of the mouth, pharynx, or œsophagus, although there may be evidence of corrosion in the stomach. Its degree of absorption is relatively slight.

Chemical tests (see p. 724).

Silver nitrate.

This likewise is corrosive both in solid and liquid form. A fatal case is recorded in which a piece of solid stick nitrate became lodged in the throat while the tonsil was being cauterised.

Chemical tests (see p. 722).

Chromic acid and **Potassium** and **Sodium chromates** are also corrosive in action when in solid, and concentrated liquid, forms. Diluted, they act as irritant poisons, having a special action upon the nervous system. In chrome workers, the injuries may assume the form of skin lesions, such as acne, eczema, burns or perforations, called "chrome-holes" by the workmen, eye lesions, and nasal lesions, especially atrophy of the mucosa, and ulceration or perforation of the septum. Chrome ulceration is notifiable and its occurrence must be reported to the Home Office (see p. 33).

Chemical tests (see p. 713).

References

1. Brit. Med. Jour., Vol. II, 617, 1902.
2. Lancet, Vol. II, 413, 1933, Willimott.
3. Canad. Med. Ass. Jour., Vol. XLVI, 615, 1942, Miller.
4. Pharm. Jour., 156, 321, 1946.

CHAPTER XIX

METALLIC AND NON-METALLIC IRRITANTS

General action of metallic and non-metallic irritants.

THE onset of signs and symptoms, which is variable, is usually within half an hour to an hour. They are indicative of gastro-intestinal irritation, and consist of severe pain over the epigastrium and abdomen, associated with, or followed by, continuous and painful vomiting and diarrhœa. The vomited matter at first consists of normal stomach contents, but later becomes bilious in character and may be of a "coffee-grounds" appearance due to altered blood. Over the track of the œsophagus there is a feeling of heat and constriction which provokes thirst, but drinking only induces further vomiting. The diarrhœa consisting at first of loose stools, and later of stools mixed with blood, accompanied by tenesmus, is severe and urgent. After some time the patient begins to show signs of shock, the pulse becomes thready and irregular, the skin clammy and cold, and cramps may affect the muscles of the limbs. During this period the mind of the patient remains clear, but before death unconsciousness, preceded or succeeded by convulsions, usually heralds the fatal issue. The period before death is variable, depending upon the quantity of poison taken, and the condition of the patient at that time. Death may be due to cardiac failure through involvement of the myocardium, by the direct action of the poison, or indirectly by the effects of general prostration.

Fluid is lost from the blood which becomes concentrated, and the blood-pressure falls. The fluid loss is due to vomiting, diarrhœa, sweating, and inability to drink.

Post-mortem appearances.

These consist of inflammatory changes chiefly in the stomach, and frequently in the duodenal and rectal portions of the intestinal tract, especially in the latter.

Arsenic.

Arsenious acid, arsenious oxide, or white arsenic, is the substance most commonly employed in homicidal poisoning. As met with in commerce, it is either in the form of cakes, broken

lumps, or a white, gritty powder. It is very insoluble in cold water, which will only dissolve $\frac{1}{2}$ to 1 grain per ounce. When, however, it is mixed with boiling water and is boiled for some time, about 55 grains per ounce will be held in solution so long as the water remains hot, but after the water has become cold only about 12 grains per ounce remain dissolved, the remaining 43 grains being deposited in the vessel. By reason of this insolubility, when the fine powder is placed in water or other liquid, some of it forms a whitish film on the surface of the fluid, and the heavier particles fall to the bottom of the vessel. Experiments have shown that 100 grains of arsenious acid mixed with two teaspoonfuls of cocoa, milk, and boiling water in a teacup, could not be detected either by appearance, taste, or smell, but that on standing, the milk curdled and the arsenic sedimented. With arrowroot and gruel similar results were observed.

Inorganic arsenic.

Arsenic is used extensively in industry for dyeing, artificial flower-making, taxidermy, and paper-making. It is used therapeutically, also, for a variety of purposes. It is sold as a constituent of vermin-killers and of fly-papers. In medicine, and in fly-papers, the arsenic usually exists in the form of sodium or potassium arsenite, since arsenious acid is soluble in alkaline solutions. Arsenic in the form of a soluble sodium arsenite is a common constituent of weed-killers, and cases of poisoning by weed-killer have often occurred. It is an occasional ingredient of insecticide solutions used for spraying fruit trees. Arsenic may also be found as an accidental adulterant of many substances. It has also been found in glycerine, and local authorities have been advised, by the Royal Arsenical Commission, to take action where arsenic is found to exist in glycerine over $\frac{1}{100}$ grain per pound. For arsenic in relation to food poisoning, see p. 690. Sheep dips may also contain arsenic (see p. 509).

Fatal toxic effects may also be produced by arsenic in the gaseous form of arseniuretted hydrogen (see p. 578). Arsenical poisoning in industry is usually caused by exposure to dust of the solid compounds, or to arseniuretted hydrogen gas. Industrial arsenical poisoning is notifiable, and its occurrence must be reported to the Home Office (see p. 33).

Symptoms of poisoning.

Acute.

The intensity of the symptoms is regulated to some extent by the physical form in which the arsenic is taken, whether in solid form, in suspension, or in solution, and by the quantity ingested. When the amount taken is a large one it is suggestive of suicide or accident rather than homicide. The onset of symptoms is also regulated by these and other factors, including whether or not a meal has been recently taken. Usually, however, the symptoms appear

within about an hour, although in some cases, where even large doses have been given, they may be tardy in appearing. In one case, a drachm of powdered arsenious acid was taken when the stomach was empty and symptoms did not appear until after two hours. In a second case, where the quantity was similar, the symptoms did not develop for seven hours, while in a third case, ten hours intervened before symptoms appeared. When the poison is introduced into the rectum or vagina, days may elapse before the symptoms manifest themselves.

The symptoms of poisoning are often initiated by nausea, faintness, and a burning pain in the stomach and epigastrium, which is increased by pressure. These are followed by retching and vomiting, which become severe, continuous, and persistent. The vomited material varies in appearance during the course of a case. At first, it consists of stomach contents, but later becomes blackish or greenish in colour, due to the presence of bile, and latterly consists of mucus mixed with altered blood in varying quantity. Insistent and painful diarrhœa, accompanied by tenesmus, is common, the stools being streaked or tinged with blood; indeed, they may be almost completely composed of blood. The patient may complain of a sense of oppression, coldness, and collapse. There is usually an intense thirst, the gratification of which, however, only increases the vomiting. Painful cramps may be present in the muscles of the lower limbs. The pulse becomes small, rapid, feeble, irregular, and almost imperceptible, the face appears anxious and pinched, the skin becomes cold and clammy, and death is usually preceded by restlessness, convulsions, or coma, although it may supervene suddenly from cardiac failure. When successive sublethal doses of arsenic have been taken, the clinical picture of the patient may be one of subacute or chronic poisoning.

In subacute poisoning the symptoms are less intense.

Chronic.

When small repeated toxic doses of arsenic have been taken over a period, or when several sublethal doses, with short intervals between them, have been given, symptoms of chronic poisoning are likely to appear. Such doses are highly suggestive of homicidal administration. The general symptoms are remarkably constant in their character. Probably one of the earliest signs is gradual loss of weight due to malnutrition, and accompanying this there may be falling out of the hair and pigmentation of the skin. The eyes are suffused and watery, the conjunctivæ being congested, and there are symptoms of coryza. The tongue is either reddened or is covered with a thick white coating. Eczematous eruptions may be present on the body, usually accompanied by increased pigmentation of the skin which may assume the form of dark brown or blackish patches, thought to be due to a melanin derivative, or the skin may assume an earthy or jaundiced appearance. The patient will probably complain of general malaise, nausea,

disinclination for food, and diarrhœa or diarrhœa alternating with constipation. There may be intermittent vomiting. There is frequent complaint of numbness of the hands and feet, and the patient may walk with an unsteady and uncertain gait, if able to walk at all. These symptoms are due to peripheral neuritis. The palmar surfaces of the hands and the plantar surfaces of the feet are frequently the seats of hyperkeratosis. There may also be mental hebetude, or delusions.

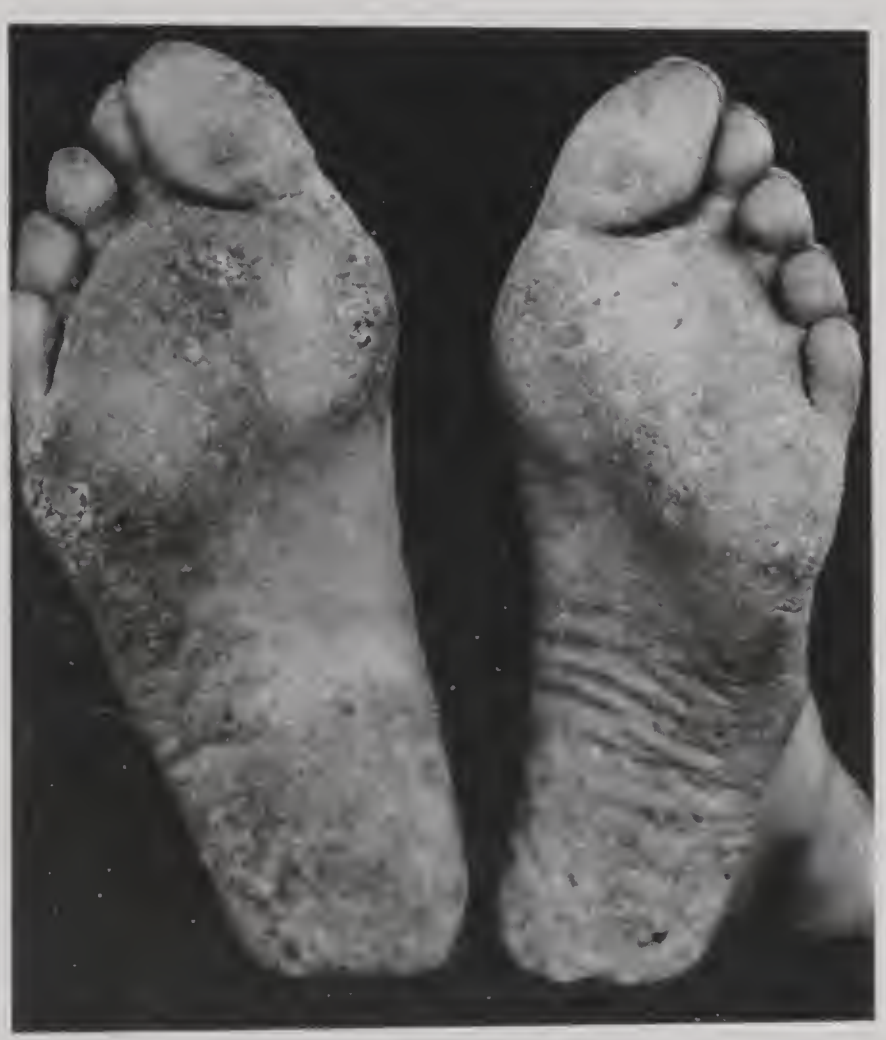


Fig. 223

Chronic arsenical poisoning showing hyperkeratosis.

Organic arsenic.

Organic preparations of arsenic in combination with a carbon atom are also poisonous, but less toxic than inorganic arsenical preparations. Cacodylic acid, although containing 54·3 per cent. of arsenic, equivalent to 71·6 per cent. of arsenious acid, is relatively non-toxic, as are also its salts or cacodylates.

There are numerous organic arsenical preparations available for therapeutic treatment, which consist of arsenic either in trivalent or pentavalent compounds. Sodium cacodylate and tryparsamide

are examples of the second class. The trivalent forms are the more potent for protozoa, and the most commonly employed of these preparations are arsphenamine and neo-arsphenamine. The proprietary names of these substances in common use include neo-salvarsan, novarseno-billon, or N.A.B., mapharside, neo-kharsivan, acetylarsan, and stabilarsan.

These preparations are used in the treatment of such diseases as syphilis, neuro-syphilis, pyorrhœa, yaws, rat-bite fever, and anthrax. Such treatment sometimes gives rise to symptoms of arsenical poisoning and fatal cases occasionally occur. As the result of exhibition of these drugs, there is a liability to occurrence of jaundice, especially in sensitive subjects. As a rule, the arsenic is stored in the body tissues, is slowly disintegrated and excreted without manifestation of toxic symptoms.

The usual method of administering these organic preparations is by the intravenous route. In the treatment of syphilis with arsphenamines, either mercury or bismuth is given as simultaneous or alternating treatment. Deaths have followed the administration of arsphenamines. Should an immediate collapse follow the use of these drugs, a few minims of adrenalin hydrochloride should at once be injected subcutaneously or ephedrine should be administered orally. The toxic symptoms which may follow the injection of salvarsan are rigors, pyrexia, reaching to 104° F. or higher, prostration, headache, giddiness, faintness, vomiting, colic and diarrhœa, loss of appetite, rapidity of pulse, cardiac symptoms, and dyspnœa. Jaundice is rarely severe, but acute yellow atrophy of the liver may occur and cause death. There may also be dryness and irritation of the throat and larynx, psychic and motor restlessness, severe sweating, conjunctivitis, salivation, skin eruptions, including urticaria and erythema, and extensive exfoliative dermatitis which may prove fatal. Encephalitis hæmorrhagica may develop two to three days after an injection, with headaches, vomiting and convulsions, and these are followed by coma and death a day or two later. For the treatment of jaundice, intravenous injections of 25 to 50 millilitres of 25 per cent. glucose should be given daily, or hepolon may be used. For dermatitis, sodium thiosulphate is given intravenously or intramuscularly, 5 to 15 grains on alternate days, with 25 millilitres of 25 per cent. solution of dextrose on each of the intervening days. More recently preparations of vitamin C have been used with reported success in the treatment of dermatitis due to arsphenamine.

British Anti-Lewisite (B.A.L.), dimercaptopropanol, is now employed as an arsenical antidote in cases of exfoliative dermatitis. It has been shown that the mean duration from the first injection to the time when healing was complete, or nearly complete, was twenty-one days. The therapeutic dose in this country has been 1·5 mg./kg. One preparation of B.A.L. consists of a sterile 5 per cent. solution of 2. 3-dimercaptopropanol in arachis oil containing 10 per cent. benzol benzoate. The substance is administered by deep intramuscular injection into the thigh or gluteal region.

B.A.L. is contraindicated in the treatment of post-arsphenamine hepatitis.

In one of our cases, exfoliative dermatitis involved almost the entire body surface with fatal termination.

The precise cause of arsenical dermatitis is not clearly established although there is usually a systemic background of predisposing causes, one or more of which may contribute to its production by an exciting cause. Osborne has suggested that the probable cause is the deposition of arsenic about the capillaries of the skin. Even with the greatest care, arsenical dermatitis is sometimes an unavoidable complication.

Tryparsamide is frequently used in the treatment of neurosyphilis, but owing to its recognised ability to produce optic atrophy, a contraindication to its use is the presence of optic neuritis.

Salvarsan and analogous substances used for the specific treatment of infective disease are included in the Therapeutic Substances Act, 1925, and Regulations (see p. 514).

Elimination of arsenic from the body.

Arsenic, quickly absorbed, is stored in the liver, and from there passes into the general circulation. It is slowly excreted by the urine, fæces, and sweat. Although the intestinal tract eliminates some of the arsenic from the body, the kidneys are the principal excretory organs. After a single dose, arsenic may be found in the urine in less than an hour and may continue to be present for about ten to fourteen days or even longer. After a series of doses, excretion of arsenic may persist for about three months. The presence of arsenic in the urine may be intermittent. Absorbed arsenic is found in greatest quantity in the liver, and in the course of a few days produces fatty changes. In two to three weeks following recovery from a single dose, all traces of the poison may have left the body with the exception of the hair, nails, skin, and bones. Arsenic will probably not be found in the hair until about a week after ingestion, but its excretion into the hair may continue for long periods even after all traces have left the organs. The amount of the poison found in different parts of the intestinal tract is highly important in fatal cases in relation to the interval of time which has elapsed between ingestion and death. When it is present in the upper part of the small intestine, the interval would be about three to six hours approximately. About ten hours would be necessary for it to reach the lower part of the small intestine and about twelve hours to reach the cæcum. It must not be forgotten that arsenic, by its irritant action, hastens peristalsis.

The hair and nails show a selective absorption, and an irreversible fixation of arsenic from the blood stream. Since these keratin tissues are slow in growth, the portions that are formed during the time the arsenic is being excreted will retain arsenic. Hair grows at the rate of about $\frac{1}{2}$ inch a month and it has been stated that the weekly

growth is from 1 to 3 millimetres. The rate of growth of the nails is about $\frac{1}{8}$ inch a month. Sir E. Sharpey-Schaefer [1] investigated the rate of growth of nails and found that the rate is faster on the right hand than on the left, both in winter and summer. He also found that there was a great variation in the rate of growth both in individuals and in the several finger-nails. Thus the trapping of arsenic in these keratin structures enables the analysis of successive short lengths of hair, from the base to the tip, and of portions of the nails, from the base to the tip, to give an approximate indication of arsenic dosage or the intermittent periods of such administration. It is necessary to ensure that these structures have not been contaminated by arsenical fluids from the body from which they have been removed or, when exhumation has taken place, by contamination of the soil. Both hair and nails should be cleansed prior to analysis.

Hair when soaked in an arsenical solution is capable of absorbing arsenic, but such absorption will show a more or less even distribution as opposed to natural excretion into growing hair, when, if the administration has been intermittent, the deposit of arsenic will be irregular in distribution. Experiments have shown that arsenic which has been excreted into the hair cannot be removed by prolonged steeping in cleansing fluids.

Normal presence of arsenic in human tissues.

Arsenic is so universally found that it may frequently be present in the human body, although in very minute amounts. Authorities differ as to the presence of minute amounts as normal constituents of human viscera, but the prevailing view is that arsenic is not a normal constituent of the human body. On the other hand, small quantities from innocent sources may be detected in a body after death. In order, therefore, to establish that death resulted from arsenical poisoning, the history of the symptomatology prior to death must receive careful consideration, and there must be a sensible amount of arsenic isolated.

The procedure adopted by the Director of the Chemical Laboratory, The Medico-Legal Institute, Cairo, is that when no part of the viscera or discharges contains more than $\frac{1}{50}$ milligram in 100 grammes of material, the chemist, with the consent of the Director, may consider the case as negative, and may report accordingly, although he will keep a record of the actual quantity found. When the greatest quantity detected is less than $\frac{1}{10}$ milligram per 100 grammes, but more than $\frac{1}{50}$ milligram, the chemist must consult the medico-legal expert in charge of the case and, if he agrees that there is no definite evidence of arsenical poisoning, the chemist will state in his report that only negligible traces of arsenic were detected. Quantities in excess of those mentioned are recorded in the report following analysis. Arsenic is generally present in varying quantity in Egyptian soil.

The administration of arsenic on therapeutic grounds may be a

cause for arsenic found in a body after death, and this possibility must receive attention.

Treatment.

The stomach should be emptied without delay, either by the use of an emetic or preferably by a stomach tube. Gastric lavage must be thorough, and for the purpose about 2 gallons of water should be employed. Precipitated ferric hydroxide should first be added to the fluid. This is prepared by adding sodium carbonate to 2 ounces of a solution of ferric chloride until effervescence has ceased. The fluid should then be filtered and the precipitate used. Four ounces of arsenic antidote (Antidotum Arsenum B.P.C.) should be left in the stomach and the dose repeated if necessary. To prepare this substance, two solutions should be ready for use, namely, a strong solution of ferric chloride (288 minims) mixed with $2\frac{1}{2}$ ounces of water, and also light magnesium oxide ($87\frac{1}{2}$ grains) triturated to a smooth paste with water and diluted to 15 ounces. Before being employed, $3\frac{3}{4}$ ounces of the suspension of magnesium oxide, after shaking, should be added to 400 minims of the ferric chloride solution.[2] As a substitute, 1 ounce of tincture of ferric chloride in 4 ounces of water with 1 ounce of sodium carbonate may be given. The object of the foregoing treatment is to form insoluble ferric arsenite in the stomach. The use of intravenous sodium thiosulphate has been advocated. The amount recommended, in the acute stage of poisoning, is 15 grains in 10 millilitres of sterile distilled water every four to six hours during the first twenty-four hours. In the treatment of chronic arsenical poisoning a similar injection should be given two to three times per week over a period of weeks. In acute cases, measures should be directed to combat shock, and adequate warmth should be maintained. On account of possible dehydration, physiological saline should be given intravenously whenever indication arises. Five per cent. glucose solution may also be administered intravenously. Castor oil or magnesium sulphate may be used when vomiting subsides. Demulcent drinks may be given freely. Caffeine sodium benzoate (2 grains), hypodermically, should be exhibited when collapse threatens, and $\frac{1}{4}$ grain of morphia may be given hypodermically to alleviate pain. Additional symptoms should be treated along general lines.

Post-mortem appearances.

The character of these appearances depends very largely upon the quantity taken and the period which has elapsed between ingestion and the occurrence of death. The stomach is primarily the seat of post-mortem appearances, although the poison may have entered the body by means other than the mouth. When the poison has been swallowed the mucous membrane will show a varying degree of inflammation, with injection of the vessels, which is patchy in distribution and corresponds to the deposits of arsenic, round which the inflammatory changes centre. Arsenious oxide

is converted into the yellow sulphide in the stomach and particles may be observed either with or without the aid of a magnifying glass. When seen, certain of the particles should be collected for subsequent chemical examination. The surface of the membrane may be thinly coated with mucus and streaked with blood. Submucous petechial hæmorrhages are very often seen. The contents of the stomach are frequently of a darkish-brown colour. The cardiac and pyloric ends of the stomach are often congested. Congestion of the wall of the intestinal tract is frequently found, and the tract as a whole may contain a considerable quantity of dark fluid. Congestion of the vessels of the lining membrane of the pharynx and larynx is also common. Subendocardial hæmorrhages in the left ventricle are rather typical. The liver is very often enlarged and pale in colour, due to fatty degenerative changes. The kidneys and myocardium are similarly affected in many cases.

The above appearances persist for variable intervals after death, and arsenic may be recovered from the stomach after relatively long periods, despite vomiting, due to its tenacious association with the gastric mucosa.

While the foregoing may be taken as descriptive of the appearances in the average fatal case when the poison has been ingested, their incidence is but little changed no matter by what other channel the arsenic enters the body. They are found, for example, in cases of respiratory poisoning by arseniuretted hydrogen, and even when the arsenic has been introduced by the vagina.

The preservative influence of arsenic upon the tissues of those poisoned by this substance has been repeatedly observed and noted following exhumation, despite assertions to the contrary.

Fatal dose.

The smallest recorded fatal dose is 2 grains. Recovery has, however, occurred after large doses. A woman, intent on suicide, took about 230 grains of arsenious acid, was violently ill three hours later with vomiting, and seven hours after swallowing the poison, with watery diarrhœa. Despite these symptoms, her general condition remained fairly good. On the third day she developed a hæmatemesis and melæna, but on the fourth day improvement commenced, and her recovery was complete within a fortnight.

Fatal period.

Death has occurred in individual cases at the end of twenty minutes and ten hours, respectively, but the fatal period is very variable and may range from several hours to several days. In one case, for example, when 220 grains were swallowed, death did not ensue until the seventh day. Twelve to thirty-six hours, however, may be taken as an average period.

Chemical tests (see p. 708).

ILLUSTRATIVE CASE

The practical significance of many points, already described, will be clearly seen in the details of the following case.

Post-mortem findings.

The mucous membrane of the pharynx, epiglottis, and larynx was congested and the vessels were injected. Both chambers of the right side of the heart were considerably dilated. The left auricle was also dilated. A number of small subendocardial hæmorrhages were present in the left ventricle over an area situated immediately below the posterior cusp of the mitral valve. The liver was pale in colour and enlarged. The stomach contained about 1 pint of darkish, yellowish-brown fluid. There were numerous patchy groups of petechial hæmorrhages in the submucosa. These were most marked nearer the pyloric end of the stomach. The intestinal tract, as a whole, was more or less filled with darkish-brown fluid. The lining of the small intestine showed slight, generalised congestion. That of the large intestine showed more marked congestion which was patchy in distribution although generalised in extent. Microscopical examination of the liver and kidney tissue showed the presence of fatty degenerative changes.

The brain, stomach, stomach contents, intestinal tract, intestinal contents, liver, spleen, kidneys, pancreas, heart, adrenals, lungs, and also the urine and bile were retained for analysis. Subsequent exhumation of the body was made, to obtain additional samples of hair, nails, muscle tissue, skin, and bone. Specimens of earth adjacent to the coffin, together with a portion of the coffin and shroud covering the body were also taken.

Analytical examination.

A preliminary test by the Gutzeit method on portions of the liver and kidney, and a mixed sample of stomach tissue and stomach contents showed the presence of a large quantity of arsenic. This was confirmed by a Reinsch test, and crystals of arsenious oxide were obtained by sublimation.

An investigation was then carried out to ascertain if sufficient arsenic was present to raise the presumption that death was due to arsenical poisoning.

To determine the distribution of the arsenic throughout the body, representative portions of all the specimens were heated with nitric and sulphuric acids to destroy the organic matter, and an estimation of the arsenic was made by the Gutzeit method (see p. 709), often in triplicate, on aliquot portions of resulting solutions, every precaution being taken throughout the long series of experiments to ensure accuracy. All apparatus and chemicals used were rigorously tested, blank determinations accompanying each batch of specimens analysed. The large amount of arsenic present in the stomach and intestinal contents permitted a gravimetric estima-

tion being carried out and complete agreement was obtained with the results of the Gutzeit estimations. The distribution of the arsenic in the various tissues and fluids is given in the following table:—

	Total weight received.		Arsenic in tissues, etc.	
			Arsenic in total weight of organ submitted.	Expressed as parts per million.
	Lbs.	Ozs.	Grains.	
Brain	3	1·0	0·018	0·9
Stomach tissue	...	13·0	0·143	25·0
Stomach contents	...	14·0	5·18	861·0
Intestinal tissue	3	6·0	1·29	54·0
Intestinal contents	1	12·0	5·88	486·0
Liver	4	3·0	2·33	80·0
Spleen	...	4·0	0·009	5·8
Kidney	...	9·0	0·103	27·0
Pancreas	...	4·0	0·002	1·2
Heart	...	12·0	0·011	2·0
Adrenals	...	0·6	0·003	9·6
Lungs	1	3·0	0·026	3·2
Urine	...	0·6	0·004	15·0
Bile	...	1·0	0·003	5·7

All results throughout are expressed as arsenious oxide.

Microscopical examination of the stomach and intestinal contents showed the presence of arsenious oxide crystals. Portions were centrifuged, selected particles picked out, and identified as arsenious oxide by chemical and microscopical means.

In view of the large quantity of arsenic in the stomach and intestinal contents, it was considered advisable to isolate at least a poisonous dose of arsenious oxide. The difficulties likely to be encountered in carrying this out by a mechanical process are almost insuperable, and a chemical process was therefore adopted, by which 5·47 grains of arsenious oxide were isolated from approximately half the mixed stomach and intestinal contents, a quantity considerably in excess of what is commonly accepted as a poisonous dose.

It will be observed from the foregoing table, showing the distribution of the arsenic in the various tissues, that the average arsenious oxide content of the whole intestinal tract was 54 parts per million.

As some portions of the intestinal tract showed a greater degree of inflammation than others, a further examination was made.

Selected portions which showed definite inflammation were removed for further examination. These portions had been in contact with the intestinal contents for a period of approximately forty days after removal from the body. The selected inflamed portions of the tract were found on analysis to contain the following amounts of arsenic :—

1. Terminal, 5 inches—lower portion of pelvic colon	75	parts per million.
2. First portion of ascending colon	80	,, ,, ,,
3. Ileum, 39 inches	83	,, ,, ,,

To determine whether any post-mortem absorption of arsenic by the tissues had occurred the following experiment was carried out.

Portions of intestinal tissue were obtained from a post-mortem examination of a person who had died from causes other than arsenical poisoning. These were tested for the presence of arsenic, with negative results. The remainder of the intestinal tissue was then immersed for forty days in the intestinal contents obtained from the body of the deceased, and in which arsenic was present. At the end of that period the arsenic found in the tissue was as follows :—

1. Descending colon	8·3	parts per million.
2. Ascending colon	8·7	,, ,, ,,
3. Middle portion of small intestine	6·5	,, ,, ,,

While no general conclusion can be drawn from these results, it is evident that in this particular case, although some post-mortem absorption had taken place, it occurred only to the degree shown, and was very much less than the arsenic found in the intestinal tissues of the deceased at the end of a period of forty days.

Forty-seven days after death the body was exhumed and the following specimens were submitted for analysis :—

Earth adjacent to the coffin, portions of the shroud and coffin lining, head hair, portions of the left femur, muscle and skin overlying the left femur, skin from the soles of the feet, and the nails from the hands and feet.

Weighed portions of the earth were boiled with (*a*) 25 per cent. hydrochloric acid, and (*b*) water, the respective solutions being filtered and the amount of arsenic determined in the filtrate. In the water extract arsenic was absent, while in the hydrochloric acid solution only a trace, amounting to 0·25 parts per million, was present.

The shroud and coffin lining were damp and discoloured, and these were not washed or dried. Arsenic was found to be absent.

The hair, in two bundles, was wet, and showed no evidence of the presence of crystals of arsenious oxide microscopically.

The larger bundle, which measured $3\frac{1}{2}$ inches in length, was divided into three portions, each being washed three times with water. These washings were free from arsenic.

The smaller bundle, which measured 2 inches, was divided into two portions of 1 inch, each being washed as before. The washings were arsenic free.

The whole of the left femur, except the uppermost portion, was utilised, and after removal of the marrow, the hard bone substance was washed with water. The bone and marrow were separately examined for arsenic.

The epidermis was separated from the dermis of each foot and each layer was examined separately.

Each finger-nail was examined microscopically for the presence of crystals of arsenious oxide, with negative results. The nails were cleaned and washed with water. Each nail was measured and divided into two equal portions transversely, a root end and a tip end. The root ends of the nails from both hands were then mixed, weighed, and the arsenic determined, the tip ends being similarly treated. The same procedure was adopted in the case of the nails of the feet.

The results are given in the following table :—

		Arsenic found (in parts per million).
Hair	(*a*) Scalp end—1 inch	10·0
	Middle portion—1 inch	3·8
	Free end—1½ inches	3·3
	(*b*) Scalp end—1 inch	10·0
	Free end—1 inch	8·0
Left femur	(*a*) Marrow	0·81
	(*b*) Hard bone substance	1·2
Left thigh	(*a*) Muscle	3·4
	(*b*) Overlying skin	2·8
Right foot	(*a*) Epidermis	5·0
	(*b*) Dermis	8·0
Left foot	(*a*) Epidermis	5·4
	(*b*) Dermis	5·4
Finger-nails	(*a*) Root end	57·0
	(*b*) Tip end	46·0
Toe-nails	(*a*) Root end	64·0
	(*b*) Tip end	46·0

In consequence of the results obtained in the examination of the specimens, some further investigations were made.

Twenty-one specimens of head hair were obtained from various patients who had died from causes other than arsenical poisoning, none of whom had received any form of arsenic during their illness prior to death. The average arsenic content of these specimens was 0·72 part per million. The arsenic content of fourteen of these specimens lay between 0·1 and 0·7 part per million, of five specimens, between 0·7 and 1·3 parts per million, and of two specimens, between 1·3 and 1·7 parts per million.

Nail clippings were also obtained from nine persons, all of whom were laboratory workers, and the average arsenic content was found to be 8 parts per million of the mixed clippings.

Opinions.

These included :—

The large amount of arsenic present in the entirely fluid contents of the stomach (5·18 grains) and especially the large amount in the entirely fluid contents of the intestine (5·88 grains), together with the amount in the liver (2·33 grains) were consistent with a massive dose of arsenic having been swallowed shortly before death, certainly within twenty-four hours. The presence of arsenic in the distal portions of the hair was consistent with the poison having been taken within a minimum of six months before death. The presence of arsenic in the proximal and distal portions of nails and proximal, middle, and distal portions of hair was consistent with it having been taken throughout the minimum period of six months before death. The relative amounts of arsenic present in the various portions of nails and hair were consistent with larger amounts having been taken towards the end of life.

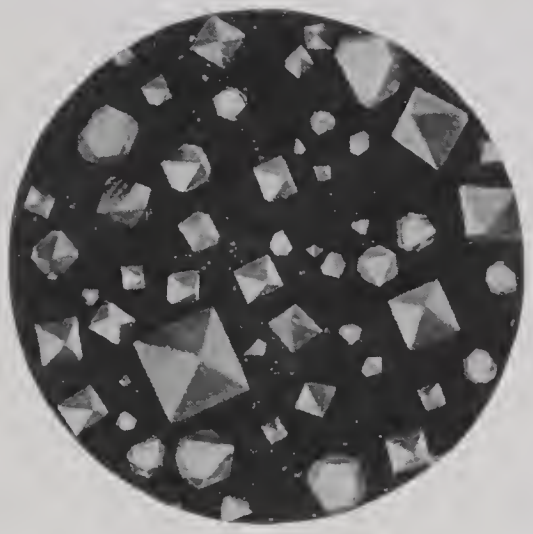


Fig. 224
Arsenious oxide crystals.

Antimony.

The principal salt of antimony with which cases of poisoning have been associated is antimony-potassium tartrate, or tartar emetic, which has a diaphoretic and emetic action. Tartar emetic occurs in the form of a whitish, or whitish-yellow powder, which contains between 33 and 35 per cent. of antimony. It is soluble in boiling water, one in three, is about six times less soluble in cold water, and is insoluble in alcohol. Intravenous administration is employed in the treatment of filariasis, bilharziasis, kala-azar and of certain other diseases.

Untoward results may follow an intravenous injection either as the result of susceptibility or of anaphylaxis. Asphyxial signs developed in the case of a woman following an intravenous injection of 1 grain in a 6 per cent. solution, although recovery took place following an intracardiac injection of adrenalin.

Trichloride of antimony is corrosive in action (see p. 537).

Acute poisoning.

The following signs and symptoms are common :—An astringent metallic taste in the mouth ; a sensation of heat and constriction in the throat ; dysphagia, to some extent ; gastric pain followed by vomiting of an incessant character and accompanied or succeeded

by diarrhœa; faintness and profound prostration: there may, or may not, be intense thirst; the pulse is small, rapid, and weak: the skin is covered with a cold, clammy perspiration: there may be cramps in the abdomen and limbs, which before death may assume almost a tetanic character: partial or complete coma usually precedes death.

Chronic poisoning.

The symptoms consist of marked nausea followed by vomiting, and sooner or later the stomach contents show the presence of bile-stained mucus; watery diarrhœa, or diarrhœa alternating with constipation; the pulse gradually becomes weak; there is progressive weakness and loss of weight; the patient develops a loathing for food, since vomiting is sometimes associated even with the sight of it; cachexia accompanied by prostration generally precedes death.

Treatment.

The first line of treatment is to empty the stomach with a stomach tube. If vomiting is not frequent, and when the tube is not available, aid the emesis by simple emetics, and give strong tea or tannic acid to form the insoluble tannate. When the tube is used, warm water alone is sufficient for lavage. Stimulants should be administered in the event of threatened collapse, and the patient should be kept warm. Sedatives should be used when improvement in the pulse and general condition indicate that the patient is out of immediate danger. Careful dieting is necessary for some time following recovery from the immediate effects of poisoning.

Post-mortem appearances.

Acute poisoning.

The mucous membranes of the pharynx, larynx, œsophagus, and stomach are congested. The gastric mucosa is frequently covered with tenacious mucus, and scattered, irregular areas of submucous petechial hæmorrhages may be seen. These are prevalent on the greater curvature and at the cardiac orifice, although they may be distributed generally. The mucous lining of the duodenum shares the same appearances. The contents of the stomach are usually scanty in amount, are dark in appearance, and sometimes blood-stained.

Chronic poisoning.

In chronic poisoning the findings may not be well marked. The tongue is usually dirty and the buccal mucous membrane may show aphthous patches. The gastro-intestinal tract will show evidence of irritation, and patches of ulceration may be found either in the stomach or intestines, or in both. There is considerable emaciation of the body.

Fatty degenerative changes involving the liver, kidneys, and myocardium are frequently found.

Fatal dose.

The amount is indefinite, and depends both upon the constitution of the individual and upon the fact that the emetic action of the drug readily induces vomiting. Ten grains have killed a boy of five, and a similar quantity a girl of three. However, recovery has followed doses of 60, 170, and over 400 grains.

Fatal period.

Sixty grains produced death in an adult in ten hours. In the boy and girl above mentioned, 10 grains proved fatal in eight and twelve hours, respectively.

Chemical tests (see p. 707).

Mercury.

Mercury was at one time freely used in the trade of mirror-silvering, but its use has been largely supplanted by other processes. It is still used, however, by furriers, felt hat manufacturers, and the makers of many scientific instruments, such as thermometers and barometers. The trade processes which expose to intoxication are numerous and include the preparation and use of lead, mercury, and solder, and the manufacture of the salts by the dry method.

Cases of industrial mercurial poisoning, some of them fatal, occur from time to time. Poisoning by mercury in industry is notifiable and its occurrence must be reported to the Home Office (see p. 33). When mercury enters the system in the metallic state it is comparatively innocuous unless in a finely divided form, when it will produce a toxic result. Inhalation of mercury dust or vapour may induce poisoning. The mode of absorption from the lungs is in doubt, but it is probable that the mercurial vapour dissolves in the fluid of the moist surfaces and so is absorbed by the blood. It has been reported that the problem of mercurial poisoning in a Californian mine has been overcome by spraying all mine floors, walls, timber, piping, wire, and ladders with lime-sulphur solution, thus converting the free mercury vapour and dust present into insoluble sulphide. Mercurial salts are largely used clinically for both external and internal treatment. Mercuric chloride or corrosive sublimate, which is used as a disinfectant, is a frequent source of poisoning. It has an acrid and metallic taste, and one part of it is soluble in about fourteen parts of water at ordinary temperature.

Vaginal douching with corrosive sublimate may produce serious poisoning with fatal result.

The preparation of corrosive sublimate in the form of soloids has given rise to several cases of accidental poisoning.

The other poisonous mercurial salts are mercuric oxide or red precipitate, mercuric ammonium chloride or white precipitate, mercuric potassium iodide, mercuric nitrate, and mercuric cyanide.

A number of fatal and almost fatal reactions following the intravenous injection of mercurial diuretics have been recorded.

The reaction in some of the cases was so sudden that the patient was dead before there was time to remove the needle from the vein, while in others it was characterised by convulsions, dyspnœa, cyanosis, coma, and death. The cause of death remains obscure, although a number of theories have been advanced. Some consider that the reaction is anaphylactic and connected with alteration of blood protein in nephrosis, and others that these drugs exercise a deleterious effect on the heart. It has also been thought that excessive dosages have been given, that the drugs have been continued after they have already produced toxic effects, or that the drugs have been administered to patients likely to die at any moment. It is advised that, before administering this treatment, the relatives should first be warned of the risk, and such restoratives as adrenalin and coramine should be available before the injection is given.

Poisoning may also arise from absorption of mercurial preparations applied to the skin.

Symptoms of acute poisoning.

The symptoms frequently assert themselves soon after the poison is swallowed. A metallic, bitter taste is perceived in the mouth, with a sense of constriction or suffocation in the throat, accompanied by a feeling of heat, which extends down the œsophagus to the stomach. Pain is experienced in the region of the stomach, which is increased by pressure, followed by nausea and constant vomiting. After the ordinary stomach contents have been evacuated, the vomit consists of blood-stained tenacious mucus. There is profuse diarrhœa, with severe tenesmus, the stools being composed latterly of blood-stained mucus. The pulse becomes small, feeble, and irregular, the skin cold and clammy, respiration difficult, and syncope, convulsions, or general insensibility usually precedes death. Urinary secretion is either entirely suppressed or scanty in amount. The urine frequently contains albumin and blood. Elimination of the poison occurs through the mucosa of the intestinal tract and by the kidneys, and these organs are therefore much affected by irritation. Elimination also occurs by the salivary glands, bile, and skin. Uræmic convulsions are rare.

Symptoms of chronic poisoning.

The principal signs and symptoms are :—Progressive anæmia, gastric disorders, salivation, inflammation and tenderness of gums, with ready bleeding, and tremors chiefly affecting the muscles of face, hands, and arms. Some measure of paralysis may also be present. When workers are exposed to fumes of mercuric nitrate, the teeth become blackened, eroded, and loosened, due to recession of the gums. Later they may fall out. Mercury vapours may be inhaled by workers in certain processes in connection with preparation of metals.

When the poison is administered internally in repeated sub-lethal doses, the following signs and symptoms manifest themselves.

The patient experiences a metallic taste and suffers from colicky abdominal pains, anorexia, nausea, vomiting, chronic diarrhœa, and general depression. The gums are swollen and may slough in patches, salivation is present, the teeth become loosened, and the salivary glands are swollen. An inflammatory line on the gums round the upper teeth may be seen, although this is frequently absent, and there is often much fœtor from the breath. The patient becomes emaciated, suffers from general muscular tremor, and the effects of fully developed peripheral neuritis. Nervous manifestations frequently supervene and include, morbid fears, mental depression, loss of memory, acute mania, and paralytic dementia. Renal complications are common. Before death the patient may be subject to mental disturbance accompanied by hallucinations. There may be skin eruptions.

Treatment.

Treatment should be employed without delay, since this poison is rapidly absorbed and damage to the kidneys may occur quite quickly. The stomach should be emptied by gastric lavage, but should a stomach tube not be at hand emesis should be induced. White of egg should be given very freely, but it must be removed from the stomach after a very limited period since the albuminate of mercury formed by it is soluble in excess of albumen and sodium chloride. Medicinal charcoal in suspension is probably more effective, but it also should be removed from the stomach.

Leschke states that the fixation of mercury may be effected by the administration of $\frac{1}{2}$ litre of skimmed milk to which have been added 50 grammes of glucose, 20 grammes of sodium bicarbonate, and three eggs. These ingredients should be beaten into a mixture. He recommends absorption by means of animal charcoal, Carbo medicinalis (Merck), since 1 gramme of this substance is capable of binding 850 milligrams of corrosive sublimate.

The use of sodium formaldehyde sulphoxylate has been introduced as a chemical antidote which reduces the perchloride to metallic mercury, and its action may be accelerated by the addition of 5 per cent sodium bicarbonate. A 5 to 10 per cent. solution of freshly prepared sulphoxylate may be used for lavage, and about 200 millilitres should be left in the stomach. Immediately afterwards, 10 grammes, dissolved in from 100 to 200 millilitres of distilled water, should be injected intravenously during a period occupying twenty to thirty minutes. In severe cases, after four to six hours, a further intravenous administration of 5 to 10 grammes may be repeated. Should colitis develop later, high colonic irrigation with 1 in 1,000 solution of sulphoxylate should be carried out once or twice daily. If sulphoxylate treatment is to be of value as an antidote it must be administered almost immediately after the ingestion of the perchloride of mercury. Rosenthal,[3] who recommends the above treatment, reports that sodium formaldehyde sulphoxylate has saved nine out of twelve dogs from a fatal dose of corrosive mercuric chloride. When used in ten human cases of

acute poisoning, recovery occurred without appreciable kidney damage. British Anti-Lewisite (B.A.L.), dimercaptopropanol, is stated to be an effective antidote for mercury poisoning (see p. 543).

Symptomatic treatment should be given as indications arise. A lowered blood chloride level should be treated by intravenous administration of physiological saline. For the treatment of acidosis (see p. 637). In certain cases, saline with 5 per cent. dextrose administered intravenously may prove very beneficial.

Post-mortem appearances.

The tongue is white and sodden in appearance, and the mouth generally has a whitish colour. The mucous membrane of the pharynx, œsophagus, and stomach is more affected than that of the mouth. It may show a whitish or bluish-grey colour, and is inflamed or ulcerated in parts. In the stomach, especially, there is more or less general evidence of inflammation, there may be considerable extravasation of blood, and the muscular coats are often so softened that it is difficult to remove the organ without rupturing them. The mucosa of the intestines more or less shares in the inflammatory condition, and the cæcum, colon, and rectum may be the seat of marked inflammatory action. Perforation of the stomach is rarely found. Histological changes, involving renal tissue, will probably be present in the endothelium of the vessels situated in the glomeruli, and the epithelial cells of the uriniferous tubules may show a varying degree of necrosis. Ogilvie [4] has recorded the results of an investigation respecting the condition of rabbits poisoned by corrosive sublimate. He found general congestion of the kidneys. The convoluted tubules and ascending limbs showed grades of injury which varied from cloudy swelling to coagulative necrosis and calcification. The liver was also congested and cloudy. Swelling and sometimes hydropic degeneration were present. Congestion of the spleen was accompanied by some destruction of the lymphoid cells. The changes in the myocardium consisted of congestion, cloudy swelling, and general patchy, fatty degeneration.

ILLUSTRATIVE CASES

A girl, aged seventeen, swallowed 1 ounce of acetic acid, and an unknown amount of added red oxide of mercury. The amount taken was thought to have been 30 grains. Her symptoms were stertorous breathing, vomiting of frothy fluid, without blood, pain in the throat and stomach, and frequent blood-stained evacuations from the bowel, very often copious, clots being passed. The patient was conscious, but collapsed. There was very slight corrosion of the mouth. There were no convulsions.

She died within thirty hours after taking the poison. A post-mortem examination was made eight hours after death. The œsophagus was inflamed and congested, and the stomach intensely so. At the pylorie end was a large blackened patch of erosion, the

mucous membrane being destroyed. The duodenum showed patchy congestion, but was not ulcerated.

Millar[5] records the case of an unmarried woman who was in the habit of using mercuric chloride tablets dissolved in water as a vaginal douche, and inserted one tablet containing 8·75 grains of that salt without water into the vagina at bedtime. By 10 A.M. next day she complained of pain and swelling of the vulva and by 1 P.M. was suffering from severe abdominal pain, followed by persistent vomiting and diarrhœa. At 7 P.M. the pain, diarrhœa, and vomiting had become worse. Next day a considerable amount of blood was passed in the stools and there was suppression of urine. She was thirsty and very drowsy. On the following day the gums were swollen, inflamed, spongy, and of dark colour, the breath being fœtid. Diarrhœa, pain, and vomiting persisted. The symptoms became progressively worse and swelling of the salivary glands appeared, with active salivation. She died on the evening of the sixth day. Post-mortem examination showed slight jaundice, some evidence of irritation of, and a few small hæmorrhages in, the stomach, also necrosis and gangrenous ulceration of portions of the mucous layer of the intestines, with submucosal hæmorrhages.

A young man swallowed, with suicidal intent, twenty-four or twenty-five soloids, each of which contained 1 grain of mercuric potassium iodide. Vomiting quickly ensued, and was encouraged. After treatment, including emetics, oils, and white of egg, he recovered.

Dérobert[6] reports a case of poisoning in a man of eighty-five by self-administered mercuric sulphate. Post-mortem examination did not disclose evidence of irritative lesions of the œsophagus, larynx, or trachea. The gastric mucosa was of brown colour, congested, and hæmorrhagic.

Lewinski[7] records details of a case of suicide by swallowing a quantity of a 1 per cent. solution of mercuric cyanide. Typical symptoms of mercurial poisoning followed and the woman died from uræmia after five days. Autopsy showed evidence of subacute mercurial poisoning.

Fatal dose.

Three grains of corrosive sublimate have killed a child, and a similar dose an adult. This amount may be reckoned as the minimum fatal dose. Recovery has, however, followed much larger doses, after 9½ grains, 19 grains, 30 grains, and after still larger doses; and of the red oxide, 30 grains. Much depends on the extent of the prevention of absorption of the mercury by its being bound by albumen and the amount which has been removed by emesis or gastric lavage.

Fatal period.

The time is very variable, and usually extends to several days.

Chemical tests (see p. 718).

Lead.

The principal salts of lead which produce toxic effects are the acetate, the oxide converted into the oleate, in the form of diachylon, the carbonate or white lead, the tetroxide or red lead, the yellow chrome or chromate of lead, and lead arsenate. The chloride and nitrate, not being easily procured by the public, do not bulk so largely in medico-legal work. All lead salts are less irritant in action than those of arsenic and the other metals, already considered, but the acetate and chromate are more irritant than the other lead salts. Chronic lead poisoning is quite common in the industries in which lead is used or handled in one form or another, for example, in lead-grinding works, potteries, paint factories, diamond-cutting, japanning, lacquer works, dye-works, in which lead chromate is used, coach-building, tinning, and enamelling works, plumbing, file making, and in electric accumulator works. Lead may gain access to the body by inhalation, ingestion, or by the skin when damaged. Lead poisoning is a notifiable disease and its occurrence must be reported to the Home Office (see p. 33). Inhalation of lead dust or fumes is most frequent in industry, more especially in the manufacture of white lead, the smelting of metals, and ship-breaking in which oxy-hydrogen burners are in use. It has been estimated that workers may inhale lead fumes to the extent of 0·215 gramme of lead in eight hours' work with oxy-hydrogen flame upon paint, when employed in breaking up ships, thus severe symptoms of lead intoxication may appear quickly. Lead vapour is liberated from lead when it is heated to temperatures above 700° C. Cholak and Bambach [8] have found that in 1,052 normal persons with no occupational lead hazard, the mean lead concentrations were 0·030 milligram per 100 grammes of blood, 0·027 milligram per litre of urine and 0·28 milligram per twenty-four hours' sample of fæces. In a communication from the Government Laboratory, the lead content of normal urine was given as varying from nil to 0·133 milligram per litre.[9] In lead poisoning an average rate above 0·21 milligram per litre is usual.

On account of the use of diachylon or lead plaster as an abortifacient, it has been placed under Schedule I of the Pharmacy and Poisons Act, 1933, and is therefore a substance to which special restrictions in sale apply. Poisoning has frequently been caused by its use.

Machle [10] has reported two cases of lead poisoning from the lodgment of bullets in the body. In one of the cases, a bullet was embedded in the head of the right femur for a period of about six to seven years. The man suffered from giddiness, headache, and malaise. His blood and urine showed an abnormally high lead level. After removal of the bullet the lead concentration in the blood and urine slowly fell to normal.

We have seen two cases of lead poisoning due to cracks in a frying-pan having been soldered with lead.

The following case of attempted murder by poisoning shows some unusual features. A man was charged with having poured white lead into a cup of milk which he placed beside his wife, so that she might drink it; having given her a chocolate filled with metallic mercury; and having on previous occasions filled a number of pills, intended for his wife, with brass filings, white lead, and copper wire, which were swallowed by her.

The bottle of milk was found to contain 86·11 grains of white lead, and the chocolate, over 3 grains of metallic mercury. The interior of the pills had been drilled out, and filled either with finely powdered brass or copper and then plugged or entirely filled with white lead. The amount of lead in the pills depended upon the size of the plug or the amount of filling, and varied from $\frac{1}{5}$ grain to over 1 grain in each pill.

The symptoms from which the woman suffered were abdominal pains with constipation and, later, continuous diarrhœa, loss of appetite, and cachexia.

The accused was found guilty on the charges except that of administering the chocolate.

Symptoms of acute poisoning.

Lead acetate and other lead salts in large doses produce an astringent metallic taste in the mouth, a burning, pricking sensation in the throat and œsophagus, and a similar sensation in the stomach, with pain, succeeded after an interval by nausea and vomiting, the vomited matter being latterly streaked with blood. Diarrhœa may supervene early. There is dryness of the fauces and great thirst. Colicky pains develop in the abdomen, the abdominal walls are tense and contracted, and the pains are relieved by pressure. Instead of looseness of the bowels, there may be constipation, the stools being black, and having a very offensive odour. Symptoms of collapse set in before death. Should, however, the case be protracted, arthralgia may develop, and there are likely to be numbness and paralysis of the limbs. Convulsions, delirium, or coma may precede death.

Symptoms of chronic poisoning.

These may be summed up as follows:—Colic, which is very intense; general malaise, prostration, anæmia, debility, and loss of weight; obstinate constipation; gradual emaciation; anorexia; sallowness of face; in most cases, there is a blue line on the gums, but this is not constant. In the presence of oral sepsis, the line appears close to the gum margin. The line is not seen in the presence of healthy teeth, in edentulous subjects or at points where teeth are absent. The blue colour is due to the action of sulphuretted hydrogen generated by decomposing protein food around septic teeth in the presence of circulating lead. A blue line may also be met with in mercury and bismuth poisoning. The pulse is usually slow and hard and the blood pressure elevated, the result of constriction of the vessels caused by the action of lead on the neuro-

vascular system. The nervous symptoms, almost characteristic, are "wrist-drop," due to paralysis of the extensors and supinators of the forearms, accompanied by muscular atrophy, tremors, cramps, and shooting pains in the limbs. We have seen several cases, occurring among pottery-dippers, in which the scapular muscles and those of the arms were very atrophied. There may be paralysis of the limbs.

Early mental changes may manifest themselves and include headache, irritability, and lethargy. Lead encephalopathy sometimes occurs, especially in young subjects. This disturbance of cerebral function may give rise to recurrent convulsions, and progressive mental deterioration.

In a large number of cases of acute and chronic poisoning, punctate basophile staining of a number of the red cells will be found, and there is diminution of hæmoglobin and of the red cells. Punctate basophile staining may occasionally be seen in poisoning by benzol and aniline. The polymorphonuclear leucocytes are decreased and lymphocytes increased. There may be evidence of vacuolation of the red cells and poikilocytosis.

Treatment of acute poisoning.

The stomach must be emptied by emetics, or preferably by stomach tube. The ingoing water should be mixed with magnesium sulphate, 2 ounces of sulphate to 2 gallons of water. This forms the insoluble sulphate. Sodium thiosulphate, 5 millilitres of 10 per cent. solution, given intravenously, has been recommended. After the preliminary treatment has been completed, demulcent drinks and milk should be given, and the remainder of the treatment regulated as symptoms indicate. High colonic lavage may be usefully employed. Morphia may be given to relieve abdominal pain.

Behaviour of lead in the body.

When large quantities of lead are taken into the body, excretion does not maintain an equilibrium and lead becomes stored. The poison is mainly excreted into the colon and, to a lesser extent, through the kidneys. If the rate of excretion equals that of absorption there may be no signs of poisoning. Only a part of the lead which is swallowed is absorbed into the general circulation. Non-soluble compounds are excreted in the fæces but soluble compounds are carried to the liver, where some is stored and the remainder distributed to other body tissues. Lead is stored in the bone marrow as triple phosphates, and when the acid content of the blood is increased, becomes soluble and in part may be excreted through the ordinary channels. In chronic lead poisoning there is substitution of lead for calcium, the result of the retention of lead in the bones, and an increased elimination of calcium in the urine. There appears to be a close analogy between lead and calcium with regard to distribution, storage, mobilisation, and excretion, and it has been shown that the state of calcium nutrition, the acid-base equilibrium, and the state of activity of the parathyroids are important factors in

determining whether lead is stored in bone without toxic effect or is passed into general circulation with resultant tissue damage.

To establish the absorption of lead in the body by its presence in the fæces is fallacious since such a finding only establishes exposure to lead. Its absorption can only be established by its detection in the blood serum and urine in amounts which exceed those that could occur in persons not exposed to lead (Hamilton and Johnstone,[11] vide infra).

Treatment of chronic poisoning.

The patient should be given large doses of ammonium chloride, a capsule containing 15 grains, six times daily at regular intervals, followed by large quantities of water and a low calcium diet. This brings about a negative calcium balance and increases calcium and lead excretion, in fæces and urine, or " de-leading."

By raising and lowering the calcium intake carefully, the excretion of lead, deposited in the bones, can be controlled and its elimination so regulated as to obviate serious recurrent symptoms of lead poisoning during the process. Parathyroid extract, Parathormone (Lilly), given intramuscularly, to mobilise lead and calcium from the bones by producing a negative calcium balance, has also been recommended.

Holmes, Amberg, and Campbell[12] have recommended the use of vitamin C (ascorbic or cevitamic acid) in the treatment of chronic lead poisoning, and hold the view that it reacts with toxic lead ions to form a poorly ionised and much less toxic compound.

Some are of opinion that a low calcium diet combined with a high phosphorus diet aids excretion of lead.

Constipation should be treated with magnesium sulphate which facilitates the removal of excreted lead from the intestines.

Hamilton and Johnstone[11] recommend that in cases of severe intestinal colic the patient should be hospitalised and intravenous calcium instituted. This is continued until the more violent colic has subsided, at which time calcium by the mouth is given. All calcium therapy is stopped upon the cessation of intestinal discomfort. It is their considered opinion that once the calcium has caused the colic to cease, its utility is ended. " The almost instantaneous relief of lead colic following the use of calcium leaves little doubt that this drug has a physiological effect upon the smooth muscle of the intestinal tract, but the action is too sudden and dramatic to cause anyone to believe that the quick relief is due to the immediate storage of lead within the bones of the body." Hamilton and Johnstone believe that the treatment of lead intoxication requires therapy for the acute condition only, and that attempts to store lead or to delead a patient are unnecessary and, in the case of deleading, may be dangerous.

Post-mortem appearances.

After acute poisoning, the appearances are chiefly those of gastro-enteritis. The gastric mucosa is congested. There may be eroded

patches. In chronic poisoning, the kidneys are of small, granular, and contracted type. The left ventricle in such cases is frequently hypertrophied and the vessels show arterio-sclerotic changes.

Chemical tests (see p. 717).

Lead tetra-ethyl.

This compound is employed in fuel for motor cars, with which it is mixed to about 0·1 per cent. by weight to counteract "pinking," and on inhalation in sufficient quantity is extremely toxic. It is a colourless fluid with a sweetish odour and emits a vapour at ordinary temperature. Apart from inhalation, poisoning may be induced by absorption through the skin surface following contamination with the liquid. Poisoning in acute form chiefly affects the central nervous system and includes such symptoms as headache, nervousness, irritability, mental excitement, vertigo, tremor, insomnia, delusions, hallucinations, maniacal attacks, delirium, and convulsions. There may be slowing of the pulse rate, falling of the blood pressure and body temperature, nausea, vomiting, and diarrhœa which is more frequent than constipation. Muscular pains and twitchings, fatigue, and general weakness are common. The symptoms in chronic poisoning are very similar to those of chronic lead poisoning.

In the treatment of severe cases, there should be a liberal intake of fluids, and normal saline or a 5 per cent. solution of dextrose in normal saline will be found beneficial. Intravenous administration of from 2 to 4 grammes of a 2 per cent. solution of magnesium sulphate has been recommended in cases of delirium.

Post-mortem findings are not specific. The brain and lungs are often congested. The liver and kidneys may show degenerative changes.

Thallium.

The compounds of thallium, a heavy metal, may be found in zinc salts or in crude sulphur. Industrially, it is used in dyeing and glass manufacture. It is also an ingredient of certain vermin poisons. Therapeutically, it has been advocated for the treatment of ringworm in the form of thallium acetate and it is used as a depilatory in certain preparations, occasionally causing poisoning.

Repeated small doses have a cumulative effect. Chronic poisoning injures the endocrine glands. Peripheral neuritis and alopecia are outstanding manifestations. General malaise, headache, drowsiness, depression, and insomnia are other symptoms.

Acute poisoning, resulting from swallowing the substance, is characterised by stimulation of the heart followed by depression which causes death from cardiac failure. There may be nausea, sickness, colic, diarrhœa, and albuminuria. Hyperæsthesia, impaired vision, ataxia, convulsions, and delirium or dementia complete the clinical picture.

Gleich [13] reports two cases of thallium acetate poisoning in the treatment of ringworm affecting the scalp. Both children showed myelo-encephalic symptoms and were dead within twelve days.

Treatment.

The stomach should be emptied either by stomach tube or by an emetic. A purgative dose of magnesium sulphate should be given. Intravenous injection of potassium iodide is recommended. Sodium thiosulphate, slowly administered, intravenously, 20 millilitres of a 3 per cent. solution, daily, will cause gradual elimination of thallium in the urine.

Fluids, especially milk, should be given by mouth, and the patient should be kept warm.

Chemical tests (see p. 723).

Copper.

Copper salts when taken in large doses produce toxic effects. Probably the most poisonous are the sulphate or blue vitriol, and the subacetate or verdigris. They are seldom used for criminal purposes because of their striking blue or green colour. The sulphate, however, has been taken with the hope of inducing abortion. Workers in copper are said to suffer occasionally from toxic effects produced by inhalation of copper dust.

A case of poisoning after the injection of copper sulphate into a tuberculous fistula in a boy of six has been reported. Death occurred.[14] Nine millilitres of a 10 per cent. solution were injected. Chemical analysis showed almost the total amount of injected copper in the liver. There was severe parenchymatous injury of the heart, liver, and kidneys (see p. 538).

Symptoms.

If it be assumed that $\frac{1}{2}$ ounce of sulphate or subacetate has been taken, the following are signs and symptoms which may be looked for: pain in the mouth, œsophagus, and stomach; bluish or greenish coloration of the mucous membrane of the mouth; sickness, which produces a bluish or greenish vomit; there may be diarrhœa accompanied by colicky abdominal pains; convulsions and cramps of the limbs may sometimes occur. If the case terminates fatally, death is preceded by convulsions, paralysis, or coma. In chronic poisoning, which is but rarely observed even among workers in the metal or its salts, the main indications are on a parallel with salts of lead. There are evidences of progressive emaciation, gastro-intestinal irritation, vomiting, loss of appetite, diarrhœa or constipation, a coloured line on the gums, and perhaps a coloration of the teeth, the colour being bluish, greenish, or purplish, may be found. The evidences of implication of the nervous system are also very similar to those from lead poisoning, namely, peripheral neuritis, and atrophy of the muscles of the shoulder, arm, and forearm, with wrist-drop in some cases.

Treatment.

Empty stomach by emetic or tube. Potassium ferrocyanide, 10 grains in water, is recommended for its antidotal properties. Give albuminous and demulcent fluids. Treat symptoms along ordinary lines.

Post-mortem appearances.

Perhaps the most striking appearance is the bluish or greenish coloration imparted to the gastric mucosa. The mucous membrane is congested and injected, and occasionally shows eroded patches. The intestinal mucous membrane may share the same appearances. The coloration by bile may be mistaken for that of copper, but on touching copper staining with ammonia the blue colour becomes intensified or the green is changed to blue.

Fatal dose.

Half an ounce of subacetate, and 1 ounce of the sulphate, have caused death. The minimum dose has not been ascertained with any degree of precision.

Fatal period.

Four hours, in a child who swallowed an unknown quantity of the sulphate. Twelve hours, in the case of a man who took 1 ounce.

Chemical tests (see p. 714).

Zinc.

The toxic effects of zinc are confined to the use of two salts of the metal, namely, the sulphate, the action of which is irritant, and the chloride, which is corrosive. Zinc fumes also exercise a toxic action upon persons engaged in such occupations as zinc and copper smelting. The commonest form of sickness by a compound of zinc is the so-called "brass-founders' ague" or "metal fume fever" which results from the inhalation of freshly sublimed zinc oxide. This condition, however, is not specific to zinc, since copper, iron, beryllium, magnesium, and other metals may cause the same condition.

Ingestion of zinc compounds is not usually fatal on account of their effective emetic action.

Symptoms.

From the sulphate.

These are principally of gastro-intestinal irritation, and consist of vomiting, pain in the stomach and abdomen, diarrhœa, and, when a considerable quantity has been swallowed, symptoms of collapse. Zinc sulphate has been taken in mistake for Epsom salts.

From the chloride.

The symptoms are of a somewhat different nature on account of the corrosive character of this salt. These are an immediate burning sensation in the mouth, throat, œsophagus, and stomach, with vomiting, diarrhœa, and symptoms of shock.

The symptoms of chronic poisoning are closely allied to those of lead and copper.

Treatment.

Gastric lavage is the first procedure. Sodium bicarbonate in tepid water should be administered freely. Milk and egg drinks should also be given. In addition, the case should be treated symptomatically.

Post-mortem appearances.

When zinc sulphate has caused poisoning, the gastro-intestinal mucosa will show patches of congestion, variable both in degree and extent. There may be a yellowish discoloration of the œsophagus and stomach. In some cases, the stomach may show patches of submucosal hæmorrhage and erosion. The small intestine may be similarly affected. When zinc chloride has been swallowed, signs of corrosion may be present. The action of this salt is mainly local and corrosive, the tissues with which it passes into contact being affected. The degree of absorption is relatively slight and is in inverse proportion to its corrosive action. Taken by mouth, in certain cases, there may be no definite signs of corrosion of the mouth, pharynx, or œsophagus. In one case, the mucous membrane of the mouth, throat, and œsophagus was white and opaque, the stomach was hard and leathery, and contained a white curd-like fluid.

Chemical tests (see p. 724).

Tin.

The chief danger of poisoning by this metal arises from canned goods (see p. 690).

Symptoms.

The salts of tin cause gastro-intestinal irritation such as nausea, vomiting, pain in the abdomen, diarrhœa, and prostration. A metallic taste may be experienced.

Treatment.

Gastric lavage, demulcent drinks, and symptomatic measures.

Chemical tests (see p. 723).

Iron.

Of the cases in which poisoning has occurred by the ingestion of iron preparations, ferrous sulphate and the tincture of perchloride

of iron have been the responsible agents, although such cases are few. Forbes[15] has published an interesting paper in which he describes details of two fatal cases of acute ferrous sulphate poisoning in children aged three years and one year. In the first of these the child was thought to have swallowed fifty tablets each containing 3 grains of ferrous sulphate and in the second, thirty to thirty-five tablets of the same preparation. Death occurred after some fifty-three and thirty hours, respectively. In both cases some tablets were returned in the vomitus (see p. 538).

Symptoms.

The clinical manifestations usually consist of vomiting, thirst, restlessness, and collapse. The vomited material may be of a brownish colour or may, after a short period, contain fresh or clotted blood.

Hurst[16] reports the case of a woman, the subject of severe simple achlorhydric anæmia following gastro-jejunostomy, who was treated by the administration of 160 grains of iron and ammonium citrate daily, and who suffered an acute cerebral attack, closely resembling lead encephalopathy, after she had been under treatment for three weeks.

Although a similar case has not previously been recorded, the symptoms were regarded as due to iron poisoning. The patient completely recovered and was subsequently given smaller doses of iron, when finally the anæmia disappeared.

Treatment.

An emetic should be given and the intake of bland fluids encouraged. In addition, the application of warmth and the use of cardiac stimulants form the principal line of treatment. When breathing is embarrassed, the use of oxygen is frequently beneficial. A course of penicillin treatment should be rapidly initiated when pneumonic complication threatens.

Post-mortem appearances.

The stomach may contain coffee-ground material, since it is very often the seat of an acute hæmorrhagic gastritis. The intestinal tract shows a varying degree of vascular engorgement and œdema, while the contents reveal blackish coloured staining. Degenerative changes, verging from cloudy swelling to complete necrosis, are found in the liver, which may have assumed a yellow tint. The kidneys are frequently in a state of cloudy swelling, while small, irregular hæmorrhages may be present on the surface of the heart, lungs, thymus, and aorta. Pneumonic consolidation of one or more lobes of the lungs is not an infrequent finding.

Manganese.

Manganese is used in the chemical, ceramic, glass, dye and varnish industries. It is also used as a deoxidising and desulphurising agent, and in the preparation of manganese steel. Industrial

poisoning may occur through handling, or by the inhalation of dust, especially during the process of grinding. Manganese is also used in the manufacture of galvanic cells. Industrial poisoning by this substance is notifiable and its occurrence must be reported to the Home Office (see p. 33).

The signs and symptoms of chronic poisoning are lassitude, drowsiness, slight dyspnœa, weakness and trembling, Parkinsonism, and disturbanees of gait and speeeh. The voice is dull and monotonous, the face mask-like, and the gestures stereotyped. Steppage gait and Rombergism may be evident. Other symptoms include excessive salivation, irritability, impairment of sexual function in the male, and emotionalism. Manganese toxæmia produces progressive bulbar paralysis, amyotrophic lateral sclerosis and diffuse nodular hepatic cirrhosis. Differential diagnosis between post-encephalitic Parkinsonism, multiple sclerosis, and chronic manganese poisoning is sometimes necessary, and a history of exposure will help to elucidate the matter. In the Annual Report of the Chief Inspector of Factories, 1943, reference is made to a fatal case of poisoning in a man engaged in welding tram lines. He used an electrode eontaining 14 per cent. manganese under poor conditions of ventilation.

Potassium permanganate is so widely used that its possibility as a toxic agent is often overlooked. It is a powerful oxidising and corrosive agent and fatal poisoning has occurred in a number of cases by ingestion either in the solid state or in strong solution. Dilute solutions may cause gastro-intestinal irritation. In concentrated form, this substance may affect the cardio-vascular system or the central nervous system and may produce mild corrosion. Willimott [17] reports a fatal case following urethral injection of 200 millilitres of a 10 per cent. solution, equivalent to 20 grammes of the solid salt. Ascending from the bladder to the umbilicus was adhesive peritonitis, and the bladder mucosa was the seat of corrosions varying in size from a pin-head to a half-crown piece.

Treatment.

When the substance has been swallowed gastric lavage, using charcoal, should be employed. Calcium bromide, intravenously, or calcium gluconate, intramuscularly, has been recommended.

Chemical tests (see p. 717).

Boracic acid and borax.

Acute poisoning by boric acid is infrequent, but chronic poisoning following internal administration or bladder irrigation is perhaps more common. Borates act as depressants on the central nervous system, and may produce necrosis of the gastro-intestinal tract and of the renal tubular epithelium.

A teaspoonful, taken in error, has caused death. Following operation, a woman was given an intravenous injection of 600 millilitres of a 2·5 per cent. solution of boric acid in 10 per cent.

dextrose in error for a solution of 10 per cent. dextrose. She recovered.[18]

Symptoms.

These include malaise, nausea, hiccough, vomiting, diarrhœa, an erythematous skin eruption, fall of temperature, convulsions, coma, and collapse. The erythematous rash may be accompanied by papules, vesicles developing between the fingers and on the backs of the hands. In one of our cases, in which 5-grain doses were administered internally for a chronic bladder condition, the patient developed symptoms of weakness out of all proportion to the ailment, and a papular rash over certain parts of the body. Nephritis occasionally develops and there may be a few submucosal hæmorrhages. Sanders recounts the facts of his personal experiences of toxic effects of boric acid. Suffering from dysentery, a solution of the acid in warm water was used as a rectal douche daily for some time. He developed a rash, resembling bromide rash, over the entire body, became delirious, had a feeble pulse, and was unable to sleep. After a fortnight, during which his condition was serious, he commenced to recover.

Fifty per cent. of the acid is excreted in the urine within twelve hours, the rest remains in the body up to three to four days, and thus may accumulate as the result of repeated dosage.

For the use of boric acid as a food preservative, see p. 691.

Treatment.

Empty the stomach with a stomach tube, or by means of an emetic, and thereafter give a purgative dose of magnesium sulphate.

Chemical tests (see p. 711).

Cadmium.

Therapeutically, this substance is of no importance, but occasionally a case of poisoning occurs industrially, since it is used in the process of ore smelting, and thus workmen have to handle bags containing cadmium oxide. It is also used in electro-plating. Poisoning may occur from ingestion, or inhalation of fumes. The signs and symptoms of poisoning by inhalation consist of headache, irritating cough, metallic taste in the mouth, nausea, vomiting, sometimes accompanied by diarrhœa, and tachycardia. After some twenty-four to thirty-six hours, breathlessness, pain in the chest, and more marked gastro-intestinal symptoms make their appearance. The urine may assume a brownish colour. When death occurs, post-mortem examination usually shows congestion of the gastro-intestinal and respiratory tracts. Bronchitis, nephritis, and fatty changes in the heart and liver may be found. When cadmium is swallowed, its emetic action usually obviates fatality, but vomiting, with or without diarrhœa, is marked.

Chemical tests (see p. 711).

Gold.

Preparations have been used intramuscularly in the treatment of rheumatoid arthritis. These have various proprietary names, such as sanocrysin, aurobin, allochrysine, krysolgan, and solganal. Toxic and even fatal results have resulted from the use of some of them. The toxic reactions comprise diarrhœa, vomiting, albuminuria, exfoliative dermatitis, aplastic anæmia, and occasionally agranulocytosis. Fatzer [19] has reported two cases. Both patients, elderly men, suffered from rheumatoid arthritis of long standing. One was treated with allochrysine and the other with solganal. The first patient died of agranulocytosis with secondary necrotic angina, the second recovered following treatment with liver extract, irradiation, and nucleotide, although he died a month later from another cause. Idiosyncrasy is quite common. In addition to the foregoing manifestations, focal or confluent hepatic necrosis, necrosis of the renal tubular epithelium, peripheral neuropathy, and blood dyscrasias should be mentioned.

The treatment consists in the administration of sodium thiosulphate either orally or by injection. British Anti-Lewisite (B.A.L.), dimercaptopropanol, is an effective antidote (see p. 543). In addition, treatment has to be directed along general and specific lines.

Chemical tests (see p. 715).

Aluminium.

The soluble salts, when taken in large doses, may produce gastro-intestinal symptoms. The use of aluminium vessels is generally regarded as innocuous, and reported cases of toxæmia from their use may have been due to idiosyncrasy.

Chemical tests (see p. 706).

Bismuth.

Metallic bismuth is the least toxic form, and the soluble compounds are more toxic than the insoluble preparations. Roe [20] reports the case of a child one month old who, suffering from diarrhœa, had become dehydrated. Ten grains of bismuth subnitrate were given every two hours, and death occurred two days later. Methæmoglobin was found in the blood, and there were small hæmorrhages in the cortex of the brain. Dowds [21] describes three deaths due to subcutaneous injections of sodium bismuth tartrate. In two of these there was intense stomatitis, and the urine was loaded with albumin and contained bismuth. Bismuth is used in the treatment of syphilis and yaws.

The toxic effects include stomatitis, colitis, and nephritis. Bismuth compounds are contraindicated in cardiac disease, since bismuth is liable to induce weakness and irregularity of the action of the heart.

Treatment.

Sodium thiosulphate, 0·5 gramme in a 10 per cent. solution given intravenously is recommended. Symptomatic and general treatment should be employed.

Chemical tests (see p. 711).

Barium.

In its metallic form there are no toxic manifestations, but some of the soluble compounds in large doses are poisonous. They may affect the cardio-vascular, gastro-intestinal, or nervous system. The principal salts are the chloride, nitrate, and the carbonate, the first being the most poisonous. These are constituents of certain rat poisons. Morton [22] describes severe poisoning of eighty-five British soldiers in Persia by barium carbonate, intended for use as rat poison, which was accidentally mixed with flour and made into pastry. Sauer [23] records the case of a woman who was given about 108 grains of barium chloride in error. Recovery followed treatment. When seen three hours after taking the dose she was collapsed and her pulse, which was thready, was 120 per minute. Four teaspoonfuls of crystalline magnesium sulphate were given in water followed by a hypodermic injection of $\frac{1}{100}$ grain of atropine sulphate and $\frac{1}{4}$ grain of morphine sulphate. The pain was relieved almost at once, and on the following day the patient felt perfectly well.

Symptoms.

Nausea and vomiting of a watery mucus ; convulsive movements of hands and feet ; general convulsions. To these symptoms must be added diarrhœa with tenesmus, grave signs of shock, loss of motor power and sensation. The heart is embarrassed in its action, and the breathing laboured and slow.

Treatment.

Employ the stomach tube and wash out stomach with a solution of magnesium sulphate (2 ounces in 2 gallons of water). Give 1-ounce dose of sodium sulphate, well diluted, and repeat if neeessary. Treat general condition along usual lines. Cardiac or respiratory stimulants may be necessary.

Fatal dose.

The fatal dose is variable. One teaspoonful has caused death. A man, who took a mouthful of a solution containing 130 grains of chloride, died. The nitrate and earbonate have also caused fatal results, although cases have recovered from the latter salt.

Fatal period.

From two hours upwards.

Chemical tests (see p. 711).

Potassium nitrate.

This salt has frequently caused death. Taken accidentally, as it usually is in mistake for Epsom salts, the quantity is generally large.

Symptoms.

The signs and symptoms consist of vomiting accompanied by severe gastric pain, and sometimes diarrhœa with blood-stained stools; symptoms of collapse, lividity of face and insensibility are followed by death.

Treatment.

Free lavage; demulcent drinks; stimulants; warmth to body; and treatment of prominent symptoms.

Post-mortem appearances.

The mucous lining of the stomach is bright red or brownish-red in colour. The mucous membrane is injected.

Fatal dose.

The usual fatal quantity is from 1 ounce upwards.

Fatal period.

A few hours, to thirty-six hours.

Chemical tests (see p. 721).

Potassium and sodium chlorate.

In large doses these salts act as acute hæmolytic poisons and produce signs and symptoms which may be summarised as follows:— Severe vomiting, pain in the epigastrium and abdomen generally, profuse diarrhœa, dyspnœa and deep cyanosis, lowered blood-pressure and cardiac weakness, also headache, giddiness, muscular weakness, restlessness, coma, and death. Oliguria or anuria are manifestations and any urine passed is first dark in colour, then reddish-brown, and contains hæmoglobin, methæmoglobin, hæmatin, blood cells, casts, and albumin. The urine on standing shows a considerable quantity of chlorates. The blood corpuscles are destroyed and spectroscopic examination of the blood, which frequently is of chocolate colour, shows the spectrum of methæmoglobin. The sclerotics are sometimes icteric. Asphyxia occurs when the methæmoglobin prevents the blood from carrying the available oxygen. In subacute cases, death usually results from renal changes following obstruction of the renal tubules with fragmented red cells. The body, after death, may vary in colour from white to bluish-green or chocolate, due to methæmoglobin.

The following cases, which include post-mortem findings, are typical:—

A woman took, by mistake for Rochelle salts, two table-spoonfuls of chlorate. Within twenty-four hours she was pro-

foundly prostrated. Her temperature was 99° F., and the pulse and respiration rates were 136 and 32, respectively. The body surface was cyanotic, the breathing rapid but not laboured, and the pulse rapid but not feeble. She had vomited freely, and was still vomiting after admission to hospital. Two hours later, the temperature rose to 104° F. Three dark brown motions had been passed, and dark-coloured urine was voided involuntarily. The urine contained blood cells, methæmoglobin, and copious albumin. Next day, the skin, conjunctivæ, and lips were of chocolate tint. She died thirty-seven hours after the poison had been taken. Coma preceded death. On post-mortem examination, the blood in the large vessels was of chocolate colour. The heart was soft and flabby and the lungs were normal, but on section brown in colour. The spleen was of chocolate hue and the kidneys were large and contained chocolate-coloured blood. The bladder contained 3 ounces of urine, darkish brown in colour. Spectroscopic examination of the blood showed a spectrum of methæmoglobin. Microscopic examination of the heart muscle showed extensive fatty degeneration, and the tubules of the kidneys were filled to distension with disintegrated corpuscles and blood-pigment, giving a peculiar striped appearance of the pyramids.

A man, aged fifty-three, took an overdose of this salt. He died in coma. On post-mortem examination the appearances were as the foregoing, and in addition, the gall-bladder was full of thick, very dark green bile.

Gastro-enteritis may be present and the liver may show evidence of fatty degeneration.

Treatment.

Evacuate the contents of the stomach by free lavage. Transfusion of blood may be necessary to replace the destroyed blood cells. Intravenous sodium bicarbonate or methylene blue has been recommended. Diuresis should be encouraged by the administration of alkaline drinks. Administer oxygen, also cardiac and respiratory stimulants, if necessary.

Fatal dose.

About 380 to 390 grains of potassium chlorate and upwards. The smallest single adult dose was 210 grains.

Fatal period.

This usually extends into days.

Chemical tests (see p. 721).

Potassium and sodium bromide.

As sedatives, their use is widespread in a variety of conditions and occasionally toxic manifestations appear. These may be mild, but severe and even fatal cases sometimes occur. The rate of excretion is dependent chiefly on the efficiency of the kidneys and

the daily fluid and sodium chloride intake. Bromides replace the chlorides in the body. The symptoms show a wide variation and may be acute or chronic. There is also personal idiosyncrasy. The symptomatology is not peculiar to bromide intoxication, and diagnosis with certitude can only be established by ascertaining whether the blood bromide is raised and by the gradual disappearance of the symptoms when the bromide administration is withheld.

Symptoms.

Furred tongue, anorexia, constipation, slurring speech, listlessness, giddiness, gingivitis, lachrymation, coryza, tachycardia, irregular pulse, occasional diarrhœa, alteration in the tendon reflexes, rash, tremors, and toxic-delirium states with confusion and hallucinations.

Tod and Stalker's [24] authoritative contribution points out that "to prescribe bromide without ascertaining the chloride intake or the bromide saturation is the same as letting the patient take as much or as little bromide as he pleases," and that there is no constant relationship between the amount of bromide ingested and the appearance of the toxic symptoms, since the body is unable to distinguish between bromides and chlorides.

Treatment.

Stop bromide. Give large quantities of bland fluid and 30-grain sodium chloride capsules, four hourly. Five millilitres of suprarenal cortex, daily, by intramuscular injection is a recommended line of treatment, in conjunction with sodium chloride, to expedite the disappearance of eruptions and mental symptoms. Bowel action should be kept free. In cases of delirium sedatives are dangerous but, if imperative, paraldehyde is recommended.

It may take several weeks for the symptoms to disappear.

Chemical tests (see p. 722).

Pyrogallic acid.

This substance is commonly used in photography, and in the manufacture of hair dyes and marking inks. It has very toxic properties, not only when swallowed but even when it is absorbed through the skin. Cases of poisoning from both causes are recorded. Its toxic effect is serious since, like several other poisons, it has a hæmolytic action, destroying the red corpuscles and liberating the hæmoglobin into the plasma. Dyspnœa, subnormal temperature, coma, and altered blood in the urine, which is brown in colour, are outstanding manifestations. On post-mortem examination the blood is generally of a brownish-red tint.

Treatment.

Free lavage of stomach; inhalations of oxygen; stimulants; maintenance of body heat; and transfusion.

Chemical tests (see p. 722).

Phosphorus.

There are two kinds of phosphorus, crystalline, and mixed crystalline and amorphous. The former, found in commerce as yellow, translucent, waxy-looking sticks, and usually kept submerged in water to prevent oxidation, is poisonous. It is slightly soluble in alcohol, ether, and other liquid solvents, but freely soluble in carbon disulphide. When exposed to the atmosphere it gives off dense white fumes composed of phosphoric and phosphorous acids. The mixed form is of red colour, is insoluble in carbon disulphide, and is non-poisonous. It does not give off fumes or become luminous in the dark as does the other form.

Poisoning from phosphorus is infrequently homicidal, but is often accidental or suicidal. Since prohibition of the use of yellow phosphorus in matches, the incidence of suicidal poisoning, through swallowing phosphorus from this source, has been relegated to the past. Yellow phosphorus is an ingredient of certain vermin exterminators. Industrial hazard arises in the production of phosphates by the treatment of bones with sulphuric acid, and in the further processes associated with the manufacture of phosphorus. Industrial workers exposed to phosphorus fumes suffer from necrosis of the jaws and caries of the teeth, the necrosis occurring at points where the teeth are carious. The fumes enter the system through defective teeth or mucous membrane, due to injury or disease which exposes bone. A worker does not develop necrosis if the teeth and gums are intact, since the presence of septic bacteria is essential for its production. The development of necrosis of the jaw is slow, and there may be as long a period of exposure to the fumes as two to five years before the bone becomes affected. Workers who handle phosphorus frequently develop burns and caustic lesions, which heal slowly, together with dermatitis, conjunctivitis, itching, or nasal irritation. Industrial phosphorus poisoning is notifiable and its occurrence must be reported to the Home Office (see p. 33).

Acute symptoms.

Phosphorus, an active protoplasmic poison, which depresses cellular oxidation and causes widespread fatty degeneration when swallowed in poisonous dose, causes pain in the stomach, which may, however, not be marked, but which is usually succeeded by vomiting. The patient may complain of a garlic taste in the mouth, and a garlic odour may be perceived in the breath. There is an acrid, burning sensation in the throat and œsophagus, accompanied by great thirst. The vomited material is usually dark in colour, dark green, coffee-coloured, or black, has a garlicky odour, and, if exposed in the dark becomes luminous or phosphorescent. The fæces are also dark and luminous. Diarrhœa may or may not be present. Signs of shock develop, the pulse becomes small, irregular, feeble, and, at times, imperceptible, there are cold, clammy skin, anxious, pinched face, and subnormal temperature. Coma or convulsions usually precede death. In most cases, however, the progress

is not continuous to a direct fatal termination. There is usually an intermission in the severity of the symptoms, so much so, at times, as to give the impression that recovery has taken place.

Sooner or later, it may be two or more days, fresh symptoms appear. Jaundice develops, the epigastric pain, not yet passed away from the original attack, increases in severity, and the abdomen becomes distended. On examination the liver will be found enlarged, as probably also the spleen. Vomiting returns. Hæmorrhages from the nose, or from other mucous surfaces, or subcutaneous hæmorrhages appear. The urine becomes scanty, strongly acid in reaction, high-coloured, and contains blood, albumin, bile, and sometimes sugar. The nervous system becomes implicated, and sleeplessness, pains in the head, together with other manifestations, appear. The fæces are pale or clay-like in colour. Gradually the patient becomes more prostrate, the pulse becomes weaker, irregular, and more compressible, and some degree of hyperpyrexia sets in. Prior to death convulsions or coma may appear. The clinical picture may be suggestive of acute yellow atrophy of the liver.

Chronic symptoms.

These usually result from inhalation rather than from ingestion. Gastro-intestinal symptoms are prominent, excreta and breath have a garlic odour, the red and white cell counts are increased, and the liver is enlarged. Anæmia, jaundice, fragility of bones, bronchitis or broncho-pneumonia, necrosis of jaw, "phossy-jaw," and general prostration may, and probably will, be evident in the more severe cases as time passes. "Phossy-jaw" is a condition of suppurative osteomyelitis of the maxilla or of the mandible with multiple sinuses and resultant considerable disfigurement.

Treatment.

Early use of the stomach tube, and free lavage with a 1 per cent. solution of potassium permanganate will exert an oxidising effect upon the phosphorus. A dilute solution of copper sulphate, 15 grains to 2 gallons of water, may be used instead. Evacuation of the bowels is important, and the most prominent symptoms must be treated. Alkaline drinks and dextrose should be given, but oils, fats, and eggs should be withheld.

Post-mortem appearances.

The appearances generally are those of a highly irritant poison, consisting of inflammation or erosion of the mucous membrane of the pharynx, œsophagus, stomach, and intestines. The stomach may contain dark-coloured fluid, consisting of mucus and altered blood, which may have a garlicky odour. The contents may be luminous in the dark. The liver, enlarged and usually of yellowish colour, is the seat of marked fatty degeneration and quite frequently shows a softened condition. Its colour varies. On microscopical examination, extensive destruction of the liver cells is found, and

evidence of fatty degenerative changes is marked. Hæmorrhagic extravasations are commonly found. Fatty degeneration of the musculature of the heart, of voluntary muscle fibres, and of the kidneys is present.

Blaxland[25] reports the case of phosphorus poisoning, due to an explosive German bullet, in an airman wounded over enemy territory. Four days after the injury he became semi-comatose with a lemon tint of skin ; there was anuria and he died two days later. A post-mortem showed extensive necrosis of the liver with fatty degeneration. The kidneys showed catarrhal nephritis and the cells of the convoluted tubules were in many instances necrotic. The gross destructive lesion of the liver was typical of acute phosphorus poisoning. The bullet contained $3\frac{1}{4}$ grains of phosphorus and had exploded, probably on striking the iliac bone, so scattering the phosphorus.

Chemical tests (see p. 720).

References

1. Proc. Roy. Soc., Edin., Vol. LI, 8, 1931, Sharpey-Schaefer.
2. " Extra Pharmacopœia," 22nd ed., 210, 1941.
3. Jour. Amer. Med. Ass., Vol. CII, 1273, 1934, Rosenthal.
4. Jour. Path. & Bact., Vol. XXXV (2), 743, 1932, Ogilvie.
5. Brit. Med. Jour., Vol. II, 453, 1916, Millar.
6. Med.-Leg. Rev., Vol. V., Pt. II, 227, 1937, Dérobert.
7. Ibid., 228, 1937, Lewinski.
8. Jour. Industr. Hyg., Vol. XXV, 35, 1943, Cholak and Bambach.
9. Analyst, 725, 1929.
10. Jour. Amer. Med. Ass., Vol. CXV, 1536, 1940, Maehle.
11. " Industrial Toxicology," 663 *et seq.*, Hamilton and Johnstone, 1945. Oxford Medical Publications.
12. Science, N.S., Vol. LXXXIX, 322, 1939, Holmes, Amberg, and Campbell.
13. Jour. Amer. Med. Ass., Vol. XCVII, 851, 1931, Gleich.
14. Med.-Leg. Rev., Vol. VI, Pt. II, 209, 1938, Behrens.
15. Brit. Med. Jour., Vol. I, 367, 1947. Forbes.
16. Guy's Hosp. Reports, 81, 243, 1931, Hurst.
17. Brit. Med. Jour., Vol. I, 58, 1936, Willimott.
18. Jour. Pharmacol., Vol. LX, 113, 1937.
19. Med.-Leg. Rev., Vol. IV, Pt. IV, 331, 1936, Fatzer.
20. Jour. Amer. Med. Ass., Vol. CI, 352, 1933, Roe.
21. Lancet, Vol. II, 1039, 1936, Dowds.
22. Ibid., 738, 1945, Morton.
23. Jour. Amer. Med. Ass., Vol. CII, 1471, 1934, Sauer.
24. Edin. Med. Jour., N.S., Vol. XLV, 561, 1938, Tod and Stalker.
25. Brit. Med. Jour., Vol. II, 664, 1942, Blaxland.

CHAPTER XX

GASEOUS AND CERTAIN VOLATILE POISONS AND ANÆSTHETICS

IRRITANT gases have been classified as follows, largely on the basis of their solubility.[1]

Sub-group I includes those gases which act primarily upon the upper respiratory tract, for example, ammonia, hydrochloric acid fumes, sulphuric acid fumes, hydrofluoric acid gas and formaldehyde.

Sub-group II includes those which act upon the respiratory tract, but also spread their action to the deeper structures, for example, sulphur dioxide, chlorine, bromine, iodine and hydrogen sulphide.

Sub-group III includes those which act primarily on the lungs and only to a much less extent upon the upper respiratory tract, for example, nitrous fumes, phosgene, phosphorus trichloride and arsenic trichloride.

Sub-group IV includes those which are not altered or destroyed by contact with the tissues of the respiratory tract, for example, the fatty hydrocarbons, their alcohols, ethers and halogen substitution products, where important symptoms are induced by their action following absorption into the blood.

In the descriptions of the gaseous and volatile poisons which follow, we have not adhered to any classification.

Arseniuretted hydrogen or arsine.

This gas is formed when acids react with metals having an arsenic content, or by the action of water on calcium, magnesium, or sodium arsenide, or of dilute acid on other metallic arsenides.

It is a colourless and inflammable gas, possessing a disagreeable odour of garlic, and burns with a bluish-white flame.

Poisoning by this gas has arisen in a variety of industrial and scientific pursuits.

Arseniuretted hydrogen exerts its poisonous action after absorption into the blood stream through the lungs. Exposure to a concentration of 1 part per volume in 20,000 for an hour is dangerous and to a concentration of 1 part in 100,000 for twelve hours is fatal.

When arseniuretted hydrogen is absorbed into the blood stream, gradual and progressive hæmolysis of the red cells follows, with

liberation of hæmoglobin and methæmoglobin. The conversion of these substances by the liver into bile pigments, excessive in amount, produces jaundice. Hæmoglobin and methæmoglobin are also excreted by the kidneys with resultant tubular degeneration of the kidneys. The continued hæmolysis of the corpuscles leads to progressive anæmia (see p. 607).

Symptoms.

Malaise, shivering, headache, and great weakness, giddiness, faintness, pains in the head, epigastrium, and loins, sense of oppression in breathing, with, perhaps, some cyanosis, nausea, sickness, and vomiting. These are followed by continuous vomiting, with bile or blood in the vomit, jaundice with enlargement of the liver, varying in tint from golden-yellow to mahogany, thirst and dryness of the throat, hæmorrhages from one or more parts of the body, and hæmoglobinuria or hæmaturia, oliguria, or complete anuria. The urine varies in colour from red to reddish-brown depending on the amount of unaltered hæmoglobin, methæmoglobin, or bile pigments. It contains albumin and casts. The patient, who is on occasion delirious, gradually becomes comatose and there is a progressive deterioration of the pulse until death ensues.

Treatment.

Blood transfusion, about 500 millilitres on each occasion, is most beneficial, but should be given slowly. Diuresis must be encouraged by the administration of fluids by the mouth or rectum to avoid blockage of the renal tubules by cellular debris. A close estimate of the intake and output of fluid must be made to exclude the possibility of œdema. Sodium or potassium citrate is recommended : 3-4 drachms during each twenty-four hours should be given to ensure an alkaline reaction of the urine. Glucose, with or without insulin, should be given, and this line of treatment is of special importance when there is necrosis of the liver. Oxygen inhalations are necessary in the condition of excessive anæmia. Other treatment must follow lines indicated by individual cases.

Post-mortem appearances.

Jaundice is usually present. The lungs are congested. In the pericardium and pleuræ, reddish or reddish-brown coloured serum may be found. The myocardium very frequently shows fatty degenerative changes, and subendocardial hæmorrhages may be present. The liver is enlarged, and the gall-bladder distended with bile. The colour of the liver is often abnormal, ranging from yellow to deep indigo. The kidneys are also changed in colour, from dark red to indigo, and are enlarged. The spleen is very frequently enlarged and altered in colour. In the stomach there are congestion of the mucous membrane, submucous petechial hæmorrhages, and, occasionally, patches of erosion, in which changes the intestines often share. Fatty degenerative changes are also found in the liver

and kidneys. The urine contains hæmoglobin, methæmoglobin, red blood cells, casts, bile pigments, and albumin. Microscopical examination of the blood shows a considerable reduction in the number of red cells per cubic millimetre, an alteration in the shape and colour of the red cells, and the presence of "shadow cells." The blood-spectrum is frequently that of methæmoglobin.

Fatal quantity.

Generally regarded as small. Wignall,[2] however, has estimated the amount of arsenic excreted in the urine of patients poisoned by arseniuretted hydrogen over three to four weeks during detention in hospital, and holds the opinion that a greater amount of this gas may be taken by inhalation without causing death than has hitherto been believed. In one case, 796 milligrams of arsenious oxide per 100 millilitres of urine were found a few days following exposure, and four weeks later the urine contained less than 10 milligrams of arsenious oxide per 100 millilitres. This patient made a slow recovery.

Fatal period.

About five to seven days.

Antimoniuretted hydrogen.

This gas is not so toxic as arsine. In the treatment of ores, however, there can be little doubt that, from those which contain both arsenic and antimony, an operation which will liberate the one will also free the other.

Phosphoretted hydrogen.

This gas, heavier than air, is colourless, irritating, possesses a most unpleasant odour, reminiscent of rotten fish, and is highly toxic. Poisoning may occur industrially when the gas is liberated as a by-product. Ferrosilicon, used in the production of steel, may liberate both phosphoretted hydrogen and arseniuretted hydrogen when brought into contact with moisture. Fatal results have been caused in this way. Poisoning may also occur in those engaged in the preparation and use of calcium phosphide. The onset of symptoms is rapid after inhalation. The symptoms include oppression over the epigastrium, where a burning or piercing sensation may be felt, and breathing becomes rapid at first, but, later, slow and laboured. Nausea, vomiting, an odour of the gas in the breath, giddiness, profuse perspiration, convulsions, delirium and coma are frequently present. Death occurs from either cardiac or respiratory failure. There may be bronchitis, inflammation, or œdema of the lungs. The presence of gas may not be detected in the body after death. The post-mortem appearances are engorgement of the lungs, liver, and covering membranes of the brain, also engorgement of the heart with dark-coloured blood.

✓ Carbon monoxide.

This is a colourless, odourless, non-irritant gas which cannot be perceived by the senses. The sources are many and varied. Among the principal are gases from ignition of explosives, from blast furnaces, the slow combustion of blaes or iron-waste heaps, coal-gas itself, and coal-gas admixed artificially with variable proportions of water-gas, the fumes from geysers in bathrooms, lime-kiln burning, the air of mines, and exhaust gas from internal combustion engines. Explosions in confined spaces, such as in mines, produce poisonous quantities of carbon monoxide.

We have seen many cases of poisoning from coal-gas. Most of these were suicidal, some accidental, but in one or two instances they were homicidal. Illuminant gas is very commonly employed for the purpose of committing suicide. In the investigation of cases of death due to carbon monoxide, it must not be forgotten that this gas may be used for the purpose of homicide as well as of suicide, and, for this reason, great care should be exercised. In one case, for example, the dead body of a man was found lying on a bench in his workroom, and the gas jet above his head was turned on. A piece of gas-tubing with an attachment for fixing to the jet lay beside the body. Examination of the blood showed that it was almost completely saturated with carbon monoxide. The skin on various large areas of the body was of cherry-red colour. Post-mortem examination revealed a fracture on each side of the lower jaw. Two men were apprehended and charged with murder. A watch, chain, and money, belonging to deceased, were acknowledged by one of the accused to have been stolen by him. Owing to insufficient evidence connecting the accused with the assault and with the gas incident, one was found guilty of robbery and the other was discharged.

"Town's gas" contains a very considerable percentage of carbon monoxide, varying according to the method of production. In the vertical retort, steaming is introduced and the percentage is high, often reaching 22 per cent. Carbon monoxide is an odourless gas, but when mixed with ethylene gas the odour of the latter is always perceptible. A considerable number of deaths has been caused by the use of gas geysers in bathrooms, since, occasionally, they may generate carbon monoxide. The fractured gas main is a further source of poisoning.

As the result of lime-burning, deaths from this gas are also common. Exhaust gas from internal combustion engines contains from 1 to 7 per cent. of carbon monoxide depending on the richness of the mixture. A twenty horse-power motor-car engine will produce sufficient carbon monoxide, within five minutes, to render the atmosphere of a single car garage deadly, provided the engine has been run with the garage doors closed.[3] It is therefore evident that fatalities may readily occur in this way. Defects in the exhaust pipe of a motor car may permit the entry of carbon monoxide into the car and, depending upon the amount, may produce symptoms.

Choke-damp, which is a mixture of carbon monoxide and carbon dioxide, is liable to form in all collieries at "dead-ends," where the ventilation is imperfect. Blasting in mines and pit explosions are relatively common causes of fatal poisoning by carbon monoxide. In burning buildings the carbon monoxide in smoke is the cause of a considerable number of deaths. Occasionally cases occur under unusual circumstances. We made a post-mortem examination of the body of a man found dead in a boiler which he had been repairing, and it showed evidence of death by carbon monoxide.

Dangerous effects may also result from coke-stoves, and the use of flueless braziers. Owing to the use of gas in industry, chronic poisoning by carbon monoxide also occurs. Signs of chronic poisoning may become manifest in persons who have been exposed to regular small yet toxic amounts of this gas over a considerable period, but these are very variable in character and degree.

There is a definite susceptibility to carbon monoxide under certain conditions. This is seen at extremes of age, in anæmic subjects, and those who suffer from cardiac and respiratory conditions. Chronic alcoholics are also susceptible.

The total number of fatalities in Scotland due to carbon monoxide poisoning amounted to two hundred and twenty during the year 1946.

Symptoms of acute poisoning.

The symptoms of poisoning by carbon monoxide vary, depending upon the degree of saturation of the blood. Haldane has found that when the blood has absorbed carbon monoxide to the extent of 20 per cent., the symptoms only manifest themselves on exertion when there is a slight degree of shortness of breath and giddiness. Thirty per cent. saturation of the blood gives rise to symptoms when the individual is at rest. When 50 per cent. saturation is reached, the afflicted person shows inco-ordination, and staggers; loss of consciousness may be induced by exertion. Death occurs when the saturation of the blood reaches from 60 to 80 per cent. The action of carbon monoxide is cumulative, and even although there is only a small percentage of the gas present in the atmosphere, it is progressively linked up with the hæmoglobin, with displacement of oxygen, until a condition of oxygen starvation is established by the formation of carboxyhæmoglobin. When a person is at rest, 0·1 per cent. of carbon monoxide in the atmosphere inhaled may produce a blood saturation of 50 per cent. in two and a quarter hours, but with exercise, an hour would suffice. Carbon-monoxide poisoning in mines is usually a gradual process induced by small percentages of this gas. When the amount of oxygen is 300 times greater than carbon monoxide, half of the hæmoglobin can combine with the carbon monoxide and half with the oxygen, and this has been estimated as about the degree of blood saturation at which unconsciousness occurs. As the concentration of carbon monoxide in the air rises, the saturation of the hæmoglobin increases and the oxygen carrying capacity of the blood progressively diminishes

until symptoms of anoxæmia occur. Only in this respect can carbon monoxide be regarded as cumulative in action. The onset of symptoms of poisoning may be insidious; a feeling of lassitude may merge into a state of drowsiness with a dulling of the senses which is succeeded by unconsciousness. On the other hand, the prodromal symptoms may be giddiness, palpitation, breathlessness, weakness of limbs, which may preclude escape, and, finally, unconsciousness.

The affinity of carbon monoxide for hæmoglobin has been estimated as 300 times greater than that of oxygen.

Henderson and Haggard [4] give the following equations for the calculation of the physiological response to various concentrations :—

Time (in hours) × Concentration (parts per million) = 300, no perceptible effect.

Time (in hours) × Concentration (parts per million) = 600, just an appreciable effect.

Time (in hours) × Concentration (parts per million) = 900, headache and nausea.

Time (in hours) × Concentration (parts per million) = 1,500, dangerous.

In cases of acute carbon-monoxide poisoning the signs and symptoms are: deep coma; frequently the presence of a rosy tint affecting the lips and cheeks, but sometimes the face is pale and bedewed with perspiration or it may show, especially in deep coma, a leaden tint; the eyes are staring and glassy, the pupils are dilated and fixed; a small quantity of frothy fluid is usually present at the mouth; the breathing is shallow and quiet; the pulse is weak and almost imperceptible; subnormal temperature; coldness of the body. The patient suffers from anoxæmia and his state of oxygen lack may be as severe as that of blue-black victims of obstructive asphyxia (see p. 147). The rosy colour of the victim, which is due to the presence of carboxyhæmoglobin, is very misleading, especially since the action of respiration is apparently unembarrassed due to anoxic depression of the respiratory centre. Should the patient recover from the severe symptoms, it may take months before he is restored to his former health. Pneumonia is by no means an uncommon complication in severe cases and may cause death within a few days following exposure to the gas. In the various stages of acute poisoning, the symptoms might be confused with those due to drunkenness or acute alcoholic poisoning.

Shillito, Drinker, and Shaughnessey [5] have recorded data based on an investigation of nervous and mental sequelæ in carbon-monoxide poisoning. The New York Metropolitan area was chosen for this study, since at least 21,142 acute exposures occurred there in a ten-year period. For the same period, a survey of the state mental institutions serving this area showed thirty-nine certain cases of sequelæ of carbon-monoxide poisoning. When nervous or mental damage occurred, the intoxication had been extreme. None of the cases followed chronic poisoning from exposure over a long period.

In two-thirds of these cases, the symptoms started immediately following exposure, and in the remaining third, within one to three weeks. The mental sequelæ consisted of confusional psychosis, with disorientation, lack of judgment, and amnesia. Nervous sequelæ were ranged from altered reflexes to advanced Parkinsonism. Out of a total of forty-three cases, twenty-three patients made a complete recovery, nine suffered permanent nervous or mental sequelæ, and eleven died.

Martland [6] states that in cases showing post-asphyxial encephalitic syndromes, due to prolonged anoxæmia, the appearance of bilateral degeneration in the globus pallidus of the lenticular nuclei consists of elongated, brownish areas, grossly resembling thrombotic softening and measuring 1 to 2 centimetres in diameter.

Symptoms of chronic poisoning.

When there has been exposure to repeated small yet toxic doses of carbon monoxide, the following comprise the commoner symptoms :—Headache, giddiness, gastro-intestinal disturbances, anæmia, tachycardia, palpitation, præcordial pain, faintness, breathlessness on exertion, muscular cramps, depression, irritability, and general nervousness. In women the menstrual periods may be irregular.

Gettler and Mattice [7] have shown that the average carbon-monoxide content of the blood of eighteen persons, living in New York City under conditions of minimal exposure, was from 1 to 1·5 per cent. saturation. The average content of the blood of twelve persons, confined to a state institution in an ideal rural surrounding, was less than 1 per cent. The blood of twelve New York street cleaners was about 3 per cent., and the blood of two taxi-cab drivers was found to have between 8 to 19 per cent. on several occasions. Hanson and Hastings [8] report that, in a short series of normal individuals who did not smoke, the hæmoglobin was saturated with carbon monoxide to the extent of 1·5 per cent. In subjects who were smokers, the saturation varied from 3 to 5 per cent.

It would appear that such factors must receive consideration in the interpretation of the results of blood estimations.

Treatment.

In a case of carbon monoxide gas poisoning, it should be remembered that the linkage with hæmoglobin is reversible in the presence of oxygen. The patient must be removed from the vitiated atmosphere at once. Often this is followed by collapse, more especially if he is taken into the open air. The reason for this lies in the fact that the atmospheric carbon dioxide is much less than in the vitiated atmosphere, and since carbon dioxide plays an important part in carbon monoxide dissociation, the effects of deprivation are shown clinically. The body should be well wrapped up after hot-water bottles, suitably covered, have been applied. This is important

since carbon monoxide disturbs the heat regulating centre with reduction in the processes of oxidation. To commence with, artificial respiration should be applied if breathing is only slightly embarrassed or irregular. If respiration is poor, a mixture of 93 per cent. oxygen and 7 per cent. carbon dioxide should be given with a mask, in order to stimulate breathing and to break down the carbon-monoxide-hæmoglobin molecule. This treatment should be continued until normal respiration has been restored, and thereafter careful observation of the patient is necessary to detect an early indication of relapse. Before applying this treatment the airway should always be clear. Blood transfusion is of little benefit unless performed within an hour of the patient's removal from the poisoned atmosphere. The position of the patient should be changed from time to time to prevent accumulation of fluid in the bronchi and dependent parts of the lungs.

Haggard and Greenberg[9] state that intravenous injection of methylene blue is contra-indicated, that it is not an antidote, that its use probably exerts deleterious effects, and that it may produce fatalities which would not otherwise occur.

In serious cases a slow intravenous injection of 5 millilitres of 25 per cent. coramine may prove valuable.

Stimulants administered hypodermically will also prove of service. Prolonged rest is essential.

FIG. 225

Left, blood containing carbon monoxide. Note cherry-red colour.

Right, normal blood.

Post-mortem appearances.

There is usually a pink coloration of the face and a similar, but patchy, colour of various surfaces of the body. Post-mortem staining is of pinkish hue. The blood is of a bright red colour. The lungs are congested, often œdematous, and some frothy fluid is usually present in the upper air passages. The muscles and organs also show this bright red colour. Although the proximate fatal factor in illuminant gas poisoning is the contained carbon monoxide, the rosy-coloured markings on the body may not always be found since the unconsciousness and symptoms of asphyxia may be produced from the effects of combined gases, in varying quantity, which compose the illuminant gas. Petechial hæmorrhages may be seen on the meninges or surface of the brain, or both, and similar hæmorrhages may be present in the cortical substance and in the corpus striatum. The ventricles may contain blood-stained fluid. The brain may be œdematous. Microscopical examination of sections may show lenticular degeneration. Carbon monoxide is quite

rapidly eliminated from the blood of those who survive exposure, and therefore a sample of blood for examination should be secured as quickly as possible. In fresh air, and more especially when respiration has been stimulated by oxygen and carbon dioxide administration, about half the total quantity of carbon monoxide will be eliminated probably during the first hour, the remainder in greater degree in the next six to ten hours, and all traces of it will probably have disappeared within twenty-four hours. Thus long continued administration of carbon dioxide and oxygen may be unnecessary. After death, carbon monoxide disappears from the blood very slowly and may be detected after long intervals. It has

FIG. 226

Accentuated colouring of hypostasis, or post-mortem staining, in carbon monoxide poisoning. Note areas of contact flattening.

been found 210 days after death by chemical and spectroscopic examination. A sample of blood should be taken from the heart. There is no evidence that carbon monoxide is formed during the process of putrefaction. The gas cannot be absorbed by the blood in the deep vessels or heart after death.

Spectroscopic and chemical tests (see p. 712).

Nickel carbonyl.

Tetracarbonyl of nickel is a compound of nickel and carbon monoxide prepared by passing a current of carbon monoxide over finely divided metallic nickel. It is a gaseous compound which can be condensed into a liquid and becomes volatile at room temperature. This gas has been estimated as being fifteen times more toxic than carbon monoxide. In the presence of moisture, the inhaled gas becomes dissociated and thus finely divided particles of nickel are deposited over the respiratory epithelium and are absorbed into the circulation. Poisoning occurs in the industrial process of manufacture by inhalation of fumes or by working with the fluid. It is highly toxic when inhaled and, as a constituent of the atmosphere, to the extent of even less than 0·5 per cent., is dangerous to life.

Symptoms.

Headache, lassitude, sickness, and constriction over the chest are premonitory symptoms. Visual disturbances, tremors, insomnia, and metallic taste are also experienced. Dyspnœa, bronchitis, and broncho-pneumonia, with cough and blood-stained sputum, frequently follow. Death usually occurs between the fourth and twelfth days after exposure. Elimination of the poison occurs through the intestines and kidneys.

Post-mortem appearances.

These may be summarised as including congestion and œdema of the lungs, which show hæmorrhagic areas ; fatty degeneration of the heart, liver, and kidneys ; and hæmorrhagic areas, with degenerative changes and thrombi in some of the vessels in the brain, especially in the region of the corpus callosum, medulla, and in the upper part of the spinal cord. The presence of nickel may be found in the brain and lungs.

Carbon dioxide.

This gas, devoid of colour and odour, is often associated with carbon monoxide, as in choke-damp, lime-burning, and brick-burning. It is the cause of fatalities in workmen associated with well-sinking, well-cleaning, breweries, fermentation vats, and aerated water factories. In such circumstances, when it exists in large concentrations, over 30 per cent., it quickly produces powerlessness, insensibility, and death. When smaller percentages are present, the manifestations include somnolence, lassitude, and breathlessness. Constant exposure to air of ill-ventilated apartments produces anæmia, malnutrition, and other manifestations.

The post-mortem signs are those of asphyxia.

Treatment.

Fresh air ; artificial respiration ; oxygen inhalations ; restoration of body heat.

Carbon disulphide.

The vapour from this substance, when inhaled, rarely causes fatal acute poisoning, but chronic poisoning is by no means infrequent among those employed in occupations in which it is used, as, for example, in rubber works, especially in the "curing-rooms," or in the manufacture of artificial silk by the viscose process. Carbon disulphide poisoning when contracted industrially is notifiable and its occurrence must be reported to the Home Office (see p. 33). Acute poisoning is usually due to swallowing the liquid. It is held that a concentration of 1,150 parts per million is dangerous after an exposure lasting half an hour to one hour.

Symptoms.

The signs and symptoms in the acute form are: Unconsciousness, dilated and fixed pupils, embarrassment of breathing, with cyanosis, general symptoms of shock, due to the absorption of liquid, and an odour of carbon disulphide in the breath. Acute mental disturbances, including mania, may occur.

Chronic poisoning.

The symptoms of chronic poisoning are many and varied.

Any part of the central or peripheral nervous system may become affected.

Nausea, vertigo, headache, fatigue, insomnia, irritability, anæmia, and a perceptible odour in the breath are commonly found. If the exposure is continued, there may be impairment of vision and memory, tremors, evidence of optic neuritis with amblyopia, polyneuritis, impotence, emaciation, depression, and mental disturbances including acute hallucinatory psychoses. Pylorospasm may develop. As a rule the prognosis is good and mental disturbances subside rapidly with restoration approaching normal in about three to four weeks.

Treatment.

When the poison has been swallowed, gastric lavage should be used until the returned fluid is non-odorous. Stimulants may be given. When inhaled, administer oxygen.

Post-mortem appearances.

Acute poisoning.

When the poison has been swallowed, the manifestations include an odour of carbon disulphide in the cavities of the body, especially in the stomach and abdomen generally. The lungs and other organs are congested and there may be gastric submucosal hæmorrhages. When fumes have been inhaled, the respiratory system shows evidence of congestion and there is often œdema of the lungs.

In chronic poisoning there may be changes in the ganglion cells of the cerebral cortex, and parenchymatous degeneration of the peripheral nerves.

Fatal dose.

About $\frac{1}{2}$ ounce when swallowed.

Chemical tests (see p. 711).

Carbon tetrachloride or tetrachlormethane.

Carbon tetrachloride is a clear volatile liquid, with a rather heavy, unpleasant odour, which is used as a rubber and paint solvent. It is also contained in certain chemical fire extinguishers, is a non-inflammable dry cleaner, and is employed in the chemical industry.

It is further used in the treatment of ankylostomiasis. Poisoning may result from drinking the fluid or by inhalation of its fumes. A hundred parts per million is the maximum allowable concentration, and this is a dangerous concentration for continued exposure. When the fluid is sprayed on burning material the chief decomposition products include phosgene and chlorine (see pp. 596, 591). Thus when carbon tetrachloride is used as an extinguisher, poisoning may result from its fumes or those of decomposition products. When swallowed or inhaled it exerts a soporific action, depressing both cardiac and respiratory centres. The chief symptoms of poisoning are nausea, vomiting, pain in the epigastrium, faintness, loss of consciousness, and convulsions. Clinical manifestations of impaired renal function are common and vary in degree. The urine may be dark-coloured and this manifestation is frequently followed by jaundice. The urine may be loaded with casts and albumin, and death may result from uræmia. The liver may also be damaged, with accompanying symptoms of jaundice, bradycardia, hæmatemesis and melæna.

Treatment.

Treat respiratory failure by artificial respiration in the open. If the poison has been swallowed, stomach lavage must be employed without delay. The patient should be kept warm and symptoms treated along usual lines. McGuire [10] states that serious cases should be treated with adrenalin, caffeine sodium benzoate, and digitalis, together with calcium lactate. He holds the view that calcium deficiency and the use of alcohol render the individual more susceptible, and that calcium therapy is particularly indicated in treatment. A beneficial line of treatment is the administration of 10 millilitres of 10 per cent. solution of calcium gluconate intravenously two or three times daily with 12 grammes calcium gluconate, or 8 grammes of calcium lactose by the mouth.

Post-mortem appearances.

These vary with the mode of administration. A characteristic odour is usually perceived, and there may be fatty degeneration of heart, liver, and kidneys. The larynx, bronchi, and lungs will be congested, in varying degree, and evidence of broncho-pneumonia may be present if the fumes have been inhaled, while the gastric mucosa and the lining of the œsophagus will probably show evidence of inflammatory irritation with or without areas of hæmorrhage, when the drug has been swallowed. Jaundice, following liver involvement, and acute nephritis are frequently present.

Sulphuretted hydrogen and sulphur dioxide.

The principal irrespirable gases of sulphur are sulphuretted hydrogen and sulphur dioxide, the latter, because of its extremely pungent character and its consequent irritant action upon the mucous membrane of the respiratory tract, and the former, chiefly

because of its action on the blood after absorption. Sulphuretted hydrogen is a colourless gas with an odour resembling rotten eggs and may be evolved whenever organic material decomposes. It is heavier than air and tends to collect in vats and cellars. When mixed with air it is inflammable. The oil industry is a common source of accidental poisoning. Poisoning by inhalation occurs in workmen employed in chemical works, and in connection with sewers. While sewer-gas is a mixture of several gases, sulphuretted hydrogen is undoubtedly the most lethal. When sewers are badly ventilated, oxygen is reduced, nitrogen increased, and carbon dioxide is present in large amount, together with the sulphides of hydrogen and ammonia. The symptoms of poisoning depend on the proportion of those gases present. When concentrated mixtures are inhaled, there is profound depression of the central nervous system and death may be immediate. If diluted, lividity is usually well marked, and there are fixation of pupils and insensibility, often accompanied by convulsions. Even in small percentages, sulphuretted hydrogen prejudices the health of those constantly exposed. A concentration of between 100 and 400 parts per million causes irritation of the eyes, nose, and throat, one of 500 parts per million will induce headache, giddiness, excitement, inco-ordination in walking, diarrhœa and painful micturation, after about thirty minutes, a concentration exceeding 500 parts per million will cause severe poisoning, while concentrations of 1,000 to 3,000 are rapidly fatal.

Sulphur dioxide is a colourless gas which has a pungent odour and an irritant effect when inhaled. Poisoning may occur during the process of manufacture of this gas, or as the result of its employment in certain metallurgical and chemical works, or when the gas is used either for the purpose of disinfection or refrigeration. It is not as toxic as sulphuretted hydrogen.

Sulphuretted hydrogen or hydrogen sulphide.

Symptoms.

This is both an irritant and general poison. Its immediate lethal toxicity is not much below that of hydrocyanic acid. The symptoms of acute poisoning develop quickly and consist of dyspnœa, cyanosis, and other asphyxial signs, due to stimulation of the vagus nerve, and direct irritant action upon the alveolar mucous membrane of the lungs which causes a progressive pulmonary œdema. Convulsions are frequent. Sulphuretted hydrogen does not combine with oxyhæmoglobin, but only with methæmoglobin, and the presence of sulphæmoglobin is largely the result of post-mortem changes. The gas also acts upon the nervous system, and in cases of subacute poisoning, psychic manifestations may develop and accompany bronchitis, and other symptoms of respiratory tract irritation. In cases of acute poisoning, the cause of death is asphyxia, the result of paralysis of the respiratory centre.

Sulphur dioxide.

Symptoms.

Fifty to one hundred parts per million is the maximum concentration permissible for a period of from half an hour to one hour.

Irritation of the eyes, nose, and respiratory tract, an acid taste in the mouth, bronchitis, broncho-pneumonia, œdema of the larynx and lungs, a livid colour of the mucous membranes, and symptoms of gastro-intestinal irritation are usually found.

Sulphuretted hydrogen or hydrogen sulphide.

Treatment.

In acute cases the principal line of treatment is the administration of artificial respiration and the use of oxygen combined with 5 to 7 per cent. of carbon dioxide. Bronchitis and broncho-penumonia require the usual lines of treatment when they arise as complications.

Post-mortem appearances.

These are chiefly asphyxial signs, but the blood has a darker hue than the ordinary asphyxial cases, an odour of the gas will be perceptible, and putrefaction is rapid. There will be evidence of respiratory irritation and probably the lungs will be congested and œdematous. In some cases the tissues and liver may present a greenish-gray or ashen tint due to the post-mortem combination of hydrogen sulphide and methæmoglobin.

Sulphur dioxide.

Treatment.

Removal to fresh air; artificial respiration, which should be continued for a very considerable period, even in apparently hopeless cases; oxygen inhalations with 5 to 7 per cent. carbon dioxide, restoration of bodily warmth, and stimulants.

Post-mortem appearances.

There will be asphyxial signs and evidence of irritation of the respiratory passages.

Chemical tests (see p. 723).

Chlorine.

Chlorine is a greenish-yellow gas, two and a half times heavier than air, with a smell like that of bleaching powder. When inhaled, it exercises both an irritant and suffocative effect. Industrial poisoning is caused by accidents in the course of its manufacture and from its use in the chemical industry. Chlorine inhalation is not

an uncommon occurrence in workers engaged in the manufacture of hydrochloric acid and bleaching powder, but fatal results in such occupations are rare, and when they occur are more often due to pulmonary complication than to general constitutional effect. Exposure to 1,000 parts per million will, after a short period, produce death.

The results of acute poisoning are frequently fatal.

The symptoms of acute poisoning are intense irritation of the eyes and mucous membrane of the respiratory passages, with extreme dyspnœa, nausea and vomiting, and spasm of the glottis. Death is caused by cardiac failure following inflammatory œdema of the lungs and pulmonary congestion. Should the patient survive for forty-eight hours, septic broncho-pneumonia may be a later and fatal complication. Chlorine inhalation in repeated small amounts may give rise to a chronic condition which may result in anæmia, cachexia, dental caries, and phthisis. Bronchitis and emphysema are common.

Treatment.

Rest and maintenance of body warmth are imperative; the chief danger is acute pulmonary œdema; oxygen from an inhaler should be administered promptly and continuously while œdema is present and until there is no return of cyanosis after withdrawal; venesection is necessary when there is deep cyanosis and venous engorgement; 300 to 400 millilitres of blood should be withdrawn up to a total quantity of 700 millilitres, if necessary; contra-indications are leaden grey colour with circulatory failure, low blood-pressure, and a small, rapid, or impalpable pulse; transfusions and infusions are contraindicated in pulmonary œdema, but intravenous injections of saline may otherwise be given to reduce the high viscosity of the blood; expectoration should be encouraged by posture and not by expectorant mixtures; camphor and coramine should be given in collapse; atropine, strychnine, digitalis, and alcohol are contraindicated; morphia should be restricted to $\frac{1}{6}$-grain doses; when infective bronchitis or broncho-penumonia develops, sulphonamide drugs or penicillin should be given.

Post-mortem appearances.

These are principally asphyxial due to acute pulmonary œdema. The lungs are heavy and water-logged. On section, considerable congestion, occasional thrombosis of the network of pulmonary vessels, an abundant outpouring of fluid, and disruptive emphysema of weakened lung tissue, due to coughing, may be observed. Petechial hæmorrhages may be visible on the pleural surfaces, and the pleural cavities will probably contain a variable quantity of serous or blood-stained fluid. Infective bronchitis, broncho-penumonia, or pleurisy may be present. The chambers on the right side of the heart will be dilated and contain a considerable quantity of blood, as will the associated large vessels.

Iodine.

Liquid preparations of iodine, such as the liniment or tincture, have been used for suicidal purposes, and cause corrosion of the parts with which they come in contact on swallowing. Iodine vapour is very irritant to the respiratory tract. Its potassium salt, largely used in medicine, has given rise upon occasion to dangerous symptoms, but in the cases recorded, the doses were not exceptionally large, and its exaggerated action must be associated with personal idiosyncrasy.

When iodine is swallowed the symptoms are those of a corrosive and irritant poison. Inflammation of the mucous membrane of the mouth, pharynx, œsophagus, and stomach, accompanied by dark brown or yellowish staining of the parts, an intense, burning pain in the mouth, œsophagus, and stomach, vomiting, and signs of shock commonly occur.

One drachm of the tincture has caused death, but recovery has followed the taking of 1 ounce.

Treatment consists of free gastric lavage with starchy fluids, and the rest of the treatment should be conducted along general lines.

Chemical tests (see p. 716).

Iodoform.

Iodoform is employed as an antiseptic and has a characteristic, penetrating odour. It has caused toxic symptoms when absorbed from a large raw surface, and has caused death when dissolved in ether and injected into large tubercular abscesses. The post-mortem findings include nephritis and fatty degenerative changes in the liver. The symptoms are those of gastro-intestinal irritation, accompanied by skin eruptions and a rise of temperature. Following injection, there may be delirium, coma, and death.

Chemical tests (see p. 716).

Bromine.

This is a dark brown liquid which emits a pungent, acrid odour. When the gas is inhaled, it is highly irritant in its action and involves the entire respiratory tract. The mucous membranes show a brownish-yellow discoloration. Œdema of the glottis sometimes results, and œdema of the lungs, with fatal result, is common. For treatment, see p. 592.

Methyl bromide.

This substance is used as one of the components in the manufacture of pigments, in the preparation of antipyrin, in certain fire extinguishers, and as a fumigant for insects. It is practically odourless, but its thermal decomposition products are almost

irrespirable. When the vapour is inhaled toxic symptoms occur. The early symptoms are headache, giddiness, vomiting, and drowsiness. Following these, there is usually a period of intermission, which may last for quite a long interval, without evidence of illness. After this, there is full development of the signs of poisoning. The poison produces irritation of the central nervous system, and the symptoms include motor and sensory paralysis, inco-ordination, visual disturbances, slurring speech, mania, delirious states, convulsions, pulmonary œdema, with dyspnœa and cyanosis, and death. The urine may show the presence of volatile halogen and methyl alcohol.

The use of adrenalin or glutathione has been recommended in the treatment of this form of poisoning.

Since methyl bromide affects the vasomotor system, all the organs, including the brain, will be found in a state of congestion on post-mortem examination Two interesting cases of fatal poisoning have been reported. One of a boy who came into contact with an accidental discharge from a fire extinguisher. He was dead when found. The other, of a man who had been engaged, for three days in the open air, in emptying methyl bromide fire extinguishers. He collapsed, went into coma, took convulsions, and died. His blood contained 34 milligrammes of methyl bromide per 1,000 millilitres of blood. The post-mortem findings consisted of multiple minute hæmorrhages scattered throughout the brain, myocardium, spleen, and lungs. Degenerative nephritis and broncho-pneumonia were present.

Sodium and potassium bromide (see p. 573).

Chemical tests (see p. 722).

Bromoform.

This is a clear, heavy, oily liquid which volatilises at low temperature. Deaths have occurred from its use. It has an odour of chloroform, and its vapour produces symptoms similar to those caused by chloroform inhalation.

Fluorine.

Fluorine is a member of the halogen group, and its compounds hydrofluoric acid and sodium fluoride are important toxicologically. Hydrofluoric acid gas or fluorine is a greenish-yellow gas which exerts a very irritant effect; 50 to 250 parts of this gas per million parts of air are dangerous for even short exposure. In the presence of aqueous vapour it is converted into hydrofluoric acid. This acid is used extensively in the process of etching upon glass and metals, and is sometimes used for cleaning sandstone, marble, and porcelain enamel. Sodium fluoride and sodium silico-fluoride are constituents of some insecticide powders and fluorides are used in smelting steel and aluminium. The fumes from the acid are extremely irritant

to the respiratory passages and lungs and cause pain in the throat, laryngitis, and bronchitis. There is also vomiting and collapse. Ulceration of the conjunctivæ, nose, mouth, and gums is often produced. When the gas comes into contact with the skin, severe burns, which heal slowly, result. Sodium fluoride or sodium silicofluoride, is a protoplasmic poison which paralyses cellular respiration and deprives tissue of calcium by forming non-ionisable calcium fluoride. When it is swallowed, symptoms of gastro-intestinal irritation in acute cases may be very severe, vomiting, purging, and griping being common manifestations. There is dyspnœa, and respiration becomes rapid. The pulse becomes weak, and muscular cramps in the legs, sensory disturbances, paralysis, tremors, and convulsions may follow. Albuminuria is common. Unconsciousness precedes death from respiratory failure. When the acid is swallowed, the symptoms are similar, but there is usually a greater involvement of the respiratory passages and lungs due to the liberation of fumes.

Lidbeck, Hill, and Beeman[11] report a case of acute sodium fluoride poisoning from the accidental use of "roach" powder instead of powdered milk in mixing scrambled eggs at a hospital. The mistake caused two hundred and sixty-three cases of acute poisoning, forty-seven of which ended fatally. In many cases blood was present in the vomitus and stools. Death in most cases occurred two to four hours after ingestion. In some cases in which death was delayed and in some which recovered, there was paralysis of deglutition, spasm of the extremities, and carpopedal spasm. The egg mixture contained 3·2 to 13 per cent. of sodium fluoride.

In 1930, smoke from a number of factories became mingled with a fog which hung over the Meuse Valley. Several thousand persons developed pulmonary symptoms and there were sixty deaths, chiefly among the elderly members of the community. Fluorine was regarded as the probable chemical cause of this, the "Belgian Fog Disaster."

In cases of chronic fluorism, mottling of the dental enamel is characteristic, and sclerosis of the bones may result from fixation of calcium due to fluorine. Gastric symptoms may also present themselves.

Treatment.

When fluorides have been swallowed, gastric lavage with lime-water, milk, or medicinal charcoal in suspension, should be employed. Copious bland fluids should be given, and castor oil is a suitable purgative.

Post-mortem appearances.

When fluorides have been swallowed, there is usually a hæmorrhagic gastritis with œdema and corrosion of the gastric mucosa and a dark crimson discoloration of the rugæ. The upper part of the small intestine is frequently involved.

The acid produces such signs as ash-grey bleaching of the buccal mucosa, and denudation of the epithelium of the tongue, pharynx,

and œsophagus. The gastric mucosa is in a similar condition to that already described, but there may also be marked corrosion and blackish discoloration, with submucosal hæmorrhages. When the fumes have been inhaled, the respiratory passages and lungs are chiefly implicated and pulmonary congestion and œdema, together with congestion of the larynx and trachea, will be present.

Chemical tests (see p. 715).

Phosgene or carbonyl chloride.

This gas is used in the preparation of dyes, in chemical and pharmaceutical industries, in the preparation of several metallic oxides, and is met with as a decomposition product of trichloroethylene and carbon tetrachloride, used in certain types of fire extinguishers. Rapidly fatal results will follow exposure to over 25 parts of the gas per million parts of air. Phosgene owes its toxicity to the fact that it is readily split up, in the presence of water, into hydrochloric acid and carbon dioxide. Evolution of hydrochloric acid occurs in the smaller bronchi and in the alveoli. The essential lesions produced are pulmonary œdema, rupture of the pulmonary alveoli, and concentration of the blood, with an increased viscosity and tendency to thrombosis. For treatment see pp. 592 and 597.

Ammonia gas.

Poisoning from ammonia fumes occurs industrially in the course of the manufacture of ammonia, the handling of containers, and among other sources, in working with refrigeration plant. This gas affects the tissues with which it is brought into contact and is not absorbed into the system. A concentration which is dangerous for an exposure of half an hour is 2,500 to 4,500 parts of the gas per million parts of air. The excellent report on forty-seven cases of ammonia gas poisoning published by Caplin [12] has covered the salient points in this connection, and we take the liberty of quoting freely from this authoritative contribution.

The cases in question occurred during a heavy air raid in London, as the result of a connecting pipe of an ammonia condenser in a cellar having been damaged by a piece of flying metal. Caplin divides the cases into three groups, according to the nature of the symptoms shown by the poisoned persons, namely, mild, moderate, and severe. The mild group comprised nine cases. These persons showed irritation of the conjunctivæ and upper respiratory passages. They also suffered from inflammation of the lips, buccal mucosa, and swelling of the fauces. The moderate group, which included twenty-seven cases, in addition to the above symptoms, suffered from hoarseness, dysphagia, and bronchitis. Eleven cases, relegated to the severe group, showed evidence of shock, cyanosis, intense dyspnœa, persistent cough, with frothy sputum, and suffered from bronchitis and pulmonary œdema. All cases, classified as mild,

recovered. Three cases in the moderate group died after thirty-six hours from pulmonary œdema ; nine others developed broncho-pneumonia, and three of these cases also died within two days. Of the cases relegated to the severe group, four died within forty-eight hours following admission to hospital, and three other cases died later. The cause of death in all instances was broncho-pneumonia.

The treatment was directed to shock. Electric cradles and morphia ($\frac{1}{6}$ to $\frac{1}{4}$ grain in severe cases) were used. The dose of morphia was reduced when dyspnœa and cyanosis were present. Oxygen inhalation by intratracheal catheter was administered to relieve cyanosis. When moist sounds in the lungs were pronounced, $\frac{1}{100}$ grain of atropine was given and repeated at two-hourly intervals. To neutralise the ammonia on the buccal mucous membrane, diluted vinegar was first used as a gargle and mouth-wash, and later liquid paraffin was applied. For pain and œdema of the mouth and throat, a cocaine and adrenalin spray was employed. The condition of the eyes was relieved by irrigation with boracic solution and the instillation of a few drops of castor oil.

Nitrous fumes.

Nitric acid when exposed to air emits both nitrous and nitric oxide fumes and these when inhaled produce a caustic effect on the respiratory tract. The liberation and inhalation of nitrous fumes occur during many industrial processes, including the treatment of metal with nitric or sulphuric acid and oxyacetylene welding under conditions of deficient ventilation. A considerable quantity of a mixture of oxides of nitrogen is liberated when nitro-explosives are incompletely detonated or subjected to a process of slow combustion. Such nitrous fumes are dangerous when they vitiate a confined space. The fumes have either a yellow or reddish-brown colour, and form nitrous acids in the presence of moisture and oxygen. In one case, with which we were associated,[13] a number of men were employed in a ship in heating a heavy steel crosshead. Two large oxyacetylene burners were used, each consuming about 200 cubic feet of oxygen and acetylene per hour. The compartment was of about 5,000 cubic feet capacity and was ventilated by air injected from a blower together with two compressed-air jets. After about forty minutes, several of the men collapsed and one of them died in hospital about thirty-two hours later. The symptoms were typical of nitrous fumes. In autogenous welding, due to the generation of great heat, the oxygen and nitrogen of the atmospheric air combine to form oxides of nitrogen. The post-mortem findings consisted of massive œdema with commencing pneumonia. The blood was darker than usual. The lungs were of yellowish-brown colour.

The principal danger of inhalation of nitrogen oxide is that dangerous or fatal œdema of the lungs may follow an inhalation, not regarded as significant at the time on account of the fact that respiratory irritation has not been experienced since only the deeper parts of the respiratory tract are involved. When the fumes come

into contact with moisture in the respiratory system they are converted into nitric and nitrous acids, and thus a caustic effect is produced with resultant congestion of the lungs and inflammatory œdema. In such cases nitric acid may be found in the blood.

The usual symptoms are severe cough, pain in the chest, vomiting, dyspnœa, cyanosis, and a reddish-brown sputum. In addition to massive inflammatory œdema of the lungs, there may be œdema of the brain, swelling of the liver, and necrosis of the kidneys (see p. 528).

Treatment should be along lines similar to that for other irritant gaseous poisons (see p. 592). In addition, the following lines of treatment have been recommended. For pulmonary œdema, intravenous 50 per cent. dextrose, 50 to 100 millilitres, up to 200 millilitres daily. When oxygen is used, it is suggested that it should first be passed through a bottle containing 2 grammes ammonium carbonate to 1·5 ounces of water.

Chemical tests (see p. 718).

Acetylene.

This is a colourless gas with a garlic odour produced by the interaction of water and calcium carbide. It is irrespirable and inflammable, and in crude form may contain both arseniuretted hydrogen and phosphoretted hydrogen.

Frequently used in the oxyacetylene burning process and less frequently as a source of domestic illumination, poisoning has occurred. Acute symptoms include giddiness, nausea, vomiting, headache, palpitation, tachycardia, tremors, cramps, excitability, bronchitis, and unconsciousness.

Hydrocyanic acid or prussic acid.

Hydrocyanic acid, when pure, is a colourless, volatile liquid, evolving a strong odour of peach-blossom, laurel-water, or bitter almonds. It is feebly acid and reddens litmus paper slightly. The pharmacopœial preparation contains about 2 per cent. of the anhydrous acid, and that of Scheele, about 4 per cent. The vapour, when inhaled, causes a sense of constriction in the throat. The acid has a pungent or bitter taste. It has never been found as a natural constituent of the body, although traces of sulphocyanides are found in the saliva. There is no convincing evidence that it may be a product of putrefaction.

Amygdalin and emulsin exist in the kernels of various fruits, such as peaches, plums, and bitter almonds, and react upon one another in the presence of water to form hydrocyanic acid. It is also found in the leaves of cherry-laurel, a preparation of which is used medicinally, and which contains about 0·08 to 0·1 per cent. In oil of bitter almonds it is present in much larger percentage than in preparations from other fruits, the quantity of acid varying up to 10 per cent., but, for pharmacopœial purposes, the amount of acid is adjusted to contain from 2 to 4 per cent. Eating bitter almonds has produced toxic effects on more than one occasion.

Hydrocyanic acid combines with bases of several metals to form cyanides. Those of the alkalis and alkaline earths readily give off hydrocyanic acid in the presence of even the feeblest acid. The cyanides which are formed with the bases of heavy metals, such as mercury, zinc, lead, copper, and others, have no odour, are insoluble in water, but give off hydrocyanic acid in the presence of mineral acids. Hydrocyanic acid, in combination with metals, forms cyanides, such as potassium ferrocyanide and ferricyanide, and, in combination with sulphur, sulphocyanides. All of the sulphocyanides, although odourless, are poisonous, and give off hydrocyanic acid when distilled with acids.

Sodium and potassium cyanide are used industrially in connection with electro-plating, coating silver, case hardening of steel and iron, and in tanning. Calcium cyanide is utilised as a fertiliser.

Of preparations likely to cause poisoning, oil of bitter almonds, potassium cyanide, used in photography, electro-plating, and gold-recovery, and hydrocyanic acid itself are the most common. Laurel-water has also caused poisoning.

Hydrocyanic gas, or hydrogen cyanide, in high concentration is a fulminant poison which causes very rapid death by paralysing the respiratory centre in the brain. Henderson and Haggard state that in low concentration it may be detoxicated in the body as quickly as it is absorbed, that the gas is capable of being absorbed by the skin, especially when moist with perspiration due to its ready solution in water, and that it is dangerous to remain too long in a high concentration even when wearing a respirator.

The fumigation of premises with hydrogen cyanide is a common practice. It is recommended that the gas should be combined with a lachrymatory gas, such as cyanogen chloride, in order to warn those who are taking part in the process of fumigation that hydrogen cyanide is present. By the Hydrogen Cyanide (Fumigation) Act, 1937, which is applicable to both England and Scotland, whenever any accident occurs which occasions loss of human life or personal injury as the result of the fumigation of any premises or article, the person by whom, or by whose agent, the fumigation was carried out shall forthwith send, or cause to be sent, to the Secretary of State, notice of the accident and of the loss of human life or personal injury.

In a case with which we were associated, a ship's fireman was found dead in his bunk. The ship was berthed and had been fumigated with hydrocyanic gas. A sample of blood, the liver, heart, and lungs all showed the presence of hydrocyanic acid.

Two men, employed in a chemical works near Glasgow, were engaged in cleaning out sodium cyanide tanks after removal of the fluid. The men, one inside the tank to fill buckets with the deposit and the other outside to empty them, were overcome by hydrocyanic fumes, and although promptly rescued and resuscitative measures were employed, they quickly died.

Symptoms.

These, to some extent, depend on the preparation which is swallowed, and its quantity. In large doses, the acid produces death almost immediately. There is rapid loss of consciousness and muscular power. Prolonged and deep, but jerky, respirations, cyanosis, irregular pulse, palpitation, coldness of extremities, dilated pupils non-reactive to light, convulsive seizures, and death form the usual clinical picture. A small quantity of fine froth may appear at the mouth.

In poisoning by potassium cyanide the rush of events is not quite so sudden, and minutes elapse before the symptoms commence. The principal symptoms include giddiness, dyspnœa, dilated and fixed pupils, convulsive seizures, and insensibility. The face is cyanosed, the jaws may be tightly clenched, the respirations are jerky, and the pulse is small and rapid.

When a high concentration of hydrogen cyanide is inhaled a fatal result ensues in a few minutes. Vertigo, palpitation, and dyspnœa commence after a few breaths have been taken, and the subject falls to the ground in a state of unconsciousness. Convulsions usually occur, and death supervenes from paralysis of the respiratory centre and circulatory failure. When low concentrations are inhaled, there are usually transient symptoms of headache, vertigo, nausea, and mental confusion.

Hydrocyanic acid and the cyanides must be regarded as true protoplasmic poisons since they arrest the activity of all forms of living matter by inhibiting tissue oxidation and suspending vital functions. Asphyxia is caused by inhibition of tissue respiration, which accounts for the bright colour of the blood in the venous system, since oxygen is not abstracted from it by the tissues and it thus returns to the veins in arterial condition. Such poisons not only inhibit the enzymic activities, but also act upon the central nervous system.

Repeated exposure to non-lethal doses of cyanide may produce such clinical manifestations as headache, vomiting, diarrhœa, chronic cachexia, and mental disturbances.

Treatment.

When the poison has been swallowed, there should be immediate use of the stomach tube, and free lavage with warm water either alone or containing a 5 to 10 per cent. solution of sodium thiosulphate, or a mixture of the sulphates (ferrous and ferric) of iron followed by a solution of potassium carbonate, to form Prussian blue, which is inert. Hydrogen peroxide, in 3 per cent. solution, or a 0·2 per cent. solution of potassium permanganate, may be used, and emetics, if a tube is not available. Stimulants, such as strychnine, $\frac{1}{8}$ grain, or caffeine sodium benzoate, 2 grains, may be given hypodermically. Ammonia inhalations, artificial respiration, and oxygen, with or without carbon dioxide (5 to 7 per cent.), should be given to stimulate

respiration. Atropine hypodermically should be tried with a view to stimulating the respiratory centre. Sodium thiosulphate may be administered intravenously (10 to 50 millilitres of 20 per cent. solution), provided there is sufficient time for it to act, or methylene blue (50 millilitres of 1 per cent. solution). Gettler and Baine [14] express the view that methylene blue, when administered intravenously, is of definite value in at least prolonging life for an hour or more and that when supplemented by other lines of treatment, there is a chance of recovery, provided the treatment is instituted as soon as the cyanide has been taken. Intravenous administration of 1 millilitre of 0·1 per cent. Merck's medicinal methylene blue, in Ringer's solution, per kilo of body-weight is recommended. Methylene blue should not be dissolved in physiological solution of sodium chloride since precipitation occurs. They also recommend stimulation of the cardiac and respiratory centres by injections of lobeline and cardiazole. Geigor [15] has used methylene blue intravenously in cases of potassium cyanide poisoning with very beneficial and successful results. He states that the dye can be used in quantities up to 100 millilitres of a 1 per cent. solution within a period of half an hour without untoward symptoms, and that by using this full amount, measurable quantities of free methæmoglobin were not produced. The methylene blue treatment appears to act by producing methæmoglobin which in turn combines with the cyanide to decrease the poisonous effects of the latter. Jetter considers that the use of methylene blue should now be regarded as obsolete and that the principle to be aimed at in treatment is to convert hæmoglobin into methæmoglobin so that the cyanide will combine with the latter compound to form non-toxic cyanmethæmoglobin. He is of the opinion that methæmoglobin is best produced by the inhalation of amyl nitrite, substitutes being sodium nitrite and sodium thiosulphate. P-aminopropiophenone, or PAPP, has recently been recommended as an antidote since it apparently acts by forming methæmoglobin which then removes the cyanide ions from the tissues by the formation of cyanmethæmoglobin.

When hydrogen cyanide has been inhaled, the patient should be speedily removed from the contaminated atmosphere. The use of artificial respiration, and oxygen with carbon dioxide, is of the highest value. In desperate cases, a slow venous injection of 5 millilitres of a 25 per cent. solution of coramine has been recommended.

Cyanide is metabolised quite rapidly to thiocyanate and in this form it is excreted.

Post-mortem appearances.

The face, including the lips, are of reddish colour and the body surface frequently shows pinkish, irregular patches which are not as pronounced as in carbon monoxide poisoning. Internally, the signs are remarkably negative, except that the lungs and right side of the heart are engorged. The odour of prussic acid will probably be perceived on opening the chest cavity, and from the contents

of the stomach. The mucous membrane of the stomach may be of a reddish colour, and the other organs, together with the blood, are usually of the same shade. The cause of the bright red colour of the blood has already been explained. Cyanides are rapidly altered in the body after death since they unite with sulphur to form sulphocyanides.

Fatal dose.

The smallest dose which has proved fatal is $\frac{1}{2}$ drachm of the pharmacopœial acid, or 0·6 grain of anhydrous hydrocyanic acid. Recovery has followed the use of much larger doses, namely, 1·3, 2·3, and 4·8 grains of anhydrous acid, respectively. It may be taken that 1 drachm of the pharmacopœial acid will kill. Two drachms of the oil of bitter almonds killed a man in seventeen minutes. Five grains of the cyanide of potassium have produced death, but recovery has followed larger doses.

120 to 150 parts of hydrogen cyanide per million parts of air is a dangerous concentration in half an hour to one hour; 3,000 parts per million prove rapidly fatal.

Fatal period.

When a large dose of the acid is swallowed death may occur almost immediately. In acute poisoning, two to five minutes may elapse, but when smaller doses of the cyanides have been taken, death may not result for from twenty minutes to an hour. In a case of suicide, in which a woman swallowed a weak preparation of the acid, death did not ensue for forty minutes. In another suicidal case, life was prolonged for three and a half hours after swallowing the poison which was mixed with milk.

Chemical tests (see p. 715).

Formaldehyde.

Formaldehyde, and formalin, which is a 40 per cent. watery solution of formaldehyde, are used for aerial disinfection of rooms and for preservative purposes. The former is used commercially in the aniline colour industry, also in the photographic and chemical industries. The vapour is colourless, has a very pungent odour, and provokes intense irritation of the mucous membranes and conjunctivæ. Dyspnœa, bronchitis, and œdema of the lungs may also occur. Death from the effects of inhalation of fumes is exceptional. When strong solutions are brought into contact with the skin, dermatitis will result. Anuria is an occasional complication. A number of cases of poisoning by swallowing formalin have occurred, and the details of two will provide a picture of both symptoms and post-mortem signs.

A woman swallowed about 4 ounces of a 40 per cent. formaldehyde solution. She was collapsed and drowsy, but when roused, complained of great pain in the mouth, throat, and epigastrium. There was a strong odour of formalin from her breath.

The stomach was washed out with ½ pint of water containing 2 drachms of liquid ammonia to the pint. Later, she vomited some blood-tinged fluid. She then became restless and emotional, and the pulse deteriorated. Later in the evening she became unconscious and died. Post-mortem examination showed a bleaching of the surface of the tongue, with eroded areas and hæmorrhagic patches. The upper air-passages were markedly congested and definitely œdematous. The right chambers of the heart were engorged with dark blood. Both lungs and bronchi were acutely congested and œdematous. The gastric mucosa was congested and showed patchy desquamation. It was somewhat leathery to the touch.

In the second case, a man swallowed a quantity of formalin. Post-mortem examination showed that the mucous lining of the lower end of the œsophagus was discoloured, excoriated, and indurated. The stomach, in a similar condition, was dark grey in colour, the walls were contracted, and the mucosa, which showed patchy desquamation, was thrown into folds. A few small hæmorrhages were present. The first part of the duodenum showed similar changes. The odour of formalin was detected on opening the body and was perceived in the stomach. Both lungs were congested. The blood-vessels of the brain were also congested and well-marked punctate hæmorrhages were visible in the substance of the brain. Death was due to comato-asphyxia.

Treatment.

Free gastric lavage with tepid water and a solution of ammonium acetate or diluted ammonia (1 drachm to 1 pint of water). Some of this solution should be left in the stomach to combine with formaldehyde to form hexamine. When fumes have been inhaled, treat as for other irritant gases (see p. 592).

Chemical tests (see p. 715).

Methyl chloride.

This is a colourless gas, which is non-irritating, has an aromatic odour, and is used in chemical processes and also in domestic and commercial refrigerators.

The onset of symptoms is insidious and weakness and staggering are frequent. Nausea, vomiting, confusion, abdominal pain, coma, and convulsions complete the picture of serious poisoning. The post-mortem signs include congestion and œdema of the lungs, petechial hæmorrhages in the pericardium and pleuræ, hyperæmia of the gastro-intestinal vessels, and cloudy swelling of the kidneys, together with congestion and œdema of the brain.

Treatment.

Artificial respiration may be required, together with the administration of oxygen and 5 to 7 per cent. carbon dioxide. Since methyl chloride, when taken into the body, is decomposed into methyl

alcohol and hydrochloric acid, the general treatment is similar to that of poisoning by methyl alcohol (see p. 637).

Chemical tests (see pp. 702 and 715).

Dimethyl sulphate.

This is a dimethylic ether of sulphuric acid, emitting a highly toxic gas which is used in the colour industry. It is a yellow oily fluid which liberates a vapour. The symptoms of acute poisoning result from involvement of the central nervous, or cardio-vascular, system. This gas also affects the kidneys and blood. When the fumes are inhaled, convulsions, paralysis, and coma result. Poisoning may also result from swallowing the fluid. A notable feature of poisoning by this substance is the delay in the onset of the symptoms following exposure and the rapidity of death after the manifestations of toxic symptoms.

Dinitro-orthocresol.

Chronic poisoning may follow inhalation of the fumes and of dust or dry powder. The symptoms are giddiness, faintness, vomiting and weakness, bronchitis, yellow coloration of the skin, sweating, breathlessness, unconsciousness, and death. The post-mortem appearances include a yellow coloration of the skin, buccal mucosa and hair, together with hyperplasia of the bone marrow. Within recent times, two deaths have occurred from making and using this substance, now employed as a fumigant and insecticide. During the period 1942-45, ten non-fatal cases of poisoning have been reported.

Petroleum distillates.

Petrol, benzine, paraffin, and kerosene.

Petroleum contains gaseous and liquid constituents. When distilled fractionally, ether, petrol, paraffin, and kerosene are obtained. The fractions which distil below 150° C. include ether, petrol, naphtha, and benzine, which are poisonous when swallowed or when their vapours are inhaled. Benzine is much less toxic than benzene. When the fumes from the more volatile of these fluids are inhaled, nausea, headache, vertigo, and sometimes vomiting occur, and unconsciousness may supervene. On regaining consciousness, or even prior to losing consciousness, there may be mental confusion or excitement with or without violence. Convulsions sometimes occur. In fatal cases, cyanosis, unconsciousness, and profound coma precede death. When these fluids are swallowed, the signs are those of gastro-intestinal irritation, and in fatal cases a state of coma will merge into death.

Nunn and Martin [16] have recorded seven cases of petrol poisoning in children. In two of the fatal cases, the outstanding clinical picture was increased respiration and rapid pulse-rate. Cyanosis, stupor or

coma, and moist râles throughout the lungs were present. Death occurred within an hour and a half of swallowing the fluid, the quantity of which was not ascertained. Post-mortem examination showed œdema of the lungs, the alveoli of which were filled with fibrin and serous exudate. Fatty degenerative changes were present in the liver, and the stomach contents emitted a strong odour of petrol. They have also reported sixty-five cases of poisoning by kerosene in children. 9·2 per cent. of the cases were fatal, the children lived from two to eighteen hours after swallowing, or aspirating the substance, and in all there were definite clinical evidences of pathological changes in the lungs, namely, moist râles, rapid and shallow respiration and cyanosis. Convulsions occurred in two fatal and in two non-fatal cases. The treatment administered, in the various cases, consisted of removing the kerosene by emesis or by gastric lavage, together with the administration of laxatives.

Treatment.

When inhaled.

Artificial respiration or oxygen and carbon dioxide inhalations are recommended, also atropine sulphate and caffeine sodium benzoate.

When swallowed.

Free gastric lavage. Patient should be kept warm. Colonic irrigation may be of value.

Post-mortem appearances.

Evidence of acute gastro-enteritis following ingestion.

Benzene or benzol.

Benzene is a single chemical substance obtained by distillation of coal tar and purified by redistillation. Benzene produces toxic effects either when drunk or by inhalation of its vapour.

Benzene poisoning may occur in such industries as glove-cleaning, waterproofing, dyeing operations, and in aniline factories. It is used as a solvent for paints, lacquers, varnish, rubber, fats, in colour printing, as a motor fuel, and in the chemical manufacturing industry. It may enter the body by ingestion, inhalation, or by the skin. Industrial benzene poisoning is notifiable and must be reported to the Home Office (see p. 33).

When benzene is taken by the mouth, there is gastro-intestinal irritation, and cerebral symptoms quickly supervene. Coma usually precedes death.

The effects are more common, however, after inhalation of the vapour.

Acute poisoning follows the inhalation of a large quantity of the vapour, and the symptoms are giddiness, nausea, vomiting, muscular prostration, dyspnœa, delirium, convulsions, coma, and death, due to severe hæmorrhage or toxæmia. In fatal cases, purpuric spots

may be present on the mucosa of the nose, mouth, and throat, and sometimes on the uterus.

Chronic poisoning is characterised by general malaise, headaches, giddiness, impairment of vision, nervousness, gastro-intestinal disturbances, anæmia, loss of weight, insomnia, loss of memory, and neuritis. The blood picture shows a leucopenia and poikilocytosis of red cells. Benzene is a bone-marrow poison.

Treatment.

When inhaled.

Oxygen inhalation, intravenous saline and dextrose and symptomatic treatment.

When swallowed.

Gastric lavage.

Nitro- and amino-derivatives of benzene and its homologues.

The nitro- and amino-derivatives are the nitrated or aminated compounds of benzenes, toluenes, xylenes, etc. These derivatives comprise a very large number of substances and the symptoms are more or less similar. The nitro-derivatives chiefly affect the central nervous system and the amino-derivatives exert a toxic action upon the blood. The substances which cause fairly frequent cases of poisoning include : Nitrobenzene, dinitrobenzene, trinitrotoluene or trotyl or T.N.T., dinitrophenol, trinitrophenol or picric acid, dinitrocresol, aniline, phenylenediamine, pyridine, and nitroglycerine.

Toluene.

Industrial poisoning may occur as the result of fumes, since toluene is used in the manufacture of munitions, lacquer, and rubber. Toluene has a narcotic action, and the inhalation of its fumes produces irritation of the mucous membranes, and depresses the central nervous system. The bone marrow is also affected.

Nitrobenzene.

Pure nitrobenzene has been used as a scenting agent, and on account of its odour is known as Oil of Mirbane. It is prepared by the nitration of benzene by nitric acid and is used in the manufacture of aniline dyes, soaps, and explosives. Its vapour is of yellowish colour. When inhaled it is an active poison and affects the hæmoglobin and central nervous system. The hæmoglobin is converted into methæmoglobin, with resultant bluish-grey cyanosis of the skin and mucous membranes, and darkening of the blood in the arterial system. The poison may also be absorbed by the skin, but when introduced in this way is less toxic.

Symptoms of acute poisoning.

The symptoms of poisoning show a wide range of variation, and since the substance is a typical example of a nitro-derivative of benzene, they are given in some detail to avoid repetition when analogous substances are dealt with.

A sense of fullness and throbbing in the head with flushing of the face is experienced. There is often a burning sensation in the throat, a feeling of tightness in the chest, and there may be visual disturbances. In cases of acute poisoning, an ashen-blue cyanosis, progressive in character, develops. Nausea, vomiting, dyspnœa, giddiness, prostration, convulsions, coma, and death, describe a fatal issue. Methæmoglobin is present in the blood which is of dark colour, and a reduction in the number of blood cells, which show degenerative changes, will be found.

Henderson and Haggard in discussing the effects of methæmoglobin in blood, state that its stability prevents it from performing the normal function of hæmoglobin, which is the transportation of oxygen in the body. On this account tissue asphyxia is caused. If methæmoglobin is in large amount, part of the hæmoglobin is irremediably damaged, the red cells are broken up and their contents are diffused in the blood plasma. Profound anæmia may follow even a single intense exposure. Some of the methæmoglobin passes into the urine and produces a characteristic reddish-brown or chocolate colour. Hæmoglobin loss through the kidneys is particularly serious since it causes a depletion of the body's store of iron, necessary for the formation of new hæmoglobin and fresh corpuscles.

Symptoms of chronic poisoning.

The symptoms, although similar to those above, are less aggressive. Blood and bile pigments may be present in the urine.

Treatment.

Inhalations of oxygen with 5 to 7 per cent. carbon dioxide. Blood transfusions. Intravenous administration of 5 to 10 per cent. glucose in normal saline. The stomach may be washed out with water containing magnesium sulphate.

Post-mortem appearances.

These include marked congestion of the meninges and brain, a dark or chocolate colour of the blood, with engorgement of the chambers on the right side of the heart. The scented odour of nitrobenzene may be perceived in the lungs and stomach. The substance can be recovered from the body fluids by distillation.

Dinitrobenzene.

Persons engaged in the manufacture of this substance and of explosives are liable to experience acute or chronic toxic effects. Tonite contains dinitrobenzene, gun cotton, and barium nitrate, also

roburite, dinitrobenzene and ammonium nitrate. Dinitrobenzene is readily absorbed by the skin when handled with unprotected hands. The toxic symptoms are similar to those caused by nitrobenzene.

Headache, drowsiness, loss of appetite, nausea, dyspnœa, palpitation, cyanosis, and pains in the legs, due to peripheral neuritis, are the main symptoms. Jaundice does not occur usually, although the liver may be involved in some cases. There is a conversion of some of the oxyhæmoglobin into methæmoglobin, and, in severe cases, degeneration of the erythrocytes may occur. There may also be a decrease in the number of polymorphonuclear cells and an increase in the lymphocytes.

Trinitrotoluene, trotyl, or T.N.T.

This substance, an extensively used explosive in all forms of ammunition, is produced by the action of mixed concentrated nitric and sulphuric acids upon toluene which is converted into dinitrotoluene, and by further treatment into trinitrotoluene.

Trinitrotoluene is crystallised out in pans and then either broken into small pieces, ground into powder, or shaved into flakes.

Persons employed both in its manufacture and its uses in dry and molten forms frequently suffer from toxic symptoms, since absorption may occur by inhalation of vapour or dust, through the skin by handling, or by ingestion.

The principal channel of absorption is the air-passage by inhalation of fine dust.

A sago-grain, irritating dermatitis with exfoliation is produced by handling the powder or mixtures which contain it, particularly when the skin is perspiring, and is prone to occur in workers at the commencement of their employment. Mixtures of trotyl with hygroscopic salts, such as amatol, a mixture of trotyl and ammonium nitrate, may be absorbed through the skin and cause poisoning. The toxic action of trinitrotoluene is similar to that caused by the other nitro- and amino-derivatives of toluene and benzene. It is capable of causing toxic jaundice.

Among the early symptoms are drowsiness, headache, and nausea. Some measure of cyanosis, which is of a bluish-grey colour, due to anilism, affects the lips. Later, there are likely to be abdominal pain, loss of appetite, gastritis, a feeling of oppression over the chest, and dyspnœa. Staggering gait, palpitation, and rapid pulse-rate may be added to the picture. The advent of toxic jaundice makes the prognosis serious since this symptom indicates liver damage or necrosis. In very acute cases, unconsciousness may take the place of drowsiness, with perhaps convulsions, and there is likely to be marked toxic jaundice. Occasionally during the early stage of recovery from an attack of a subacute nature, amblyopia, with limitation of the field of vision and colour, has been observed. Aplastic anæmia may occur among T.N.T. workers.

The period of employment before the manifestation of toxic symptoms has been found to vary from several days to weeks, the time being related to circumstances of the working surroundings and individual conditions.

It has been suggested that the selective effect of T.N.T. on the different organs should be attributed to acquired differences in tissue susceptibility.

The action of this particular poison is primarily centred on the blood and is hæmolytic. Methæmoglobin is present. Microscopically, the red cells assume forms closely resembling those found in pernicious anæmia. Neutrophil leucopenia and lymphocytosis are common features. The leucoblastic function of the bone marrow is progressively involved. Should the methæmoglobin in the blood become considerable, the liver is unable to remove it and methæmoglobinæmia is established, the blood tending to become chocolate brown in colour, and hence the cyanosis of the patient is deepened in tint (see p. 606).

In fatal cases the liver is frequently the seat of yellow and red necrosis. In a number of instances, this organ, although enlarged in the earlier stages, is found to be very atrophied and to weigh as little as from 20 to 30 ounces when examined post-mortem. In a reported series of cases showing jaundice, about 27 per cent. were due to pure T.N.T., 67 per cent. to amatol, and 6 per cent. to ammonal,[17] a mixture containing aluminium, ammonium nitrate, and trinitrotoluene.

The dermatitis of T.N.T. resembles vesicular eczema, accompanied by swelling and itching of the affected parts. It commonly appears first on the skin of the hands, wrists, and forearms, but it may also be present on parts, usually covered by clothing, to which the dust has gained access.

Treatment.

Oxygen and carbon dioxide to combat cyanosis, intravenous calcium gluconate, and a high calcium diet. Purgation.

Chemical tests (see p. 724).

Dinitrophenol.

This is a substitution product produced by nitration of phenol. Its chief importance toxicologically is that, when taken internally, it increases the metabolic rate and therefore has been employed extensively, especially in America, for slimming. Many cases of poisoning have occurred. It aids the combustion of fat without exerting much effect upon the protein or nitrogenous constituents of the body. Certain persons are intolerant to its use. It is usually taken in the form of sodium dinitrophenol. The symptoms of poisoning include flushing, with a feeling of warmth, sweating, and dyspnœa in acute cases. In the more chronic form of poisoning, debility, impaired vision, due to cataract, and peripheral neuritis are frequent.

In acute poisoning, pulmonary congestion and œdema, mild nephritis symptoms, and tachycardia have been reported. The hepatic and renal cells are frequently injured.

Treatment.

This consists of gastric lavage with a 5 per cent. solution of sodium bicarbonate, or a solution of 1 in 2,000 potassium permanganate. Dextrose and normal saline should be given intravenously. For cataract, ascorbic acid has been recommended. In certain cases, oxygen may be administered to relieve dyspnœa, and an ice pack employed to reduce febrile symptoms. The use of pentnucleotide has been suggested. Purvine[18] reports the post-mortem findings in a suicidal case of a woman of thirty-six. These included congestion of the lungs, spleen, and kidneys. The gastric mucosa and the mucosa of the duodenum and upper part of the ileum showed small hæmorrhagic areas. The tubular epithelium of the kidneys was swollen, the cells of the tubules were degenerated, and there was congestion of the glomeruli. Chemical analysis of the blood showed a concentration of 0·12 gramme of dinitrophenol per 100 millilitres of blood. A few cases of granulopenia have been reported. Tainter and Wood[19] have reported that in one case the onset of rigor mortis was rapid, the body being rigid within ten minutes after death. In their experience, ecchymotic hæmorrhages in the pericardium, endocardium, and pia-arachnoid membrane, may be found.

Trinitrophenol or picric acid.

Picric acid is used in the manufacture of explosives and fireworks and in the dye industry. Industrial poisoning usually occurs by inhalation of fumes or dust. Clinically, it is used in the treatment of burns and certain skin conditions, but poisoning from this cause is unusual.

The symptoms are of a catarrhal nature, accompanied by epigastric pain, giddiness, and stomatitis. When swallowed, the symptoms are those of gastro-intestinal irritation. The skin will show a yellowish colour in varying degree.

Treatment.

This consists of gastric lavage and the administration of albuminous fluids.

Dinitrocresol, orthocresol, and metacresol.

Dinitrocresol is similar in its use and action to dinitrophenol. The proprietary preparation is Dekrysil. Its use has caused death.

Chronic poisoning may be produced by inhalation of fumes of any of these substances.

The symptoms are nausea, vomiting, giddiness, faintness, debility, sweating, dyspnœa, and a yellowish coloration of skin. Bronchitis may develop, accompanied by rise of temperature, and unconsciousness usually precedes death.

Aniline, aniline oil, or phenylamine.

This is a colourless, oily fluid, which, after standing, may develop a brown shade. It has a peculiar odour, and very toxic properties, not only when swallowed but when inhaled or absorbed through unbroken skin. It has a selective toxic action on the heart. Dangerous concentrations of vapour may occur in such trade processes as dye manufacturing, dye-using works, leather-polish works, rubber works, and in the manufacture of certain chemicals. After several hours of exposure to concentrations of 7·0 to 26·0 parts per million slight symptoms are produced. Serious disturbances follow exposure to concentrations exceeding 105·0 to 160·0 parts per million for more than an hour.

Industrial aniline poisoning is notifiable and must be reported to the Home Office (see p. 33).

The symptoms in acute poisoning consist of bluish-ashen coloured cyanosis of the lips, ears, fingers, face, and mucous membranes, headache, giddiness, nausea, vomiting, palpitation, tachycardia, and shortness of breath. The action of the heart becomes enfeebled and the temperature is subnormal. An odour of aniline may be perceived in the breath and from perspiration and urine. Methæmoglobin is formed in the blood, and the urine becomes dark in colour (see p. 606). Chronic poisoning presents such symptoms as anorexia, headache, digestive disturbances, anæmia, and neurasthenia. A papillomatous growth of the bladder which undergoes carcinomatous degeneration has occurred with some frequency in aniline workers.

Treatment.

Free gastric lavage should be employed and cardiac treatment may have to be administered without delay. Use artificial respiration and oxygen inhalations if necessary. In certain cases, benefit may be obtained from the use of intravenous saline or blood transfusions.

Post-mortem appearances.

The post-mortem signs are not characteristic. There will be evidence of either cardiac or respiratory failure. The blood is dark and the peculiar colour of post-mortem lividity will attract attention. The heart, liver, and kidneys will show a varying degree of fatty degenerative change.

Chemical tests (see p. 707).

Phenylenediamine and toluenediamine.

These substances, used as dye intermediates in dyeing hair and furs, are toxic. As a hair dye, a 2 per cent. solution, with a 1·5 per cent. solution of sodium hydroxide, is applied, followed by an oxidising agent such as a 5 per cent. solution of ferric chloride, if a brown shade is desired, or a 3 per cent. solution of hydrogen peroxide when a black colour is required (see p. 114). The use of phenylenediamine

in dyes may cause local dermatitis and serious toxic manifestations. Israels and Susman [20] point out that an attempt to remove the dye with hydrogen peroxide, or sodium thiosulphate, should never be made, since by the production of aniline the symptoms are intensified. The general symptoms of poisoning are giddiness, weakness, insomnia, epileptiform convulsions, coma, and death. Toluene-diamine produces destruction of the red blood cells, methæmoglobin, and toxic jaundice with accompanying degenerative changes (see p. 608). The amino compounds are also absorbed by inhalation, but the toxic effects are less serious than those of the phenyl compounds. The risk of poisoning industrially is present in the manufacture of colours and in the process of dyeing furs.

Pyridine.

This substance is obtained from coal-tar, subsequent to the extraction of phenol, and has a yellow, oily appearance and penetrating, nauseating odour.

When swallowed, it acts as a gastro-intestinal irritant and frequently causes evidences of asphyxia. Cerebral symptoms are common. In fatal cases the cause of death is asphyxia.

If the fumes are inhaled the symptoms chiefly affect the respiratory system. The upper air-passages and lungs become congested, an irritating and persistent cough is a frequent accompaniment, and the eyes become suffused.

Nitroglycerine or glyceryl trinitrate.

This is prepared from glycerine and used in the manufacture of dynamite. It is a pale, oily fluid without characteristic odour. Either concussion or heat will cause it to explode. Its toxic effects are the cause of headache, gastro-intestinal irritation, faintness, insomnia, dryness in throat, dyspnœa, bronchitis, and mental excitement.

In chronic poisoning, tremors and neuralgic pain are common.

It has the capacity of being absorbed through intact skin, and handling may produce ulceration at the finger-tips and below the nails. Absorption may also occur through the mucous membranes.

Cordite.

This substance is a combination of nitrocellulose and nitroglycerine, with the addition of about 5 per cent. of mineral jelly. It is used as an explosive. Poisoning from cordite frequently occurs and is shown by such symptoms as tachycardia, palpitation, irregularity of the action of the heart, giddiness, dyspnœa, especially on exertion, and general debility.

The cause of death in fatal cases is asphyxia. To evade service in the Forces, malingerers have been known to suffer from toxic manifestation due to self-administration.

Amyl nitrite, sodium nitrite, and ethyl nitrite.

These are common compounds used therapeutically. Amyl nitrite is a yellowish, volatile fluid used clinically as a vasodilator. It may be swallowed, or the fumes may be inhaled. Cases of poisoning are rare. Toxic doses produce vomiting, cyanosis, dyspnœa, Cheyne-Stokes breathing, coma, and perhaps death due to cardio-vascular collapse.

Sodium nitrite is obtained by reduction of sodium nitrate on fusing it with lead. It has a vasodilator action similar to that of amyl nitrite, but is slower and more prolonged. Deaths have occurred after swallowing it in error for salt.

The symptoms are muscular weakness, injection of the conjunctivæ, coldness of the extremities, rapid respiration, darkening of the buccal mucosa, and albuminuria.

Excessive dosage of these drugs causes hæmoglobin to be converted into methæmoglobin.

Treatment.

This consists of gastric lavage, injections of adrenalin or ephedrine, to counteract the vasodilating effect, artificial respiration, and oxygen inhalations with, or without, 7 per cent. carbon dioxide. To counteract the formation of methæmoglobin, small amounts of methylene blue should be administered. When such doses are small they bring about the conversion of methæmoglobin to hæmoglobin; thus exercising the opposite effect of methylene blue in high concentrations.

Post-mortem appearances.

The post-mortem findings are usually œdema of the lungs and brain, lividity of slatey-grey colour, and a brownish colour of the blood due to methæmoglobin (see p. 611). The kidneys may show degenerative changes.

Amyl acetate.

This substance is used as a solvent in many industries, and the fumes have an odour resembling that of pears. On excessive inhalation, such symptoms as irritation of the throat, giddiness, nausea, palpitation, and fainting may occur.

Naphthalene.

Naphthalene is a solid substance, obtained from the middle fraction of distillation of coal-tar, which has chemical properties similar to benzene and is very volatile. It is used chiefly in the manufacture of naphthols, naphthylamines, and sulphonic acids, employed in the dyeing industry. It is utilised because of its antiseptic and preservative properties in the preservation of woollen goods, and as a deodorant in lavatories.

Symptoms.

The symptoms of poisoning are abdominal pain, frequency of micturition, cyanosis, muscular twitchings, drowsiness, and brown coloration of the urine. An ataxic gait is not uncommon in the earlier stages.

Treatment.

Gastric lavage, a purgative dose of magnesium sulphate, and demulcent drinks, which are devoid of oils and fat, form the principal lines of treatment.

Chlorinated naphthalene.

When naphthalene is chlorinated a waxy substance is produced, which is used in industry as an insulating coat on electrical wires and for limiting the action in plating metals. "Seekay" wax is used in waterproofing, flameproofing, for insect pests and fungus, also

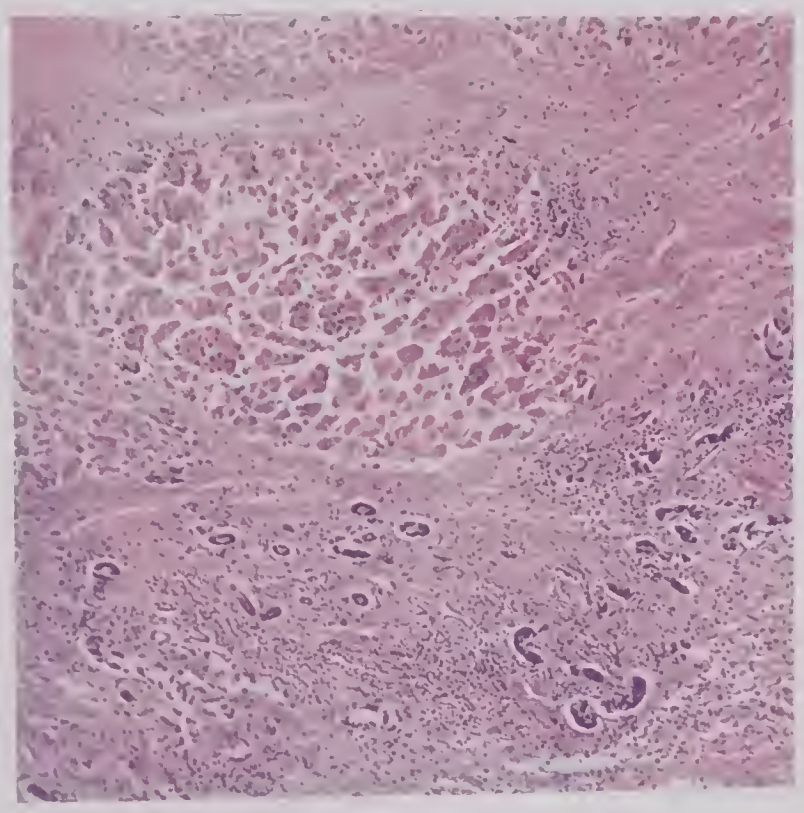


FIG. 227
Acute necrosis of liver in chlorinated naphthalene poisoning. ×60.

for bitumen blending, sealing and the impregnation of condensers, coils, etc. Overheating of the wax causes increased quantities of the fumes to be evolved (see p. 30).

Symptoms.

Chlorinated naphthalene waxes frequently give rise to inflammation and irritation of the skin, especially acne, and breathing of

their vapours or air containing fine particles of the wax may cause serious illness. Persons with liver damage are more susceptible to poisoning. Toxic jaundice is not an infrequent manifestation produced either by an acute or subacute necrosis of the liver, or by acute yellow atrophy. Continued exposure gives rise to jaundice, and slight œdema of the eyelids and ankles. When the liver is seriously involved, the area of dullness is diminished, and the urine becomes dark brown in colour and contains bile. Purpuric rashes, involving the lower abdomen and legs, may be seen.

Post-mortem appearances.

Post-mortem findings include bile staining of the kidneys and minute areas of necrosis in the pancreatic fat. The chief lesion is found in the liver, usually greatly reduced in size, which may vary in weight from 1,200 grammes to 650 grammes or less. The capsule is usually wrinkled, and histological examination shows the acute stages of yellow atrophy.

Tetrachlorethane.

This substance, called "dope" by workers, is widely employed in industry. It is used in the manufacture of cinematograph film and as a varnish solvent, in addition to many other uses. Tetrachlorethane is a colourless, heavy, volatile liquid which yields a vapour heavier than air, consequently it is apt to fall to lower levels of working places and does not readily mix with the atmosphere.

It is exceedingly toxic to workers, probably being the most toxic of all the chlorinated derivatives, and a large number of cases of poisoning have occurred from its use. Poisoning may be acute or chronic. Several cases of suicide have been caused by drinking "silk cleaning fluid."

Symptoms.

The symptoms, which are many and varied in character, are most frequently produced by inhalation of the fumes. They consist of malaise, anorexia, drowsiness, headache, mental confusion, nausea, and sometimes vomiting. When the toxic effects are severe, the symptoms shown are stupor, delirium, convulsive twitchings, nephritis, and coma, preceded or accompanied by a hæmo-hepatogenous jaundice. Cholangitis affects the finer bile-ducts and produces bile-flow obstruction, with fatty degeneration and necrosis of the liver cells in some instances. Prognosis is always grave in severe cases. Dermatitis and visual disturbances may be present.

Treatment.

Treatment consists in the administration of oxygen, intravenous saline, sodium carbonate or calcium gluconate, blood transfusion, and catharsis.

Post-mortem appearances.

On post-mortem examination, the liver is usually the chief organ affected. When the poison has been swallowed, the gastric mucosa will show congestion, with or without small erosions, and there will also be congestion of the lungs, kidneys, and intestinal tract. When death has been rapid the liver may be only congested, but when death has been delayed, necrosis or fatty degenerative changes will probably be present.

Trichlorethylene or trilene.

This is a volatile substance with a characteristic odour used in commerce as a solvent for tar, rubber, and in dry-cleaning. It is also employed for the extraction of oils and fats, degreasing metals, enamelling, cleaning photographic plates, and textile manufacture. Cases of trigeminal neuralgia have been treated by the inhalation of 10 to 20 minims. Trilene is used as an anæsthetic.

The fumes, if containing disintegration products, when inhaled may affect the central nervous system and give rise to such symptoms as headache, vertigo, fainting, drowsiness, cardialgia, parasthesia of the limbs, coma, and death.

Treatment.

In acute poisoning, inhalation of oxygen with 6 to 7 per cent. carbon dioxide. When pulmonary œdema threatens, oxygen should be given.

Post-mortem appearances.

Post-mortem findings may consist of encephalitis, subpleural petechial hæmorrhages, and desquamation of the renal tubular epithelium with fatty degenerative changes.

Diethylene dioxide.

This is a volatile liquid sold under the name of Dioxan. It is used as an intermediate in the synthesis of certain other compounds, as a solvent for waxes, resins, fats, oils, greases, celluloid, cellulose acetate or nitrocellulose, as a laequer diluent, and sometimes as a fumigant. The fumes are irritating and have narcotic properties. The irritation affects the eyes and respiratory tract. Poisoning occurs when there is exposure to air in close proximity to the liquid contained in tanks and vats since high concentrations accumulate there.

Barber [21] reports five cases in workers who died from the effects of hæmorrhagic nephritis and necrosis of the liver following Dioxan poisoning, after an acute illness lasting five to eight days (see p. 29).

ANÆSTHETICS, GENERAL AND LOCAL

Chloroform.

Death by chloroform may result from drinking the fluid or from inhalation of the vapour. Chloroform is, however, rarely taken in liquid form for suicidal purposes, but cases have been recorded where death followed at variable periods of time, after doses of 1, $1\frac{1}{2}$, 2, and 3 ounces, and recovery ensued after the use of 4 and 5 ounces.

An interesting suicidal case was that of a man who fatally chloroformed himself by means of a service respirator, the inlet of which he closed with cellophane. Both the inlet to the face-piece and the outlet had been stuffed with cotton-wool impregnated with chloroform. The amount of hydrocarbons expressed as chloroform in the liver and kidneys was 125 milligrammes per 1,000 grammes.

A few cases of homicidal administration have been recorded.

When inhaled, chloroform has a paralysing effect upon respiration, and causes a fall of blood-pressure. Some hold the view that respiratory failure is the cause of circulatory failure, but acknowledge that in some cases circulation ceases before respiration. It is generally agreed that the drug exercises a direct toxic action on the myocardium. Experiment has shown that safety or danger lies in the concentration of chloroform vapour administered. A small quantity may kill when in concentrated form, while a comparatively large amount may be safe in a concentration below 2 per cent.

Gaddum [22] writing on the subject of ketosis following chloroform anæsthesia states that the toxic action affects the liver, heart, and kidneys, which undergo fatty changes, and the patient develops ketosis and vomits. Ketosis is due to the incomplete oxidation of fats which leads to the accumulation of acid ketone bodies, such as aceto-acetic acid and hydroxybutyric acid. The occurrence of this condition following anæsthesia is possibly a consequence of the fact that anæsthetics have a specific inhibitory action on carbohydrate metabolism, which leads to a compensating increase of fat metabolism.

Much has been written regarding the condition of status lymphaticus as a cause of death under general anæsthesia. Many cases have been recorded, but the consensus of present-day opinion would appear to be that, although this condition has frequently been associated with sudden deaths during the administration of anæsthetics, the question of cause and effect has not yet been clearly defined. The condition seems to be accompanied by a lowered resistance toward shock.

As already mentioned, drinking chloroform may cause death.

Hayward records a fatal case of a woman who drank 2 ounces of chloroform. Her face was blanched, the lips and fingers were livid, the body and extremities were cold, the pulse was imperceptible, the heart sounds very feeble, the breathing shallow but regular, the corneæ insensitive, and the pupils were equal, semi-dilated,

and feebly responsive to light. She had vomited. Under treatment her condition at first improved, but when she recovered consciousness, a sudden and fatal collapse ensued.

Post-mortem examination showed that the right side of the heart contained dark, uncoagulated blood. The stomach contained about 1 ounce of dark, chocolate-coloured fluid which emitted an odour of chloroform, the walls were slightly congested, and a few scattered extravasations of blood were present. The small intestines were intensely congested throughout, the mucous membrane being swollen, velvety, and of dark cherry-red colour with numerous extravasations, and contained a quantity of dark red fluid smelling strongly of chloroform.

When chloroform has been swallowed, gastric lavage and cardiac treatment must be employed without delay. Artificial respiration and oxygen inhalations should be used as necessary.

Chemical analysis (see p. 713).

Ether.

The use of ether as an intoxicant is not uncommon, and, like alcohol, a tolerance to quantity may be established by repeated use. It is much less toxic and much more rapidly stimulant in action than chloroform.

Those who begin the habit take at first doses of from 2 to 4 drachms, but when the habit is confirmed, the amount may increase to ounces.

Death while under the influence of ether, employed as a general anæsthetic, is less frequent than in the case of chloroform and is brought about by paralysis of the respiratory centre. Only occasionally does death result from syncope as a direct result of ether administration.

Pulmonary complications, including bronchitis, bronchopneumonia, and, rarely, pulmonary œdema, sometimes follow ether administrations.

Mixtures of ether and chloroform are employed for anæsthetic purposes.

Death has resulted from nitrous ether. A druggist's maid-servant was found dead in bed, the air of the room being charged with the vapour of this substance, liberated by the breaking of a 3-gallon jar of sp. etheris nitrosi. Her appearance when found was that of a person in deep natural sleep. Post-mortem examination showed that the gastric mucosa was markedly congested, and that the lungs were engorged with blood.

Ethyl chloride.

Like chloroform, it enters into chemical union with the blood. It acts as a stimulant on the central vasomotor system. In 10 per cent. concentration and upwards it produces paralysis of the myocardium comparable with that produced by chloroform in one-twentieth of this concentration.

It is used as a general anæsthetic for minor operations of short duration, or for induction, prior to anæsthesia being maintained by ether when breathing has become regular.

Nitrous oxide.

This is the commonest anæsthetic for dental work, and is perhaps the safest anæsthetic for any short-period anæsthesia. The mortality arising from its use has been reckoned to be about one per million. The use of continuous nitrous oxide and oxygen, with or without ether, is also a prevalent form of anæsthesia for certain operations.

Divinyl ether.

The vapour, which is highly inflammable, is capable of producing a rapid anæsthesia with good muscular relaxation. Post-anæsthetic complications are rare following its use, but it should be noted that prolonged anæsthesia may bring about liver damage, and thus patients with liver insufficiency should be regarded as unsuitable subjects.

Cyclopropane.

This anæsthetic is supplied as a liquid in cylinders. It becomes a vapour at atmospheric temperature and pressure, and is inflammable. Cyclopropane is used in a closed circuit apparatus and is a powerful anæsthetic which in low concentration does not irritate the respiratory passage, but has a tendency to depress respiration. It is much less toxic than chloroform, and anæsthetists hold the opinion that respiratory failure precedes failure of circulation.

Basal anæsthetics.

Tribromethyl alcohol or avertin.

This is a basal narcotic, used for the purpose of anæsthesia, and is administered by the rectum. One-half to two-thirds of a grain per pound of body-weight is the amount given. Toxic dosage causes death by respiratory paralysis. A number of deaths have been recorded. (For treatment, see p. 639).

See barbiturates, p. 641.

Curare.

The combination of curare injection with light general narcosis for surgical operative purposes has been in use for some time, and the view that such procedure gives the advantages of a high spinal block with practically none of the disadvantages is held by many. A small dose of curare is recommended since this can be added to subsequently if there is indication. Prostigmine is the physiological antidote for overdosage. The following preparations of curare, among others, are available, intocostrin, curarine chloride, and

tubarine. Myanesin has been recommended as a synthetic curare substitute. The use of curare is not without danger and its use should be restricted to those who are fully experienced in the administration of anæsthetics (see p. 669).

Spinal anæsthetics.

Spinal anæsthetics comprise synthetic drugs having a similar composition to cocaine, for example, stovaine and novocaine, also nupercaine, a drug allied to quinoline. Of these, nupercaine is regarded as probably the least toxic. Death has resulted from the use of these drugs as spinal anæsthetics. Death from spinal anæsthesia usually occurs as the result of a fall in blood-pressure following vasomotor paralysis, or from diminished respiration due to abdominal or intercostal paralysis rather than medullary ischæmia.

Local anæsthetics.

These are administered by means of nerve blocking or by local infiltration. Cocaine on account of its toxicity is never injected and its use is confined to surgery of the eye, nose, mouth, and throat where it may be applied to the surfaces in a diluted solution. Local anæsthetics are cocaine derivatives as procaine or novocaine and amethocaine, or quinine derivatives such as quinine and urea hydrochloride or quinine hydrochloride and a urethane. Frequently adrenalin is combined with a local anæsthetic.

Death during anæsthesia.

The expectation of death under an anæsthetic has been estimated at about 1 in 1,000.

It is the duty of the authorities who are charged with the investigation of the causes of sudden deaths to make inquiry regarding every death which occurs during the administration of an anæsthetic, or following the administration of an anæsthetic, or under circumstances in which the anæsthetic may be regarded as a contributory cause of death. For the purpose of inquiry the term anæsthetic includes all general, spinal, and local anæsthetics. It is most desirable that the public should be assured from such inquiries that due care has been exercised prior to, and during, the administration of the anæsthetic, and that appropriate measures have been taken to resuscitate the patient. In Scotland, deaths under anæsthesia must be reported to the Procurator-Fiscal, and those in England to the Coroner. An inquiry in accordance with the rights and duties of his office is held by each official. With the announcement of the death to the Fiscal in Scotland a report by the patient's doctor is furnished on a form, supplied from the office of the Fiscal, in which are specified the circumstances of the death, including the following details:—(1) Full name of patient; (2) Age: (3) Address; (4) When admitted to Hospital or Nursing

Home; (5) Nature of disease or ailment; (6) Date of operation and hour of death; (7) When, and by whom, patient was informed that anæsthetic was necessary; (8) What precautions were used in preparing patient for its administration; (9) When, and by whom, patient was examined as to condition of the heart, lungs, and urine, and what was their condition; (10) Anæsthetist, anæsthetic, method of administration: time during which patient was under influence of anæsthetic: (11) What physicians were present at operation; (12) Who was the operator. This report is sent by the Procurator-Fiscal to a medico-legal examiner, who, from the facts found on inquiry into the circumstances attending the death, and from the post-mortem examination, gives his opinion on the following points in his report:—(1) The cause of death: (2) Whether the patient was medically examined before it was decided that an anæsthetic should be administered: (3) Whether all due precautions were observed during the actual administration of the anæsthetic: (4) Whether there were any symptoms about the patient which might and should have been discovered by examination indicating that the administration of an anæsthetic would be attended by special risk to life; and (5) whether prompt and adequate resuscitative measures were employed.

It is difficult, if not, indeed, impossible, to lay down a rule regarding the question of apportionment of responsibility between the surgeon who operates and the anæsthetist who administers the anæsthetic, because there are several conceivable sets of relationships between these persons. The general position would appear to be that the decision to operate rests with the surgeon, that the control of the operation is in his hands, but that with a skilled anæsthetist, the assessment of the fitness, or otherwise, of the patient to take the anæsthetic must rest with the anæsthetist. The circumstances of each individual case must determine this issue.

Responsibilities of an anæsthetist.

An anæsthetist is only bound to show a reasonable degree of skill and care (see p. 11). He is not responsible for an error in judgment. The skilled anæsthetist is an independent expert. The duties of an anæsthetist include the satisfactory working of the anæsthetic assembly as well as the administration of the anæsthetic, and thus he is responsible to ensure that the cylinders are in their correct positions and that the gas passing through each flowmeter is actually the one which the flowmeter bears. With the use of so many and varied anæsthetics used singly and in combination, it is inevitable that the apparatus for their administration has become complicated in design. Special precautions are therefore necessary to avoid the risk of accidents, including explosion arising from defective circuits or static sparks. Burning of a patient by contact with liquid ether or chloroform is yet another hazard to be avoided. The responsibility of the anæsthetist extends to the question of a patient's safety prior to removal from the operating

theatre at the conclusion of an operation, and such points as the prevention of partial or complete obstruction to breathing due to vomiting, falling back of the tongue, dropping of the jaw, and respiratory depression, shock, and collapse are matters for close attention.

Causes of death under anæsthesia.

Most anæsthetic deaths fall into one of three groups :—

1. Deaths primarily due to the condition of the patient.
2. Deaths incidental to the anæsthetic, such as vomiting.
3. Deaths primarily due to the anæsthetic.

The principal accidents which occur under general anæsthesia comprise asphyxia caused by vomiting, failure of respiration, and failure of circulation. Of the many cases which we have investigated, a large percentage of the patients were in a critical condition prior to anæsthesia.

Before it can be established that the anæsthetic was the principal factor in the cause of death, it must be established that the general health of the patient was good before the operation, that the operative shock could not be regarded as the major factor in the death, and that post-mortem examination did not disclose any condition which might have proved a contributory factor. In most cases, the anæsthetic may be regarded as a contributory factor.

Primary cardiac failure is most frequently due to the advent of ventricular fibrillation, while primary respiratory failure may be brought about by depression of the respiratory centre, deficient oxygen intake, or obstruction of the airway as a result of a variety of causes.

Cardiac failure may result from overaction of the sympathetic nerves, or vagus nerve, or from primary depression of the cardiac musculature.

Treatment.

In primary respiratory failure, having ensured a clear airway, artificial respiration with oxygen administration, combined with carbon dioxide, should be promptly initiated and maintained for a prolonged period. Tongue traction should be undertaken. Hypodermic medication, to stimulate the respiratory centre, may usefully be adopted.

When cardiac failure threatens, artificial respiration and direct cardiac massage are the two most important measures to be employed. To be successful, cardiac massage must be undertaken within five minutes of cardiac arrest. When the circulation has ceased for any length of time, anoxæmia of the brain cells is thought to bring about irreversible changes in the central nervous system. It has been strongly recommended that for cardiac arrest the ventricles should be pricked within three-quarters of a minute of ascertaining that the heart has ceased to beat, and that in the

event of this simple procedure being ineffective, the heart should be massaged forthwith. Others recommend auricular puncture since it is considered that the auricle is more sensitive than the ventricle to mechanical stimulation.[23] Artificial respiration should be performed from the earliest possible moment and maintained for a considerable period.

Post-mortem appearances.

The findings are similar in both forms of death. In cases of primary respiratory failure, lividity of the exterior of the body and visceral congestion are usually more marked than in cases of primary cardiac arrest.

References

1. "Noxious Gases," Henderson and Haggard, Amer. Chem. Soc., Monograph Series, New York, 125, 1927.
2. Brit. Med. Jour., Vol. I, 826, 1920, Wignall.
3. "Noxious Gases," Henderson and Haggard, Amer. Chem. Soc., Monograph Series, New York, 105, 1927.
4. Ibid., 109.
5. Jour. Amer. Med. Ass., Vol. CVI, 669, 1936, Shillito, Drinker, and Shaughnessey.
6. Ibid., Vol. CIII, 645, 1934, Martland.
7. Ibid., Vol. C, 92, 1933, Gettler and Mattice.
8. Ibid., Vol. C, 1481, 1933, Hanson and Hastings.
9. Ibid., Vol. C, 200, 1933, Haggard and Greenberg.
10. Ibid., Vol. XCIX, 988, 1932, McGuire.
11. Ibid., Vol. CXXI, 826, 1943, Lidbeck, Hill, and Beeman.
12. Lancet, Vol. II, 95, 1941, Caplin.
13. "Industrial Accidents," Vol. IV, 14, 1934, H.M. Stationery Office.
14. Amer. Jour. Med. Sc., Vol. CXCV, No. 2, 182, 1938, Gettler and Baine.
15. Jour. Amer. Med. Ass., Vol. CI, 269, 1933, Geigor.
16. Ibid., Vol. CIII, 472, 1934, Nunn and Martin.
17. Brit. Med. Jour., Vol. II, 842, 1916.
18. Jour. Amer. Med. Ass., Vol. CVII, 2046, 1936, Purvine.
19. Ibid., Vol. CII, 1147, 1934, Tainter and Wood.
20. Lancet, Vol. I, 509, 1934, Israels and Susman.
21. Guy's Hosp. Report, LXXXIV, 267, 1934, Barber.
22. "Pharmacology," Gaddam, 104, 1940, Oxford Univ. Press.
23. Lancet, Vol. I, 5, 1947, Hamilton Bailey.

CHAPTER XXI

THE ALCOHOLS

Ethyl alcohol.

THERE are several forms of alcohol of importance to the medico-legist. The popular term alcohol refers to ethyl alcohol. It is present in varying strengths in the numerous alcoholic beverages in common use, and the approximate percentage of alcohol in these beverages[1] is in Whisky (30 per cent. U.P.), 40 per cent. of volume; in Brandy, 45·5 to 48·5 per cent.; in Rum, 50·0 to 69·5 per cent.; in Gin, 40 per cent.; in Beers, 4·6 to 6 per cent.; in Sherry and in Port, 16 to 20 per cent.; in Liqueurs, 34·0 to 59·0 per cent.; and in Wines, 10 to 15 per cent.

The abuse of alcohol, leading to acute alcoholic poisoning, is common, but it is unusual to meet cases of acute fatal poisoning.

Physiological considerations.

While alcohol is usually absorbed by way of the gastro-intestinal tract, instances have been recorded of intoxication following inhalation of alcoholic fumes, or as the result of absorption from large areas of burning of the skin to which alcohol has been applied. Little or no absorption takes place from the mouth or œsophagus.

The most rapid absorption is from the stomach and small intestine. In fasting subjects, it has been estimated that about 60 per cent. of the alcohol ingested is absorbed in one hour, over 90 per cent. in one and a half hours, and 95 per cent. in two hours, complete absorption taking place within two and a half hours. This rate of absorption is modified by many factors, including the presence of food in the stomach, the condition of the stomach wall, and the exhibition of certain drugs. In an individual habituated to alcohol, absorption may be more rapid than in the normal person. The drug is rapidly diffused after absorption and passes to all the organs and tissues except bone and fatty tissue. As the concentration in the blood increases, the concentration in the tissues rises proportionately. In the brain, spinal cord, and cerebrospinal fluid, the rise in concentration is slower and more persistent than in the blood.

The absorbed alcohol is oxidised to carbon dioxide and water slowly within the body. It has been estimated that the maximum amount which can be metabolised in one hour is about 15 millilitres (12 grammes), equivalent to half a glass of whisky. The ability to deal with alcohol depends upon the power of oxidation of the alcohol by the tissues. It would seem that the average rate of metabolism is probably 6 to 10 millilitres of alcohol per hour.[2] Renal and hepatic insufficiency may influence adversely the meta-

bolism of the drug. The rate of destruction of the alcohol is constant, and is independent of the concentration in the blood and tissues.

Alcohol is excreted in the urine, sweat, milk, saliva, and expired air. Its presence in these products can be demonstrated, and chemical methods have been devised for estimating the concentration (see p. 702). From the medico-legal point of view, the concentration in the blood and urine is of especial importance, as giving information from which the amount of alcohol consumed can be estimated (see p. 634). The amount excreted in the urine is not more than 5 per cent. of the total ingested.

Symptomatology.

In the earliest stages of alcoholic intoxication symptoms may be few or absent. There is a wide variation in the reaction of different individuals to alcohol and in the time interval before the onset of symptoms. The effects manifest themselves in the mental and physical processes, these changes being accompanied by some degree of toxæmia. While it is common to describe several definite stages of intoxication, there is really no sharp demarcation of these stages, and the rate of transition from one to another is inconstant.

At the outset there is some disturbance of behaviour, resulting from diminished control of the higher centres. This shows itself in actions suggesting a feeling of well-being and confidence. As intoxication progresses, this condition may change to one of excitement, aggressiveness, rudeness, moroseness, or obscenity, and the individual may fail to appreciate his position or company. In some cases a surprising degree of mental clarity may exist in the presence of well-marked intoxication as evidenced by motor disturbances and signs of toxæmia. The physical disturbances are seen in the inability to perform simple actions dependent upon the fine co-ordination of motor and sensory functions and a degree of clumsiness develops, at first slight, but becoming increasingly more obvious as intoxication develops. The gait becomes unsteady, there is inability to perform simple balancing tests correctly, and lack of co-ordination in the upper limbs may occur. Muscular fatigue becomes more generally evident and the individual's reaction time to stimulation is lengthened. The speech becomes slurred in character as intoxication progresses until, in the more advanced stages, incoherence is present.

The pupillary reactions are of great importance. When alcoholic intoxication is well established, the pupils are frequently found dilated and sluggish in their reaction to light and accommodation. In many instances, however, and especially in the comatose stage of intoxication, the pupils are contracted and small. In comatose cases it has been noted that stimulation of the patient, by slapping or shaking, will cause a dilatation of the pupil, the individual remaining comatose, with a slow return to the contracted state, when the stimulation is withdrawn (Macewen's test). This reaction is not seen in head injuries and other comatose states and is only absent in alcoholic cases when, from previous operation or disease, the pupil has become altered in shape or fixed by adhesion.

Tachycardia is usually present, but sometimes even in marked intoxication there is only slight increase in pulse rate. The blood pressure is stated to be raised in intoxication. Flushing of the face and conjunctival congestion may occur. Dryness of the mouth and tongue are frequently noted and the latter may be furred. Sometimes there is excessive salivation.

The memory is affected by diminished mental activity, and events, together with their sequence within the preceding hours, cannot be recalled with accuracy. The power to estimate time intervals is also defective, and errors of one hour or more are not uncommon.

The breath in most cases will smell of alcohol, and it should be noted whether the odour is that of fresh or stale alcohol. Generally speaking, it is not possible to state with certainty the nature of the alcohol taken as judged by the odour of the breath. A deodorant is sometimes used, for example, peppermint, but is not usually completely effective.

It is not uncommon to meet cases where the individual passes into coma without any of the preceding stages of intoxication being noticed. This can occur when large quantities are consumed within a short period, especially when the stomach is devoid of food.

In the comatose stage the chief signs are : deep unconsciousness ; pallor of face, with occasionally some degree of cyanosis of lips ; subnormal temperature ; regular deep, but not stertorous breathing, and contracted pupils which become dilated with stimulation of the patient.

In the paralytic stage of acute alcoholic poisoning, there is commencing medullary paralysis, shown by such symptoms as slow, stertorous respiration, cold, clammy, cyanotic skin, dilated pupils, abolished reflexes, and almost imperceptible pulse. Death occurs from paralysis of the cardiac, or respiratory centres, or from the effects of pulmonary œdema (see p. 635).

In the recovery phase, after an alcoholic bout, depression, remorse, and gastro-intestinal irritation are commonly present. Some persons recover sobriety more quickly than others following the consumption of a quantity of alcohol. It is unwise for an examiner to express an opinion as to the possible state of the examinee, in relation to sobriety or insobriety, at any time other than that of the examination.

Diagnosis of insobriety.

In the final diagnosis of alcoholic intoxication, on clinical grounds alone, it must be appreciated that there is no single symptom or sign peculiar to alcohol. The diagnosis depends upon a combination of a number of symptoms and signs which can have no other interpretation than that of alcoholic intoxication.

There is no statutory definition of drunkenness in the laws of Great Britain. This is due no doubt to the difficulty of defining the phenomena of alcoholism as exhibited by different individuals, or by the same individual at different times. It is well known that the

same individual can react differently to the same amount of alcohol under different circumstances, and that the same quantity of alcohol will produce different effects on different people under the same circumstances. Moreover, certain factors, such as the presence of food in the stomach, physical or mental fatigue, and the administration of certain drugs, can materially influence the absorption and action of the drug.

The usual effects of alcohol in their grosser form can commonly be diagnosed by the ordinary individual, but phenomena arising from other causes may be confused therewith. For the determination as to whether an individual is "drunk" or "under the influence of alcohol," the reports of a Committee of the British Medical Association contain important and practical information.[3] Among the many recommendations are :—

That the word "drunk" should always be taken to mean that the person concerned was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation in which he was engaged at the material time.

That it is desirable that a medical practitioner should base his opinion on the following considerations :—

Whether the person concerned has recently consumed alcohol.

Whether the person concerned is so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time. (Under the Road Traffic Act, 1930, the term used is "under the influence of alcohol to such an extent as to be incapable of having proper control of the vehicle.")

Whether his state is due, wholly or partially, to a pathological condition, which causes symptoms similar to those of alcoholic intoxication, irrespective of the amount of alcohol consumed.

That in the absence of any pathological conditions a person is definitely under the influence of alcohol if there is a smell of alcoholic liquor in the breath, and/or in the vomited matter (if any), provided there is a combination of all or most of the following groups of signs or symptoms :—

A dry, furred tongue, or, conversely, excessive salivation.

Irregularities in behaviour, such as insolence, abusive language, loquacity, excitement or sullenness, and disorder of dress.

Suffusion of the conjunctivæ and reaction of the pupils. The pupils may vary from a state of extreme dilatation to extreme contraction, and may be equal or unequal. (In the opinion of many police surgeons, when alcohol in toxic quantity has been consumed the pupil reflex to ordinary light is absent, whereas the pupil will contract in a bright light and remain contracted for an abnormally long time, indicating delayed action of the pupil.)

Loss or confusion of memory, particularly as regards recent events, and appreciation of time.
Hesitancy and thickness in speech, and impaired articulation.
Tremors and errors of co-ordination and orientation.

That there is no single test by itself which would justify a medical practitioner in deciding that the amount of alcohol consumed had caused a person to lose control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time. A correct conclusion can only be arrived at by the results of the consideration of a combination of several tests or observations, such as :—

General demeanour.
State of clothing.
Appearance of conjunctivæ.
State of the tongue.
Smell of the breath.
Character of speech.
Manner of walking, turning sharply, sitting down and rising, picking up a pencil or coin from the floor.
Memory of incidents within the previous few hours, and estimation of their time intervals.
Reaction of pupils.
Character of breathing, especially in regard to hiccough.

That the following are tests upon which, taken by themselves, little stress should be laid in deciding whether or not a person is under the influence of alcohol :—

Presence of tachycardia.
Repetition of set words and phrases.
Character of handwriting.
Walking along a straight line.
Failure of convergence of the eyes.

That in ordinary circumstances any person accused of "drunkenness" should be able to rely upon being seen by a doctor, if he so desires, within half an hour of the time at which he is charged.

The tests usually employed may be divided into two main groups :—

Data tending to establish the fact that alcohol has been taken.

Smell of alcohol in breath.
Suffusion of conjunctivæ.
Flushed face.
Tachycardia.
Admission of having taken alcohol.
Certain abnormal conditions of tongue and lips.
Certain abnormal conditions of pupils.
Presence of tremors.

Tests designed to determine whether or not the amount taken has disturbed the normal behaviour of accused.

Memory tests. Accused is asked simple questions, such as date and time of day; where he lives, what he was doing prior to arrest, and where he now is.

Visual tests.

Speech test. Accused is induced to talk, and his manner of talking is noted.

Co-ordination tests. These include approaching and picking up a small object from the floor or table; selecting a chair and sitting upon it, and getting up again; ability to stand steady with heels together and eyes shut; walking away and returning; walking along a chalk line.

Writing tests.

From these recommendations it will be seen that a complete medical examination of the individual must be made to exclude the possibility of some other clinical condition being the sole or partial cause of the symptoms noted. Cases of disseminated sclerosis, epilepsy, cerebral syphilis, cerebral thrombosis, syringomyelia, hyperinsulinism, and drug idiosyncrasy, in persons accused of being under the influence of alcohol, have been met with in our experience, the diagnosis being complicated in most cases by the fact that alcohol had been consumed recently (see p. 632).

Under the Road Traffic Act, 1930, section 15, medical men are called upon to examine persons accused of being under the influence of alcohol when in charge of a motor vehicle, and it becomes the responsibility of the doctor to determine whether the individual is under the influence of drink or a drug to such an extent as to be incapable of having proper control of a motor vehicle. It must be remembered that to be under the influence of a drug, while in charge of a motor vehicle, to such an extent as to be unfit to have proper control of the vehicle is an offence of equal gravity. Such cases are rare, and for that reason the following instance is both important and interesting.

In 1937, an omnibus driver was tried at Airdrie Sheriff Court on a charge of having driven a motor omnibus while under the influence of a drug, namely, insulin. Evidence by the bus conductress was to the effect that on starting the journey about 11.32 A.M. the accused, the driver, was quite normal and was driving properly. Later, about 12.11 P.M., it was noticed that his behaviour was irregular and he appeared pale and dazed. There was no smell of alcohol, and in point of fact he was a teetotaler. As he was thought to be under the influence of drink, he was examined medically at 1.15 P.M., when his condition was considered to be consistent with an overdose of insulin. He admitted having taken insulin between 10 and 11 A.M. He was considered unfit to have control of the vehicle. Medical evidence was led at the trial to the effect that the accused was a diabetic, being treated with insulin and a balanced diet. He had been taking insulin for three years, and had been

warned to carry sweets as against overdosage. The sheriff said he was compelled to convict the accused who had been driving for twelve years, although he realised that this was not a matter of his own seeking. Accused was fined £3, or alternatively, twenty days' imprisonment, with endorsement of his driving licence.

In 1945, a motorist was charged with driving while under the influence of insulin. Counsel for the defence insisted that insulin was not a drug within the meaning of the Road Traffic Act, 1930, and that even if it was, the influence under which his client was driving was not that of insulin, but of carbohydrate starvation. The judge refused to accept this argument, but the case was dismissed under the Probation Act. The narrator of this case [4] adds that the possibility that the courts might have to decide a case in which a motorist had an accident through suffering from hyperglycæmia must be kept in view, and that it had been suggested that in this event, the Road Traffic Act would not be applicable since the accused would not have been under the influence of a drug, and that diabetes is not a disease which on application for a driving licence has to be disclosed. The writer concludes by stating that such an incident is improbable for clinical reasons, but that it is believed to have happened in the case of an engine-driver and thus it was not impossible that it might happen to a motorist.

The terms of the medical certificate given in cases under Section 15 of the Road Traffic Act, 1930, are such that, in certain instances, it is almost inevitable that medical opinion should be at variance, if the opinion as to the degree of intoxication present is based on clinical grounds alone. As an adjuvant to the clinical examination, the estimation of the blood or urine alcohol concentration can provide very useful information (see p. 632).

In 1948, a full bench of seven judges in the High Court of Justiciary, Edinburgh, decided that in the normal case of a person suspected of driving while under the influence of drink, he should be asked by the police for his consent to medical examination and tests, and should be made to understand that the result may be used as evidence, and that he is not compelled to submit. In a normal case, before being examined by the police doctor, the suspect ought to be charged and cautioned in the usual way. The accused should be informed of his right to summon his own doctor, and be afforded facilities for doing so, but the police examination should not be delayed until that other doctor is present. The examination should normally proceed outwith the presence of police officers and any interrogation of the accused by the doctor with regard to recent events should be directed solely to testing his memory and coherence and not to detecting information bearing on his guilt. Any information which might be incidentally elicited must not be communicated to the police. If consent to examination was refused, that fact could be proved in evidence, but the doctor, if summoned, should confine himself to observation without examination or test. So far as the police are concerned, in future any departure from the principles laid down by the court would require to be justified.

In 1948, because an accused man had not been warned that if he wished he could refuse medical examination, a police surgeon's evidence was excluded at the trial of a man in Edinburgh Sheriff Court on a charge of having driven a car while under the influence of drink.

The ruling that a police doctor in England is not one who is acting as the mere hand of the police emerged from the case of R. v. Nowell and was laid down by the Court of Criminal Appeal.[5, 6] In this case, a police doctor had examined the appellant, in connection with an alleged offence involving drunkenness while driving a motor car, who was informed that he could have present a doctor of his own, and he was asked to give the name of a doctor who would come. The appellant did not at that time give the name of a doctor and the police doctor examined the man and certified him as unfit to drive a motor car. The appellant was convicted and on appeal it was submitted on his behalf that the appellant did not wish to be examined by the doctor, who asked him several times to let him examine him. The doctor was a little persistent and explained that it might be in the appellant's own interests that a doctor should be allowed to examine him. The appellant reluctantly agreed to be examined. It was not suggested that the doctor had used force.

The court said that they would deal with the situation when force was used, should it ever arise for decision. It held that the doctor from beginning to end behaved perfectly reasonably in the interests of the person he was examining. The court referred to a Scottish case (Reid v. Nixon, Sess. Notes, 17, 1948) according to which it would appear that in Scotland the police doctor is not regarded as an independent medical referee but as the hand of the police. The court could only say that it did not agree that this state of affairs existed in England whether it existed in Scotland or not. Their view was that the evidence of a doctor, whether he was a police surgeon or anyone else, should be accepted, unless the doctor himself showed that it ought not to be, as the evidence of a professional man giving independent expert evidence with no other desire than to assist the court. The appeal against the conviction was dismissed.[6]

From the examination of the blood and urinary alcohol it can be shown—

that the individual has consumed alcohol very recently ;
that there is present in the blood or urine a concentration of alcohol which is, or is not, consistent with a state of insobriety ;
that a certain mimimum amount of alcohol has been consumed.

Experiments by Widmark, Hoffmann, Koller, Elbel, McGrath, and others have shown that the blood-alcohol content of a fresh blood sample, provided that the container is securely closed, will remain substantially unchanged for a considerable period, two weeks at least. Even complete hæmolysis of the specimen in a warm room for a period of sixteen days has not effectively altered the concentration.

For the chemical estimation of alcohol, the methods of Kozelka and Hine and that of Gettler are recommended. The minimum quantity of blood or urine required for the former test is 2 millilitres, and for the latter, 150 grammes (see p. 702).

When alcohol has been consumed and absorbed, its maximum concentration in the blood is reached within half-an-hour to an hour after ingestion. This level is maintained for another hour and thereafter the concentration falls steadily, the rate of fall depending upon the power of the tissues to oxidise the alcohol. The form in which alcohol is taken has an influence on the blood alcohol in that concentrated alcoholic drinks produce a higher blood alcohol than more dilute forms. Numerous investigations have been carried out to determine the degree of concentration of alcohol in the blood which is indicative of insobriety, and in consequence certain standards have been established. It is generally accepted that with a blood alcohol of 60 milligrams alcohol per 100 millilitres blood and under, the individual may be regarded as sober. Between 60 to 120 milligrams alcohol per 100 millilitres blood, the possibility of intoxication increases. Between 120 and 180 milligrams alcohol per 100 millilitres blood, about 50 per cent. of individuals will probably be sober, while over 180 milligrams per 100 millilitres blood will probably indicate intoxication. There seems no doubt that with concentrations of 400 milligrams alcohol per 100 millilitres blood or over, there is likely to be coma or impending coma. In this connection, Jetter is of the opinion that a dose of alcohol, sufficient to produce a blood concentration of 500 milligrams alcohol per 100 millilitres blood, and over, is likely to be a lethal dose.[7]

The relationship of the urine alcohol concentration to the blood alcohol concentration is of interest and practical importance. According to Jetter, the urine alcohol is higher than the blood alcohol in the proportion of 1·23 : 1. Moreover, the urine alcohol concentration, at any given time after the maximum concentration in the blood has been reached, will be higher than the blood alcohol concentration at that moment, since the specimen of urine examined will have been secreted from the blood at some earlier period. For this reason, if it is desired to obtain from the urine alcohol a reliable estimation of the blood alcohol at the time of the examination, the specimen of urine examined should be secreted at the time of the examination. If the bladder is emptied at the beginning of the examination, and again at the end, the second specimen will give an indication of the blood alcohol at the time of the examination. The examination of the urine will, however, prove whether alcohol has been recently taken and, if so, the minimum amount consumed can be estimated. The standard given above for blood alcohol and the indications to be drawn therefrom, regarding the question of intoxication, can also be applied to the urinary alcohol concentration, having regard to the facts that the urine alcohol will be higher in the proportion of 1·23 : 1, and that the urine alcohol, except under the circumstances mentioned above, is not an indication of the blood alcohol at the time of the examination. The condition of diabetes

may play a part, but only a small part, in the estimation, and routine tests should always be employed to exclude the presence of sugar, acetone, diacetic acid and beta-butyric acid in the urine.

From these estimations the probability, or otherwise, of intoxication being present can be gauged, but the estimation by itself cannot always be taken as a certain indication that a condition of insobriety exists. The clinical examination must still remain the determining factor in arriving at a final diagnosis, and the laboratory observation is to be considered as an adjuvant.

Blood alcohol in relation to total body alcohol.

Widmark has originated the following simple formula for the approximate calculation of the quantity of alcohol in the body, after equilibrium between the blood and tissues has been reached :—

$$a = cpr.$$

a = the amount of alcohol expressed in grammes.
p = the weight of the body in kilograms.
r = a constant which is 0·60 for men and 0·50 for women (minimum).
c = concentration of alcohol found, by analysis, in blood, expressed in grammes per kilogram.

(r = body alcohol ÷ blood alcohol.)

Urinary alcohol concentration is higher than blood alcohol concentration in the proportion of 1·23 : 1. A man of 70 kilograms can oxidise 10 millilitres of alcohol per hour.

The table on p. 634 should prove of considerable value since it includes the body alcohol content, and indicates the minimum quantity consumed in the forms of pure ethyl alcohol, whisky, wines, or beer.

Since it is possible, from the body-weight and the alcohol concentration in the tissues or blood, to determine the minimum amount of alcohol consumed, a method of investigation is available, for use at post-mortem examination, to investigate the possibility of intoxication having been present before death (see p. 634). Alcohol can be separated from the tissues for some time after death and even in the presence of putrefactive changes. The question, however, of possible vitiating factors must arise. Decomposition in a dead body does not cause alcohol to be formed in the tissues, although it has been suggested that certain yeasts, and perhaps a few rare bacteria, might play a part in the formation of alcohol from the blood glucose. The amount, if it could be produced in this rare event, would be negligible in the estimate. The estimation of blood alcohol in the cadaver would err on the side of safety, since more alcohol might have been present in the living state than after death as the result of a certain amount of alteration following death. When putrefaction is present in advanced degree, the determination of blood alcohol, likely to have been present at the time of death, becomes difficult.

Amount of ethyl alcohol per 100 millilitres of blood (tissues and blood concentrations being in equilibrium).		Amount of ethyl alcohol in a man of 70 kilograms in weight (11 stones).		Minimum amount of liquor consumed, in one of the following forms, by a man weighing 70 kilograms (11 stones).			Approximate time required for complete removal of alcohol from the body.
				Whisky. Alcohol 40 per cent. by Vol. = 30° U.P.	Wine. Alcohol 16 per cent. by Vol.	Beer. Alcohol 3·28 per cent. by Vol.	
Milligrams.	Millilitre.	Millilitres.	Fluid Ounces.	Fluid Ounces.	Fluid Ounces.	Pints.	Hours.
50	0·063	26·4	0·9	2·3	5·8	1·4	2·5
100	0·126	52·9	1·8	4·6	11·6	2·8	5·0
200	0·252	105·8	3·7	9·3	23·2	5·6	10·5
300	0·378	158·7	5·5	13·9	34·9	8·5	16·0
400	0·504	211·6	7·4	18·6	46·5	11·3	21·0
500	0·630	264·6	9·3	23·2	58·2	14·1	26·5
600	0·756	317·5	11·1	27·9	69·8	17·0	32·0

Taking the matter as a whole, it is fairly definite that the alcohol determined as present in the blood after death would have been present during life. On the other hand, it is questionable if it would be justifiable to conclude that the amount of alcohol found in the blood after death represented the total amount present in it at the time of death. Where possible, the blood should be properly collected within not more than two to three days after death. The post-mortem estimation of blood alcohol concentration can be of considerable value in cases of fatal assaults and accidents, where intoxication may have been a contributory factor to the fatal issue. For the determination of alcohol, blood is a more suitable material for analysis than urine, since in the latter instance the bladder may have contained a large amount of urine prior to the alcohol being excreted into it and thus the percentage of alcohol in the urinary specimen would be inaccurately low and misleading. In the case of R. v. Watson, an estimation of the alcohol concentration in the blood after death was admitted as evidence and went far to show that the deceased woman had been markedly intoxicated before death (see p. 306).

Acute alcoholic poisoning.

Treatment.

The stomach should be emptied and washed out by the stomach tube; the patient should be roused to prevent coma and stimulated by rectal injections of coffee and hypodermic injections of strychnine or digitalin. In cases with dangerous respiratory depression, inhalation of 10 per cent. carbon dioxide and 90 per cent. oxygen has been recommended to establish and maintain normal respiration until natural colour is shown. This concentration of carbon dioxide should be used only as emergency treatment. General measures should also be adopted, including restoration of bodily warmth.

Post-mortem appearances.

In fatal cases there are congestion of the brain and internal organs, some of which may show cloudy swelling. On opening the cavities of the body, a distinct vinous odour is frequently noted. The gastric mucosa is congested and the contents have a vinous odour, especially after washing the stomach with warm water. The bladder is usually distended. On microscopical examination of the organs, fatty changes may be seen.

Methyl alcohol or methanol.

Methyl alcohol is synthesised on a commercial scale from water-gas under pressure in the presence of a catalyst, such as metallic copper containing 10 per cent. of zinc oxide. It is used as a solvent for shellac and gums in a variety of varnishes. When pure, it is almost colourless, and is practically devoid of odour or taste. Its toxicity is increased by such impurities as acetone, ketone, and

aldehyde. Pyridine or furfurol may be added. The commercial fluid, known as wood naphtha, pyroxylic spirit, or wood spirit, is 60 to 90 per cent. pure, and contains acetone and other impurities. Industrial methylated spirits is a mixture of 95 parts by volume of ethyl alcohol and 5 parts by volume of wood naphtha. Mineralised methylated spirits contains ethyl alcohol, wood naphtha, crude pyridine, and methyl violet for colouring purposes.

Methylated spirits, mixed with cheap port or red wine, was a frequent alcoholic drink used by a certain class of persons and was commonly known in Glasgow as " Red Biddy."

By the Methylated Spirits (Sale by Retail) (Scotland) Act, 1937, it is unlawful to sell by retail any methylated spirits, or surgical spirit, unless the seller is an authorised seller, or the seller's name is on the local authority's list, and an entry is made in a book kept for the purpose, and the purchaser signs the entry or delivers a signed order.

Surgical spirit contains industrial methylated spirit with castor oil, methyl salicylate, or mineral naphtha, and ethyl phthalate.

The symptoms of acute poisoning by methyl alcohol include drowsiness, nausea, vomiting, vertigo, headache, sometimes epigastric pain, and visual defects, frequently succeeded by blindness. There are cardiac depression, dyspnœa, cyanosis, cold sweats, subnormal temperature, confusion, delirium, convulsions, and unconsciousness, followed by coma and death. Many fatal cases of poisoning have occurred.

Methyl alcohol frequently remains in the body for several days and is excreted in the breath and in the urine. It is oxidised into formic acid and formaldehyde and large quantities of lactic and other organic acids are formed. Thus it is that the patient suffers from marked acidosis. Death has followed the ingestion of 1 ounce, but cases have recovered after drinking more than ten times this quantity. It is generally held that the consumption of ethyl alcohol counters the effects of methyl alcohol and a reason advanced is that the former displaces the latter from its intracellular attachments and thus checks its oxidation to formic acid, the substance held to be primarily responsible for the acidosis. After taking methyl alcohol there is usually a latent period of twelve to twenty-four hours before the manifestations of poisoning are disclosed.

In 1942, a number of fatalities, due to drinking synthetic methyl alcohol mixed with industrial spirit obtained from leaking casks, occurred in Glasgow. In some of the cases, many hours elapsed before the onset of acute illness accompanied by severe abdominal pain. The patients passed into coma and death resulted from paralysis of the respiratory or cardiac centres. In one of the Glasgow hospitals 18 cases were treated, but 7 of these proved fatal. Four eyes were examined, post-mortem, and the chief histological findings were œdema of the retina and optic nerve head, but no changes were seen in the optic nerve itself.

Duke-Elder, in connection with eye changes in methyl alcohol poisoning, states that it is probable that the primary retinal damage is to the ganglion cells and that optic atrophy is secondary.

In suspect cases, a sample of blood should be retained for the determination of methyl alcohol, since the post-mortem signs show considerable variation and a reliable history is often not obtained.

Treatment.

Gastric lavage with 4 per cent. sodium bicarbonate and the administration of copious fluids. Protection of the eyes from strong light. Treatment should be that of acidosis due to the production of formic acid. Chew, Berger, Brines, and Capron [8] founding on their experience recommend the use of massive doses of sodium lactate. To check the downward progress of the carbon dioxide combining power of the plasma, 160 millilitres of a molar solution of the lactate, in a litre of an isotonic solution of the three chlorides, is given intravenously. When sodium lactate is not at hand, freshly prepared 5 per cent. sodium bicarbonate in 250 millilitres can be substituted. They suggest, however, that the lactate is preferable since it yields the sodium ion more slowly into the system. Sodium bicarbonate is given by the mouth in doses of 4 grammes every fifteen minutes for four doses or, if the patient is unconscious, by stomach tube. This routine may have to be repeated three to four times to raise the combining power from 10–20 to 40–50 volumes per cent. Where there are no facilities for checking the plasma bicarbonate, the urinary reaction may be used as a guide to the administration of the alkali. Continued treatment with sodium bicarbonate, 2 grammes an hour is recommended while the urinary pH is below 7·0, and 2 grammes every two hours when the pH has reached 7·0. Of 31 cases so treated, after taking an average of 222 millilitres of methyl alcohol, only five died, of whom four were moribund when first seen. It is advised that, for the present time, this form of treatment should be adopted as a routine. Cardiac stimulants, and strophanthin or caffeine sodium benzoate are recommended. When there is dangerous respiratory depression, inhalation of up to 10 per cent. carbon dioxide, and 90 per cent. oxygen, has been recommended in order to establish and maintain normal respiration until the natural colour has been fully restored.

Amyl alcohol.

Amyl alcohol is obtained by purification of fusel oil. It consists of a mixture of about 90 per cent. of primary isoamyl alcohol and 10 per cent. of primary active amyl alcohol. Cases of poisoning from amyl alcohol are comparatively rare.

The symptoms of acute poisoning may be delayed for several hours and unconsciousness may then rapidly supervene with accompanying signs of collapse. An odour resembling that of pears may be detected from the breath.

Treatment.

Restoration of body warmth and free gastric lavage. Artificial respiration and oxygen inhalations, with 7 per cent. carbon dioxide,

will prove of high value in the treatment of respiratory embarrassment. Stimulants such as cardiazol may be usefully employed.

Post-mortem appearances.

Asphyxial signs will be present. In the stomach, the characteristic odour of amyl alcohol is likely to be perceived.

The mucous membrane of the stomach will be congested.

References

1. " A Review of the Effects of Alcohol on Man," The Report of a Medical Committee, 279, Gollancz, 1931.
2. " The Method and Significance of Blood Alcohol Estimations," Reprint, Irish Jour. Med. Sc., July 1939, McGrath.
3. Brit. Med. Jour., Vol. I, Supp. 53, 1927.
4. Medical Annual, 4, 1946.
5. Police Jour., Vol. XXI, No. 4, 246, 1948.
6. 1 All E.R., 794, 1948.
7. Amer. Jour. Med. Sc., Vol. CXCVI, 475, 1938, Jetter.
8. Jour. Amer. Med. Assoc., 130, 61. 1946, Chew, Berger, Brines, and Capron.

CHAPTER XXII

HYPNOTICS AND ANTIPYRETICS

Paraldehyde.

THIS substance, which is a colourless liquid, is used for inducing sleep, and is also administered rectally as a basal hypnotic for the purpose of anæsthesia. Moderate doses, given orally, produce sleep which is not accompanied by any marked change in circulation, respiration, or sensibility. When administered rectally, as a basal hypnotic, the usual dose is 1 drachm per stone of the body-weight, up to 8 drachms. It is probably one of the safest basal hypnotics. Paraldehyde not infrequently becomes a drug of addiction, despite its unpleasant taste and penetrating odour. It is excreted unchanged in the breath and urine. Its use produces tolerance. Death from overdosage is infrequent, and recovery has followed the ingestion of 2 ounces. Confusion between drachm and ounce doses is sometimes the cause of mishap, and accidental poisoning occurs from confusion between paraldehyde and a dilute solution, namely, " haustus paraldehydi " or " paraldehyde draught." It is recommended that bottles containing draught or the pure solution must have the dose clearly stated on the label. The draught is not an official preparation, and its use is one of the reasons for mistakes. In one of our cases, death resulted from accidental overdosage. In error, 5 ounces were given rectally instead of 5 drachms. Post-mortem examination showed marked congestion of the meningeal vessels, brain, and lateral ventricles, which contained an excess of clear fluid, and of the lungs and abdominal organs. All the chambers of the heart showed dilatation, more marked on the right side. Death occurred forty-three hours after the administration. The clinical picture of overdosage is that of coma. On opening the body at autopsy, there is usually perceived an aggressive and typical odour of paraldehyde. Death occurs from respiratory failure. Treatment consists of gastric lavage and the use of oxygen with 5 to 7 per cent. carbon dioxide. The airway must be kept clear. Every effort should be made to arouse the patient, but he should not be submitted to muscular exertion. Strong coffee, with 5 per cent. dextrose, may be given by rectum. Small doses of strychnine may be injected hypodermically with benefit. The patient should be kept warm.

If the administration of paraldehyde has been by rectum, high colonic or rectal lavage should be employed as soon as possible after the purpose of the paraldehyde has been served.

Sulphonal.

This is a sulphone hypnotic which does not produce tolerance by its use. It is cumulative, since its excretion from the body is slow. Many cases of poisoning have been recorded from overdose. In cases of poisoning, the urine is frequently of a dark bluish-red colour, due to the presence of hæmatoporphyrin, and when it is examined spectroscopically, the appearance of the spectrum will depend upon whether the urine is acid or alkaline. When acid in reaction, there is a narrow band between C and D, close to the D line, and a second much broader band between D and E. If the reaction is alkaline, there is one band between C and D, two distinct bands between D and E, and a fourth broad band between E and F (see p. 335).

When coma is prolonged, there is always the danger of complication by broncho-pneumonia. Death results from respiratory failure.

Thirty grains have killed but, on the other hand, recovery has followed the ingestion of 275 grains and more.

The treatment of acute poisoning includes free gastric lavage. Sodium bicarbonate in diluted solution should be freely given. Stimulants such as strong hot coffee or strychnine will prove efficacious. Coramine, 5 to 15 millilitres of 25 per cent. solution intravenously, artificial respiration, intravenous injection of normal saline with 5 per cent. dextrose, lumbar puncture with drainage, and oxygen inhalations, with 5 to 7 per cent. carbon dioxide, are other lines of treatment which may be employed beneficially as the condition indicates.

The post-mortem signs are chiefly those already mentioned under paraldehyde (see p. 639).

The spectroscopic detection of hæmatoporphyrin in the urine affords strong suspicion of sulphonal poisoning.

Chemical tests (see p. 723).

Trional, or methylsulphonal, and tetronal.

Trional is an oxidation product of mercaptol, and the strength of its hypnotic action lies between that of sulphonal and tetronal. The toxic effects and treatment of trional and tetronal are the same as those of sulphonal (see above).

Chloral hydrate.

This hypnotic, which imparts a burning feeling to the mouth and exerts an irritant action on the stomach, should be given in well diluted form. It is of special value in the treatment of nervous and mental conditions, is sometimes used for suicidal purposes, and has been employed criminally by mixing it with alcoholic beverages, with the object of stupefying persons for purposes of robbery. In a case of suicide of an intemperate man, seen by us, the quantity taken was 220 grains.

Occasionally, chloral hydrate has caused death by having been taken, or given, accidentally. In one case, ½ ounce of the drug, instead of salts, was taken in gin.

Symptoms.

The principal manifestations are drowsiness, merging into unconsciousness, slow, shallow respiration, with cyanosis of lips, and gradual weakening of the pulse. Chloral hydrate is a central nervous system depressant which, in toxic doses, paralyses the respiratory and cardiac centres.

Treatment.

Free gastric lavage should be employed and the body heat maintained. Subcutaneous injections of strychnine and caffeine sodium benzoate should be given. Artificial respiration and oxygen inhalations, with 5 to 7 per cent. carbon dioxide, may be necessary.

Post-mortem appearances.

These are not typical.

Fatal dose.

Very uncertain. While so small a quantity as 20 grains has killed an adult, a boy of sixteen has recovered from the effects of swallowing 1 ounce.

Fatal period.

Is variable. It may be comparatively short, or it may be prolonged for two or three days. Usual period is from ten to fifteen hours.

Chemical tests (see p. 713).

Barbiturates.

In this series is included barbitone, and malonylurea or veronal, also known as diethylbarbituric acid. A large number of barbituric acid derivatives are extensively used as hypnotics under various trade names such as Luminal, Amytal, Soneryl, Nembutal, Evipan, Phanodorm, Dial, Ipral, Allonal, Noctal, and Pernocton. Evipan, Amytal, Nembutal, and Pernocton are rapid in action but shorter in effect. The barbiturates used for anæsthetic purposes as basal narcotics include sodium Amytal, Nembutal, Pernocton, sodium Soneryl, and Hebaral sodium. Those used for induction or as complete intravenous anæsthetics include Evipan sodium, Pentothal sodium, Eunarcon, Narconumal, and Venesetic. Basal narcotics such as Avertin and Pentothal sodium are administered per rectum, Nembutal and, for children, sodium Soneryl, by mouth, and Pentothal sodium, intravenously. The use of barbiturates as hypnotics, for pre-anæsthetic medication and for the treatment of psychiatric conditions has steadily increased (see p. 617). They have a depressant effect upon the central nervous system and affect the cardiac and respiratory centres. Respiration becomes shallow and

rapid or slow and laboured. When narcosis is of long duration, pulmonary œdema or broncho-pneumonia is prone to develop. In some cases there is idiosynerasy, and in all cases, if the administration is continued, the risk of addiction is ever present. In cases of fatal poisoning, death is usually caused by respiratory failure. Barbiturates are contra-indicated when there is impairment of the liver or kidneys, since excretion is normally slow and these drugs are cumulative.

Many cases of suicidal barbiturate poisoning have been recorded. A case of suicidal overdose of veronal by hypodermic injection has been reported.

Symptoms.

The following signs and symptoms are indicative of poisoning :— Giddiness, indistinct articulation, occasional nausea, diplopia, stupor, coma, shallow and rapid or slow and laboured respiration, slowing of the pulse-rate and subnormal temperature. The reaction of the pupils varies. As poisoning advances, the face tends to become progressively cyanotic. Oliguria may be present. Coma usually continues for one or two days, but the period of survival may be much shorter. When œdema of the lungs, or broncho-pneumonia, complicates the condition, death may result from cardiac failure. Many cases of fatal poisoning have occurred from an overdose of veronal and other barbiturates. Richards [1] has wisely directed attention to the danger of self-administration, and points out that the patient having already become confused by an initial dose of a barbituric compound may continue, on the same occasion, to take further and possibly fatal quantities of the drug without realising the danger. In his opinion, this may explain some of the acute and even fatal cases of poisoning which might otherwise appear unaccountable. The memory of the patient is so affected after taking the drug that he does not realise that he has already satisfied the need and automatically repeats the dose at intervals. Doses should therefore be set aside for the patient so that they cannot be exceeded in the manner described. Richards describes three cases by way of illustration.

In cases of chronic addiction to certain of the barbiturates, such as soluble barbitone, withdrawal convulsions have been reported. These usually occur about four to five days after withdrawal of the drug. They do not follow acute intoxication.

Treatment in acute poisoning.

The stomach should be washed out thoroughly by means of the stomach tube, using a dilute solution of potassium permanganate. About 60 grammes of magnesium sulphate should be left in the stomach. The bowels should be evacuated as soon as possible, and the airway must be kept clear. Colonic lavage is recommended with the view of removing any undissolved tablets of the drug. Stimulating drugs such as caffeine sodium benzoate (15 grains), strychnine ($\frac{1}{30}$ grain), ephedrine ($\frac{5}{6}$ grain), or cardiazol may be administered. The patient should be kept warm and roused, but

not submitted to muscular exertion. Coramine, 5 to 15 millilitres of 25 per cent. solution, or picrotoxin, in doses of 5 milligrams in a 2 per cent. solution of aqueous or normal saline, may be administered intravenously. Small and repeated doses of the latter are preferable to one or two massive doses in combating the cerebral and medullary depression. Picrotoxin is now accepted as the drug of choice on account of its antipathy to barbiturates and its powerful stimulation of the medullary centres, especially the respiratory centre. The drug should be given either intravenously, or intramuscularly in small doses and at intervals of 15 to 30 minutes. Overdosage shows itself by the onset of convulsions which can be controlled by slow intravenous administration of a soluble barbiturate. In cases of barbiturate poisoning, very large doses of picrotoxin have been administered with success.[2] The largest amount given appears to be 2,134 milligrams in all. It has been recommended that the treatment should commence with an initial dose of 10 milligrams intravenously, to be followed two hours later with 5 milligram doses in an aqueous solution intramuscularly at fifteen minute intervals for two to four days, depending upon the degree of poisoning and the clinical response to treatment. Fresh solution should be prepared daily. Cardiazol has also been employed in doses of 2 millilitres of 10 per cent. solution given intravenously, together with 2 millilitres intramuscularly at the same time, to rouse patients from narcosis. It is reported that sodium succinate has been used therapeutically with apparently excellent results. The rationale of the treatment is based upon the hypothesis that barbiturates act by inhibiting brain glucose metabolism prior to the succinate stage and that the replacement of succinate restores the complete carbohydrate metabolic cycle.

Artificial respiration and intratracheal oxygen should be utilised when indications present themselves. The bladder should be catheterised.

An airway should be inserted. A drip-feed dextrose-saline and lumbar puncture are important in certain cases. Willcox has recommended cisternal drainage in serious cases, and this method should be borne in mind to supplement other measures.

The post-mortem signs are not in any way characteristic. The general picture is that of comato-asphyxia. There may be evidence of commencing, or established, broncho-pneumonia, or of œdema of the lungs.

Recovery has followed doses of 125 and 300 grains of veronal, 270 grains of sodium barbital, and 150 grains of soneryl.

When there has been a considerable interval between the ingestion of a poisonous dose and death, analysis of the organs may only disclose traces of barbiturates, due to excretion and decomposition changes. Careful consideration must be given to the clinical picture and post-mortem findings, in conjunction with the results of analysis, before expressing the opinion that death was due to barbiturate poisoning.

Chemical tests (see p. 710).

Acetanilide, antifebrin, or phenylacetamide.

This synthetic drug, so commonly used in analgesic or headache powders, not only gives rise occasionally to toxic symptoms but is taken by patients as an acquired habit. Tolerance can be acquired.

Acetanilide poisoning has occurred not only from swallowing the drug, but also from its inadvertent application to raw healing surfaces. Idiosyncrasy may sometimes exist.

Large doses may cause cyanosis due to the formation of methæmoglobin, the result of hæmolysis. The drug forms para-aminophenol which is excreted in the urine. Chronic poisoning causes debility, tachycardia, breathlessness, cyanosis, dermatitis, and depression. In cases of acute poisoning, in which the foregoing symptoms are intensified, treatment consists of gastric lavage, maintenance of body warmth, stimulation, oxygen inhalations, and dextrose-saline by the intravenous route. Fisher [3] writes of a patient who had been taking 120 grains of acetanilide, and 45 grains of phenacetin, daily over a long period. Typical symptoms of chronic poisoning developed, including cyanosis, weakness, subnormal temperature, mental and physical debility, and methæmoglobinæmia. Typical withdrawal symptoms also developed when the drugs were withheld, but recovery followed.

Chemical tests (see p. 702).

Phenazone or antipyrin.

This substance is an analgesic and antipyretic and is more toxic than phenacetin. The occurrence of toxic symptoms is less frequent than those of acetanilide, which they resemble. The treatment for poisoning is the same as that employed in acetanilide poisoning (see above).

Chemical test (see p. 719).

Phenacetin or acetophenetidin.

This substance is used for the relief of pain and reduction of temperature in certain febrile conditions. It is the safest of the antipyretics, but idiosyncrasy is not infrequent.

In the fatal cases which have been recorded, the prominent feature was marked cyanosis, of pronounced bluish-black colour, affecting skin and mucous membranes. The temperature was subnormal and the urine was chocolate-coloured, due to the presence of methæmoglobin.

Chemical test (see p. 719).

Exalgin or methylacetanilide.

This drug has also given rise to toxic symptoms.

The doses which caused symptoms varied from 6 grains, taken three times daily for a week, to 17½, 150, and 248 grains, respectively,

taken in a single dose. In none of these instances did the patient die. It is used for analgesic and anti-neuralgic purposes.

The symptoms include unconsciousness, delirium, convulsions, cyanosis, and profuse perspiration. Methæmoglobin is found in the blood. Death is caused by respiratory paralysis.

The treatment is the same as for acetanilide poisoning (see p. 644).

Amidopyrin, amidofebrin, or pyramidon.

This substance is a dimethylamido derivative of antipyrin, and is a compound similar to antipyrin. It is analgesic and antipyretic, and is therefore also similar in action to antipyrin. Amidophen contains amidopyrin, phenacetin, caffeine, and hyoscyamus extract. Compral is composed of amidopyrin combined with trichlorethylurethane. Gardan is a combination of amidopyrin and novalgin, while Veramon is a combination of amidopyrin and a molecular compound of amidopyrin with barbitone. Allonal, eibalgin, and veropyron also contain amidopyrin in conjunction with a barbiturate (see p. 641).

Amidopyrin, when taken over a period, or in the case of a susceptible person, may give rise to a serious form of poisoning the outstanding feature of which is agranulocytosis. The red cells are not usually involved. Accompanying symptoms usually consist of pyrexia, ulcerative stomatitis, and possibly pharyngitis, accompanied by marked debility. Fatal results have been numerous. Benjamin and Biederman [4] report the case of a woman who, during a period of good health, was given 10 grains of amidopyrin which produced all the symptoms of leucopenia within forty-eight hours. This is a typical case of hypersensitivity to the drug.

The treatment for poisoning is that used for poisoning by acetanilide. The condition of agranulocytosis may require such measures as blood transfusion, intramuscular injections of liver extract, and the administration of pentose nucleotide (see p. 682).

Acetylsalicylic acid or aspirin.

Aspirin is well known to the public and is used for a variety of conditions, including headache, influenza, and rheumatism. This drug has a mild soporific action. It has also antipyretic and anti-rheumatic properties. In very excessive doses, or as the result of idiosyncrasy, toxic effects may ensue.

Symptoms.

The symptoms of poisoning include vomiting, giddiness, buzzing in the ears, œdema of the face and eyelids, skin rash, cyanosis, and acidosis, due to reduction of alkali reserve. In acute poisoning, perspiration is profuse, and collapse, with subnormal temperature and a weak pulse, occurs. Delirium is present in certain cases. Large doses of aspirin are well tolerated, and as much as 1,500 and 1,250 grains, respectively, have been swallowed with recovery, but susceptibility must not be forgotten. Death of an infant occurred

after swallowing two dessertspoonfuls of a solution containing 1,500 grains in a pint of water. Deaths from aspirin poisoning in England over a period of five years totalled 272 cases.

Treatment.

Treatment consists in free gastric lavage, or active emesis, and the administration of an alkali, such as sodium bicarbonate in 5 per cent. solution. In very serious cases, the withdrawal of cerebrospinal fluid may prove highly beneficial. The patient must be kept warm and stimulants given when indications arise. Dextrose in saline may be administered intravenously.

The following case, for the details of which we are indebted to Drs Glen, Millar, and Shanks, Victoria Infirmary, Glasgow, is illustrative of most of the findings in aspirin poisoning :—The patient swallowed 750 grains of aspirin, in addition to two sleeping powders of unknown composition. She was admitted to hospital, about seven hours afterwards, when she was mentally clear and rational. Prior to admission, she had suffered from violent vomiting and diarrhœa, also buzzing in the ears. The pulse-rate was fast, the extremities were moist and cold, but there was no cyanosis although breathing was rapid. Shortly after admission, she had a convulsion, of short duration, at first tonic and later clonic. Following this, she became rather confused and perspired freely. Her condition deteriorated and she gradually sank into coma. Breathing became progressively more acidotic and ultimately there was extreme "air-hunger." She showed cyanosis and died within twenty-six hours of admission.

The treatment employed consisted of gastric lavage, which yielded a dirty green result, but no tablets were seen. Shortly after this, the patient was able to retain four-hourly doses of alkali, 40 grains. She was also given four-hourly rectal saline with glucose.

Post-mortem examination showed moderate cyanosis of the face and finger-tips. The right heart was dilated. The myocardium was soft. The lungs were intensely congested. The liver, pancreas, and kidneys were also deeply congested.

Chemical tests (see p. 702).

References

1. Brit. Med. Jour., Vol. I, 331, 1934, Richards.
2. Amer. Jour. Med. Sci., 196, 46, 1938, Rovenstine.
3. Jour. Amer. Med. Ass., Vol. C, 736, 1933, Fisher.
4. Ibid., Vol. CIII, 161, 1934, Benjamin and Biederman.

CHAPTER XXIII

VEGETABLE POISONS

AN alkaloid is a complex chemical substance which is found in various plants. It has received the term because it behaves chemically like an alkali in that it constitutes a base which unites with acids to form salts. The method for separating the alkaloids of vegetable poisons from mixtures, containing organic matter, such as stomach contents or viscera, is the Stas-Otto process. This is an intricate procedure which falls within the province of the analytical chemist rather than that of the medico-legist. The reader who desires detailed information is referred to appropriate works upon this subject (see p. 524).

Opium

(Nat. Ord., Papaveraceæ).

Opium consists of the inspissated juice of Papaver somniferum, and contains a large number of alkaloids, including morphine, narcotine, codeine, narceine, papaverine, thebaine, apomorphine, anarcotine, and others, several of which are highly poisonous, also the neutral substance called meconin, and meconic acid. The principal alkaloids used in medicine, and obtained from opium, are morphine, codeine, and apomorphine, of which the first two act as narcotics or sedatives, and the last chiefly as an emetic. Opium and morphia enter into the composition of a number of official preparations as well as of proprietary medicines, as, for example, chlorodyne, Nepenthe, and others. Heroin is a derivative of morphine.

Cases of poisoning may arise from any of the preparations which contain opium, but laudanum, chlorodyne, and of the preparations of morphia, any soluble salt of the Pharmacopœia, are the commonest sources.

Symptoms.

These appear earlier from the use of morphia than from crude opium or its preparations, and they may be divided into two groups, namely, those indicative of excitation of the higher nerve-centres, and those indicative of narcosis. When a case calling for treatment comes under notice, the former symptoms will usually have passed away, and the latter are fully developed. The latter symptoms especially, therefore, call for consideration. They consist of an overpowering drowsiness which gradually becomes deeper until it ends in profound coma from which no stimulus can arouse the

patient. In the earlier stages, however, the person may be partially roused to consciousness. In the later stages the muscles become relaxed, the pulse is small and weak, and the breathing laboured, noisy, and perhaps irregular, and finally, shallow and slow. The temperature is subnormal. The face is pale, cold, and sometimes bedewed with clammy perspiration, and the pupils are strongly contracted, almost to pin-head size. Death may follow the deepening of narcosis, or it may be preceded, especially in children, by convulsive seizures.

It occasionally happens that there may be a remission of the symptoms for a short period, but when this occurs they usually return in their original severity, and the person may die.

The symptoms of morphia poisoning may be complicated or masked by morphia having been taken with other drugs.

Infants and young children are most susceptible to the influence of opium and morphia, and the administration of these drugs to children should be avoided, and the use of patent medicines which contain them strongly denounced. Very small doses of laudanum have proved fatal.

Self-administered injections of morphia in overdose may also produce poisoning. One must therefore be alive to this form of administration, and examine for evidence of puncture marks on parts of the body available for self-administration.

Morphinism is more prevalent in persons who have a psychopathic tendency, or an unstable nervous system. After a period there is usually evidence both of physical and moral deterioration. Addicts develop a marked tolerance to the drug and are able to take quantities which would have fatal consequences in those unaccustomed to its use. Untruthfulness, dishonesty, and mental deterioration are commonly found. When under the influence of the drug, addicts appear calm and composed; when the effect wears off, they become restless, irritable, and cantankerous. Withdrawal symptoms include delirium and hallucinations.

Treatment.

This must be regulated by the circumstances of each individual case. If the poison has been swallowed, there must be free lavage of the stomach, first with warm water, the return being retained for analysis, and then with warm water in which a suitable quantity of potassium permanganate has been completely dissolved. Thirty grains of potassium permanganate should be used in 2 gallons of water. Lavage should be continued for several hours.

Even in cases in which morphia has been administered hypodermically, the permanganate treatment should be adopted since morphia is excreted by the stomach. It is important to remember that permanganate must not be used in too strong solution as it is slightly corrosive. The use of medicinal charcoal is also beneficial.

Although potassium permanganate will oxidise the alkaloid present in the stomach, it will also be necessary to combat the toxic effects of the amount absorbed.

Atropine sulphate ($\frac{1}{100}$ grain) may be injected hypodermically, but it must be used with caution to prevent depression following stimulation of the respiratory centre.

In all cases of poisoning the body warmth must be restored by appropriate measures, and it is unwise to subject a patient to vigorous or prolonged muscular exercise. It is more helpful to rouse him by sustained attention. In the immediate absence of a stomach tube, emesis should be promoted and encouraged by the readiest emetic. Stimulation can be effected by the use of strong coffee, caffeine sodium benzoate, 2 grains hypodermically, or strychnine, $\frac{1}{30}$ grain. Coramine, 5 to 15 millilitres of 25 per cent. solution, may be given intravenously. Artificial respiration and inhalations of oxygen with 5 to 7 per cent. carbon dioxide, or oxygen alone, may be administered as circumstances indicate. The airway should be kept clear and the patient should be catheterised.

Post-mortem appearances.

The post-mortem signs will be those of coma, or comato-asphyxia. If the case has been untreated, and opium or laudanum has been swallowed, the odour of the drug may be perceived in the stomach. It should not be forgotten that opium may be introduced to the body by channels other than the stomach, as, for instance, by the rectum.

Fatal dose.

Infants.

One drop of laudanum has killed on more than one occasion, and compound tincture of camphor containing $\frac{1}{19}$ grain of opium has produced the same result. Children have, however, recovered from large doses: from $7\frac{1}{2}$ grains of opium, from one and two teaspoonfuls of laudanum, and from 60 minims of liq. morphinæ. We have seen recovery in a child of nine months, after 15 drops of laudanum, without recourse to treatment.

Adults.

Four grains of opium and 1 grain of morphia, respectively, may be regarded as probable fatal doses in those not addicted. Recovery has, however, followed treatment when 360 grains of opium and 55 grains of morphine acetate, respectively, have been taken. In a case, which we treated, recovery followed the swallowing of the contents of one and a half bottles of Collis Browne's Chlorodyne.

Fatal period.

Death may occur within forty-five minutes. This happened in the case of a woman who had swallowed 1 ounce of laudanum. The usual period is from nine to twelve hours, but it may extend to two days. In one case where 10 grammes of a liquid preparation of opium were injected into an adult instead of ten drops, death did not occur till two days later.

Chemical tests (see p. 719).

Atropine

(Nat. Ord., Solanaceæ).

Nearly all parts of the plant, Atropa belladonna, yield the alkaloids atropine and hyoscine. This plant is popularly called Deadly Nightshade. The root and leaves are used medicinally.

Eating the berries has caused poisonous and fatal effects on several occasions. When poisoning has been caused in this way there may be a long interval between ingestion of the berries and the appearance of symptoms.

Cases of poisoning by belladonna liniment, taken accidentally or suicidally, are recorded. In one, a wineglassful was taken; in a second, 2 ounces and 2 drachms were swallowed; and in a third case, a boy of two and a half years drank a quantity equal to about 4 drachms of the extract. Hamilton and Sclare [1] report a case of accidental belladonna poisoning which presented as an acute confusional state after ingestion of 10 minims of the liquid extract. Visual symptoms commenced within half an hour, and general symptoms after three hours. All of these patients recovered following treatment.

From the use of belladonna as an external application in the form of liniment and plaster, and as an enema, serious and fatal results have occurred. We have seen toxic symptoms after the use of a 2-grain pessary.

Cases of accidental and suicidal poisoning by atropine are comparatively rare, and criminal poisoning still rarer. Comroe [2] reports a case of recovery following poisoning with 7½ grains of atropine sulphate, despite the fact that the stomach was not evacuated until one and a half hours after ingestion of the drug.

The action of atropine, in overdosage, is first to stimulate the higher centres, including the motor centres, but this is followed by depression. The secretory mechanism is inhibited. As the result of depression of the respiratory centre, death ensues from asphyxia.

Symptoms.

These are giddiness, drowsiness, mental confusion, great thirst, due to parching of the mouth and throat, the result of diminished secretion, indistinct articulation and difficulty in swallowing. In the earlier stages, the patient is talkative, chattering, active, and in a state of delirium which is often turbulent or violent. The face is flushed, the pupils are widely dilated and immobile, and the temperature is elevated. In cases which end fatally, the drowsiness lapses into deep sleep, then coma, during which the face usually assumes a livid colour. Finally, there are convulsions, and death.

Carter [3] describes an interesting case of a man, aged seventy-three, who swallowed 5 drachms of belladonna liniment, equivalent to 1-2 grains of atropine sulphate, in error for a cough mixture. No treatment was given for ten hours. After taking the poison, the man experienced an unusual taste in his mouth which was accompanied by a burning sensation. He went to sleep and in the morning his wife found

him in a convulsion. On admission to hospital he was unconscious, flushed, and breathing stertorously. There was slight excoriation of the lips, the tongue and mouth were dry and leathery, the pupils were fully dilated and inactive to light, and there were occasional convulsive movements of the legs and arms, together with occasional bilateral twitching of the lower part of the face. The temperature was 99·6, respirations 26, and pulse-rate 120. There were some extra-systoles. The bladder was distended. Following active treatment, the patient was discharged, fully recovered, eleven days after admission, and when seen two months later was in excellent health.

About half the quantity of atropine absorbed into the body is excreted unchanged in the urine in the course of some thirty-six hours.

Treatment.

Gastric lavage first with water and then with a solution of 1 in 5,000 potassium permanganate should be carried out as quickly as possible. A dilute solution of tannic acid may be employed as an alternative. Strong coffee may be left in the stomach with benefit. It is advisable finally to leave a solution of magnesium sulphate in the stomach to promote purgation. Morphia ($\frac{1}{4}$ grain) and physostigmine ($\frac{1}{50}$ grain) may be given hypodermically. Pilocarpine should not be used. When respiratory action is depressed, morphia should be withheld. Excitement may be controlled by the use of sedatives or mild hypnotics. Lumbar puncture may prove beneficial in certain cases. Artificial respiration, or the use of oxygen, may become necessary.

When the berries of atropa belladonna have been eaten and have remained for some time in the stomach, they tend to resemble raisins with which they should not be confused, since the seeds of the former are smaller and darker than raisin seeds. In such cases it is expedient to use an emetic rather than a stomach-tube since the berries may readily block the tube.

Post-mortem appearances.

In the case of children, the stomach contents should be carefully examined for portions of berries or seeds. The post-mortem signs are generally those of comato-asphyxia, and œdema of the lungs may be present.

Fatal dose.

A rectal injection of a decoction of 80 grains of the root, one of a drachm of the extract, and a swallowed dose of the same quantity of the liniment have proved fatal. Death has occurred from the external application of a belladonna plaster to a raw surface. Two grains of atropine have killed. Recoveries have followed $\frac{1}{2}$ ounce of the liniment, 8 to 12 grains of the extract in a child, and doses of $3\frac{1}{2}$, 4, and $5\frac{1}{2}$ grains of atropine. An ointment containing about 3 grains of atropine applied to the abraded skin caused death. Thirty berries of atropa belladonna have caused severe poisoning in children.

Chemical tests (see p. 710).

Datura and hyoscyamus

(Nat. Ord., Solanaceæ).

Belonging to the natural order, Solanaceæ, are many poisonous plants (see p. 653), including Datura, Hyoseyamus (Henbane), Solanum tuberosum, or the common potato, Solanum dulcamara, or Woody Nightshade or Bittersweet, Solanum nigrum, or Garden Nightshade, and other Solanums. Datura is poisonous in all its parts and contains the alkaloids lævo-hyoscyamine, atropine, and hyoscine or scopolamine. Farnwrith, Anderson, Henderson, Begg, and MeNaughton [4] state that the lethal dose of all these three alkaloids is about 1 grain for adults and $\frac{1}{16}$ grain for children. They consider that the action is slow and that a fatal issue may not occur for hours or even days. The symptoms of poisoning are dryness of the mouth and throat, visual hallucinations, mental excitement, temporary lapse of memory, nausea, vomiting, abdominal pains and distension, and dimness of vision. Only in rare instances, and at certain periods of growth, is the potato responsible for mild toxic manifestations due to the presence of small quantities, or traces, of atropine and solanine. The symptoms are chiefly those of gastro-intestinal irritation. Solanum dulcamara and Solanum nigrum contain also the alkaloid solanine and traces of atropine. The mydriatie alkaloids, hyoscyamine, hyoscine (scopolamine), daturine, duboisine, atropine, and others, which in common possess the power of dilating the pupil, are practically identical so far as their symptoms are concerned. Both hyoscine and hyoscyamine are less deliriant in their action than atropine (see p. 650).

Hyoseine is rarely used homicidally, but in the Crippen case it was believed to have been the cause of death.

About 2 grains of hyoscyamine and $\frac{1}{2}$ grain of hyoseine have caused death.

The post-mortem findings in, and the treatment of poisoning by, hyoseine and hyoseyamine are closely similar to poisoning by atropine (see p. 651).

Chemical tests (see p. 710).

Aconite

(Nat. Ord. Ranunculaceæ).

Aeonitine is obtained from the dried root of the plant Aconitum napellus, or Monkshood. The root has caused poisoning, having been eaten in error for horse-radish.

Aconite root when tasted produces tingling and numbness of the tongue, mouth, and lips, and when eut slowly reddens. Horse-radish root when tasted is hot and pungent, but does not cause tingling, and when eut remains white.

From the fresh leaves and flowering tops of the aconite plant an extraet is made, and from the root, a tineture and a liniment.

Aconitine is usually a white amorphous powder. Other alkaloid derivatives have been obtained from the aeonite elass.

FIG. 228.—**Poisonous berries and seeds.**

1. Solanum nigrum (black nightshade). 2. Solanum dulcamara (bitter-sweet or woody nightshade). 3. Bryonia dioica (common bryony). 4. Ligustrum vulgare (privet). 5a. Fruit of colocynth. 5. Citrullus colocynthis (colocynth or bitter apple).

FIG. 229.—**Poisonous berries and seeds.**

6. Laburnum vulgare (laburnum)—*a*, leaves; *b*, ripe pods; *c*, ripe pod; *d*, ripe seed; *e*, unripe pods; *f*, unripe pod. 7. Prunus lauro cerasus (cherry-laurel). 8. Daphne laureola (spurge laurel). 9. Convallaria majalis (lily of the valley). 10. Taxus baccata (yew). 11. Atropa belladonna (belladonna). 12. Fruit of belladonna.

Symptoms.

The signs and symptoms may be described as follows :—Their onset develops from a few minutes to an hour after taking the poison : numbness, burning, and tingling of the mouth and throat ; a feeling of constriction and burning in the throat ; severe pain and tenderness in the stomach ; nausea and vomiting ; numbness, loss of power, and pain in the limbs ; giddiness, singing in the ears, deafness, and impairment of vision ; indistinct articulation, and ultimate loss of the power of speech ; unconsciousness, convulsive gasps, or convulsions ; contraction and later dilatation of the pupils. The cause of death is either cardiac or respiratory failure due to paralysis of the centres in the brain.

Poisoning is relatively more common from accident than from suicide or homicide. We are acquainted with the facts of a case where a man swallowed, shortly after a meal, $\frac{1}{2}$ ounce of A.B.C. liniment, and although the symptoms of poisoning did not actively supervene for about one hour after and he eventually recovered, his life was in great danger for some hours. Treatment had, however, been promptly and energetically applied.

Treatment.

Free gastric lavage with a solution composed of 180 grains of tannic acid in 2 gallons of water. Give 20 grains of tannic acid in 6 ounces of tepid water followed by animal charcoal suspended in water and administer stimulants. Restoration and maintenance of body warmth are essential. A hypodermic injection of strychnine, $\frac{1}{8}$ grain ; atropine sulphate, $\frac{1}{100}$ grain ; or digitalin, $\frac{1}{200}$ grain, are recommended. Respiratory embarrassment may necessitate the use of artificial respiration and oxygen inhalations.

Post-mortem appearances.

These may be as follows :—Pallor of the mucous membrane of the mouth ; congestion or engorgement of the brain and lungs ; inflammation of the mucous membrane of the stomach ; congestion of the liver and kidneys.

Fatal dose.

About 1 drachm of root, 4 grains of extract, or 25 drops of strong tincture. Of the alkaloid, $\frac{1}{15}$ grain has caused death, but recovery has followed 2 teaspoonful, and 1 tablespoonful, doses, of the tincture and a dose of over 2 ounces of the liniment.

Fatal period.

The shortest period is three-quarters of an hour, the longest, twenty hours, and the average about eight hours.

Test (see p. 702).

Strychnine

(Nat. Ord., Loganiaceæ).

From the seeds of Strychnos nux vomica an official tincture and extract are prepared for medicinal purposes. The seeds are flattish, almost round, less than an inch in diameter, slightly convex on one side, concave on the other, and are covered by fine, short, silky, yellowish-grey hairs. They are very hard, and when a small portion is chewed, an intensely bitter taste is experienced in the mouth. From this seed the alkaloids strychnine and brucine are obtained. The alkaloid strychnine has an intensely bitter taste, and 1 grain will impart a perceptible bitterness to a gallon of water (1 in 70,000).

Symptoms.

Strychnine has a selective action on the central nervous system and produces a condition of irritability of the reflex centres of the spinal cord. When a poisonous dose has been swallowed, the following symptoms appear after an interval which may vary from a few minutes to an hour. An intensely bitter taste is perceived in the mouth and soon afterwards there is a sensation of suffocation, accompanied by twitching of the muscles of the neck, body, and limbs, followed by severe tetanic convulsions which involve all the muscles of the body. During this state the muscles become stiff and rigid, so that the body is thrown into an arch, only the back of the head and heels touching the bed or ground. This condition is known as opisthotonos. In some cases, the body-curve is in the opposite direction, called emprosthotonos, or the body may be curved laterally, pleurosthotonos. Owing to the tetanic contraction of the thoracic muscles, breathing becomes difficult and imperfect, and the face in consequence becomes markedly cyanosed. As the result of the contraction of the muscles at the angles of the mouth risus sardonicus is produced, the lower jaw becomes firmly shut or fixed by the contracted condition of the masseters, the fingers are clenched in the palms of the hand, the feet arched inwards, and the eyes staring and wild looking. This tonic spasm lasts from half a minute to two minutes, and then there is a remission of symptoms. During a spasm the patient is in complete possession of his senses, and experiences acute pain. During the remission he lies in a calm but weakened condition, and may fall asleep. During the convulsive seizure the pupils become dilated, but in the period of remission they resume their normal condition, which, relative to the former state, appears to be one of contraction.

After a variable interval, depending upon the severity of the toxic effects and often as the result of a very trivial cause, such as someone walking across the floor, a touch, or even a draught of cold air, another attack similar to the first comes on. In cases proceeding to a fatal issue the intervals of remission are short, in less severe

cases they may be longer. Death usually supervenes either during a spasm from asphyxia induced by fixation of the chest wall, or from exhaustion due to the repetition of attacks. Death, therefore, may follow, very shortly after the spasms appear, or it may be delayed for some hours.

In tetanus, or lockjaw, fixation of the lower jaw is one of the earliest and most enduring symptoms, but in strychnine poisoning it is only a part of the general tetanic contraction of the body muscles, and passes off with the muscular relaxation during the period of remission. Indeed there is no other set of phenomena, from disease or poison, which is exactly comparable to that which follows the absorption of strychnine in the body.

Dixon has reported a case of intermeningeal spinal hæmorrhage, ascertained on post-mortem examination, which by reason of the similarity of the symptoms to strychnine poisoning compelled him to decline to grant a certificate of death without a post-mortem examination.

Symptoms of strychnine poisoning appear even if the poison is applied externally and is absorbed. In one case, the application of $\frac{1}{12}$ grain of strychnine to the eye produced toxic effects in about three to four minutes.

Strychnine poisoning has occurred through mistakes in prescriptions by chemists.

Homicidal administration has given rise to some well-known cases, including those of William Palmer, William Dove, Silas Barlow, George Horton, Walter Horsford, Thomas Neill Cream, and Jean Pierre Vaquier.

In the body, part of the strychnine is destroyed by the liver and some of it becomes detoxicated by loose combination with other substances. A portion of it is excreted in the urine unchanged and this excretion continues for two or three days.

Treatment.

Chloroform should be administered as soon as possible in view of the fact that convulsive seizures are induced by almost any form of treatment. As soon as the patient is under its influence, the stomach tube should be used and free gastric lavage performed, with a solution of potassium permanganate in water, 60 grains in 2 gallons. To follow, medicinal charcoal and water are recommended, and these may be freely given and renewed from time to time. Light chloroform anæsthesia should be continued for long periods, if necessary, and for as long as the convulsions threaten to return. Sedatives such as chloral hydrate may be administered. Haggard and Greenberg [5] assert that there is true antagonism between the action of sodium phenobarbitone and strychnine. Kempf, McCallum, and Zerfas [6] report eleven cases successfully treated with sodium amytal, or pentobarbital sodium, given intravenously. They advise that only a quantity sufficient to put the patient to sleep should be given, or if convulsions are present, only enough to stop them. They are of the further opinion that, when such drugs are

administered, gastric lavage is both unnecessary and inadvisable until after the patient is asleep. A mild convulsion should be awaited before repeating the dose of amytal. Stalberg and Davidson [7] state that intravenously sodium amytal is a definite life-saving measure. In conjunction with this treatment, they also use avertin anæsthesia by rectum, and this reduces the quantity of sodium amytal required. One patient received five injections of sodium amytal, a total of 35 grains, intravenously, and four rectal injections of avertin, each 4 grains. Five capsules of sodium amytal were also given by the mouth. The patient had taken $1\frac{3}{4}$ grains of strychnine. The convulsions lasted for five and a half days and did not commence until forty-nine hours after ingestion of the strychnine. Wheelock,[8] following the treatment of a suicidal case, states that the results were so dramatic that in future cases sodium amytal and sodium phenobarbitone should at least be tried. The patient had taken 1 grain of strychnine.

Artificial respiration and oxygen, with 5 to 7 per cent. carbon dioxide, inhalations may also be necessary. Other symptoms should be treated as they arise.

Post-mortem appearances.

The most constant appearances of the internal organs consist of engorgement of the lungs and vessels of the brain and spinal cord, but there are no typical signs. These appearances indicate death by comato-asphyxia.

Fatal dose.

Although alarming symptoms have on more than one occasion been initiated by $\frac{1}{12}$ grain, $\frac{1}{4}$ grain is the smallest fatal dose which has been recorded. Half a grain has also produced fatal results. Recoveries after prompt treatment have, however, followed large doses.

Fatal period.

Death may supervene almost immediately. This happened in the case of a druggist who took $1\frac{2}{3}$ grains, along with nux vomica powder. Life may, however, be prolonged for several hours after the onset of symptoms.

Chemical tests (see p. 723).

Brucine.

This differs in action from strychnine. It produces at first slight convulsive seizures, but these pass off quickly, the chief action being paralysis of the motor nerves and narcotism, thus resembling the action of curare or conium. It possesses only about one-eighth of the toxic effect of strychnine.

Chemical tests (see p. 711).

Digitalis

(Nat. Ord., Scrophulariaceæ).

The glycosides of digitalis are obtained from the plant Digitalis purpurea. Its leaves, root, and seeds contain the toxic glycosides, digitalin, digitoxin, digitalein, and digitonin, and other principles. From the leaves is obtained the glycoside digitalin, which, when pure, consists of fine white acicular crystals, has no odour, and possesses a bitter taste. Digitalin and digitoxin are the principal poisonous constituents. Digitalin is a cumulative poison. Its action is upon the heart, with prolongation of the systolic period in the cardiac cycle. It regulates the rhythm of the heart by depressing both excitability and conductivity. In toxic doses there is increased excitability of the heart with extrasystoles. It has been used homicidally at least upon one occasion.

Allen [9] describes the case of a child of three who swallowed twelve tablets of digitalin (Nativelle) totalling $\frac{1}{50}$ grain of digitalin. Five hours later he vomited and passed loose motions. Twenty-two hours later the pulse-rate was 84 and there were irregular systoles. Despite treatment, the pulse-rate fell to 54. The interesting part of the case lay in the response to treatment. Atropine was more effective than strychnine, and both more than caffeine sodium benzoate in the raising of the pulse-rate from the state of digitalis bradycardia.

Symptoms.

These are nausea, vomiting, which may not, however, commence for two or three hours after taking the poison, followed by abdominal pains, and, perhaps, diarrhœa. The pulse-rate becomes remarkably slow and irregular. Consequent upon this condition of the heart, there is a feeling of faintness and præcordial oppression, the respirations become slow and sighing, the patient becomes drowsy, and the condition may gradually deepen into coma. Convulsions may precede death.

Treatment.

Gastric lavage with a weak solution of tannic acid is necessary. The patient should be kept warm and at rest in bed. Atropine, $\frac{1}{100}$ grain, should be given hypodermically, and stimulants or sedatives used as indicated by the condition of the patient.

Chemical tests (see p. 714).

Strophanthus

(Nat. Ord., Apocynaceæ).

The glycosides of strophanthus, namely, strophanthin, and incine, are obtained from the dried seeds of Strophanthus kombé. Strophanthus has a similar effect to that of digitalis, but acts more

rapidly. Its use is contraindicated in arteriosclerosis, and in both acute and chronic nephritis.

Symptoms.

Headache, præcordial oppression, bradycardia, cardiac arrhythmia, nausea, vomiting, restlessness, and elevation of blood-pressure.

Treatment.

As for poisoning by digitalis (see p. 659).

Post-mortem appearances.

These are not characteristic.

Chemical tests (see p. 723).

Cocaine

(Nat. Ord., Linaceæ).

Cocaine is derived from the alkaloid of Erythroxylum coca, and chemically is methyl benzoyl-ecgonine. It is a crystalline substance, without colour, having a bitter taste, which imparts a numbness to the tongue and mucous membrane of the mouth. It is slightly soluble in water, but dissolves freely in alcohol. It has the property of desensitising the terminal nerves of the part into which it is injected or absorbed, and in a large dose produces paralysing effects upon the central nervous system. When injected for minor operations, such as extractions of teeth, it has produced toxic effects and death. We have investigated a number of deaths which have occurred (see p. 620). Many synthetic drugs have been substituted for cocaine and are employed as local and spinal anæsthetics. These include novocaine, nupercaine, stovaine, neocaine, and planocaine. In some persons, hypodermic injection of cocaine has become a confirmed habit and fatal poisoning has resulted.

Symptoms.

Pallor of the face, dilatation and immobility of the pupils, dyspnœa, headache, giddiness, cramps, vomiting, fainting, rapid unconsciousness, convulsions, and pulse abnormally rapid at first, but quickly slowing down and becoming intermittent and feeble. In fatal cases, asphyxial manifestations supervene, and death results from depression of the respiratory and cardiac centres.

A fatal case is recorded in which, for temporary relief of a urethral stricture to enable a catheter to be passed, ½ drachm of a 10 per cent. solution of cocaine hydrochloride was injected into the urethra, the meatus of the penis being held to prevent regurgitation of the fluid. In a few minutes the patient, a man of fifty-six, was in a state of clonic convulsion. His jaws were moving spasmodically, and he had bitten his tongue. The face was somewhat cyanosed, the breathing very spasmodic and slightly stertorous, the eyeballs

fixed, and eyelids half closed. A pulse could not be felt at the wrist, but the heart was beating. About a minute later, respiration ceased, the cyanosis increasing. Artificial respiration was at once commenced, and other measures employed, but the patient was dead in about three minutes.

Cocaine addiction.

Addicts usually have an initial psychopathic tendency, or an unstable nervous system. The stimulating effect of the drug is responsible for the acquired habit. When the effects wear off, however, there are irritability and restlessness. In addition to the signs of physical degeneration which are apparent, addicts frequently suffer from psychical degeneration, which commonly assumes the form of insanity. Anorexia is a frequent symptom. In such persons, complaint is often made of a sensation in the skin as if grains of sand or insects or other small bodies were lying in, or under, it. Further, they become tolerant to the drug, the dosage of which is gradually increased. The cause of addiction may be attributed to the fact that cocaine quickly banishes fatigue and mental exhaustion, which are replaced by a feeling of mental and physical vigour. The effects of cocaine lead to increased erotic tension in women with the removal of normal inhibitions, which may ultimately lead to a state of nymphomania. In men the physical component decreases while the mental component is increased, this condition leading to many sexual perversions. Hallucinations may also be present. Cocaine may be taken either by the nose, in the form of snuff, or by hypodermic injection.

Treatment.

The stomach should be washed out with a dilute solution of potassium permanganate or tannic acid. Medicinal charcoal may also be employed. If the drug has been administered hypodermically these remedies will be ineffective. Strychnine, $\frac{1}{8}$ grain, may be given hypodermically and inhalations of ammonia may prove beneficial. Coramine, 5 to 15 millilitres of 25 per cent. solution, intravenously, will combat threatened cardiac failure. To control convulsions, sodium amytal, 3 to 10 millilitres of a 10 per cent. solution given intravenously, at the rate of 1 millilitre per minute, has been recommended. Artificial respiration and oxygen should be resorted to in the event of threatened asphyxia. If poisoning has resulted from the contact of cocaine with the mucous membrane, an endeavour should be made to remove as much of the substance as possible by washing the affected surface.

Post-mortem appearances.

In cases of acute poisoning, these are valueless as indicative of the character of the poison. Death being due to asphyxia, or cardiac failure, the signs of these forms of death are likely to be found.

Chemical tests (see p. 713).

Physostigmine

(Nat. Ord., Leguminosæ).

Physostigmine is the alkaloid obtained from the ripe seed, Calabar bean, of Physostigma venenosum. The seed or bean consists of a shell and kernel, the former hard, brittle, and dull claret-coloured, the latter white in colour, and without smell or taste. Physostigmine, or eserine, is used principally in ophthalmic practice. The alkaloid is either colourless or slightly yellow in colour and has a bitter taste. Physiologically, it causes increased irritability of the vagus and oculomotor nerves. For the latter reason, the pupils are contracted. It also produces increased peristalsis, depression of the central nervous system, and twitching of the voluntary muscles. In poisonous doses, it causes giddiness, paralysis of the voluntary muscles, and muscular twitchings. Death is due to asphyxia, resulting from paralysis of the respiratory centre.

There is no recorded case of criminal poisoning, but there are several cases of accidental poisoning.

Treatment.

Gastric lavage with a solution of 60 grains of potassium permanganate in 2 gallons of water. Medicinal charcoal may be administered. Hypodermic injection of atropine sulphate, $\frac{1}{50}$ grain, should be given as a physiological antidote. Stimulants and artificial respiration may be utilised in accordance with clinical indications.

Chemical tests (see p. 721).

Pilocarpine

(Nat. Ord., Rutaceæ).

This alkaloid is obtained from the leaves of Pilocarpus jaborandi. It promotes the flow of saliva and perspiration. Accidental poisoning has occurred. The toxic signs and symptoms are increased lacrimation, perspiration, and excretion of mucus, giddiness, thirst, dyspnœa, laboured breathing, convulsions, and death, which is brought about by paralysis of the respiratory centre.

Treatment.

Gastric lavage with a solution of potassium permanganate, 60 grains to the gallon. Atropine, $\frac{1}{100}$ grain, should be given hypodermically. Atropine is the physiological antidote. Stimulants should also be given.

Post-mortem findings.

These are not characteristic.

Chemical tests (see p. 721).

Cannabis indica, Indian hemp, or hashish

(Nat. Ord., Urticaceæ).

Hashish is produced by compressing the resinous exudate and dried flowering tops of Cannabis sativa into hard masses or slabs. The active principle, cannabinol, which is not an alkaloid but a lipoid-soluble, non-nitrogenous substance, is contained in the resin. The drug is extensively used by the natives of the Near and Far East on account of its aphrodisiac, hypnotic, and analgesic actions. It is sold in native bazaars under different names and in different forms, for example, it may be smoked mixed with tobacco or taken otherwise as an adulterant in confections, coffee, or capsules. Much tolerance is not acquired and although a drug of addiction, it does not frequently lead to physical deterioration or to withdrawal symptoms.

Symptoms.

Taken in very large doses, acute poisoning with fatal results may ensue. In small doses, it produces symptoms similar to the early stages of inebriation, namely, a sense of elevation and superiority. With increased doses, drowsiness is likely to supervene, and addicts relate that during this stage erotic and other visual impressions are usually experienced. After large doses, the stage of drowsiness is accompanied by numbness and a sense of loss of power of the limbs, feeble but quiet respiration, weak and irregular pulse and faint heart action, dilatation of pupils and deep unconsciousness. Fatal cases are unusual. Acute mental disorders are much more frequent than chronic ones. People with excitable temperaments may become violent if interfered with while under the influence of the drug.

Treatment.

Hypodermic injection of strychnine, when the patient is deeply unconscious. Stimulants may be given by rectum, or by mouth if the patient is able to swallow, and artificial respiration should be employed, if indicated. The general line of treatment should follow that for other narcotics (see p. 648).

Gelsemium

(Nat. Ord., Loganiaceæ).

The poisonous alkaloids, gelsemine and gelseminine, are obtained from the dried root of Gelsemium sempervirens or yellow jasmine. The latter has the greater activity.

Symptoms.

In poisonous doses it produces impairment of sight, double vision, and sometimes total blindness, with ptosis. The pupils are dilated and usually insensible to light. Speech is impaired. The gait is staggering, due to muscular inco-ordination. The skin is warm and moist, although the extremities may be cold. There

is marked muscular relaxation of the body, but the muscles of the face and neck may, in some cases, be in a state of tetanic spasm. There is general prostration. The mind remains clear for some time, but unconsciousness, sooner or later, supervenes. Death results from asphyxia due to paralysis of the respiratory centre.

Few cases of poisoning by the drug have been recorded in this country, and those which have occurred have largely been due to errors in dispensing.

Treatment.

Empty and wash out the stomach with a dilute solution of tannic acid. Warmth. Stimulants. Artificial respiration, and oxygen inhalations, with 5 to 7 per cent. carbon dioxide, as indicated.

Laburnum

(Nat. Ord., Leguminosæ).

Toxic symptoms arise when the seeds or the bark of the tree Cytisus laburnum are eaten. This sometimes occurs in the case of children. The toxic alkaloid of the bark and seeds, cytisine, belongs to the narcotico-acrid class. Death has resulted in animals and human beings from eating the bark and seeds. Two fatal cases in children, aged three and eight years, occurred after eating seeds. The elder child was attacked by vomiting and diarrhœa, pains in the head, and complete prostration, and died within fourteen hours. The younger child vomited, complained of pain in the head, became unconscious, and had convulsions for two and a half hours before death. She died within eight hours. Cytisine was found in the stomach contents of each child.

Symptoms.

Nausea, vomiting, purging, giddiness, dilatation of pupils, tachycardia, restlessness, drowsiness, subnormal temperature, coma, with convulsive twitches, and convulsions.

Treatment.

Gastric lavage, artificial respiration, and stimulants.

Post-mortem appearances.

Evidence of gastro-intestinal irritation, and asphyxial signs.

Chemical tests (see p. 717).

Colchicum

(Nat. Ord., Liliaceæ).

The corm of the plant Colchicum autumnale contains the alkaloids colchicine and colchiceine, and medicinal preparations made therefrom are poisonous. The plant is poisonous to both cattle and human beings. As the active principle of the drug is a common constituent of proprietary preparations for gout, accidental poisonings have frequently occurred.

Symptoms.

Vomiting and diarrhœa accompanied by griping, abdominal pains, and weakness. Later, convulsions and coma just before death, following paralysis of the respiratory centre. There may be partial or complete suppression of urine.

Treatment.

Treatment consists of gastric lavage with a dilute solution of tannic acid, repeated administration of medicinal charcoal, glucose-saline intravenously, atropine, $\frac{1}{100}$ grain hypodermically, and stimulants.

Post-mortem appearances.

These are of irritant poisoning (see above). The kidneys may show acute degenerative changes.

Chemical tests (see p. 714).

Privet

(Nat. Ord., Oleaceæ).

This shrub, Ligustrum vulgare, which grows in gardens and hedges, is poisonous. The toxic principles include ligustrin and ligustron. Its berries, which in autumn are purplish-black in colour, are sometimes mistaken by children for edible fruit, with development of symptoms of poisoning from which death has ensued.

Symptoms.

Vomiting, diarrhœa, weak, thready pulse, subnormal temperature, coldness of body, and muscular twitchings or convulsions.

Treatment.

As for irritant poisoning (see p. 664).

Yew

(Nat. Ord., Coniferæ).

This shrub or tree, Taxus baccata, is poisonous, because of the presence of the toxic alkaloid taxine. The alkaloid is to be found in every part of the shrub, but chiefly in the leaves and seeds of berries. Children are liable to be attracted by the scarlet berries. The plant is poisonous to both man and animals.

Symptoms.

Vomiting, gastro-intestinal pain, diarrhœa, weak, irregular pulse, muscular weakness, collapse, delirium or convulsions, coma and death from respiratory failure.

Treatment.

As for irritant poisoning (see p. 664).

Post-mortem appearances.

Examine stomach contents for portions of leaves and seeds. The stomach and upper part of the intestinal tract will show evidence of congestion.

Conium

(Nat. Ord., Umbelliferæ).

Conium maculatum, or spotted hemlock, is a common umbelliferous plant. If the leaves are bruised between the fingers a mousy odour is perceived. The contained alkaloids, coniine and coniceine, are most abundant in the fruit and have an acrid, bitter taste. A tincture, extract, and succus are made from the plant.

A number of boys who were camping mistook hemlock for an edible herb. Almost immediately twenty-four of them became ill. Emetics were freely administered and, with the exception of a boy of nine, all recovered. The deceased was very ill from the beginning and never regained consciousness.

Symptoms.

To commence with, the symptoms are those of an irritant poison, but later they manifest themselves in the form of muscular weakness and gradually increasing paralysis, due to depression of the motor nerves, during which the breathing becomes difficult and slower. Delirium, coma, or convulsions supervene, and the patient dies of asphyxia due to paralysis of the respiratory centre.

Treatment.

Free lavage of the stomach with a dilute solution of tannic acid, artificial respiration, oxygen inhalations, with 5 to 7 per cent. carbon dioxide, and stimulants hypodermically.

Post-mortem appearances.

Are those of asphyxia. The remains of leaves or root should be looked for in the stomach.

Chemical tests (see p. 714).

Cicutoxin and Œnanthotoxin

(Nat. Ord., Umbelliferæ).

Cicuta virosa, or water hemlock, and Œnanthe crocata, or water dropwort, contain the active principles, cicutoxin and œnanthotoxin, respectively. These substances produce gastro-intestinal symptoms accompanied by others involving the central nervous system. Miller [10] records a case of poisoning, in a boy aged ten, through

eating some root of Cicuta maculata, a type of water hemlock, with recovery. His symptoms resembled strychnine poisoning.

The treatment consisted of an intravenous injection of sodium amytal which was completely successful in stopping the convulsions. Later slight tremors developed, and a further, but smaller, dose was given by the same route. The patient fell asleep and on awakening the following day was quite well.

Post-mortem appearances.

These are indicative of gastric irritation, and of asphyxia.

Sparteine

(Nat. Ord., Leguminosæ).

Broom, or Cytisus scoparius, contains a volatile liquid alkaloid, sparteine. It is similar in action to coniine, but less poisonous, and resembles the action of digitalis upon the heart. It is also diuretic in its effect. In poisonous dosage it has a markedly depressant action on the central nervous system.

Veratrine

(Nat. Ord., Liliaceæ).

Veratrine is the name given to a mixture of alkaloids obtained from some of the Veratrum or Hellebore species of plants, including Veratrum sabadilla, cevadilla, album, and viride. The alkaloids composing veratrine are veratridine, cevine, cevadine, cevadilline, and sabadine. These act upon the central nervous system and on sensory, motor, and secretory nerve endings. Their action resembles that of aconitine.

Poisoning is usually accidental.

Symptoms.

A tingling sensation in the mouth, pharynx and œsophagus, which gradually becomes general, and is replaced by a sense of numbness. Vomiting, diarrhœa, colic, signs of collapse, muscular twitchings or convulsions, and coma. Death as a rule results from failure of respiration.

Treatment.

Gastric lavage, the use of medicinal charcoal, maintenance of body warmth, administration of stimulants, and artificial respiration, if necessary. Atropine, $\frac{1}{100}$ grain, may be given hypodermically.

Chemical tests (see p. 724).

Nicotine

(Nat. Ord., Solanaceæ).

The plant, Nicotiana tabacum, contains the alkaloid, nicotine, to which its toxic effects, even by smoking, are due. This alkaloid,

a colourless, transparent, volatile liquid, on exposure to air develops an amber tint, and latterly becomes a resinous substance. It has a pungent, acrid taste, and produces a greasy looking green stain on filter paper. Cases of acute poisoning are comparatively rare, but subacute and chronic symptoms of poisoning may result from the immoderate use of tobacco.

An unusual case of absorption is recorded by Gill.[11] A convict was admitted to prison, and four hours later was found in a state of collapse and suffering from nausea, vomiting, and paralysis of both legs. The cause of his illness was regarded as due to an ounce of cut tobacco concealed in his rectum, and which he had been unable to remove. The man recovered.

Nicotine poisoning may be caused by absorption either through intact, or broken skin, by inhalation or ingestion. Poisoning by absorption through the skin has followed the use of an insecticide containing 40 per cent. of free nicotine. It has also followed the swallowing of wine containing 80 per cent. of nicotine. Cases of industrial poisoning, due to commercial preparations such as fertilisers, insecticides, and fumigants, have also occurred.

Symptoms.

When nicotine is absorbed in poisonous amount, the principal symptoms are those of successive central and peripheral stimulation. Nausea, sickness, tachycardia, cardiac irregularity, præcordial oppression, severe prostration, and cardio-vascular collapse are experienced. There may also be convulsions, delirium, irregularity of respiration, dyspnœa, and coma.

Treatment.

Gastric lavage, caffeine, and adrenalin medication, and strychnine, $\frac{1}{60}$ grain, for collapse. Artificial respiration and oxygen inhalations, with 5 to 7 per cent. carbon dioxide, may be necessary.

Post-mortem appearances.

When nicotine has been swallowed, the signs are usually those of asphyxia. The mucous membrane of the œsophagus and stomach may be congested.

Chemical tests (see p. 718).

Lobeline

(Nat. Ord., Campanulaceæ).

The plant Lobelia inflata, or Indian tobacco, has toxic properties due to its active principle lobeline. Its action is similar to nicotine. It is a respiratory stimulant, of short duration, when used clinically, due to its action through the carotid sinus. It is used to revive patients from an overdose of narcotic.

Treat as for nicotine (see above).

Chemical tests (see p. 717).

Picrotoxin

(Nat. Ord., Menispermaceæ).

Picrotoxin consists of colourless, odourless crystals extracted from the fruit of Animirta paniculata, known as Cocculus indicus. It is a powerful convulsive poison which differs from strychnine in that it acts upon the medulla, and also increases the secretion of mucous and sweat glands. The symptoms consist of gastro-intestinal irritation, prostration, perspiration, rapid and subsequently slow respirations, general clonic convulsions, twitching of the facial muscles, coma, and death from cardiac arrest or respiratory failure. It has been recommended in the treatment of barbiturate poisoning (see p. 642).

Treatment.

Gastric lavage, sodium amytal, or phenobarbitone, intravenously, chloroform, potassium bromide and chloral hydrate, and maintenance of body warmth are the principal lines of treatment.

Post-mortem appearances.

The post-mortem findings are those of gastro-intestinal irritation and general visceral congestion, including congestion of the brain.

Chemical tests (see p. 721).

Curare

(Nat. Ord., Loganiaceæ).

The species Strychnos toxifera, and certain other tropical plants, contain curare which is used as an arrow poison. Curare contains curarine. This poison obtains entry to the body by an injured surface. It is not absorbed from the intestine and must be injected. It has a selective action on the motor nerve endings in voluntary muscle which become paralysed, and thus the affected person is unable to move. The muscles of respiration become involved and asphyxia follows. A curare alkaloid is now used in anæsthesia (see p. 619).

Treatment.

Prostigmine is the physiological antidote, and its intravenous administration in quantities of 2 millilitres of a 1 in 2,000 solution has been recommended. Artificial respiration, with oxygen inhalations, and 5 to 7 per cent. carbon dioxide, should be maintained for long periods during which excretion of the drug should be expedited by all available means.

Chemical tests (see p. 714).

Quinine

(Nat. Ord., Rubiaceæ, see p. 385).

Quinine is one of the alkaloids from the bark of the genus Cinchona, chiefly Cinchona calisaya. Fatal poisoning by quinine

is rare, and when it occurs idiosyncrasy plays an important part. Terplan and Javert [12] and Vartan and Discombe [13] have recorded cases. The case described by Vartan and Discombe is that of a woman who took 76 grains of pure alkaloid for the purpose of procuring abortion. The illness commenced with pyrexia, vomiting, urticarial rash, and vaginal hæmorrhage, followed by hæmoglobinuria and progressive anæmia with a leucocytosis of 26,000. The urine was of black colour and contained a high concentration of hæmoglobin. Death took place, "as a blackwater fever," from anuria, and the microscopical pathology of the organs was identical with blackwater fever.

As a rule quinine is quickly destroyed in the body. In poisonous dosage it depresses the cardiac and respiratory centres. The fatal toxic dose varies in different persons and circumstances.

The milder toxic effects comprise partial deafness with ringing in the ears, rashes, and giddiness. It is held by some that quinine does not stimulate the normal pregnant uterus and therefore does not act as an abortifacient, but the general view is that it stimulates the tone and contractions of the uterus unless in toxic doses, when it has a depressing effect. In later stages of pregnancy, especially, quinine exerts a stimulating action on the uterine musculature.

Post-mortem appearances.

The picture is one of a varying degree of gastric irritation. In certain cases hæmoglobinuria, due to blood changes, may be present.

Chemical tests (see p. 722).

Camphor

(Nat. Ord., Lauraceæ).

Camphor is a white crystalline substance obtained from Cinnamomum camphora. It may also be prepared synthetically from turpentine.

Camphor has given rise to poisoning in children who have eaten it. The Pharmacopœial preparations, the liniments and spirit, especially, have been drunk by mistake, sometimes with fatal results.

Symptoms.

Vomiting, convulsions, stertorous breathing, cyanosis of the lips, and coma. In certain cases, especially in children, there may be hyperpyrexia.

Treatment.

Gastric lavage, maintenance of warmth, a purgative dose of magnesium sulphate, well diluted, stimulants such as digitalin, strychnine, or caffeine sodium benzoate, and artificial respiration.

Post-mortem appearances.

The characteristic odour will be perceived, and there is likely to be some inflammation or injection of the mucous membrane of the stomach.

Chemical tests (see p. 711).

Turpentine

(Nat. Ord., Coniferæ).

Turpentine is obtained by distillation from the oleo-resin of Pinus sylvestris and other species of Pinus.

Spirits or oil of turpentine, being used for a variety of domestic purposes, occasionally gives rise to accidental poisoning in both children and adults. Being volatile, its vapour produces certain pathological effects upon those who are exposed to it for lengthened periods. The cause of death is asphyxia.

Symptoms.

A burning sensation in the mouth and stomach, vomiting, diarrhœa, giddiness, cerebral excitement, unconsciousness, strangury, and an odour of violets from the urine, in which blood is frequently present. Coma usually precedes death.

Treatment.

Free gastric lavage with tepid water, until the washings are free from odour. A purgative dose of magnesium sulphate, demulcent drinks, restoration of body warmth, and the application of warm fomentations over the loins. Morphia, $\frac{1}{4}$ grain hypodermically, may be administered to relieve pain.

Post-mortem appearances.

Intense congestion of the lungs and large blood-vessels. The gastric mucosa shows deeply injected patches, the meninges are frequently injected and the brain is œdematous. Degenerative changes may be found in the kidneys.

Fatal dose.

Half an ounce has killed a child; 1 teaspoonful has caused the death of an infant; 4 and 6 ounces have killed adults.

Chemical tests (see p. 724).

Eucalyptus

(Nat. Ord., Myrtaceæ).

Eucalyptus oil is obtained from the leaves of numerous species of Eucalyptus. The popular use of this drug as a remedy for nasal catarrh and its presence in households have given rise to cases of poisoning when swallowed accidentally.

Symptoms.

The toxic effects are chiefly those of gastro-intestinal irritation, accompanied by cerebral excitement, convulsions, and coma. The typical odour may be perceived from the urine. Death may occur from paralysis of the respiratory centre.

Treatment.

The treatment consists of gastric lavage and stimulation.

Post-mortem appearances.

The post-mortem findings are those of irritation of the respiratory and gastro-intestinal systems.

Chemical tests (see p. 715).

Ergot

(Micro Fungi—Pyrenomycetes—Hypocraceæ).

Ergot (Secale cornutum) is a fungus (Claviceps purpurea) which affects the ripe grains of certain cereals, especially rye, turns them black, and renders them poisonous (see p. 689). It contains at least 0·05 per cent. of ergot alkaloids calculated as ergotoxine.

The action of ergot is that of a true ecbolic (see p. 385) and emmenagogue. Among the active constituents of ergot are ergotinine, ergotoxine, ergotamine, ergotaminine and ergometrine; in addition, histamine and tyramine are present. Ergotoxine promotes contraction of the human uterus at the end of pregnancy, but has little effect during the course of gestation. By contracting the arterioles, it eventually causes gangrene. It is the most important factor in ergot poisoning. Ergotamine has similar properties. Ergometrine is the principle chiefly responsible for the action of ergot on the uterus. Within a few minutes of its administration, it causes prolonged muscular contraction of the uterine musele which is specially sensitive following delivery. It differs from the other alkaloids in ergot on account of the fact that it is less active in the production of gangrene.

McKay [14] reports the case of a woman, aged thirty, who, thinking herself pregnant, obtained a 12-ounce bottle which she was told contained ergot, for the purpose of procuring abortion. She finished the contents of the bottle in a week, but without the desired effect. She got a stronger mixture, finishing it in seven days. Before it was finished her arms began to ache, her skin to itch, and her fingers to swell. Her left index finger became cold and showed cyanosis at the tip. The remaining fingers of this hand and also those of the other hand were similarly affected, and later several of them became gangrenous as far as the distal joints. She failed to abort.

Few fatal cases from acute poisoning have been recorded, and then only when large quantities of the drug have been taken. Von Storch [15] has reviewed a number of cases in which untoward

sequelæ have developed following the prolonged use of ergotamine tartrate on therapeutic grounds. In forty-two cases the sequelæ were serious, and were similar to the milder form of convulsive ergotism. Overdosage appeared to be responsible in most instances.

Symptoms of acute poisoning.

The principal symptoms are pain in the stomach, nausea, vomiting, with or without hæmorrhage, a weak, rapid pulse, a feeling of oppression in the chest, subnormal temperature, coldness of the body, muscular cramps, with impairment of sensation, gangrene of the toes or fingers, convulsive movements, stupor, delirium, convulsions, and coma.

Symptoms of chronic poisoning.

These are indicative of gastro-intestinal irritation, of nervous exhaustion or excitement, sometimes amounting to mania, and dry gangrene of the fingers and toes.

Treatment.

Free gastric lavage with diluted tannic acid, a purgative dose of magnesium sulphate or castor oil, stimulants such as brandy or ½-drachm doses of aromatic spirit of ammonia diluted with water, inhalations of amyl nitrite, and restoration of body heat. Intravenous dextrose has been recommended on account of hypoglycæmia often present in ergotism.

Post-mortem appearances.

The internal signs are extravasations of blood in the stomach, liver, kidneys, and abdominal cavity, together with evidence of gangrene of the fingers and toes.

Chemical tests (see p. 714).

Savin

(Nat. Ord., Coniferæ).

Oil of savin is extracted from the young shoots of Juniperus sabinæ. In large doses, it sometimes produces abortion, accompanied by serious toxic manifestations, principally gastro-intestinal in character (see p. 384). Hæmaturia may be present.

Post-mortem appearances.

These are chiefly inflammation of the gastro-intestinal tract and irritation of the kidneys.

Pennyroyal

(Nat. Ord., Labiatæ).

The plant, Mentha pulegium, contains a volatile oil of pulegone which has toxic properties, and emits a strong mint odour. Several cases of its toxic effects have been recorded. It is mildly irritant to the urinary system and may cause reflex uterine contractions (see p. 384.)

Apiol

(Nat. Ord., Umbelliferæ).

Apiol is the active principle of the fruit of Carum petroselinum, or common parsley plant, and is not infrequently found as an ingredient of abortifacient preparations. It is obtained by extracting the bruised fresh or dried fruits of the plant with alcohol and distilling the solvent. Commercial apiol is a green, non-viscous, oily liquid with a peculiar odour and disagreeable pungent taste.

Apiol is usually made up in capsules or perles for internal administration, but in some preparations it is found in conjunction with other abortifacient drugs, for example, in the preparation, ergoapiol, which contains active principles of ergot and apiol, in capsule form, and is used for amenorrhœa, dysmenorrhœa, and kindred ailments. Polyneuritis, nephrosis, uræmia or, rarely, encephalopathy may follow its use due to the action of triorthocresyl phosphoric acid which it contains in an amount varying from 28 to 50 per cent. Lowenberg [16] has described severe cortical damage of the brain as the result of its use.

VEGETABLE IRRITANTS

Castor-oil seeds

(Nat. Ord., Euphorbiaceæ).

The raw seeds of the plant, Ricinus communis, produce severe toxic effects when eaten. Even a single seed may produce alarming symptoms. The toxic principle is ricin, which may be destroyed by boiling the seeds. Oleum ricini does not contain this poisonous protein. Poisoning by seeds is rare in this country. The symptoms are those of gastric irritation and prostration. Death has been reported.

Wrightson [17] reports that he has seen boys and girls, employed in shelling castor-oil seeds in a factory, who have eaten five or six seeds at a time with no worse effects than those following a dose of castor oil. He does not, however, mention whether the seeds had been boiled previously.

Croton oil

(Nat. Ord., Euphorbiaceæ).

Croton oil is obtained from the seeds of Croton tiglium. It has a bitter taste, an unpleasant odour, and is yellowish and viscid in appearance. The active principle is crotin which possesses both irritant and vesicant properties.

In the following case, the croton oil was given by a nurse in error, the amount administered being 1 drachm. Twenty minutes after, the patient became unconscious, was pale and sweating, with a weak thready and intermittent pulse. Large doses of olive oil, mixed with white of egg in a pint of water, were given and the

stomaeh was lavaged. Some vomiting followed. Ten minutes later violent diarrhœa set in. An hour after the oil was given, the patient was conscious, but collapsed, and there was continuing violent diarrhœa with blood-stained stools, aecompanied by severe pain in the abdomen. By the following morning the condition had improved eonsiderably.

Santonin

(Nat. Ord., Compositæ).

Santonin, a white powder which assumes a yellowish colour on exposure, is a glycoside, obtained from Artemisia maritima, and has a popular reputation as an aphrodisiac. It is used as an anthelmintic. Toxic symptoms and fatal poisoning have occurred.

Symptoms.

Giddiness, nausea, vomiting and diarrhœa, pain in the stomach, trembling of the limbs, frequency of mieturition, with tenesmus, a burning sensation at the urinary meatus, saffron-yellow-coloured urine when acid, and red when alkaline, with perhaps hæmoglobinuria, and disturbanee of vision. Yellow vision or xanthopsia, sometimes preceded by violet vision, may develop. In fatal cases, coma and convulsions preeede death. Chronie poisoning from santonin is observed in absinthe drinkers, the principal effects produced being degenerative ehanges in the central nervous system which are not infrequently aecompanied by psyehosis.

Treatment.

Prompt evacuation of the stomaeh contents and gastric lavage. A purgative dose of calomel. Demulcent drinks which do not contain oils or fats. Chloral hydrate to combat eonvulsions.

Post-mortem appearances.

Nothing charaeteristic is found either in the stomaeh or body generally.

Fatal dose.

About 2 grains of santonin killed a boy of five and a half years in fifteen hours, but a man who took 1 ounee, in mistake for Epsom salts, recovered.

Chemical tests (see p. 722).

Colocynth

(Nat. Ord., Cucurbitaceæ).

Colocynth is obtained from the dried pulp of the fruit, Citrullus colocynthis, or bitter apple. It is a drastic purgative, like jalap, podophyllin, and scammony. It is partly excreted by the kidneys. In overdose, intestinal irritation and exeessive purgation result. Colocynth is oceasionally taken in an attempt to produce abortion.

Aloes

(Nat. Ord., Liliaceæ).

Aloes contains the toxic principle aloin and is obtained from Aloe socrotina, A. barbadensis, and A. ferox. It is purgative in action, and in excess will produce excessive diarrhœa and intestinal irritation.

Podophyllum

(Nat. Ord., Berberidaceæ).

Podophyllin is obtained from Podophyllum peltatum, popularly called American Mandrake or May Apple. Its principal constituent is podophyllotoxin, the effects of which are similar to aloin.

Bryony

(Nat. Ord., Cucurbitaceæ).

Bryonin, the toxic principle of Bryonia dioica or common White Bryony, produces irritant symptoms.

Crocus

(Nat. Ord., Iridaceæ).

The toxic principle of Crocus sativus, or Saffron crocus, is crocin. It is responsible for irritant symptoms.

Nutmeg

(Nat. Ord., Myristicaceæ).

Nutmegs are the dried kernels of the seeds of Nux moschata or Myristica fragrans. From the relative frequency with which this substance is taken, there seems to be a popular belief that nutmeg contains abortifacient properties. The nut is commonly ground or grated into coarse powder, mixed with water, and swallowed.

The following case, for the details of which we are indebted to Dr Ernest S. Wyder, is illustrative of the effects of nutmeg poisoning. A married woman, of thirty-two, eight months pregnant, while preparing lunch consumed, in small portions and over a period of an hour and a half, a whole nutmeg due to an unusual craving. She enjoyed a hearty meal and motored to a town on shopping errands. About an hour later she suddenly felt ill, but was able to walk to her car, a distance of about a quarter of a mile. She drove home, was able to proceed upstairs to her bedroom, but required assistance into bed. Her pulse-rate was 160, breathing became rapid, and she experienced a feeling of impending death. The pulse-beat became markedly fainter, but soon became more perceptible, and she felt rather better. She experienced attacks of recovery and relapse for one and a half hours. During the relapses, she felt "as if I was drowning in inky waters and went down and down until the waters were closing over my head." This was followed by a period of unconsciousness and then "I rose to

the surface again." These attacks were replaced by what appeared to be normal sleep. The pulse-rate was still 160 and the face was flushed. She had a normal night. The following morning the pulse-rate was 135. About midday, uterine contractions commenced and lasted continuously for four days, but there was no evidence of progress in labour and the contractions gradually subsided. On the sixth day the pulse-rate fell to 90. Five weeks after her illness she was delivered of a healthy 10-pound child. It was estimated that the patient had swallowed 125 grammes of nutmeg which possibly contained about 20 minims of the oil.

References

1. Brit. Med. Jour., Vol. II, 611, 1947, Hamilton and Sclare.
2. Jour. Amer. Med. Ass., Vol. CI, 446, 1933, Comroe.
3. Brit. Med. Jour., Vol. II, 664, 1940, Carter.
4. East African Jour., XXI, 355, 1944, Farnwrith, Anderson, Henderson, Begg, and McNaughton.
5. Jour. Amer. Med. Ass., Vol. XCVIII, 1133, 1932, Haggard and Greenberg.
6. Ibid., Vol. C, 548, 1933, Kempf, McCallum, and Zerfas.
7. Ibid., Vol. CI, 102, 1933, Stalberg and Davidson.
8. Ibid., Vol. XCIX, 1862, 1932, Wheelock.
9. Brit. Med. Jour., Vol. I, 896, 1938, Allen.
10. Jour. Amer. Med. Ass., Vol. CI, 852, 1933, Miller.
11. Brit. Med. Jour., Vol. I, 1544, 1901, Gill.
12. Jour. Amer. Med. Ass., Vol. CVI, 529, 1936, Terplan and Javert.
13. Brit. Med. Jour., Vol. I, 525, 1940, Vartan and Discombe.
14. Ibid., Vol. II, 365, 1906, McKay.
15. Jour. Amer. Med. Ass., Vol. CXI, 293, 1938, Von Storch.
16. Ibid., Vol. CX, 573, 1938, Lowenberg.
17. Brit. Med. Jour., Vol. I, 1312, 1905, Wrightson.

CHAPTER XXIV

ADDITIONAL POISONS

Cinchophen.

This substance, which is phenylquinolene carboxylic acid, is known by a variety of proprietary names, such as quinophan, agotan, phenoquin, tophosan, and atophan, among others. A large number of fatal cases of poisoning have been reported, together with many cases in which its toxic effects have been shown. Its use is confined to the prevention and treatment of gout, lumbago, sciatica, and neuralgia, since it aids the excretion of uric acid in urine. This drug exercises a poisonous action upon the cells of the liver in certain cases and, in fatal cases, acute fatty degeneration has been found. Unknown factors, apparently independent of quantity taken, appear to play an important part in predisposition or idiosyncrasy. The manifestations of toxic symptoms are numerous and include cutaneous rashes, vasomotor disturbances, such as tachycardia, palpitation, fall of blood-pressure, cyanosis, and gastro-intestinal symptoms. Liver involvement is usually accompanied by jaundice. Fraser [1] records a fatal case of subacute yellow atrophy of the liver after ingestion of $37\frac{1}{2}$ grains of cinchophen, at the rate of one tablet in five successive days, and the appearance of jaundice within five days of the commencement of treatment.

The use of the drug may be attended by a reddish coloration of urine.

Treatment.

The drug should be stopped and attention directed to the system involved. In more severe cases, glucose should be given generously either by mouth or intravenously. The administration of calcium gluconate, intravenously, has been recommended.

Cantharides.

The active principle of the dried beetle, Cantharis (Lytta) vesicatoria or "Spanish Fly," found in Southern Europe, is cantharidin. Cantharides powder contains not less than 0·6 per cent. of cantharidin, which, when absorbed, is chiefly eliminated by the kidneys and intestinal tract. Popularly it is believed to have a marked aphrodisiac action in both sexes, and it is also believed to have an abortifacient action. It has been used criminally for both purposes. Among the medicinal preparations are: acetum cantharidini and tinctura cantharidis, and liquor epispasticus. Besides these, there are several proprietary blistering preparations.

It is usually administered for abortifacient purposes in the form of powder, but other preparations have been given.

Symptoms.

When cantharides is applied to the skin an inflammatory reaction results in a few hours and later vesicles form. When swallowed, there is burning pain in the throat and stomach, subsequently, difficulty in swallowing, nausea, abdominal pain, and vomiting, the vomited matter containing blood, intense thirst, and possibly diarrhœa, with perhaps blood and mucus. Pain on micturition, with great tenesmus and the passage of blood-stained urine, is a prominent symptom. In the male, priapism may occur, and in the female there may be engorgement of the vulva. In fatal cases, coma with convulsions usually precedes death in from sixteen to twenty-four hours.

FIG. 230
Cantharis (Lytta) vesicatoria, or "Spanish Fly."

Treatment.

Free gastric lavage with tepid water. After vomiting has ceased, the excretion of urine should be aided by the use of warm demulcent drinks, and other measures, and magnesium sulphate, but not castor oil, should be given. Morphia may be used to control pain.

Post-mortem appearances.

Gastro-intestinal inflammation, due to the vesicating action of the cantharidin. This is chiefly marked on the mucous membrane of the mouth, throat, stomach, and small intestine. The kidneys, ureters, bladder, and urethra show signs of congestion and inflammation, macroscopically and microscopically, and blood-stained urine may be found in the bladder. The pelvic viscera are usually engorged. In view of the fact that the powder may have been administered, the stomach and intestinal tract should be carefully examined for the remains of the shining elytra or wing-cases of the insect.

Fatal dose.

Approximately 60 grains is regarded as a fatal dose, but a fatal result may follow a smaller dose. Twenty-five grains of the powder and 1 ounce of the tincture have produced death. Recovery, however, has followed larger doses.

Chemical tests (see p. 611).

Metacetaldehyde.

Meta Fuel is the trade name for the product made from metacetaldehyde. It is used as a methylated spirit substitute, and is in the form of white tablets which children may swallow by mistake for sweets. Fatal poisoning has occurred from both accident and suicide. The substance contains about six molecules of acetaldehyde. The ingestion of six tablets has caused death in adults. The symptoms of poisoning are those of acute gastro-intestinal irritation, stupor, coma, tonic then clonic muscular contractions and convulsions. Death may be delayed for several days.

Treatment.

Free gastric lavage with an alkaline solution, saline purgation, and artificial respiration and inhalations of oxygen, with or without 5 to 7 per cent. carbon dixoide. Attention should also be directed to the treatment of symptoms.

Post-mortem appearances.

The post-mortem findings consist of gastro-intestinal inflammation, congestion of the brain, and cloudy swelling of the liver and kidneys.

Methyl salicylate or oil of wintergreen.

This fluid may be swallowed accidentally or suicidally. The symptoms are those of gastro-enteritis. Post-mortem findings include evidence of gastro-enteritis, submucous hæmorrhages in the pelves of the kidneys, petechial hæmorrhages in the renal cortex, and visceral congestion.

Townsend [2] reports a fatal case of poisoning of a man who drank about four ounces of methyl salicylate liniment in a gulp mistaking it for alcohol. He vomited and then collapsed. Two and a half hours later he was fully conscious although much distressed with vomiting. Gastric lavage and copious fluids, including sodium bicarbonate, milk, and water were the principal lines of treatment used. Next morning his condition had deteriorated and fine crepitations were present at the bases of the lungs. He became delirious and death supervened twenty-four hours after drinking the liniment.

Ethylene glycol.

This is a colourless, odourless fluid which is used as a solvent for some dyes, as an anti-freeze mixture for motor car radiators and aeroplanes, and for other purposes. Poisoning has occurred from accidental drinking of a comparatively large quantity. One hundred millilitres has been suggested as a fatal dose. Within the body it is oxidised to oxalic acid, and then to glycollic acid. In acute poisoning, the toxic effects are probably due to oxalic acid poisoning

and depression of the central nervous system. The symptoms are vomiting, cyanosis, prostration, convulsions, coma, and respiratory failure. Severe kidney damage may occur, with extensive injury to the epithelium of the convoluted tubules, and lead to urinary obstruction and uræmia. Prus and Custer [3] have reported their experience of ten cases of fatal poisoning among soldiers who drank anti-freeze solution of the ethylene glycol type (Prestone) as an alcoholic beverage. Apart from one case, death occurred in from twenty-two to forty-four hours. In two cases there was incontinence of urine and fæces, in four, severe convulsive seizures, and in three, strabismus. In four cases the spinal fluid was blood tinged. Autopsy showed in all cases extensive oxalate crystallosis of the renal tubules, the kidneys were swollen, and the liver was enlarged. In all cases there was a moderate or marked pulmonary œdema. The authors have also seen a further eight fatal cases with similar findings. When death occurred within twelve hours, the renal damage was minimal, but if the patient lived for five days after ingestion, significant renal changes were present.

Tricresyl phosphate.

This is used in industry as a plasticiser for pyroxylin lacquers, and in the recovery of phenol residues from gas-plant effluents. It is a colourless, odourless, oily-looking liquid of low volatility. Cases of industrial poisoning have occurred. It is a lethal poison if swallowed in sufficient quantity, the fatal dose being about 1 gramme per kilogram of body-weight. The first symptoms are gastro-intestinal disturbances which are followed by a symptomless period. Some ten to fourteen days after ingestion, further symptoms develop, namely, general weakness, tingling, burning, and tenseness of hands and feet. Paralysis of these extremities next supervenes, termed "Jake paralysis," which is irreparable and can only be treated symptomatically or orthopædically. Severe cases are bed-ridden. In order of frequency, the muscles affected are those supplied by the sciatic nerve, the intrinsic muscles of the hands, and the radial extensors. In 1930-31, fifteen thousand persons were affected with "ginger-paralysis" in California, due to drinking ginger liqueur containing this compound. Ten persons died (see p. 691). The substance has been found in cotton-seed oil.[4] Triphenyl phosphate apparently has similar toxic properties (see p. 29).

Sulphonamides.

The sulphonamides are used in the treatment of streptococcal infections, especially of hæmolytic type, and in a wide variety of conditions due to other infective organisms. The compounds most commonly used are sulphanilamide, sulphapyridine, the latter in pneumococcal infections, and sulphathiazole. The newer drugs of this group are sulphadiazine, sulphamethiazole, sulphacetamide, or albucid, sulphamerazine, and solucin. Acetyl and succinyl

derivatives of these compounds are also employed therapeutically. Sulphagnanidine has proved of great value in cases of acute bacillary dysentery and infective diarrhœa. The principal effect of the sulphonamides lies in their effectiveness in inhibiting the reproduction of bacteria by impeding their metabolism.

The toxic effects assume a wide variety, and include gastro-intestinal irritation, giddiness, mental depression, and disturbances of vision. Agranulocytosis is a not infrequent, dangerous, and sometimes fatal complication in the over-prolonged use of these drugs. Hepatic necrosis has occurred in a number of fatal cases. Many cases have developed methæmoglobinæmia and sulphæmoglobinæmia. These conditions cause marked cyanosis, often alarming in character, and the removal of the former takes a much longer time than the removal of the latter, which disappears in about twenty-four hours, following withdrawal of the drug.

The manifestations of cyanosis due to methæmoglobin can be obviated by the administration of methylene blue, 1 to 2 milligram kilogram, orally, every four hours, if commenced at the same time as the sulphonamide therapy. It can also be eliminated by the same treatment or by a single dose, of the same amount, given intravenously.

Jackson and Tighe [5] suggest that the best treatment for agranulocytosis is intramuscular injection of 10 millilitres of pentnucleotide, four times daily, until the white cell count has risen, and then the same dosage, once or twice daily, until the count has returned to normal. Water diuresis for the elimination of the drug is recommended. Blood transfusion is also beneficial in some cases. Cline [6] records an unusual fatal case of sulphanilamide poisoning which produced acute yellow atrophy of the liver. The young man was eighteen years of age, and there was no history or indication that any other drug or possibly toxic substance, other than sulphanilamide, had been taken by the patient. Sulphapyridine, sulphathiazole, and sulphadiazine are more prone to cause anuria than sulphanilamide. Anuria may result from deposition of crystals of the acetylated derivative in the renal tubules or pelvis of the kidneys. Hæmaturia is not uncommon. With sulphadiazine, especially, the risk can be minimised by the administration of sodium bicarbonate until the reaction of the urine becomes alkaline, and by maintaining the daily urinary output at a minimum of 3 pints. At least 4 to 8 pints of fluid intake should be enjoined. Massage over the kidneys and ureters will aid the displacement of the obstructing crystals. Ureteric catheterisation ensures quick relief in more difficult cases. Toxic manifestations are less frequent with sulphadiazine than with sulphapyridine and sulphathiazole. The following precautions should be adopted as routine with regard to sulphonamide administration. A hæmoglobin estimation and a white cell count should be made before a patient, who is seriously ill, is given the drug ; the fluid intake must be maintained at a high level ; the condition of the blood should be carefully watched ; the drug should be discontinued on the approach of serious toxic symptoms.

Dichloro-diphenyl-trichlorethane or D.D.T.

This substance is used as an insecticide and the estimated poisonous dose for man and animals is in the region of between 150 and 600 milligrams per kilogram of body-weight. Among a number of recorded cases of poisoning, the following are worthy of note. A man aged forty-six was given a 5 per cent. solution of D.D.T. in kerosene for use as an insecticide. Some of the fluid escaped from the bottle into his pocket and contaminated some chewing tobacco. About two hours after chewing some of the tobacco the man became ill and suffered from nausea, a feeling of tightness, stiffness, and pain affecting the jaws, and soreness of the throat. These symptoms increased in severity for a time, but apart from the sore throat, they had passed off by the following day.[7]

A labourer swallowed, in its concentrated form and with fatal result, 6 ounces of D.D.T. emulsion intended to be diluted four hundred times. The emulsion had been made for spraying mushrooms and contained 20 per cent. D.D.T. dissolved in methylcyclohexanone, 40 per cent. The post-mortem dissection showed pulmonary and gastro-intestinal congestion. The amounts found in the stomach were :—D.D.T. = 34 grammes and methylcyclohexanone = 2·4 ounces. The concentration of D.D.T. was in the region of 500 milligrams per kilogram of body-weight. Death may have been due to D.D.T., or to methylcyclohexanone, or to both acting in combination. Methylcyclohexanone is not a recognised industrial poison and no cases of poisoning by it have been described. The balance of evidence points to death being primarily due to D.D.T. though there can be little doubt that the solvent assisted the toxic action of the compound.[8]

Hill and Robinson [9] state that the effects of D.D.T. poisoning as observed in the case of a child and in experimental work on baboons consist of inco-ordination and paralysis of the legs immediately preceding death and pulmonary œdema on post-mortem examination.

Amphetamine sulphate or benzedrine sulphate.

This substance is a white, odourless powder which is prepared by neutralising benzedrine in alcoholic solution with sulphuric acid. It has a marked stimulating effect on the central nervous system, manifested by a diminution in the sensation of fatigue, an increase in mental activity, and euphoria, and thus its clinical use is both wide and varied. In toxic dosage collapse and even syncope may result. A case of accidental poisoning has been reported by Hertzog, Karlstrom, and Bechtel.[10] A child aged twelve months took 40 milligrammes, in addition to a number of ferrous sulphate tablets, 3 grains each (see p. 566). The baby became drowsy, cyanotic, and semi-comatose. It vomited brownish material. Gastric lavage was undertaken, but during the process there was failure of breathing which, however, was established by artificial respiration. Death

occurred nineteen and a quarter hours after admission to hospital. The post-mortem findings included reddish blotches on the skin, generalised œdema of the lungs, and pulmonary petechial hæmorrhages. The adrenals showed hæmorrhagic discoloration.

Beryllium.

Beryllium copper alloy is extensively employed in the manufacture of precision and other instruments. The fumes and the dust in the processing of the ore have occasioned industrial poisoning and Ordstrand, Hughes, De Nardie, and Carmody [11] have written an authoritative paper on the subject. They state that during the past four years, one hundred and seventy cases of poisoning have occurred among the workers in three plants producing beryllium and its alloys. The toxic manifestations recorded include dermatitis, chronic skin ulcer, and inflammatory changes in the respiratory tract. The most severe respiratory manifestation was pneumonitis, responsible for five deaths in the recorded cases. Most of the patients had contact with beryllium sulphate, fluoride, or oxyfluoride. The symptoms of chemical pneumonitis consisted of dyspnœa, substernal pain, cyanosis, and cough with blood-stained sputum. X-ray examination of the lungs some two to three weeks after the onset of symptoms showed "diffuse haziness of both lungs, soft, irregular intervals of infiltration with peribronchial maskings, and the appearance of large or conglomerate nodules in the lung fields." The lung fields cleared after one to four months. Autopsies in five cases showed atypical pneumonitis. A case of death stated to have been due to beryllium poisoning has recently been reported in Britain.[12]

Radio-active substances.[13]

In the course of industry, exposure of the workers to radio-active substances is associated with the contact of radio-active ores and the products of these ores. It may also occur in the course of preparing or applying self-luminous compound in connection with the hands and figures on clocks and watches or to various parts of other apparatus. In the manufacture of self-luminous paint, or compound, a small quantity of a radium salt is used together with zinc sulphide crystals. Industrial exposure to radio-active substances has resulted in necrosis of bone, malignant growth of bone, pulmonary carcinoma, and various primary blood diseases.

References

1. Brit. Med. Jour., Vol. II, 1195, 1934, Fraser.
2. Ibid., Vol. I, 382, 1943, Townsend.
3. Med. Leg. Rev., Parts 3 and 4, Vol. XIV, 140, 1946, Prus and Custer.
4. Lancet, Vol. I, 207, 1946, Hotston.
5. New Eng. Jour., Vol. CCXX, 729, 1939, Jackson and Tighe.

6. Jour. Amer. Med. Ass., Vol. CXI, 2384, 1938, Cline.
7. Med. Leg. Rev., Parts 3 and 4, Vol. XIV, 141, 1946.
8. Lancet, Vol. II, 235, 1946, Biden-Steele and Stuckey.
9. Brit. Med. Jour., Vol. II, 845, 1945, Hill and Robinson.
10. Jour. Amer. Med. Ass., Vol. CXXI, 256, 1943, Hertzog, Karlstrom, and Bechtel.
11. Med. Leg. Rev., Parts 1 and 2, Vol. XIV, 74, 1946, Ordstrand, Hughes, De Nardie, and Carmody.
12. "Industrial Toxicology," Hamilton and Johnstone, 1945, Oxford Medieal Publications.
13. Ibid.

CHAPTER XXV

THE EFFECTS OF THE ATOMIC BOMB

THE principal effects comprise blast effects, thermal effects, and the effects of radiation. The blast effects are similar to those following the detonation of high explosives (see p. 321), while the thermal effects fall within the category of burning (see p. 213), which is caused primarily by the infra-red and ultra-red radiation produced during fission. The process of atomic fission gives rise to radio-activity by releasing penetrating particles, neutrons, and radiation, which are directly harmful to the human subject, by fission products which are radio-active, and by materials which, penetrated by the released neutrons, become radio-active. The term radiation includes gamma radiation, beta radiation, and alpha radiation, together with the effects of neutrons. Gamma rays and neutrons have the power of penetrating the unshielded body, and give rise to both external and internal effects, while alpha rays and beta rays are harmful when the source of such rays is within the body. It should be noted, however, that beta rays in large external doses are capable of producing severe skin lesions over unshielded parts of the body, despite the fact that no direct internal radiation effects may be noted in the body.

Atomic radiation produces its effects on tissue cells by ionisation, and speaking broadly, it may be said that the effects of all these radiations may cause tissue destruction in whatever tissues they pass through. Immature cells, or cells in an active state of division, for example, hæmatopoietic and reproductive tissue, are the most sensitive to the effects of radiation. Subjects exposed to severe penetrating radiation, if protected from flashburn and from direct injury were not found to show any immediate illness, but after an interval of about twenty-four hours, characteristic symptoms of nausea, vomiting, and fever developed, although death rarely ensued in less than a week. After the onset of the illness, if the patient survived until the second week, diarrhœa, accompanied by hæmorrhage from the intestinal tract, and general malaise became marked. Later, in severe cases, the patients suffered from blood formation deficiency. Radiation exercises its effects on the primitive cells in the bone marrow and, in severe cases, it may virtually destroy the bone marrow, and thus deficiency in red and white cells, together with blood-platelets, heralds a grave condition. With reduction of blood cells progressive anæmia becomes manifest, as the white cell count declines, lowered resistance with infection of the mouth, and gangrene of the lips, tongue, and occasionally the throat supervenes, and with the reduction in platelet formation, hæmorrhages into

the skin, the retina, the intestines and kidneys constitute an additional advent. Death brought this picture to a close probably in from one to three weeks when it had reached its peak after the explosions of the atomic bombs at Hiroshima and Nagasaki. It is thought that gamma rays ultimately caused the death of nearly everyone who was fully exposed to them up to a distance of half a mile from the centre of the damage.

Regarding the effects of radiation on human reproduction, the most striking are that, at a distance up to a thousand yards from the centre of the damage, all surviving pregnant women, with pregnancies from two months onwards, had miscarriages. At a distance up to one and a quarter miles from the centre of the damage, surviving pregnant women either had a miscarriage, or gave birth to a premature child which survived its birth only for a very brief period. Even beyond this range, up to nearly two miles, only about one-third of pregnant women gave birth to what appeared to be normal children. Two months after the explosion, miscarriages, abortions, and premature births were nearly five times as frequent as prior to the liberation of atomic energy, and formed more than a quarter of all deliveries throughout Hiroshima. Investigation has also shown that a high proportion of the male population in Hiroshima, who were in the region of perhaps three-quarters of a mile from the centre of the damage, have reduced power of reproduction. It has been found that recovery from this condition occurs (see p. 684).

References

"The Effects of the Atomic Bombs at Hiroshima and Nagasaki," Report of the British Mission to Japan, H.M. Stationery Office, London, 1946.

"The Atomic Bomb," Industrial Medicine, Vol. XVI, No. 6, June 1947, S. L. Warren, F. A. Bryan, and R. J. Buettner.

"With the Atomic Bomb Casualty Commission in Japan," Published in the Bulletin of Atomic Scientists, June 1947, A. M. Brues.

CHAPTER XXVI

FOOD POISONING

FOOD poisoning in its epidemic form is properly the concern of the Medical Officer of Health, though in special circumstances medico-legal issues may be involved, particularly in the sporadic case. In its widest sense the term implies poisoning by articles of food, due either to contained toxic principles, metallic or otherwise, or to pathogenic micro-organisms: the more usual implication is that of illness due to invasion of the body by members of the Salmonella group of bacteria or their toxins. More recently it has been shown that certain other organisms including staphylococci, the dysentery bacilli, and Clostridium Welchii may be concerned in outbreaks. The severe and usually fatal illness due to the sporing and anaerobic B. botulinus is frequently regarded separately, though the mechanism of transmission of botulism is, for practical purposes, the same as that of the other bacterial forms.

Bearing in mind the wide sense of our title it will for the present purpose be most convenient to group the types of food poisoning as Food Idiosyncrasy, Vegetable Poisons including Fungi, Animal Poisons including Shellfish, Chemical Poisons, and Bacterial Food Poisoning including Botulism.

Food idiosyncrasy.

This may be defined as supersensitivity to certain articles of diet, generally protein in nature, as a result of which their consumption is followed by illness, the common symptoms of which are nausea, vomiting, diarrhœa, and the presence of an exanthem, usually urticarial in character. Victims of this condition rarely allege food poisoning.

Vegetable poisons.

These are of wide occurrence in nature though happily restricted in Britain. The leaves of rhubarb may occasion a fatal illness from their oxalic acid content (see p. 530). Certain of the vetches, notably Lathyrus sativus, appear to play a part in the causation of a malady attended by paraplegia and muscular weakness and known as lathyrism, though it is now suggested that the condition is to some extent a deficiency disease. The berries and leaves of Atropa belladonna (see p. 664) and the pods of Laburnum vulgare (see p. 650), all of which may be eaten by children, are further examples of non-fungus vegetable poisons. In one case of interest recorded

some years ago belladonna leaves became accidentally admixed with sage employed to stuff a roast and produced symptoms of atropine poisoning within ten minutes.

The toxic principle solanine is present in concentrated form in prematurely gathered and sprouting potatoes. A recent outbreak of solanine poisoning at Cyprus involving fifty persons proved to be due to the eating of potato shoots which, it transpired, contained five times more solanine than did the tubers (see p. 652).

The so-called "mushroom poisoning" results from the ingestion of certain toxic fungi which resemble Agaricus campestris, the edible mushroom. The species mainly involved in this country are Amanita phalloides, in which case the incubation period is some ten hours, and A. muscaria, with a latent period of two to six hours. The general symptoms include gastro-enteritis, prostration, delirium, and finally coma. There may in addition be nephritis or even anuria. Poisoning by A. phalloides, of which some three hundred fatal cases are on record, may be attended by a mortality of 50 per cent. Death from A. muscaria is, however, rare. The alkaloid muscarine isolated from A. muscaria and the amanita toxin of A. phalloides are probably not the sole toxic factors concerned. Muscarine, which is antagonistic to atropine, appears to stimulate the post-ganglionic parasympathetic receptors, resulting in diminution in the rate and force of the heart and contraction of the pupils, together with disturbance of accommodation.

The post-mortem appearances in fungus poisoning are marked post-mortem lividity, engorgement and inflammation of the gastro-intestinal mucosa, and hyperæmia of the meninges. Rigor mortis may be appreciably diminished.

Ergotism (see p. 672) is a specialised type of vegetable poisoning due to Claviceps purpurea, a fungus parasitic on the rye plant. The disease may occur in epidemic form, as at Manchester some years ago, when upwards of two hundred persons were affected. Symptoms, which are associated with the eating of infected rye flour, may be local, including vasomotor constriction and even gangrene of the extremities, or generalised in the nervous system with formication and paroxysmal convulsions. The suspect flour when heated with a strong solution of potassium hydrate yields the herring-brine odour of trimethylamine (see p. 714).

The general treatment of fungus poisoning is for the most part symptomatic. Emetics may be given, and lavage of the stomach either with plain warm water or with dilute solutions of potassium permanganate has been found helpful. Morphia is useful for the abdominal cramp. Oxygen may be required when asthmatic crises predominate, and subcutaneous or intravenous salines may be employed to combat collapse. Stimulants should be given in ergotism.

Poisoning by honey, which occurs from time to time, is due to toxic substances gathered by the bees from species of azalea, oleander, and rhododendron. It is of historic as well as current interest and is quoted by Xenophon.

Animal poisons.

These as here described must not be confused with certain products of decomposition which appear in dead meat and which may produce illness, but are to be regarded rather as toxic principles present in the living animal. They are perhaps most common among the fishes. The roe of pike and the musculature of sturgeon are poisonous during the spawning season.

Poisoning by Mytilus edulis, the edible mussel, has been recognised for more than a century. A mild form of the illness, showing a very short incubation period, often only a few minutes, and characterised by intensely itchy urticaria with asthma on occasion, is known in the north of England as "musselling" and is most probably anaphylactic.

Chemical poisons.

Among trade processes of food manufacture which utilise chemicals, the hydrolysis or inversion of starch to sugar by commercial sulphuric acid takes a prominent place. The acid, made from iron pyrites, which is invariably contaminated with arsenic, contains the poison and may thus transmit it to the finished food product. Beer and confectionery which may employ invert sugar have also been responsible for outbreaks of arsenical poisoning. Baking powder "improved" with acid calcium phosphate may derive arsenic from the same source. The varnish used to lacquer tinplate containers, and to polish sweets, has also yielded the poison. Arsenic may reach apples too when its solution is employed in orchards. Prosecutions have been made for the presence of prohibited amounts of arsenic, on apples, alleged to have been due to spraying of trees. Cider, beer, lemonade, or other plumbo-solvent liquid stored in lead tanks or bottled in siphons containing lead fittings may occasion poisoning by this metal. Tea, cheese, and other foil-wrapped commodities may in like manner become contaminated. Lead-glazed vessels may yield their metal to foods cooked in them. Rathmell and Smith [1] record a unique case of acute plumbism in a child, aged twenty-two months, after drinking orange juice from toy dishes which by analysis showed a lead content. Lead solder in domestic cooking utensils may occasion lead poisoning. Poisoning by tin (see p. 566) usually results from solution of the metal from tinplate containers by acid fruits or shellfish. It is worth noting in such cases that the solid part of the contents may contain more tin than the liquid. Two grains of tin may constitute a poisonous dose. Enamels containing antimony (see p. 552) in the form of Sb_2O_3, used as an opacifying agent, have sometimes been attacked and dissolved by lemonade and other acid drinks, with the production of illness. In one such outbreak, for example, sixty-five nurses were poisoned by lemonade which had been made from the fresh fruit in cheap white-enamelled jugs. There were no fatalities. Zinc (see p. 565) is rarely a cause of poisoning, but consumers of dessert

fruits such as apples which have been cooked in zinc receptacles have before now received emetic doses of the metal. Various metallic poisons formerly occurred in foodstuffs as dyes or preservatives, including salts of antimony, arsenic, cadmium, chromium, copper, lead, mercury, and zinc as colouring matters, and boron, salicylic acid, and formaldehyde as preservatives. Cases of poisoning by cadmium deriving from refrigerator fittings have lately been reported. The Public Health (Preservatives, etc., in Food) Regulations, 1925-40, prohibit all save scheduled colours and limit the preservatives to benzoic and sulphurous acids and sodium or potassium nitrite, and these only in specified amounts in certain foods.

Other examples of chemical food poisoning include illness due to the use of cutlery cleaned with cyanide plate powder, and the "ginger paralysis," which occurred in the United States and was caused by the adulteration of fluid extract of ginger with tri-o-cresyl phosphate (see p. 681).

Bacterial food poisoning.

Infections such as typhoid fever, dysentery, scarlet fever, and diphtheria may be borne by articles of diet, and are thus, technically, examples of food poisoning. The foodstuffs mainly involved are milk, ice-cream, shellfish, green vegetables, and fruits.

More often, however, the illness is true bacterial food poisoning, frequently, though not invariably, associated with canned food and formerly known as ptomaine poisoning. Eggs, especially those of the duck, are not infrequently the vehicle of infection. The term is a misnomer, since ptomaines, chemical substances related to amines, are harmless when administered by the mouth. Bacterial food poisoning is mainly due to various strains of the Salmonella group which multiply in the intestine, producing acute enteritis, and may also cause a general bacteriæmia. It is difficult to place the causal agents in an order of frequency, but the commonest in this country is B. ærtrycke, so called from its first recognition at Ærtrycke in Belgium. The bacillus of Gärtner, B. enteritidis, also occurs widely in outbreaks. More recently the Salmonella types "Newport," "Stanley," and "Thomson" have shown an increased prevalence. Man is rarely if ever a carrier of Salmonella infection, save in the most transient way during convalescence, but certain of the lower animals such as rats and mice may harbour the pathogens for considerable periods and may thus be a reservoir of infection for the human subject.

The general symptoms of Salmonella infection are abdominal cramp, vomiting, and diarrhœa. The bacteria are present in the intestine during the attack. From 50 to 100 per cent. of persons partaking of the food are affected, but the case mortality is on the whole low and varies from 1·1 to 1·8 per cent. The degree and course of the illness depend entirely on whether

the living bacillus, or merely its toxin, has been taken into the body. The differential points concerned may be summarised in tabular form :—

	When due to bacillus.	When due to toxin.
Incubation period	Two to three days.	A few hours.
Course . .	Slow.	Rapid.
Food . . .	Visible change in character occasionally but not usually present.	Not visibly affected.
Fatality . .	May occur.	Rarely occurs.

The disease is most prevalent in the summer months, particularly when the living bacterium is concerned. Toxin outbreaks do not exhibit a seasonal preponderance. When the living bacterium has been at fault it may usually be recovered from the food as well as from the vomit or fæces of the patient. When, on the other hand, the toxin has been to blame, feeding experiments with animals alone can demonstrate its presence.

It has been estimated that almost one third of food poisoning outbreaks in Britain are due to staphylococcal intoxication, and are frequently associated with such commodities as artificial cream, pastry, and confectionery.

In investigating an outbreak of food poisoning it is essential to obtain samples of the peccant foodstuff and particularly of the individual portions served to the patient, since owing to the notoriously erratic distribution of the organisms in food, the patient alone may have received a charge of the bacteria. Material such as fæces or vomit should be sent to the laboratory with the least possible delay. Agglutinins to Salmonella strains are demonstrable in the blood after the seventh day of illness, and usually persist for a few months.

In seeking to trace the source of the illness it must be remembered that all the commodities eaten at the suspect meal should be examined and not merely those which seem a likely vehicle for the organisms. When a bacterial investigation fails to confirm the diagnosis, appropriate chemical tests should be performed.

Canned foods are not infrequently the vehicles of bacterial food poisoning. When anaerobic decomposition is present the tin may be "blown." Canned foods contaminated by Salmonella organisms rarely present changes in appearance or taste and are not as a rule characterised by putrefactive odours.

It is worth noting in conclusion, that foods packed in glass are on the whole less safe than those put up in tins, since the glass cannot, unless specially toughened, be processed to the temperatures necessary to kill bacteria or spores. Processing in the canned-food

industry is the final high-temperature heating to which the tins are exposed before labelling and despatch.

When an outbreak of food poisoning is brought to the notice of a general practitioner in England and Wales, it is his duty under food and drugs legislation to notify the medical officer of health of the area. In Scotland, the practitioner should inform the local medical officer in order that the appropriate community steps may be taken.

Fig. 231

Appearance of an unopened sound tin containing food. Note concave appearance of end.

Fig. 232

Appearance of a "blown" tin containing food. Note convex appearance of end.

Cases of prosecution for food poisoning are reported from time to time.

It not infrequently happens that foreign bodies are by accident or carelessness included in articles of food. Nails, broken glass, pieces of string, cigarette stubs, cockroaches and the like from time to time appear. Their discovery, coupled with the fear that some of the foreign substance may already have been eaten, is sufficient in highly strung individuals to precipitate an attack of illness which may closely simulate food poisoning. Not uncommonly a medico-legal issue is raised in these cases and the practitioner has to be on his guard. In a case which came under the writer's notice the offending substance was a small beetle of the genus Niptus, which is commonly present in old houses. The patient, a nervous, rather delicate female, was violently sick for a week and suffered also from diarrhœa, colic, and recurring bouts of nausea lasting over a fortnight. There was no doubt that the effect was mainly psychological. A comprehensive investigation was undertaken, including a clinical and serological examination of the patient and chemical analysis of the peccant food, in this case a soda scone. All tests for bacteria and poisonous substances were negative.

Occasionally, crystals of ammonium-magnesium phosphate form in canned salmon and may be mistaken for glass. The addition of vinegar, in which the crystals are soluble, readily settles the question.

Botulism

(Latin : botulus, a sausage).

This fatal type of food poisoning is due to B. botulinus (Clostridium botulinum), Types A and B, a sporing anaerobe whose normal habitat is garden soil. The exotoxin excreted by this organism is one of the most powerful known. The disease, also known as allantiasis, is prevalent both in Middle Europe and in the United States. The outbreaks in Europe are due in most instances to meat or meat foods, whereas those in America are caused principally by vegetable products such as olives, string beans, asparagus and the like. The incubation period is usually less than forty-eight hours and may be as short as four hours. The disease commonly begins with diplopia, which is rapidly followed by progressive paralysis of the eye muscles and finally those of the lids, producing complete ptosis. Involvement of the larynx soon ensues, articulation becomes faulty, and respiratory paralysis with extension to the breathing centre closes the scene. Only in a few cases is there vomiting. The unfortunate patient is usually conscious to the end.

Though by no means present in all instances, a rancid butter odour is often discernible in the affected foodstuff.

The outstanding British outbreak occurred in Scotland at Loch Maree in 1922, when all of eight persons who partook of sandwiches made from potted duck paste died. The average incubation period was twenty hours. Six of the patients were dead within forty-eight hours and the remaining two lingered for several days. Bruce White found the duck paste rich in B. botulinus. Antitoxic sera are available which are narrowly type-specific. The appropriate type serum should be administered immediately in all suspect cases.

Reference

1. Jour. Amer. Med. Ass., Vol. CXIV, 242. 1940, Rathmell and Smith.

CHAPTER XXVII

PLANT IRRITANTS, ARROW POISONS, STINGS AND BITES

THE stinging or wounding propensity is widespread in nature both in plants and in animals. In the latter the mechanism is either defensive or is employed to stun prey.

Contact with plants.

Many plants cause severe irritation when their acrid juices come into contact with the human skin. Flowers, leaves, and even barks may be concerned in the production of the lesions, which are conveniently grouped under the general title, dermatitis venenata. Clinically, the effect is commonly an acute erythema. When, as in the case of flowers, the plant has been pressed to the face so that its fragrance may be sampled, the lesion invades the features and the accompanying œdema may result in closure of the eyes. There can be little doubt that personal idiosyncrasy plays a large part in determining the occurrence of such dermatitis. Of flowers or leaves which may produce the syndrome there may be mentioned Clematis (Traveller's Joy), Chrysanthemum, Geranium, Nasturtium, Narcissus (Daffodil), and Ranunculus (Buttercup). Special mention is required for Rhus toxicodendron the three-leaved poisonous ivy, sometimes termed sumach vine, which is a native of America and is known in this country as Ampelopsis hoggii. Rhus dermatitis is acute and is complicated by vesicles or even bullæ which at times assume a pemphigoid character. Almost equally severe upon the sensitive skin are certain species of Primula, notably P. obconica and more especially P. sinensis, the Chinese primrose. As a buttonhole, at table, or in the greenhouse, primula claims its victims. That actual contact with the plant need not be necessary is illustrated by a characteristic case quoted by Sir Norman Walker.[1]

Barks and woods which may occasion papulo-vesicular dermatitis include birch, mahogany, satinwood and teak. Sawdust of such woods employed as packing material may suffice to produce the effect.

Closely akin to the toxins of vegetable origin are the arrow poisons, of which there is a considerable range showing both geographical and tribal variation. Besides the poisonous principles obtained from plants, venoms of insects and reptiles may be employed to smear arrow heads. In East Africa the chief plant arrow poison is that prepared by decocting and concentrating

the bark of the Acocanthera shrub. Strophanthus is used in the Congo and Gold Coast. Malay tribes favour preparations of Strychnos (see p. 669) and often use them by means of dart and blowpipe. Species of Strychnos containing the active principle curare have a vogue on the Amazon. The characteristic paralyses of the voluntary muscles and the accompanying anxiety state produced by this poison are well known.

Arrow poisons may remain long active, as is instanced by the case of a housemaid who was accidentally pierced by an Indian arrow while dusting trophies. She collapsed half an hour later and showed shallow breathing with feeble pulse. Artificial respiration was performed for two hours before consciousness was fully re-established, and some drowsiness persisted for several days.

In cases of arrow or dart poisoning, treatment, which must imperatively be immediate, consists in firm proximal ligation at the site of wounding and irrigation of the lesion with 5 per cent. potassium permanganate. Stimulants are generally required and morphia is necessary as an antidote to members of the Strychnos group (see p. 669).

Contact with animals.

The animal kingdom presents many examples of the toxic propensity, ranging from the mild irritation produced by simple aquatic forms to the lethal effect of the more poisonous snakes. Lowly examples are the stinging jelly fishes, one of which, Cyanea, or "lion's mane," is familiar to bathers. Contact produces formication and erythema, often severe. In a case which recently came under the writer's notice, involving a girl aged twenty-one, contact with Cyanea occurred in the left deltoid region. Within a few minutes there was severe and spreading erythema accompanied by a burning sensation in the throat and muscular pain sufficiently severe to require morphia. Faucial spasm persisted for some hours, after which the symptoms slowly abated. The phylum Arthropoda, which ranks next above the worms, includes a variety of classes represented by the true insects, centipedes, spiders, ticks, and crustacea. The arthropods are injurious to man in various ways. The Lepidoptera include many irritant forms, mainly represented by the urticating moths whose larvæ possess hairs or spines endowed with the stinging effect. The caterpillar of the tiger moth, known as the "woolly bear," may cause severe dermatitis, especially in allergic subjects, and that of the puss tussock moths, swelling, nausea, and even paresis. Ingestion or inhalation of the hairs may cause serious internal disturbances.

The biting Diptera are numerous both at home and abroad. The slender mouth parts of Phlebotomus, the sand fly, can inflict a poisonous wound. The bite of the anopheline mosquito may produce marked irritation and even sepsis, as may also the puncture of Culex pipiens, the common British gnat. A young medical woman was thrice bitten on the forehead by insects which from their

description appear to have been culicine mosquitoes, most probably Theobaldia. Within one minute large bullæ had formed in the area of the bites. Some five minutes later generalised urticaria had developed, accompanied by œdema, drowsiness, and some dyspnœa. These symptoms persisted for about eight hours and then slowly subsided. That the blood-thirsty female of Hæmatopota, the common eleg or horse-fly, can cause a poisoned lesion is well known, and during a summer camp the following typical case came under personal notice. A boy aged twelve, wearing the kilt and open sandals, was bitten at two places on the thigh and also below the knee and on the instep. Within three hours the affected limb was highly œdematous and showed marked vesication. The inguinal glands were slightly enlarged and there was malaise. The condition took ten days to subside and there was minor sloughing at one of the bites.

Perhaps the most common offenders, however, are bees and wasps, which belong to the order Hymenoptera. Bee venom is in toxic doses both neurotropic and hæmolytic, though the worst effects are usually from œdema and shock. A single sting in the region of the throat may kill from suffocation: multiple stings, as from a disturbed swarm, are usually fatal. In supersensitive subjects, choking sensations, shallow breathing, cyanosis, and generalised pain may follow the sting. Cases of sudden death following wasp sting have been reported by Dyke.[2] A man aged forty-four was stung on the right temple. His face became suffused and he fell down almost immediately. When seen by a doctor twenty minutes later he was dead. At autopsy the lungs were found to be congested and the thymus enlarged. In another similar instance the patient, a woman of sixty, was stung in the neck. She almost immediately fell ill and decided to lie down. While proceeding to her bedroom, she collapsed in the hall and died in a short time. No evidence of disease was found post-mortem. Among the Arachnida, which differ from the Insecta by having no true jaws, the Scorpions, which are capable of inflicting a sting accompanied by respiratory paralysis, cyanosis, and shock, and certain of the spiders, whose bite may be highly toxic, are outstanding. Children in North America, and in Australia, may suffer from tick fever, which resembles poliomyelitis and results from the toxic bite of Dermacentor. The bite of Latrodectus, the "black widow" spider, is followed by abdominal rigidity which may closely simulate, and has been mistaken for, that following intestinal perforation. Opiates may be required for the severe pain.

The treatment of severe insect bites in general should consist first in the application to the part of a solution consisting of 30 grains of menthol in 1 ounce of compound tincture of benzoin, and later, in the use of a lead or zinc lotion. Minor bites often respond to sodium bicarbonate paste or Eau de Javelle. Accompanying syncope demands appropriate internal medication.

Of the poisonous fishes, Trachinus, the weever fish, and Trygon, the stinging ray, are perhaps the best known in British waters. The weever resembles the mackerel, but is more slender. Spines

situated on the dorsal fin and gill covers are the source of the toxin, which is employed to stun prey, but which is highly irritant to the skin of man.

Snakes belong to the order Reptilia, which includes the Lacertilia, or lizards, and the Ophidia, or serpents. Snake-bite is commonly regarded by the public as invariably fatal, unless treated. Actually, of some 2,000 recorded species of Ophidia, those venomous for man are relatively few and belong to two families, the Colubridæ and the Viperidæ. Snakes do not sting, as is popularly supposed. Their poison is contained in buccal glands and is inoculated by the act of biting. A few species, such as the spitting cobras, can eject the poison in a stream. The venom, of which there are two kinds, is a clear, amber-tinted fluid containing modified proteins and toxic principles such as hæmolysins, coagulins, and the like. In certain of the Australian colubrines both types of venom may occur in the same reptile. The poison of the viperine snakes acts mainly on the vascular, while that of the colubrines attacks the central nervous system. The mortality from all types of snake-bite is in the neighbourhood of 40 per cent., though the total annual death-rate from this cause in a country such as India may exceed 20,000.

The colubrine snakes, which preponderate amongst the poisonous varieties, include the cobra, the coral snake, the mamba, the krait, and the hamadryad or king cobra. A bite from one of these is attended almost immediately by severe pain, rapid œdema, and inflammation of the wound, and is soon followed by apathy, paralysis, coma, and death. Should the patient survive the paralysis, recovery may be expected.

Viperines include the European viper, or adder, the Russell's viper, or daboia, the rattlesnake, and the phoorsa, all of which may produce fatal effects. The symptoms following the bite of the daboia, for example, are local pain, œdema, and ecchymoses or other hæmorrhage, together with collapse, thready pulse, and loss of consciousness. The local lesion later becomes further aggravated and not uncommonly is the seat of suppuration, sloughing, or gangrene. These secondary local phenomena are particularly marked in the case of the rattlesnake. The British viper produces a train of symptoms resembling those due to the daboia but of milder type. Viper bites are, however, not infrequently fatal.

Lizards though fearsome are, save in one instance, non-poisonous. The toxic exception is the desert-dwelling genus Heloderma of which the two species H. horridum and H. suspectum are known as "Gila Monsters." Their bite is severe but not necessarily fatal.

Treatment of snake-bite.

Local measures such as firm proximal ligation may be of some avail, particularly in cases of poisoning by a viperine. Free incision of the wound followed by thorough suction either with the mouth or by means of a breast pump. Treatment by permanganate crystals

is now deprecated by experts, as is the use of the cautery. Adrenalin has been found helpful in paralytic cases, and artificial respiration is often required. Sucking of the unexcised wound is useless, as is the exhibition of alcohol or strychnine. Antitoxic sera, prepared from horses and known as antivenenes, are now available, but possess certain drawbacks. They must be strictly homologous. For example, viperine antivenene to, say, the daboia, is useless in cobra poisoning. The dose should be at least 100 millilitres, given intravenously. Good results are now claimed from early intravenous injection of polyvalent antivenene serum (80 per cent. colubrine with 20 per cent. viperine) in cases where the species of the offending snake is not known with certainty. Dosage of antivenenes should be in inverse proportion to body weight, children receiving several times the official adult amount.

As regards general treatment it is of the greatest importance to reassure the patient, and to combat the physical shock. Black coffee, or caffeine, is beneficial. Alcohol and morphine should in all cases be avoided.

References

1. " Introduction to Dermatology," Walker, 119, 10th ed. Green & Son, 1939.
2. Lancet, Vol. II, 307, 1941, Dyke.

APPENDIX 1

USEFUL WEIGHTS AND MEASURES

(Conversion Data)

15 grains = 1 gramme.
437½ grains = 1 ounce.
To convert ounces to grammes, multiply by 28·35.
1 ounce = 16 drachms = 28·35 grammes.
16 ounces = 1 pound = 0·453 kilogram.
To convert stones to kilograms (approx.), multiply by 6·35.
To convert kilograms into pounds (approx.), multiply by 11 and divide result by 5.
To convert pounds to kilograms (approx.), multiply by 5 and divide result by 11.
10 minims = 0·6 millilitre (approx.).
30 millilitres = 8·5 drachms = 1 ounce (approx.).
1 millilitre = 17 minims (approx.).
1 litre = 35·125 ounces = 1·75 pints = 1,000 millilitres.
To convert centimetres into inches (approx.), divide by 2·5.
To convert inches into centimetres (approx.), multiply by 5 and divide by 2.
To convert feet into centimetres, multiply by 30·5.

Temperature.

To convert degrees Centigrade to degrees Fahrenheit, multiply by 9, divide by 5, and add 32.
To convert degrees Fahrenheit to degrees Centigrade, subtract 32, multiply by 5, and divide by 9.

WEIGHTS AND OTHER DATA OF ORGANS

Adult organs.[1]

		(Average Weight)
Adrenals		5·0 to 6·0 grm.
Brain—	Female	1250·0 ,, 1275·0 ,,
	Male	1365·0 ,, 1450·0 ,,
Heart—	Female	250·0 ,, 280·0 ,,
	Male	270·0 ,, 360·0 ,,
Kidneys—	Right	140·0 ,,
	Left	150·0 ,,
Lungs—	Right	480·0 ,, 680·0 ,,
	Left	420·0 ,, 600·0 ,,
Liver		1440·0 ,, 1680·0 ,,
Ovaries		4·0 ,, 8·0 ,,
Pancreas		60·0 ,, 135·0 ,,
Spleen		155·0 ,, 195·0 ,,
Thymus—	At birth	14·0 ,,
	End of second year	26·2 ,,
	Then decreases until gland disappears.	
	A rapid diminution occurs at puberty.	
Uterus—	Nulliparous	40·0 to 50·0 grm.
	Multiparous	Increased by 20·0 ,,

NOTE.—These weights should be interpreted in relation to size of subject.

Other data.

Diameter of aorta	1·7 to 3·0 cm.

Uterus

Nulliparous Measurements.		Multiparous Measurements.
Length	7·0 cm.	
Breadth	4·0 ,,	Increased 1 cm. or more.
Thickness	2·5 ,,	
Length (cavity)	5·0 ,,	5·7 cm.

Organs of new-born child.[1]

	(Average Weight)
Brain	380·0 grm.
Thymus	14·0 ,,
Heart	20·6 ,,
Lungs (together)	58·0 ,,
Spleen	11·1 ,,
Kidneys (together)	23·6 ,,
Testicles	0·8 ,,
Liver	118·0 ,,

Reference

1. "Post-mortem Appearances," Ross. Oxford University Press, 1931.

APPENDIX II

CHEMICAL TESTS

(Note.—Substances are arranged alphabetically.)

Acetanilide.

If a small portion of the drug is tested by the sulphuric acid-potassium dichromate test a red colour changing to bluish-green is produced. Unlike phenazone, it does not react with ferric chloride.

If a portion of the extract, or residue, containing acetanilide is boiled with 4 millilitres of fuming hydrochloric acid, evaporated, cooled, and about 2 to 4 millilitres of saturated, aqueous carbolic acid solution and a few drops of freshly prepared calcium hypochlorite solution are added, a reddish-violet colour will develop and deepen on shaking; a solution of ammonium hydroxide floated on as an upper layer will assume an indigo-blue shade, while the under layer will retain a red colour.

Acetic Acid.

Distil or steam distil and make distillate alkaline with sodium hydroxide solution. Evaporate to dryness on a water bath.

Warm portion of residue with concentrated sulphuric acid when the odour of acetic acid may be detected.

To a portion of the residue add a few drops of alcohol and concentrated sulphuric acid—equal quantities—and heat mixture in a boiling water bath. An odour of ethyl-acetate will indicate the presence of acetic acid.

Acetylsalicylic Acid or aspirin.

Litmus gives an acid reaction to an aqueous solution. If 0·1 gramme is boiled with 10 millilitres of water and a drop of ferric chloride solution is added a violet-red colour will develop. A crystalline precipitate with an odour of acetic acid will form when 0·5 gramme is boiled for two to three minutes with 10 millilitres of a solution of sodium hydroxide, cooled, and an excess of dilute sulphuric acid is added.

Aconite.

Physiological Test.

After isolation, the alkaloid is tested by its effect upon the tongue and upon the frog by injection.

Alcohol.

The determination of alcohol in body fluids and tissues.

Urine and blood.

The method most commonly employed in this country for the determination of ethyl alcohol in body fluids depends on the volatilisation of the alcohol by heat and the absorption of the alcohol vapour in a mixture of potassium dichromate and sulphuric acid. The alcohol is oxidised to acetic acid with the reduction of a corresponding amount of the dichromate to chromic sulphate. The excess dichromate remaining is then ascertained by the addition of potassium iodide and by the titration of the liberated iodine with sodium thiosulphate solution. The difference gives the amount of dichromate which has been reduced by the alcohol, and the amount of the latter is calculated therefrom. The method is not entirely specific for alcohol as it will detect also certain volatile reducing substances.

Acetone, contained in the urine of diabetic subjects, has no reducing effect on the dichromate solution under the conditions of the experiment.

The following method is due to Kozelka and Hine [1] who state that it eliminates the effect of volatile interfering substances, notably acetone and formaldehyde, the latter of which may occur in the urine after the administration of certain urinary disinfectants. As mentioned above, however, acetone has no reducing effect on the dichromate solution of the strength employed and therefore does not require to be removed. Kozelka and Hine claim that their method will remove 75 milligrams of formaldehyde, an amount far in excess of that in pathological samples. The method is rapid, and accurate, and has the advantage that very low and concordant "blanks" are obtained on alcohol-free urine and blood.

Procedure.

Reagents required

Oxidising solution . .	To 1 litre of N/10 potassium dichromate, 500 millilitres of concentrated sulphuric acid are added slowly. The mixture is cooled and transferred to a bottle.
N/10 sodium thiosulphate .	25 grammes + 0·1 gramme of sodium bicarbonate per litre and standardised.
Sodium tungstate . .	10 per cent.
N/1 sulphuric acid . .	49 grammes per litre.
Mercuric chloride . .	Saturated solution.
Sodium hydroxide . .	Saturated solution.
Potassium iodide . .	20 per cent.
Starch solution . . .	1 per cent.

The apparatus is cleansed with soap and water, then treated with chromic acid and again washed with water. (The chromic acid is prepared by adding slowly, with constant shaking, about 2 ounces of potassium dichromate, finely ground, to a winchester of concentrated sulphuric acid.)

The generator flask E is filled with distilled water, and the apparatus assembled as shown in Fig. 233, excepting tubes G, F, and water bath H.

To tube G are added 10 millilitres of the mercuric chloride solution and 10 millilitres of the sodium hydroxide solution, and the contents are well mixed.

Two millilitres of urine or blood are now accurately measured into tube F, followed by 5 millilitres of normal sulphuric acid, and in the case of blood, 5 millilitres of sodium tungstate in addition to the sulphuric acid. The tubes G and F are immediately placed in position and held by the steel springs, and the condenser water is turned on. It should be noted that on collection of the sample of blood for analysis an anti-coagulant should be added, such as the addition of 10 parts of 3 per cent. potassium oxalate solution to 90 parts of blood, and a correction applied for the resulting dilution.

The water in generator flask E is now heated, and, when it has just reached the boiling-point the water bath H, containing boiling water, is placed in position just below tubes G and F, and raised at once to a point at which the level of the water in the bath reaches to about 1½ inches from the top of the ground glass joints A. The bath is maintained at 100° C. by means of a Bunsen burner placed under it. An asbestos board is inserted between the water bath and the digestion flask D in order to shield the latter from the heat.

The distillate 45 to 55 millilitres (indicated by a mark on the side of the flask) is received in the 250 millilitres conical pressure digesting flask D. To this is added from a standard pipette, 25 millilitres of oxidising solution, directed down the side of the flask, attention being given to time of drainage.

The cap is fitted firmly to the flask, the steel springs J adjusted, and the liquids mixed by rotation. The flask is now placed in a vessel containing boiling water, and the boiling is continued for half an hour. The springs and cap are removed (an outrush of air indicates that the cap has fitted tightly) and the flask cooled in running water. Distilled water is added, washing down the side of the flask, to give volume of about 200 millilitres, thus reducing acid concentration to 10 per cent. or less. Ten millilitres of potassium iodide solution are now added, the liquids mixed by rotating gently, and after a few seconds, titrated with N/10 sodium thiosulphate, using starch as indicator towards the end of titration.

Standard : Twenty-five millilitres of oxidising solution are added to an ordinary 250 millilitres conical flask, diluted to about 200 millilitres with distilled water, followed by 10 millilitres of potassium iodide solution, mixed by rotating gently, and after a few seconds, titrated with N/10 sodium thiosulphate, using starch solution towards the end of titration.

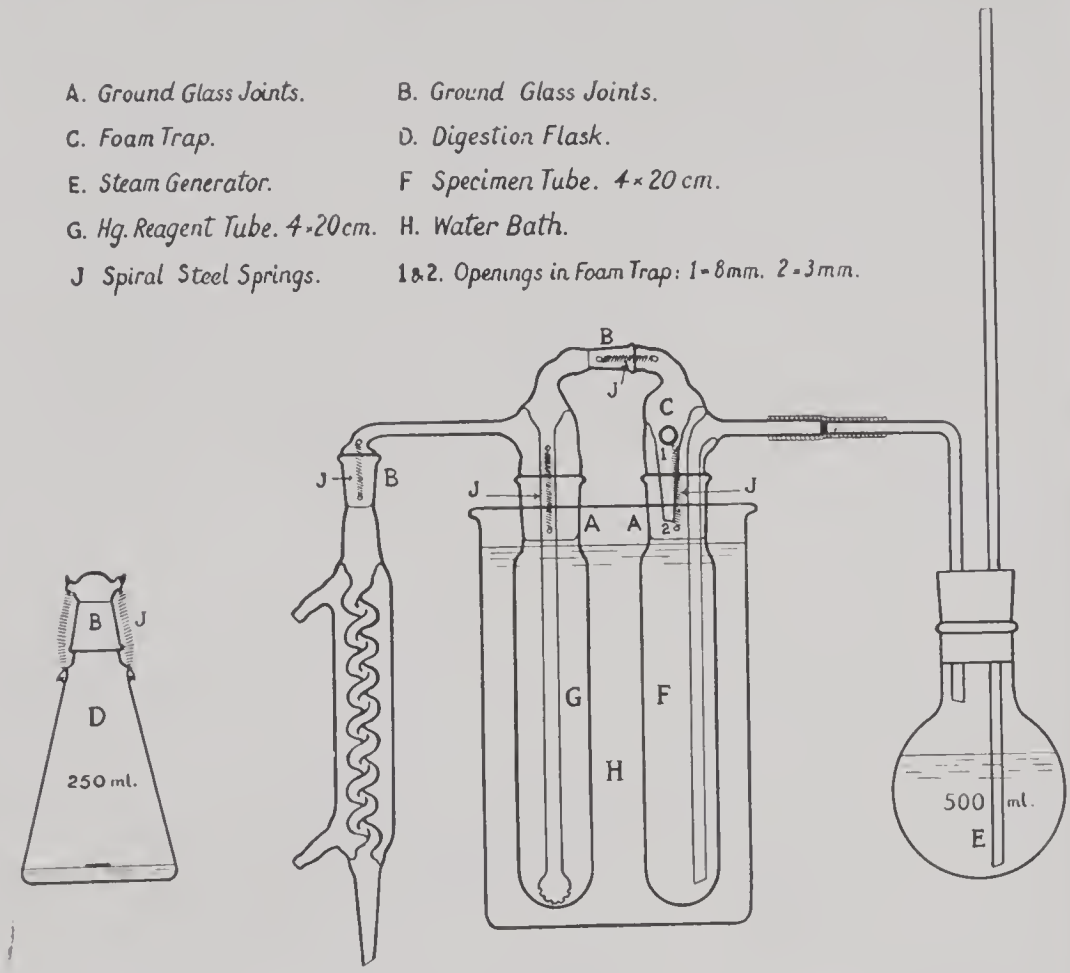


FIG. 233

Apparatus for semi-micro method of estimation of ethyl alcohol.
(Kozelka and Hine.)

1 millilitre of N/10 sodium thiosulphate = 1·15 milligrams of ethyl alcohol.
The calculation is as follows :—

$$\frac{\text{Standard} - \text{Sample} \times 1{\cdot}15 \times 100}{2} = \text{Milligrams of ethyl alcohol per 100 millilitres of urine or blood.}$$

As the thiosulphate solution does not keep, it should be frequently standardised against a known weight of iodine.

Tissues.

The following method, due to Gettler and Siegel [2] is applicable to blood and urine as well as tissues, and possesses the decided advantage for medico-legal purposes that the alcohol is isolated from the specimen as pure alcohol ; it can be identified by well-known tests, and produced in court if necessary. The original paper should be consulted for details, but the following summary gives the essentials of the process.

The tissues, or fluids, as soon as removed from the body, must be placed in a receptacle, sealed, and refrigerated. One hundred and fifty grammes are then weighed and placed in a 500 millilitre distilling flask. In the case of solids, about 200 grammes are rapidly cut into small portions, and 150 grammes placed in the distilling flask together with about 200 millilitres of water.

It is of advantage to add 0·5 millilitre liquid paraffin B.P. to minimise frothing. The contents are then steam-distilled, with great caution in the early stages to avoid frothing, until the distillate measures 200 millilitres, or thereby, the receiving flask being packed in ice.

To isolate the alcohol in the pure state, the following procedure is adopted :—

The entire steam distillate is transferred to the rectification flask A of the apparatus shown in Fig. 234. Approximately 2 grammes of granulated zinc are

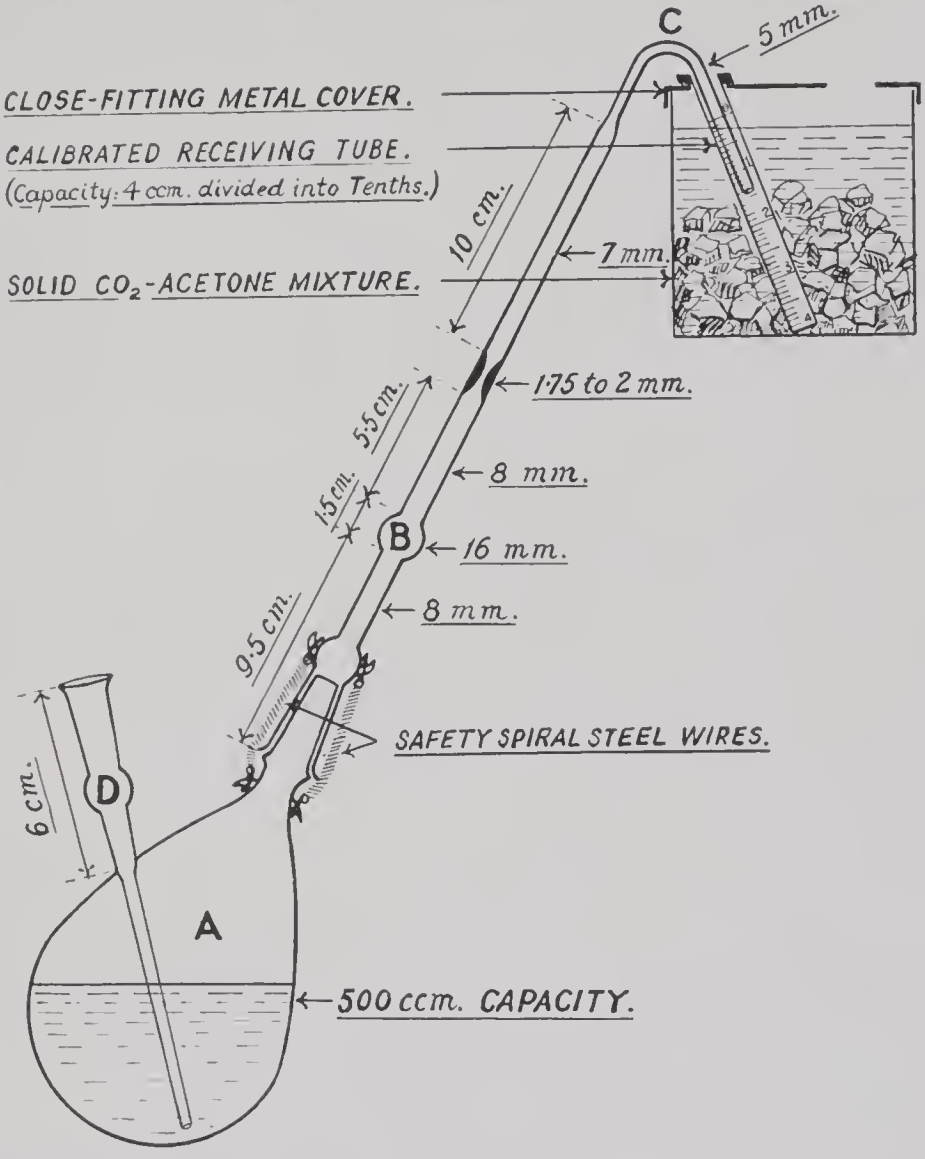


FIG. 234

Apparatus, for determination of ethyl alcohol, recommended by Gettler.

added and the flask and contents immersed in ice-water. Three hundred grammes of anhydrous potassium carbonate are added slowly, with continuous rotation of the flask to avoid localised heating, not less than fifteen minutes being taken for the addition.

The rectification arm B, which has been previously cleaned with nitric acid, washed with water, and dried, is then attached to the rectification flask and the apparatus fitted up as shown in the diagram. The flask rests on an asbestos concave-centred wire gauze and is heated with a micro-burner. The flame should be regulated so that the solution boils in fifteen to twenty-five minutes without allowing the hot vapour to rise above B. The flame is then increased so that the alcoholic ring of condensate rises from B to the bend C in from three to five minutes.

A visible ring of condensate is usually observed, but the distillation can be controlled by placing a finger on the bend C, which should feel warm (about 78° C.). If the rate of heating is correct, alcohol will distil (although little or no steam has reached C) and be condensed in the calibrated tube which is deeply immersed in a mixture of solid carbon dioxide and acetone.

As the heating is continued steam will follow, condense, freeze below the bend, and block up the tube. Back pressure will then force the solution up the safety tube D, when the distillation is discontinued. As soon as this end-point is reached, the rectification arm is lifted out of the calibrated receiver. This calibrated tube, containing the isolated alcohol, is removed from the cooling bath and kept at room temperature until any ice present is melted, after which it is placed in a bath of ice-water and $\frac{3}{10}$ millilitre of a saturated potassium carbonate solution is added, followed by a grain or two of anhydrous potassium carbonate, and the whole thoroughly mixed with a thin glass rod. If much water has distilled over, solid potassium carbonate only is added.

Centrifuge the mixture until a clear upper layer is obtained, usually this takes about one minute. Allow to stand in ice-water for about three minutes, and read the volume of the alcoholic upper layer. An excess of potassium carbonate must always be present. If these conditions are strictly observed the composition of the alcohol layer will, according to Seidell, be: ethyl alcohol, 91·9 per cent.; potassium carbonate, 0·04 per cent.; water, 8·1 per cent. (One millilitre of this layer at 0° C. weighs 0·833 gramme.) Therefore, 1 millilitre of the alcohol layer contains $0{\cdot}833 \times 0{\cdot}919 = 0{\cdot}7655$ gramme alcohol.

$0{\cdot}7655 \times$ millilitres of upper layer = grammes of ethyl alcohol in the weight of fluids or tissues taken (150 grammes sample).

$0{\cdot}5103 \times$ millilitres of upper layer = percentage of ethyl alcohol in tissue or fluid (150 grammes sample).

The purity of the alcohol thus isolated can be confirmed without further treatment (provided acetone or acetaldehyde are absent) by the determination of the boiling-point. It has been shown by many experiments that the boiling-point of the ethyl alcohol, as determined by the method to be described, approximates very closely the boiling-point of pure ethyl alcohol. Usually the amount of alcohol obtained from body fluids or tissues is so small that it is necessary to employ a micro-method for the determination of the boiling-point. The following technique is recommended:—

By means of a very fine capillary, two drops, or more if available, of the alcohol layer is transferred to a larger capillary tube, one end of which is sealed, so that a small cushion of air remains in the sealed end.

The capillary tube containing the droplet of liquid is then set up in an ordinary melting-point apparatus, and the temperature of the bath raised slowly and regularly. According to Emich, the true boiling-point of a pure liquid is the temperature at which the droplet in the capillary reaches the surface of the bath. If the droplet in the capillary is pure anhydrous alcohol, it should start to ascend at about 76° C., and should reach the surface of the bath between 77·5° and 78° C. One hundred per cent. pure ethyl alcohol can be obtained by a further process, for the details of which the original paper should be consulted.

If the boiling-point is much lower, say 66° to 76° C., it indicates the presence of lower boiling liquids such as acetone or acetaldehyde. Appreciable amounts of acetone are only met with in severe diabetics. Large amounts of acetaldehyde are only found if paraldehyde has been administered between twelve to eighteen hours before death. The presence of either of these substances can be easily demonstrated by qualitative tests. The identity of the alcohol isolated can be confirmed, if necessary, by the preparation of derivatives, such as ethyl benzoate, or by an ultimate analysis for carbon and hydrogen.

As already stated, the advantage of this method is that the substance sought for is isolated direct, purified, identified, and can be produced in court if necessary.

Aluminium.

Ammonium hydroxide solution gives a white precipitate which is slightly soluble in excess of the reagent.

Sodium hydroxide solution gives a white precipitate soluble in excess of the reagent.

Sodium phosphate solution gives a white precipitate insoluble in acetic acid, but soluble in mineral acids and in solutions of caustic alkalis.

Ammonia Gas.

See Ammonium Hydroxide.

Ammonium Hydroxide.

Test with moist red litmus paper. Colour is turned to blue.

Ammonia fumes produce a white smoke when mixed with the fumes from a rod moistened with hydrochloric acid.

Ammonium salts are volatile when heated. When treated with sodium hydroxide solution, ammonia is evolved.

When a paper moistened with mercurous nitrate solution is held over the gas it becomes blackened.

Filter paper moistened with a solution of manganous sulphate and hydrogen peroxide gives a brown colour when exposed to ammonia vapour.

A brown precipitate, or a brown or yellow colour, is produced with Nessler's reagent. This is a delicate test for traces of ammonia.

Aniline, Aniline Oil, or Phenylamine.

The poison may be obtained by rendering alkaline the vomited matter or stomach contents and distilling. The distillate is shaken up with ether, the ether is separated and allowed to evaporate, and the poison will be found in the residue.

If to a portion of the residue containing aniline a few drops of sodium hypochlorite solution are added a blue colour changing to pale red will be seen. If a few drops of dilute phenol solution and ammonia are now added a blue colour, which is stable, will develop.

A solution containing aniline when heated with one drop of chloroform and alcoholic potash gives the offensive odour of phenylisonitrile.

Bromine water will produce a flesh-coloured precipitate.

Antimoniuretted Hydrogen.

See Antimony.

Antimony.

Antimony potassium tartrate on being heated on a piece of platinum foil can be differentiated from arsenic, since the former becomes blackened and leaves a dark residue of charcoal and metallic antimony.

When antimony is present in organic fluids or in solids which have been finely divided it may be detected by the Reinsch and Marsh tests. (See Arsenic.)

Reinsch Test.

A deposit is formed upon the copper foil, the colour of which is determined by the amount of antimony present and the period of boiling. If small in amount, the colour is a violet-red or purple; if in larger amount, black and pulverulent. When the copper foil is dried and heated in a sublimation tube, the sublimate on microscopic examination is seen to consist of small amorphous particles composed of antimonious oxide. If this is dissolved in water acidulated with hydrochloric acid and sulphuretted hydrogen is added, an orange-coloured precipitate of antimony sulphide will develop.

Marsh Test.

Antimoniuretted hydrogen burns with a faintly bluish-green flame and gives a dull black spot upon a cold porcelain plate held in the flame. The deposit is insoluble in a solution of sodium hypochlorite and with ammonium molybdate in nitric acid. Antimony does not give a precipitate or a colour reaction with silver

nitrate. The deposit after being dissolved in ammonium sulphide leaves an orange-yellow stain which is soluble in strong hydrochloric acid, but is insoluble in ammonium hydroxide. If a flame is applied to the exit tube a deposit of metallic antimony occurs in the tube close to the point of application. On sublimation a white sublimate of the oxide will form.

Arsenic.

On examining the stomach and its contents, the mucous membrane should be explored for the presence of minute gritty particles of arsenious oxide. If, however, no particles are found, a small portion of the material may be submitted to the Reinsch Test. When found they should be submitted to a chemical test. Some should be washed in distilled water, dried between folds of filter paper, and placed in a small sublimation tube. The tube is heated by a small Bunsen flame and the formation of a sublimate looked for. When found it should be examined microscopically for crystals of octahedral type presumptive of arsenious oxide (see Fig. 224). The piece of tube bearing the sublimate may then be cut off, ground in a small mortar, acidulated with pure hydrochloric acid and tested in the following way.

Arsenious oxide dissolves in nitric acid and on addition of ammonium molybdate and warming a yellow precipitate forms. Phosphates behave similarly. A nitric acid solution evaporated to dryness gives a red colour with silver nitrate solution.

Reinsch Test.

This is a good preliminary test.

To the suspected fluid add about one-fifth of its volume of concentrated arsenic-free hydrochloric acid, then add two or three small pieces of pure copper foil (previously cleaned with nitric acid) about a quarter of an inch square and boil for about fifteen minutes, although the test should not be considered as negative until after boiling for thirty minutes.

It is essential that the copper foil should be proved pure. To some arsenic-free hydrochloric acid add five equivalents in bulk of distilled water and a few drops of a weak solution of ferric chloride or ferric sulphate. After boiling the mixture for a few minutes add the pieces of bright copper foil while the fluid is still boiling and continue boiling for a few minutes. If the metal is arsenic-free its original bright colour will remain unchanged, but if it is contaminated it will show a tarnished appearance. Should a darkish deposit have formed when the test is completed the pieces are dipped in water, alcohol, and ether, successively, dried, placed in a sublimation tube, and heated. When a sublimate forms the method already described for identifying its arsenical character should be employed.

It must not be forgotten that, in addition to arsenic, dissolved antimony, bismuth, gold, mercury, platinum, silver, and tin are deposited upon copper when boiled in an acid solution, but only arsenic, antimony, and mercury sublime from the copper when heated in a sublimation tube. Reinsch's test fails in its action in the presence of chlorates and nitrates and it also fails to take up all the arsenic which may be present in the material suspected.

Marsh Test.

This test is capable of detecting 0·0001 per cent. of arsenious oxide in solution.

The test depends upon the fact that soluble arsenical compounds are decomposed by nascent hydrogen which is generated by the action of pure dilute arsenic-free sulphuric acid or arsenic-free dilute hydrochloric acid on arsenic-free metallic zinc, rendered more sensitive by the addition of a small amount of arsenic-free cadmium sulphate solution, and that the arsenic unites with the hydrogen to form arseniuretted hydrogen. A few millilitres of suspected material are sufficient. About 15 to 25 grammes of pure granulated zinc and a 15 per cent. solution of pure sulphuric acid are used.

The apparatus consists of a flask, the mouth of which is closed with a tight fitting ground glass stopper in which two holes have been drilled. Through these holes are passed accurately fitting glass tubes, one of which is thistle-shaped at the top and is passed nearly to the bottom of the flask. The lower end of the other tube just passes through the stopper, its upper end remaining above the stopper.

This tube is bent twice upon itself and is connected with a calcium chloride tube, which in turn is united with a lengthy piece of hard glass tubing narrowed about 2 or 3 inches from its free end. The apparatus must be gas-tight and the calcium chloride should be both fresh and dried. It is of importance that the medium in which the arsenic is suspected to be present should be obtained free from organic matter by previous destruction with sulphuric acid, since organic matter tends to interfere with the efficiency of the test.

The granulated zinc is added to some distilled water in the flask, some pure dilute sulphuric acid or diluted hydrochloric acid is poured down the thistle funnel, and hydrogen is then liberated, which expels air from the flask. The hydrogen is then tested to ascertain the absence of arsenic by placing a burner under the ignition tube and heating it to redness. Neither an arsenic mirror nor spot should appear on the glass. If the hydrogen is proved to be arsenic-free the next step consists in pouring in some of the fluid under test and continuing the experiment. After waiting a few minutes to permit the air of the flask being replaced by the arseniuretted hydrogen, a light is applied to the issuing gas at the free end of the tube.

Arseniuretted hydrogen may be recognised by the following properties. It burns with a pale bluish, bluish-white, or lilac flame, and if a piece of white porcelain is held in front of the flame a dark spot, the "arsenic spot," forms upon the porcelain. This deposit is soluble in sodium hypochlorite solution and also in a solution of ammonium molybdate in nitric acid.

When these tests are being applied the delivery end of the tube should be dipped into a solution of silver nitrate to prevent escape of the arseniuretted hydrogen into the surrounding atmosphere with possible poisonous action on the experimenter, since this gas is very lethal.

The next step is to apply heat to the ignition tube. The effect of the application of the heat is to decompose the arseniuretted hydrogen into hydrogen and metallic arsenic, when a dark brown mirror of metallic arsenic will be deposited within the tube in the vicinity of the point to which the heat has been applied. When the tube has sufficiently cooled to enable it to be detached the mirror may again be heated, when the arsenic will sublime on the cool part of the tube in the characteristic crystalline form of arsenious oxide (see Fig. 224).

Gutzeit's Test.

Gutzeit's test is a very simple one and requires, like Marsh's test, a solution preferably free from organic matter, and the use throughout of arsenic-free reagents. Arseniuretted hydrogen is generated by the action of zinc on the dilute hydrochloric acid solution containing the arsenic and the gas, after passing through a roll of lead acetate paper, is made to diffuse through a disc of mercuric chloride paper, where a yellow or brown stain is produced, the intensity of the stain being proportional to the amount of arsenic present. Stains produced by sulphuretted hydrogen or phosphine may be differentiated from arsenic stains by immersing the paper in hydrochloric acid. The two former show little change, while the latter turns brick red. The grey stain produced from relatively large amounts of antimony fades appreciably when treated with hydrochloric acid.

The test, which may be employed for qualitative or quantitative purposes, is best carried out on a solution containing about 0·01 milligram of arsenic.

For the former purpose very simple apparatus is required. A four-ounce wide-necked bottle fitted with a rubber stopper, through which a glass tube has been passed, will serve the purpose. A roll of lead acetate paper is placed in the tube, the upper end of which is covered with a double layer of mercuric chloride paper, held tightly in position by means of a ring cut from a piece of rubber tubing. For quantitative purposes, however, it is essential that the stains be uniform in size and a more elaborate arrangement is necessary. The glass tube, 200 millimetres long and 6·5 millimetres internal diameter, tapered at the lower end and having a small hole blown in the side near the constricted part, should have the other end formed into a flange ground to a flat surface and having two glass lugs attached. After insertion of the lead acetate paper two mercuric chloride papers are placed on the flange. A cap prepared from the same tubing, having a similar flange and a double lip at the other end, is placed on top of the papers. By fastening a spring or a rubber band to one of the lugs and passing it over the double lip and under the other lug a secure attachment is made.

To carry out an estimation the arsenic solution containing 10 millilitres of hydrochloric acid is diluted to 45 millilitres with water, two drops of bromine solution are added and the excess bromine removed by the addition of a little stannous chloride solution. Ten grammes of zinc are added, the stopper carrying the prepared tube is immediately inserted, and the reaction allowed to proceed for forty-five minutes. At the expiry of this time the stain is compared with those produced from known amounts of arsenic under exactly the same conditions.

Arseniuretted Hydrogen.

See Arsenic (p. 708).

Atropine.

A drop of filtered fluid from the stomach may be instilled into the eye of an animal in order to observe whether pupillary dilatation is produced.

Ether or chloroform will extract atropine from an aqueous solution rendered alkaline with sodium hydroxide.

Gerard's Test.

Add 2 millilitres of a 1 per cent. solution of mercuric chloride in 50 per cent. alcohol to a fluid containing atropine and warm gently. A yellow precipitate, turning red on boiling, will appear. The test requires 1 milligram of atropine and may fail if there is too great dilution.

Vitali's Test.

Evaporate extract over water bath with a few drops of fuming nitric acid. Moisten residue with a few drops of potassium hydroxide in absolute alcohol, when a violet colour, changing to red, will appear. Hyoscyamine and hyoscine, or scopolamine, also give this reaction. These mydriatic alkaloids may be differentiated by the crystallisation of their gold chloride double salts.

To a solution containing any of these add a slight excess of chlorauric acid solution (gold chloride solution). The precipitate should be brought into solution by gentle application of heat. On cooling, the chloraurate will crystallise as follows :—

Atropine chloraurate : the crystals are dull and form slowly.

Hyoscyamine chloraurate : the crystals are in the form of lustrous golden-yellow leaflets.

Scopolamine or hyoscine chloraurate : the crystals are in the shape of broad, lustrous, yellow prisms.

Barbiturates.

Barbiturates are extracted by ether from acid solution and can be purified by dissolving in chloroform and extracting from the chloroform by 2 per cent. aqueous sodium hydroxide. After acidifying the alkaline solution and adding sodium bicarbonate, the pure barbiturate can be obtained by extracting with chloroform. On evaporating the chloroform solution a crystalline residue nearly always results. Proof of the presence of a barbiturate is afforded if the two following tests are positive :—

To one drop of an aqueous solution of this residue one drop of Millon's reagent is added and a white gelatinous precipitate, soluble in excess of the reagent, is formed.

When a trace of the residue (1 milligram or less) is dissolved in 2 millitres of chloroform and 0·1 millilitre of a 1 per cent. solution of cobalt acetate in methyl alcohol is added, followed by 0·6 millilitre of a 5 per cent. solution of isopropylamine in methyl alcohol, a strong violet colour is obtained.

Millon's test and the cobalt test should be applied only to the free barbiturates and not to the sodium derivatives.

Barium.

Ammonium carbonate solution gives a white precipitate soluble in acetic acid and in dilute mineral acids.

Ammonium oxalate solution gives a white precipitate slightly soluble in water, but readily soluble in mineral acids.

Potassium chromate solution gives a yellow precipitate practically insoluble in water and in dilute acetic acid, but readily soluble in mineral acids.

Dilute sulphuric acid gives a white precipitate insoluble in hydrochloric acid.

Bismuth.

Hydrogen sulphide produces a brown precipitate insoluble in cold dilute acids and in ammonium sulphide solution, but soluble in hot diluted nitric acid and in boiling concentrated hydrochloric acid.

Sodium hydroxide solution produces a white precipitate in the cold with excess of the reagent, which is easily soluble in acids. It becomes yellow on boiling. If hydrogen peroxide is added to the solution containing the white or yellowish-white precipitate a brown colour will develop.

Potassium iodide solution gives a dark brown precipitate readily soluble in excess of the reagent to give a yellow solution. On dilution an orange-coloured precipitate will form.

Boric Acid and Borates.

In the absence of oxidising agents, boric acid or borax can be detected by the turmeric test.

Acidify solution with dilute hydrochloric acid and dip a piece of turmeric paper into the fluid, removing and drying.

A red to red-brown colour changing to blue or olive green when spotted with a 1 per cent. caustic soda solution indicates the presence of boric acid.

Brucine.

Dissolve brucine in concentrated sulphuric acid and add a trace of potassium nitrate when a red colour will develop, which is discharged by adding stannous chloride. The addition of nitric acid will again produce the red colour. The red colour first produced will not be discharged by adding sodium sulphite.

Cadmium.

Acidify solution with hydrochloric acid and add hydrogen sulphide, when a yellow precipitate will form. The precipitate is soluble in hot dilute nitric acid and hot dilute sulphuric acid, but insoluble in potassium cyanide solution.

A white precipitate is formed on the addition of potassium cyanide solution. It is soluble in excess of the reagent.

Sodium hydroxide solution produces a white precipitate, insoluble in excess of the reagent.

Camphor.

The odour of camphor is characteristic. It should be noted that chloretone has a somewhat similar odour.

Cantharides.

Apply an olive oil solution of the residue to the skin and note resultant blistering or irritation.

Carbon Disulphide.

The odour is an excellent guide to its presence. Distil stomach contents and to distillate apply the following tests :—

Add a few drops of lead acetate solution, when neither a precipitate nor a colour will form. On addition of excess of potassium hydroxide solution and on boiling a black precipitate will form. This is a delicate test.

Carbon Monoxide.

To detect the presence of carbon monoxide in blood, dilute a drop of blood from the finger with water in a small test tube and prepare a drop of the suspected blood in exactly the same manner. Normal blood will show a faint yellow colour but, should carbon monoxide be present, the suspected sample will remain pink.

Warm a sample of the suspected blood in a water bath and if carbon monoxide is present a brick red deposit will be formed. Normal blood shows a dark brown deposit.

Hoppe-Seyler's Test.

Add some sodium hydroxide (sp. gr. 1·34) to the blood. A positive reaction is shown when a bright red colour develops. Normal blood tested in this way turns a dirty green colour.

Kunkel's Test.

Dilute the suspected blood with nine parts of water and add a few drops of 3 per cent. aqueous tannin. When carbon monoxide is present a pinkish-white precipitate will form. Normal blood treated in this way shows a brownish-white precipitate.

Modification of the Tannin Test.

This method is recommended by Hawke and Bergeim.[3] The blood under test is divided into two portions, each is diluted with four volumes of distilled water and placed in a separate bottle. To each bottle twenty drops of 10 per cent. potassium ferricyanide are added to convert the oxyhæmoglobin into methæmoglobin. The solutions are allowed to stand for a few minutes and then the stoppers are placed in the bottles. One of the bottles is shaken vigorously for ten to fifteen minutes, occasionally removing the stopper to admit air, the object being to free the blood of carboxyhæmoglobin. Five to ten drops of yellow ammonium sulphide and 10 millilitres of a 10 per cent. solution of tannin are added to each bottle. After a short period the contents of the bottle which were shaken will show a dirty olive-green precipitate, while the contents of the unshaken container, if the blood contains carboxyhæmoglobin, will show a bright red precipitate. This test will disclose the presence of as little as 5 per cent. of carboxyhæmoglobin.

Quantitative Estimation—Method of Hawke and Bergeim.

To 0·1 millilitre of suspected blood add 0·9 millilitre of distilled water and mix thoroughly. One millilitre of freshly prepared pyrogallic-tannic acid solution is added and mixed. This solution consists of 2 grammes of pyrogallic acid and 2 grammes of tannic acid in 100 millilitres of distilled water. After fifteen minutes the result is compared with prepared standard solutions made from proportional parts of saturated blood standards in strengths from 0, 5, 10, etc., to 100 per cent. This original saturation is obtained by putting blood in a flask and mixing thoroughly with illuminant gas. To 1 millilitre of each of these solutions, in separate tubes, 1 millilitre of pyrogallic-tannic acid solution is added and mixed thoroughly. Permanent preparations can be made by running a film of paraffin over the layer of fluid and filling up the remainder of the tube with sealing wax.

Spectroscopic Tests.

If the blood is examined spectroscopically it will be found that the spectrum of carboxyhæmoglobin, which consists of two bands between D and E, is present. These bands are nearer to the violet end than those of oxyhæmoglobin which occupy a similar position (see Fig. 184). If there is a sufficient saturation of the blood with carbon monoxide, a reducing agent such as ammonium sulphide will not cause an alteration in the two bands. When the degree of saturation is low, reduction may be effected and, instead of two bands remaining after the addition of a reducing agent, a single broad band of reduced hæmoglobin may be seen. Spectroscopic examination is negative unless the amount of carbon monoxide in the blood exceeds 20 to 25 per cent.; or a calibrated instrument is used.

The reversion spectroscope designed by Hartridge, after suitable calibration, can be recommended for the quantitative estimation of carbon monoxide present in blood. For details of this and other quantitative methods the reader is referred to "Recent Advances in Forensic Medicine," by Smith and Glaister.[4]

Chloral Hydrate.

Chloral hydrate gives a positive reaction with the phenylisonitrile and naphthol tests, as does chloroform. When a solution containing chloral hydrate is tested by adding a few drops of Nessler's reagent, a yellowish-red or yellowish-green precipitate is given on shaking.

Chloral hydrate reduces Fehling's solution if other reducing substances are absent.

Six drops of saturated resorcin and 1 millilitre of sodium carbonate are added to the distillate and the mixture is allowed to stand for half an hour. On the addition of 10 millilitres of water, a greenish fluorescence will develop. Chloroform, bromoform, iodoform, and bromal also give this test.

Chloroform.

After the viscera have been suitably prepared and acidified by the addition of tartaric acid, the isolation is effected by the process of distillation. The distillate should be tested by smell and by adding a few drops of aniline and a small quantity of alcoholic potash solution to a small quantity of the distillate when, after the application of gentle heat, if chloroform is present, the very unpleasant odour of phenylisonitrile will be produced.

This is a very delicate test since 1 part of chloroform in 6,000 may be detected.

The Naphthol test may also be employed. A few drops of a solution of alpha or beta naphthol, dissolved in concentrated potassium hydroxide solution, are placed in a tube and a drop or two of strong potassium hydroxide solution added. The mixture is warmed and some of the distillate is added when, if chloroform is present, the solution will assume a blue colour which will become red on the addition of an acid.

Chloroform does not give a reddish precipitate with Nessler's reagent.

Chromium.

A grey-green or grey-blue precipitate is given with ammonium hydroxide solution—chromic hydroxide—which is slightly soluble in excess of ammonium hydroxide in the cold, giving a violet or pink solution.

Sodium hydroxide solution gives a precipitate of chromic hydroxide which is readily soluble in acids and also in excess of sodium hydroxide in the cold, forming a green solution containing sodium chromite. If hydrogen peroxide is added to the sodium chromite solution, a yellow solution of sodium chromate is produced.

Sodium phosphate solution gives a green precipitate of chromium phosphate which is soluble in mineral acids, but practically insoluble in cold acetic acid.

Cocaine.

Cocaine is readily removed from a solution by the addition of ammonia and then shaking the mixture with ether, in which it will be dissolved. In watery solutions, cocaine gives a strong alkaline reaction to litmus, cochineal, and methyl-orange but it does not affect phenolphthalein.

The following tests may be employed for its detection in suspected solutions :—

To a suspected solution of cocaine hydrochloride add a few drops of a 5 per cent. solution of chromic acid. If cocaine is present, a precipitate will form as each drop of the solution is added but will, however, immediately dissolve. If a small quantity of strong hydrochloric acid is added a heavy yellow permanent precipitate will be formed. This distinguishes cocaine from strychnine, brucine, veratrine, and quinine.

Evaporate a moderately dilute solution of potassium permanganate on a microscope slide by means of gentle heat and by stroking the moist surface with a glass rod during the process of evaporation. A drop of

half-saturated alum solution is then put in the middle of the slide and a few crystals of cocaine are put into the drop over which a cover-slip is applied. Rectangular crystals, often overlapping and joined in tree-like formations, will form.

Physiological Test.

Local anæsthesia may be produced when a drop of the extract is applied to the mucous membrane.

Colchicum.

When concentrated sulphuric acid is added to colchicine an intense yellow colour develops. A particle of potassium nitrate added to the solution gives a green colour changing through violet to yellow. Excess of potassium hydroxide then changes the colour to red.

Conium.

When a drop or two of hydrochloric acid are added to coniine and evaporated the residue will show faint yellow crystals on microscopical examination. The characters of these crystals are acicular, columnar, or in cluster formation.

Copper.

To a neutral or faintly acid solution, the addition of potassium ferricyanide will produce a reddish-brown precipitate of cupric ferrocyanide. It is insoluble in dilute acids, but dissolves in aqueous ammonium hydroxide forming a blue solution. This is a delicate test.

Sulphuretted hydrogen gives a black precipitate of cupric sulphide in neutral or acid solutions, soluble in hot dilute nitric acid and also in potassium cyanide solution. Cupric sulphide is insoluble in hot dilute sulphuric acid.

Curare.

When dissolved in concentrated sulphuric acid, and a crystal of potassium dichromate is drawn through the solution, a reddish-violet colour is shown.

Digitalis.

Pure digitalin produces a yellow colour which turns red when concentrated sulphuric acid is added. A few drops of bromine water turn the colour to cherry-red or to a bluish-red.

When digitalin is dissolved in concentrated hydrochloric acid, a yellow colour is shown which turns red on the application of gentle heat.

Ergot.

The vomited matter or contents of the stomach are mixed with boiling alcohol and filtered. The process is repeated several times and the united filtrates are acidulated with dilute sulphuric acid. The bulk of the alcohol is evaporated off and a portion of residual liquid is examined spectroscopically. If ergot is present, a spectrum composed of two bands, one in the green and a second, broader and more marked, in the blue, will be observed. A parallel test should be made with a solution of ergot identical in colour tone to that of the residue. If another portion is rendered alkaline with caustic potash and then heated the characteristic odour of trimethylamine, suggestive of herring-brine, will be emitted.

An aqueous solution of ergot shows a blue fluorescence. If 0·001 gramme of ergot alkaloids is dissolved in 5 millilitres of 1 per cent. tartaric acid solution and to 1 millilitre of this 2 millilitres of p-dimethylaminobenzaldehyde solution (0·125 per cent. in 50 per cent. sulphuric acid) is added and the mixture exposed to a strong light for five minutes, a blue colour will develop.

When 0·001 gramme of ergot alkaloids is dissolved in 1 millilitre of glacial acetic acid containing a trace of ferric chloride and two drops of sulphuric acid are added, a purplish-blue colour will become manifest.

Eucalyptus.

The odour of eucalyptus is characteristic.

Fluorides.

On moistening an area of dry filter paper, previously impregnated with zirconium-alizarin solution with 50 per cent. acetic acid, the area will become red. If the suspected solution is added, the colour changes to yellow should fluorides be present. It should be noted that several interfering substances may be present in the solution under test.

Formaldehyde.

Schryver's Test.

This is a very sensitive test. To 10 millilitres of the formaldehyde containing liquid add 2 millilitres of a 1 per cent. solution (freshly made up and filtered) of phenylhydrazine hydrochloride; 1 millilitre of a fresh 5 per cent. solution of potassium ferricyanide is then added. On addition of 5 millilitres of concentrated hydrochloric acid a brilliant magenta colour is developed.

Gold.

Hydrogen sulphide gives a black precipitate in the cold which is insoluble in dilute acids but soluble, with difficulty, in yellow ammonium sulphide solution, from which it is reprecipitated by dilute hydrochloric acid.

Oxalic acid solution precipitates gold as a fine brown powder from cold neutral or acid solutions. Similar results are given with a ferrous sulphate solution.

Hydrochloric Acid.

Congo paper turned to blue.

Methyl-violet changed to blue, then green, and with strong acid to yellow.

Add dilute nitric acid and silver nitrate solution, when a heavy, white precipitate, soluble in ammonium hydroxide, will form. The precipitate is insoluble in nitric acid.

Hydrocyanic Acid.

Since hydrocyanic acid and its soluble compounds are rapidly decomposed in the body, examination for the detection of the poison should be made as soon as possible after death. Containers from which solutions may have been drunk should be submitted for analysis without delay. For its detection, advantage should be taken of the volatile nature of the poison.

A simple post-mortem room test may be made by using a strip of filter paper and placing it in blood previously treated with potassium chlorate. This converts the hæmoglobin into methæmoglobin of dark brown colour. When the strip is placed in the suspected fluid a positive reaction is shown by the development of a bright red colour due to cyanmethæmoglobin.

The following are good sensitive but not specific tests :—

Immerse a piece of filter paper in 0·2 per cent. alcoholic solution of guiacum and then in 0·1 per cent. solution of copper sulphate. Suspend the prepared paper over the mouth of a vessel containing the matter under test, when a positive reaction will be shown by the paper turning blue.

Prepare a piece of filter paper by immersion in a filtered solution of benzidine acetate in very dilute acetic acid and then in a weak solution of copper acetate. When tested as above the presence of hydrocyanic acid will be indicated by the development of a bluish-violet colour.

The following are specific tests :—

To detect the presence of hydrocyanic acid vapour the Prussian blue reaction may be utilised. Ganassini's paper is prepared by immersion in a mixture of 10 millilitres of 10 per cent. ferrous sulphate solution containing a trace of ferric salt and 20 millilitres of an alkaline solution of Rochelle

salt (30 grammes Rochelle salt, 10 grammes potassium hydroxide, and 100 millilitres of water). The paper should thereafter be exposed to air containing hydrocyanic acid and then to hydrochloric acid vapour, when it turns greenish-blue.

To a thoroughly cleaned test-tube add a drop of 5 per cent. potassium iodide, a drop of silver nitrate solution, 0·17 gramme per litre, and 1 millilitre of 5 per cent. solution of potassium hydroxide. A faint bluish colour of silver iodide will appear. Air is drawn through the solution under test and, if hydrocyanic acid is present, the potassium cyanide formed dissolves the silver iodide and renders the fluid perfectly clear. The test is claimed to be specific. The solution of silver iodide must be freshly prepared for each test. Using a strength of 0·0425 gramme per litre of silver nitrate, the limit of delicacy is about 1 part hydrocyanic acid in 2,000,000.[5]

Good qualitative tests for hydrocyanic acid and cyanides can also be obtained in the following ways :—

Two solutions are prepared and kept separately. The first reagent is prepared by dissolving 1 gramme of orthotolidine and ½ millilitre of glacial acetic acid in 50 millilitres of water. The second consists of 50 millilitres of an aqueous solution of copper acetate. Equal quantities of these reagents are mixed before the test and the resultant solution must be colourless. A piece of thick filter paper is moistened at one end with a few drops of the solution and then suspended over the material under test. A Prussian blue colour denotes a positive reaction in the presence of the acid or its salts.

If to a portion of the distillate, a few drops of phenolphthalin reagent dissolved in a dilute sodium hydroxide solution, and a small amount of copper sulphate solution are added, the characteristic red colour of alkaline phenolphthalein solution will develop in the presence of hydrocyanic acid. This test is very delicate.

The detection and estimation of micro quantities of cyanide may be effected by aerating a solution containing hydrogen cyanide and conducting the gas through a specially prepared test paper, secured by a pair of ground-glass flange connections. By this method it is claimed that the sensitivity of the Prussian blue test has been increased to detect and estimate as little as 0·2 microgram of cyanide. To prepare the test paper, "5 grammes of hydrated ferrous sulphate are dissolved in 50 millilitres of distilled water and any insoluble residue is removed by filtration. A single sheet of filter paper (Whatman No. 50, smooth-glazed, acid- and alkali-treated) is immersed in this solution for five minutes, then removed from the ferrous sulphate solution, suspended by means of a clamp, and allowed to dry in the air. The dried piece of filter paper is then dipped into a 20 per cent. sodium hydroxide solution. When the paper is thoroughly wetted it is removed and again allowed to dry in the air. Circular pieces of the paper having exactly the same diameter as the ground-glass flanges are cut out. These test papers will retain their usefulness for several weeks if stored in a cool dark place."[6]

Failure to detect the poison in the body may result from delay, but notwithstanding its volatility and liability to decomposition it has been detected several weeks after death. Formic acid is the main product of the metamorphosis of the poison in the body and it is important, therefore, that the presence of formic acid should be sought, and its amount estimated in those cases in which poisoning by hydrocyanic acid is suspected and where the poison as such cannot be found on analysis of the organs.

Iodine and Iodides.

Acidify solution with sulphuric acid and shake with chloroform. The chloroform will develop a violet hue if iodine is present. If no colour develops add a little sodium nitrite solution and again shake. If iodides are present iodine will be liberated and dissolve in the chloroform.

Iodoform.

To a little of an alcoholic solution of the suspected material in a small tube, add a solution of crystallised phenol in concentrated potassium hydroxide solution

and heat gently. A positive result will be shown when a red deposit forms at the bottom of the tube. The addition of a few drops of dilute alcohol will dissolve the precipitate and a carmine colour will appear.

Iodoform also gives a disagreeable odour of phenylisonitrile when warmed with aniline and alcoholic potash solution.

Iron.

Ferrous Salts.

First acidify with a small quantity of dilute sulphuric acid.

Sodium hydroxide solution gives a white precipitate of ferrous hydroxide in the complete absence of air, insoluble in excess but soluble in acids. Upon exposure to air ferrous hydroxide is rapidly oxidised, yielding ultimately reddish-brown ferric hydroxide. Under ordinary conditions it appears as a dirty green precipitate. The addition of hydrogen peroxide immediately oxidises it to ferric hydroxide.

Ammonium sulphide solution gives a black precipitate of ferrous sulphide which is readily soluble in acids with liberation of hydrogen sulphide. The moist precipitate becomes brown upon exposure to air, due to its oxidation to basic ferric sulphate.

Ammonium thiocyanate solution fails to yield a coloration with pure ferrous salts.

Ferric Salts.

Ammonium hydroxide solution gives a reddish-brown, gelatinous precipitate of ferric hydroxide, insoluble in excess of the reagent but soluble in acids.

Sodium hydroxide solution produces a reddish-brown precipitate of ferric hydroxide, insoluble in excess of the reagent.

Hydrogen sulphide reduces to ferrous salt in the presence of acid, with separation of sulphur.

Laburnum.

A solution of ferric chloride produces a red colour which disappears when hydrogen peroxide is added. The application of heat causes blue coloration.

Lead.

Dilute sulphuric acid gives a white precipitate of lead sulphate, insoluble in excess, but soluble in concentrated solution of ammonium acetate, or in ammoniacal solution of ammonium tartrate, or in a warm solution of sodium thiosulphate.

Add to a solution of lead salt a little potassium iodide solution, avoiding excess. A yellow precipitate is produced which is soluble on boiling (with the addition of more water if necessary). On cooling, golden yellow tabular crystals separate.

Sulphuretted hydrogen gives a black precipitate insoluble in ammonium sulphide, but soluble in boiling dilute nitric acid. This is a very delicate reaction.

Potassium chromate gives a yellow precipitate insoluble in acetic acid and in ammonia solution, but soluble in alkali hydroxides and in nitric acid.

Lobeline.

Lobeline is extracted from an alkaline aqueous solution by ether and gives with Frochde's reagent (one part of molybdic acid dissolved in one hundred parts of strong sulphuric acid) a deep violet colour which gradually fades.

Manganese.

Ammonium sulphide gives a salmon-coloured precipitate readily soluble in dilute acids and in acetic acid. Boiling with excess of ammonium sulphide solution converts the pink colour into a greenish colour.

Sodium hydroxide forms a white precipitate insoluble in excess, and the precipitate on exposure to air becomes brown in colour.

Ammonium chloride followed by a slight excess of ammonia produces no immediate precipitate. On shaking with air a brown flocculent precipitate forms.

Mercury.

Mercury compounds when mixed with sodium carbonate and heated in a sublimation tube yield a sublimate of minute globules of mercury.

When in organic substances, mercury may be detected by the Reinsch test. The deposit on copper foil has a silvery appearance and on heating the foil in a sublimation tube sublimation occurs.

The sublimate may be dissolved with nitric acid and the following liquid tests applied :—

Potassium iodide added to a solution of mercuric salt gives a bright scarlet precipitate, soluble in excess.

Sulphuretted hydrogen gives a precipitate which changes from white to yellow and black as more sulphuretted hydrogen is added and is insoluble in ammonium sulphide, also in boiling dilute nitric acid.

Stannous chloride gives a white precipitate, changing to grey due to formation of mercury, on adding more stannous chloride. The change of colour is accelerated by heating.

If a bright steel needle is immersed in the original solution the mercury will be deposited upon the needle as a grey film.

Nickel.

Sodium hydroxide gives a green precipitate insoluble in excess. The precipitate dissolves in ammonium hydroxide or in solutions of ammonium salts, forming a greenish blue solution.

Nickelous hydroxide is oxidised by sodium hypochlorite solution to black nickelic hydroxide.

Nicotine.

Schindelmeiser's Test.

If a drop of 30 per cent. chemically pure formaldehyde solution and a drop of concentrated nitric acid are added to nicotine a rose-red colour is shown which is not produced by coniine.

Roussin's Crystal Test.

A very small quantity of nicotine is dissolved in ether and the same quantity of ethereal solution of iodine is added. After a period Roussin's crystals will form. These are ruby-red and needle-shaped.

Nitric Acid.

Congo paper turned to blue.

Methyl-violet changed to blue, then green, and with strong acid to yellow.

Add a few drops of diphenylamine sulphate solution and with care pour the mixture upon pure concentrated sulphuric acid, free from nitric acid. If nitric acid is present a blue zone appears at the junction of the two fluids.

Mix the liquid with an equal volume of concentrated sulphuric acid and cool. Add as an upper layer a strong solution of ferrous sulphate. In the presence of nitric acid a brown ring forms at the interface.

Add a small piece of copper to nitric acid and heat. Reddish-brown fumes of nitrogen peroxide will appear.

Nitrous Fumes.

Pass the air suspected of containing nitrous fumes through 40 millilitres of 1 per cent sodium hydroxide solution. Neutralise with acetic acid and test for nitrite as follows: Add 2 millilitres of Ilosvay's solution No. 1 (0·5 gm. of sulphanilic acid, 30 millilitres of glacial acetic acid, 120 millilitres of water), followed by 2 millilitres of Ilosvay's solution No. 2 (0·5 gm. of naphthylamine, 30 millilitres of glacial acetic acid, 120 millilitres of water). A strong pink colour develops in the presence of nitrite.

The test is delicate and it is advisable to carry out a control test on the reagents.

Opium and Morphine.

If the liquid consists of comparatively clear stomach washings acidulate with acetic acid, evaporate at low temperature to small volume in a water bath, and filter. To the clear filtrate add slight excess of lead acetate which will precipitate lead meconate together with albuminous matter. After the precipitate has settled, filter through a small moistened filter and wash residue in filter with distilled water. Both the filtrate and the residue should be examined, the former for morphia, after removal of lead and extraction by ethyl acetate from ammoniacal solution, the latter for meconic acid which must be recovered from this precipitate by hydrogen sulphide. Meconic acid gives a reddish-purple colour with ferric chloride which is not discharged by boiling with hydrochloric acid or by adding mercuric chloride.

The following tests may be applied to the purified morphine extracts :—

Concentrated nitric acid produces a red colour which rapidly changes to yellow. To distinguish from brucine, stannous chloride or ammonium sulphide does not cause the yellow colour to be replaced by a blue colour.

Neutral ferric chloride will produce a temporary blue colour in a neutral solution of a morphine salt.

Marquis's Test.

A few drops of a mixture of concentrated sulphuric acid (30 millilitres) containing formaldehyde (twenty drops) when added to a morphine residue will produce a purplish-red colour passing to pure blue. Codeine and apomorphine produce a violet colour.

Oxalic Acid.

Turns blue litmus paper red.

Addition of a solution of a calcium salt produces a white precipitate insoluble in ammonia and acetic acid, but soluble in hydrochloric acid.

Calcium oxalate crystals have an envelope shaped appearance and are characteristic octahedrons.

Silver nitrate gives a white precipitate of silver oxalate soluble in ammonia and nitric acid.

Lead acetate gives a white precipitate of lead oxalate which is soluble in nitric acid but insoluble in acetic acid.

Note.—The above chemical tests, with the exception of the first, may also be employed to detect potassium binoxalate.

Phenacetin or Acetophenetidin.

Autenrieth-Hinsberg Test.

The solution or residue containing phenacetin is heated to boiling-point with a 10 per cent. solution of nitric acid, when a yellow or orange-red colour is produced. If the solution is saturated, long yellow needles will be deposited. Acetanilide and antipyrin do not show the coloration given by this test.

Phenazone or Antipyrin.

This drug gives with ferric chloride a dark red colour. Dilute mineral acid changes the red to a pale yellow. It may thus be detected in the urine of habitual users.

Phenols.

The odour is significant.

Acidify solution slightly with dilute sulphuric or tartaric acid and distil. Apply the following tests to the distillate :—

Add excess of bromine water when a whitish, or yellowish white precipitate of tribromophenol will be formed. Collect precipitate, wash and heat gently

in a test tube with sodium-amalgam and water, pour mixture into a large watch glass and acidulate, when characteristic odour of phenol will be perceived.

To a second portion of distillate add dilute ammonia and a few drops of 1 in 20 solution of freshly prepared bleaching powder and heat, when a bluish or greenish colour will develop which on acidulation will be changed to red or yellow.

To a third portion of the distillate add a few drops of ferric chloride, when a violet colour will be produced. The addition of hydrochloric acid or sulphuric acid changes this colour to yellow. The colour also disappears upon the addition of alcohol.

To a fourth portion of the distillate add one to two drops of Millon's reagent and boil mixture, when a red colour will develop.

Phosphoretted Hydrogen.

See Phosphorus.

Phosphorus.

Some dependence must be placed on the garlicky odour of vomited material, fæces, and stomach contents. Importance should be attached to the luminosity in the dark.

A portion of the material should be taken up in a separator with carbon disulphide and after separation the disulphide is allowed to evaporate spontaneously in darkness, the observer keeping watch for luminosity. Any phosphorus present will show as oily globules, which on being touched with a red hot wire will burst into a bright yellow flame.

This test is not always reliable as the luminosity may be inhibited by many organic compounds (alcohol, ether, phenol, volatile oils, turpentine, etc.; and by hydrogen sulphide and oxidising agents).

Scherer's Test.

This is a good preliminary test and depends upon the reducing effect of phosphorus fumes on silver nitrate.

The material is placed in a Gutzeit flask and cold water is added with a small quantity of cadmium sulphate. The fluid is acidified with dilute sulphuric acid and the flask is corked with a cork provided with two slits, one on each side. Into these are placed strips of test paper which should hang freely and clear of the cork. One piece is saturated with 5 per cent. silver nitrate and the other with a solution made by adding caustic soda to a 5 per cent. solution of lead acetate until the precipitate is dissolved. The flask is placed in relative darkness and warmed for some time. To interpret the results the papers should be examined after an interval of an hour or two. If the strip saturated with silver acetate is darkened and the other saturated with the prepared solution is unchanged phosphorus may possibly be present. If the first strip remains unchanged phosphorus is absent. If both strips are darkened the result may be due to sulphuretted hydrogen.

Mitscherlich's Test.

The suspected material is mixed with water acidulated with sulphuric acid and is transferred to a glass retort fitted with a long condenser. To the tube from the retort is fitted an adaptor which ends in a receiver containing a solution of silver nitrate. Distillation is made in the dark, luminosity in the tube being evidence of the poison. This process will detect one part of phosphorus in a hundred thousand parts of material.

Dusart-Blondlot's Test.

This depends upon the combination of phosphorus with nascent hydrogen and the formation of phosphoretted hydrogen. The gas is generated as in the Marsh process (see p. 708). It gives on ignition at a platinum jet a flame which is somewhat greenish in colour, particularly in its middle portion. Some of the gas may be passed into silver nitrate solution when black silver phosphide is precipitated. By the action of water this is converted to metallic silver and phosphoric acid.

Physostigmine or Eserine.

Physiological Test.

A drop or two of a solution containing physostigmine placed in the eye of a cat will cause pupillary contraction within a fairly short period.

Chemical Tests.

An aqueous solution yields a violet colour on the addition of a solution of ferric chloride.

Warm a few milligrams with several drops of dilute solution of ammonia, when a yellowish-red colour is produced. Evaporate this solution and a blue residue will remain. The residue is soluble in 95 per cent. alcohol and forms a blue solution. On the addition of acetic acid the solution appears blue by transmitted light and shows a red fluorescence, heightened by dilution with water. The residue is also soluble in sulphuric acid and assumes a green colour, which on the gradual addition of 95 per cent. alcohol changes to red but reverts to green when the alcohol is evaporated.

Picrotoxin.

A frog is injected with suspect fluid which, if picrotoxin is present, will produce convulsions of clonic type.

Concentrated sulphuric acid gives an orange colour which is discharged on the addition of a drop of concentrated nitric acid.

Fuming nitric acid gives a blue to green colour which disappears on addition of excess acid.

Concentrated sulphuric acid and benzaldehyde give a red colour which on warming turns purple.

Pilocarpine.

To the extract add a particle of potassium dichromate and 1 to 2 millilitres of chloroform and also 1 millilitre of a 3 per cent. solution of hydrogen peroxide in a test tube. Shake the contents for several minutes and this will cause the mixture to assume a dark brown colour, while the chloroform becomes bluish-violet or deep blue, depending upon the amount of pilocarpine present. Free acid or alkali prevents this reaction.

Potassium Chlorate.

Add dilute sulphuric acid and a little indigo-carmine solution until a bluish colour is obtained. Add a few drops of sulphurous acid when the blue colour will change to yellow or greenish-yellow. This is a delicate test.

Acidify the solution with an equal volume of concentrated sulphuric acid. Add two drops of a 5 per cent. solution of aniline sulphate when a blue colour will develop.

Potassium Hydroxide.

Test with moist red litmus paper. Colour is turned to blue.

Remove interfering ammonium salts by ignition or by boiling the solution with calcium hydroxide. Add acetic acid till faintly acid and then a little strong solution of sodium cobaltinitrite. Small quantities of potassium salts yield a heavy yellow precipitate.

Potassium Nitrate.

Heat concentrated solution in test tube with concentrated sulphuric acid and copper filings, when the nitrate will produce reddish fumes of nitrogen oxides.

Mix 1 millilitre of the solution to be tested with 3 millilitres of concentrated sulphuric acid and cool thoroughly. Add a trace of brucine when a red colour changing to orange will appear.

(See also tests under Nitric Acid (p. 718), and Potassium Hydroxide.)

Potassium Permanganate.

Acidify a solution with sulphuric acid and add sulphurous acid which discharges the colour. The solution will then respond to the tests under Manganese (see p. 717).

Acidify a solution with sulphuric acid and add either ferrous sulphate solution or oxalic acid solution and heat. In both cases the colour will be discharged.

Potassium and Sodium Bromide in Blood Serum or in Urine.

Hauptmann's Modification of Walther's Test.

Mix 2 millilitres of serum with 4 millilitres of water and 1·2 millilitres of 20 per cent. trichloracetic acid. After shaking allow to stand for ten minutes and filter. To 2 millilitres of the filtrate add 0·4 millilitre of 0·5 per cent. gold chloride solution. The coloured solution is compared with known standards graded in 10 milligram stages, from zero to 250 milligrams and upwards, if necessary. The results can be gauged either by the naked eye or by colorimetric methods. Hall[7] has introduced a rapid quantitative test for bromide in blood and urine. It is a spot test and utilises the reaction between bromide and gold chloride. A mixture of 20 per cent. trichloracetic acid and 0·25 per cent. gold chloride is added to a drop of serum spread on a white porcelain surface. A positive reaction is shown by colour changes from yellow to red-brown depending on the concentration of bromide present. Twenty-five milligrams bromide per 100 millilitres equals yellow colour; 50 milligrams bromide and over per 100 millilitres equals red-brown.

Pyrogallic Acid.

Extract residue with alcohol, filter, and evaporate to dryness. The residue is treated with water and shaken up with ether when it dissolves the acid. On evaporation of the ether the acid is isolated. To this apply the following tests :—

On adding lime water a purple colour is produced.

With a solution of ferrous sulphate a bluish-black colour is formed.

Quinine.

When dissolved in dilute sulphuric acid it produces a blue fluorescence which is accentuated on examination under filtered ultra-violet light.

If twenty drops of a mixture of thirty drops of acetic acid, twenty drops of absolute alcohol, and one drop of 20 per cent. sulphuric acid are added to a diluted solution of quinine and heated to boiling, after adding one drop of an alcoholic solution of iodine, green leaflets with a metallic lustre will appear.

Santonin.

Add Fehling's solution to a sample of suspected urine: a positive result will be indicated by the formation of a reddish-violet colour. On adding an excess of acetic acid the colour is converted to green.

Silver.

Dilute hydrochloric acid gives a white precipitate of silver chloride which darkens on exposure to light. The precipitate is insoluble in water and in acids, but is soluble in dilute ammonium hydroxide. It is precipitated from the ammoniacal solution by dilute nitric acid and by potassium iodide solution.

Potassium iodide gives a yellow precipitate of silver iodide which is insoluble in ammonium hydroxide solution, but is readily soluble in solutions of potassium cyanide and of sodium thiosulphate.

A red precipitate of silver chromate is given with potassium chromate solution. The precipitate is insoluble in dilute acetic acid, but is soluble in dilute nitric acid and in ammonium hydroxide solution.

Sodium Hydroxide.

As potassium hydroxide.

To the nearly neutral solution add a solution of zinc uranyl acetate. In presence of sodium a yellow precipitate forms.

Strophanthus.

When a solution of strophanthus is treated with sulphuric acid an emerald-green colour develops and changes to brown. When warmed, the colour changes to violet shades and then becomes black.

In aqueous solution the addition of some sulphuric acid and a very small amount of ferric chloride will cause a reddish-brown precipitate, which after standing for some time becomes emerald-green in shade.

The addition of some sodium nitro-prusside solution and alkali will produce a red colour when strophanthus is present.

Strychnine.

The taste test should be performed with caution. It is of value in demonstrating the presence or absence of a bitter taste even in very high dilutions.

By injecting some solution into the thoracic or abdominal cavity of a frog, tetanic convulsions will be caused if strychnine is present to the extent of $\frac{1}{30000}$ grain.

If to the crystals two drops of strong sulphuric acid are added no reaction will be noticed, but if the edge of the solution is touched with a particle of manganese dioxide or potassium dichromate a deep-blue colour will form at the point of contact. This colour rapidly changes to purple, crimson, red, orange, and then slowly fades away. The play of colours is characteristic of strychnine and can be perceived with $\frac{1}{100000}$ grain.

Sulphonal.

Test the urine after making alkaline and shaking with amyl alcohol for the presence of hæmatoporphyrin, by spectroscopic examination (see p. 335).

Sulphuretted Hydrogen.

Sulphuretted hydrogen gas in the stomach and tissues may be detected by the addition to the filtrate of a solution of lead acetate, which produces a black precipitate. The gas when found in small quantity may merely be the result of decomposition changes.

On examination of the blood a spectrum of sulphæmoglobin with a narrow band towards the red end of the spectrum, about 615, will be seen. It is distinguishable from that of methæmoglobin by the fact that the narrow band does not disappear after the addition of a reducing agent as occurs with methæmoglobin. Sulphæmoglobin produces a greenish coloration of the surface vessels of the cadaver.

Sulphuric Acid.

Congo paper turned to blue.

Methyl-violet changed to blue, then green, and with strong acid to yellow.

Sugar Test.

Concentrated sulphuric acid will produce charring.

To the diluted acid add barium chloride solution. A white precipitate insoluble in hydrochloric acid is formed.

Thallium.

A delicate test can be made by adding to a solution of thallium, a few drops of carbon disulphide, a slight excess of ammonia, and a little ammonium sulphide. Heat gently till the carbon disulphide boils, when a black precipitate changing to red appears.

Tin.

Mercury chloride with solutions of stannous compounds gives a white precipitate, but does not give a precipitate with solutions of stannic compounds.

Sodium hydroxide gives a white precipitate soluble in excess of the reagent.

Trinitrotoluene, Trotyl, or T.N.T.

Webster's Test as modified by Tutin.

Extract urine with two successive portions of ether, acidify urine with hydrochloric or sulphuric acid, and again extract with ether. The ethereal extract of the acidified urine must then be extracted twice with dilute aqueous sodium carbonate, washed with water, and treated with alcoholic potash. The characteristic violet colour will now become evident in the presence of derivatives of T.N.T. in the urine under test.

Turpentine.

A small crystal of iodine added to one drop of turpentine on filter paper will fume. If three drops of turpentine are added rapidly to a little iodine in a narrow tube the reaction is explosive.

Veratrine.

The alkaloid veratrine is taken up by chloroform from alkaline aqueous solution. After one part of veratrine has been triturated with five parts of faintly powdered cane sugar the addition of a few drops of concentrated sulphuric acid will produce a yellow colour which changes to greenish-blue.

Veratrine dissolved in concentrated sulphuric acid and gently heated will produce a yellow colour which changes to red. The change of colour is gradual.

Zinc.

In a filtrate made alkaline with sodium carbonate, the absence of a precipitate may be taken as excluding any large quantity of zinc.

To a neutral or alkaline solution add ammonium sulphide, when a white precipitate of zinc sulphide will develop. The precipitate is insoluble in excess of ammonium sulphide, in acetic acid, and in solutions of caustic alkalis, but dissolves in dilute mineral acids.

Add potassium ferrocyanide solution to a portion of the filtrate and a white precipitate of zinc ferrocyanide will form which is insoluble in dilute acids, but soluble in solutions of caustic alkalis.

The addition of sodium hydroxide solution to some of the filtrate will produce a white gelatinous precipitate of zinc hydroxide which is readily soluble in excess. The precipitate also dissolves in dilute acids.

References

1. Biochem. Jour., Vol. XVIII, 101, 1919; Vol. XIX, 737, 1919. Jour. Indust. Eng. Chem., Analytical Edition, Vol. XIII, 905, 1941, Kozelka and Hine.
2. Amer. Jour. Clin. Path., Vol. VII, 85, 1937, Gettler and Siegel.
3. "Practical Physiological Chemistry," 483-484, Hawke and Bergeim, Churchill, 1948.
4. "Recent Advances in Forensic Medicine," Smith and Glaister, 2nd ed., Churchill, 1938.
5. Science, N.S., Vol. LXXIX, 37, 1934.
6. Journal Analytical Chemistry, 270, April, 1947, Gettler and Goldbaum.
7. Lancet, Vol. II, 355, 1943, Hall.

ADDENDA.

THE FOLLOWING INFORMATION HAS BECOME AVAILABLE SINCE THE PRINTING OF THE BODY OF THE BOOK :—

National Insurance (Industrial Injuries). Prescribed Diseases.

The following have been added to the First Schedule, Part I :—

DESCRIPTION OF DISEASE OR INJURY.	NATURE OF OCCUPATION.
37. (*a*) Carcinoma of the mucous membrane of the nose or associated air sinuses. (*b*) Primary carcinoma of a bronchus or of a lung.	Any occupation in a factory where nickel is produced by decomposition of a gaseous nickel compound which involves work in or about a building or buildings where that process or any other industrial process ancillary or incidental thereto is carried on.

DEATH CERTIFICATE FORM USED IN ENGLAND. (Front)

COUNTERFOIL

For use of Medical Attendant, who should in all cases fill it up.

Name of Deceased ....

Date of Death ..... Age.....

Place of Death .....

.....

Last seen alive .....

Seen* / Not seen* after death.

Cause of death confirmed* / not confirmed* by P.M.

Cause of Death :—

I *a*.....
due to

b.....
due to

c.....

II .....

Signature .....

Date.....

* Strike out whichever is inapplicable

P.96

BIRTHS AND DEATHS REGISTRATION ACTS, 1836 TO 1929

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Registrar to enter No. of Death Entry.

.....

For use only by a Registered Medical Practitioner **WHO HAS BEEN IN ATTENDANCE** during the deceased's last illness, and to be delivered by him forthwith to the Registrar of Births and Deaths.

Name of Deceased.....
Date of Death as stated to me.....day of.....19... Age as stated to me.....
Place of Death.....
Last seen alive by me .....day of.....19... Seen* / Not seen* after death by me

The certified cause of death has* / has not* been confirmed by Post-mortem.

CAUSE OF DEATH

		These particulars not to be entered in death register. Approximate interval between onset and death.
I Disease or condition directly leading to death† . . .	*a* ..... *due to (or as a consequence of)*	.....
Antecedent causes. Morbid conditions, if any, giving rise to the above cause stating the underlying condition last.	*b* ..... *due to (or as a consequence of)*	.....
	c	.....
II Other significant conditions, contributing to the death, but not related to the disease or condition causing it.	.....	.....
	.....	.....

I hereby certify that I was in medical attendance during the above named Deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature..... *Qualifications as registered by Medical Council* .....

Residence..... *Date*.....

* Strike out whichever is inapplicable.

† This does not mean the mode of dying, such as *e.g.*, heart failure, asphyxia, asthenia, etc., it means the disease, injury, or complication which caused death.

NOTICE TO INFORMANT

I hereby give notice that I have, this day, signed a Medical Certificate of the Cause of Death

of .....

..... deceased

Signature .....

Date.....

This Notice must be given by the Certifying Medical Practitioner to the person who is qualified and liable to act as Informant for the purpose of the registration of the death. As to the person liable to act as Informant, see back.

DUTIES OF INFORMANT

The Informant must deliver this Notice to the Registrar of Births and Deaths of the Sub-district in which the Death took place, bearing in mind that the Death cannot be registered until the medical certificate has reached the Registrar. Failure to deliver this Notice to the Registrar renders the Informant liable to prosecution.

The Informant must be prepared to state accurately to the Registrar the following particulars :—

(1) The Date and Place of Death, and the place of Deceased's usual residence (2) the full Names and Surname (3) the correct Age (4) the Occupation and (5) whether Deceased was in receipt of a naval or military pension, an old age pension, or other pension or allowance from public funds.

DECEASED'S IDENTITY CARD, RATION BOOKS AND MEDICAL CARD MUST BE DELIVERED TO THE REGISTRAR.

INDEX

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NOTE.—Poisonous substances and gases will be found under "Poisoning." Certain of the commoner poisons are also included in the General Index.

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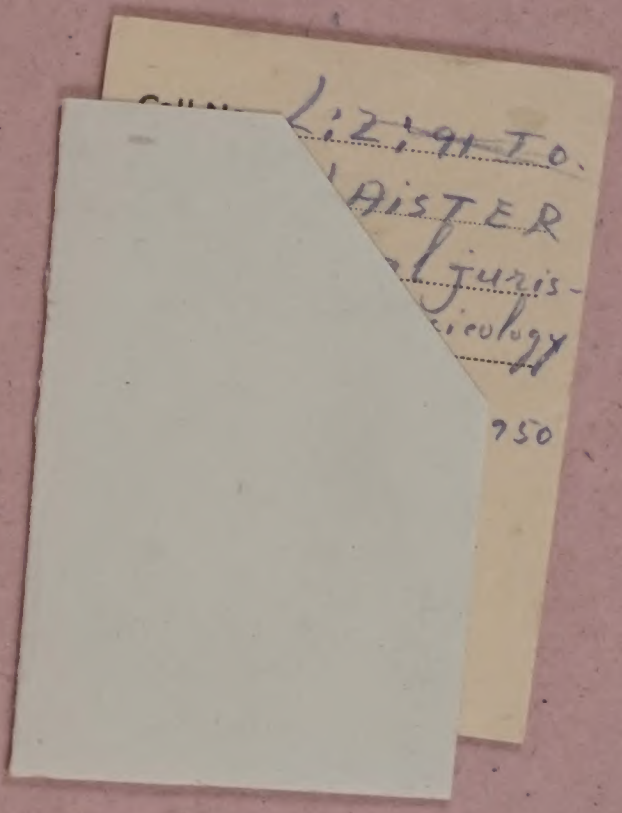
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