BLOOD AND OTHER STAINS

When blood is found at the scene of a death or on an item of evidence, the two principal questions arising are: whose blood is it? And, under what circumstances was it deposited? The first task is to establish that the stain is blood and that it is of human origin. If it is human blood, the next task is individualisation, establishing whose blood it is. The pattern and distribution of the bloodstains can provide information which allows a reconstruction of how the stains were deposited.

Presumptive and confirmatory tests

Simple, quick presumptive tests for blood are used at the scene and in the laboratory as a screening test to separate out likely bloodstains from other stains which mimic them. All of the currently used presumptive screening tests make use of the fact that the haeme group of the haemoglobin of red blood cells exhibits a peroxidase-like activity, so that it catalyses the breakdown of hydrogen peroxide to water with the release of oxygen. This peroxidase-induced reduction of hydrogen peroxide is coupled in the various tests to the oxidation of a colourless reduced dye to its coloured form. Colourless phenolphthalein used in the Kastle-Meyer test turns pink; colourless leucomalachite green turns blue-green; and tetramethylbenzidine is oxidised to its green form.

All three chemicals are highly sensitive to minute traces of haemoglobin but also give false positive reactions with chemical oxidants, particularly copper and nickel salts, and plant sources containing the enzyme peroxidase, such as apple, horseradish, potato and cabbage. Chemical oxidants will give a colour change without the addition of the hydrogen peroxide and therefore can normally be excluded by first testing whether the stain produces a colour change in the dye without the addition of hydrogen peroxide substrate. Heating the sample stain or an extract of it to 100°C for five minutes will inactivate plant peroxidases but not the peroxidase activity of haemoglobin.

For testing, the stain is lightly rubbed with a clean cotton swab or the corner of a folded piece of filter paper moistened with distilled water and then the reagent and hydrogen peroxide are added. Alternatively a small sample of the stain can be scraped away, or if it is mixed with material such as sand or soil it can be dissolved in water and the supernatant tested. A negative test result is proof of the absence of detectable quantities of haemoglobin, which constitutes most of the protein content of red blood cells. A positive colour test is not positive evidence of the presence of blood but rather an indication to proceed to confirmatory testing. Screening large areas of a scene for the presence of blood is possible by spraying with luminol (3-amino-phthalhydrazide) which is oxidised to a luminescent product visible in the dark.

Confirmatory tests, as the name implies, confirm the presence of blood. Immunological methods make use of commercially produced specific antibodies to human serum proteins and to human haemoglobin so that a positive test result is proof of the presence of specifically human blood. Positive crystal tests for the presence of haeme derivatives provide conclusive proof of blood but are not species-specific. The confirmatory crystal tests were devised by Teichmann in 1853 and by Takayama in 1912. The Teichmann test produces rhombic or prismatic dark brown 10µ crystals of haematin halide. The Takayama test produces salmon-pink pyridine haemochromagen crystals. Bloodstains 10-20 years old may still give positive crystal tests. Spectrophotometric methods are not currently in use but, in the past, demonstrating the characteristic absorption spectrum of haemoglobin was considered conclusive proof of blood.

DNA probes complimentary to primate specific DNA sequences can be used to establish that the stain is human blood. These probes are widely used in DNA laboratories to determine the amount of human DNA extracted from a sample prior to DNA typing. This testing must be complimented with a haeme identification technique to establish that the DNA was from blood and not any other human tissue or fluid.

Foetal blood has a distinct form of haemoglobin containing a gamma subunit, which is still detectable up to six months after birth using antisera specific for foetal haemoglobin, but there are difficulties in applying the technique to dried and old stains. Suggested techniques for the identification of menstrual blood are based upon the presence of high concentrations of fibrinogen degradation products and isoforms of the enzyme lactate dehydrogenase (LDH), specifically LDH4 and LDH5.

Individualisation, which is the attribution of a bloodstain to a named individual, is discussed in the chapter on genetic identification.

Bloodstain pattern analysis

Fresh blood stains on white cloth appear bright red but gradually become a reddish-brown within around 24 hours and dark brown to black within a few days, due to the conversion of haemoglobin into methaemoglobin and haematin, and then they remain that colour indefinitely.

Lacerations, incised wounds and stab wounds, whatever the circumstances of their creation, haemorrhage and may leave bloodstains at the scene, on the clothing and on the skin. Natural disease with internal haemorrhage may result in the vomiting of
blood, as with a bleeding gastric ulcer, or the coughing up of blood, as with a carcinoma of the lung or tuberculosis. All of these bloodstains can provide information on the circumstances of their formation through an analysis of their shape, pattern and distribution.

Blood spots come in different sizes and shapes. When blood passively drips off an object, such as from the tip of a bleeding nose or the fingertip of a bleeding hand, the droplet grows in size until its mass overcomes the surface tension of the blood and breaks free. The average size of such a droplet is around 0.05ml and it falls through the air in the shape of an oscillating sphere. If it strikes a horizontal surface from the perpendicular, at an angle of 90°, the blood spot formed will be round. If the surface struck is smooth, hard and non-porous, such as a glazed tile, then the stain will be neatly circular. However, if the surface struck is rough, such as wood or concrete, then the droplet will tend to break up on impact to produce radiating fine spicules at the edges of the circular stain. The same sized blood droplet falling from different heights will produce slightly larger diameter bloodstains up to a fall of 6 feet (1.8metres), after which there is no change in diameter.

When a blood droplet strikes a surface at an angle of less than 90° the resulting stain is oval, and as the angle of impact decreases so too the resultant bloodstain becomes longer and narrower. These long thin stains typically have the shape of a teardrop or exclamation mark (!), with a small cast-off droplet at one end. The pointed end of these stains, and the small cast-off droplet if present, points in the direction of travel and away from the point of origin. Similar long thin stains are commonly seen on the sides of vehicles from mud-splatting off the wheels. The angle at which the blood droplet struck the surface to produce the elongated shape is the arc sin of the width-to-length ratio (the stain width divided by the stain length). In measuring the stain length the thin trailing tail is disregarded and the length of the main oval portion of the stain is taken. Once the width-to-length ratio is calculated the arc sin can be obtained from published mathematical tables.

Thus, a blood spot indicates both the angle of impact and the direction of impact. If a piece of string is pinned to the stain and extended in the direction of origin and at the correct angle of impact, using a protractor for this purpose, then the origin of the blood stain must be along the line of the string. Using several adjacent bloodspots and running strings from them allows determination of a common point of origin where the strings cross. In this fashion complex 3-D reconstructions of several points of origin for large numbers of bloodstains can be produced at a crime scene.

Blood trails result when blood drips from a wounded person or from a bloody weapon as the person or weapon is moving above the surface. In blood trails the shape of the bloodspots may indicate the direction of movement.

Some bloodspots are formed not by passively dripping blood but as a result of dispersion due to the application of some force, such as sneezing, the rapid movement of a bloody hand, or a blow struck to a bloody head. The application of force breaks up the blood into droplets much smaller than those seen in passively dripping blood. In high velocity (high force) impact an aerosol of blood droplets, the great majority less than 1mm in diameter, is produced. These fine droplets will not travel more than 3 feet (0.9metres) from their source because of their small mass and the effect of air resistance. The resultant fine aerosol spray of high velocity impact blood spatter on objects within 3 feet is a typical result of a gunshot wound to bare skin. Sneezing, coughing and even speaking with blood in the mouth can generate an aerosol of blood mimicking the high velocity impact blood spatter from a gunshot wound.

Low velocity (low force) impact, such as from a punch, will put in flight a smaller number of somewhat larger droplets, the great majority larger than 3mm in diameter. Increasing force increases the number of blood droplets and the distance they travel from the source. However, any aerosol droplets of around 1mm that are produced will not travel more than 3 feet from the source. Thus, an examination of the size of the blood droplets gives an indication as to whether it was freely dripping blood or whether external force was applied and whether that force was a low, medium or high velocity impact.

Objects which become coated with blood and are then rapidly accelerated or decelerated as they are swung, will cast off blood in much the same way that a rapid movement can flick paint off a paintbrush, or ink off a pen. The blood drops produce on surrounding surfaces a cast off bloodstain pattern. For example, axe blows rained down upon the head of a victim in a room will result in a cast off bloodstain pattern on the ceiling of the room from the upward swing of the bloodied axe. At the high point of the arc of the swing the blood drops will strike the ceiling above at right angles leaving circular blood spots, approximately above the assailant. As the axe swings over the shoulder of the assailant the cast off blood will strike the ceiling at decreasing angles producing first teardrop and then exclamation mark blood spots in a line. The complete line of blood spots on the ceiling indicates the approximate position of the assailant below and the direction of the backwards swing of the weapon. Repeated swings, which tend not to be in exactly the same line, will result in a series of converging, fan-shaped lines of cast off pattern. From the number of lines of cast off it may be possible to determine the minimum number of blows struck. The width of a
single line of cast off generally reflects the size of the bloody object, so that cast off from a bloodied hand can leave a broad band of bloodstains. A cast off pattern may be seen on furnishings and walls and may be complex where walls meet at a corner. Although cast off is generally on the upswing of a weapon it is possible to have forward cast off occurring on the downswing when there was little blood loss from the weapon on the preceding upswing because it was a relatively slow motion, such as occurs when blows are not delivered in very rapid succession.

When an artery is severed, and the associated wound is open and not covered by clothing, the blood may spurt out of the wound under the force of arterial blood pressure. The effect is to project large volumes of blood, rather than blood droplets, which strike adjacent surfaces and then break-up into droplets which are splashed outward across the surface. When the victim is standing still the fluctuating systolic/diastolic blood pressure may produce a zigzag pattern of projected bloodstains. Large volumes of blood oozing out of a wound and falling to the ground may produce a similar pattern to projected blood. Blood which is coughed up and mixed with saliva typically contains air bubbles.

Contact or transfer bloodstain patterns arise when a bloody object contacts an unstained surface. Many contact bloodstains are nondescript but others may transfer a pattern from the bloody object, the classic example being bloody fingerprints. Other examples include bloody footwear prints and contact bloodstains where a bloody weapon has been wrapped in or allowed to lie upon fabric. Contact with bloody hair leaves a characteristic pattern from the trailing hairs, similar to a paintbrush effect but more chaotic, and may include attached bloody hairs as confirmation.

Blood stain patterns on clothing resulting from blood dripping under the influence of gravity from a wound, such as a bleeding nose or stab to the throat, may indicate, from the angle of impact, the position of the victim when the bleeding was occurring. Similarly blood flow across the body or over clothing under the influence of gravity reflects body position. Bloodstains on the body and its clothing which are of evidential value should be recorded at the scene since transportation of the bloody body may cause additional staining, obscuring the original patterns. It may be advisable to remove the clothing at the scene to preserve this evidence. Bloodstain patterns at the scene should be documented by photography with and without scales, and by sketches and notes.

Semen

Semen, male ejaculate, has an average volume of 3ml (range 1-6ml) and comprises 10-25% spermatozoa with the remainder a complex mixture of secretions from accessory glands such as the prostate, seminal vesicles, Cowper’s glands and the glands of Littre. Using ultraviolet light to scan bedding, objects and the victim of a sexual assault may disclose the fluorescence of dried semen which was not visible in ordinary light.

Identification of spermatozoa is conclusive proof of the presence of semen. Examination for motile sperm needs to be undertaken at the time vaginal or cervical samples are taken from a victim. Some of the sample, together with a drop of saline, placed on a slide and cover-slipped is examined, ideally with a phase-contrast microscope. Motile sperm may be recovered from the vagina up to 28 hours after intercourse and from the cervix up to 3 days, or sometimes up to 8 days. Non motile sperm are identified in stained smears on glass slides viewed microscopically. The maximum reported recovery times for non motile sperm are: vagina 14 hours to 10 days; cervix 7½ to 19 days; mouth 2 to 31 hours; rectum 4 to 113 hours and anus 2 to 44 hours. Microscopically spermatozoa have a distinct appearance, approximately 50-60µm in length with a flattened ovoid head 4.5 x 2.5 x 1.5µm and a 50µm tail, which may be lost to leave the isolated head.

The most commonly used screening test for semen is the Brentamine test for seminal acid phosphatase (SAP), which is present in high concentrations, and is active at an acid pH of 4.9 to 5.5. SAP testing is sensitive but not specific for semen because it is found in other tissues and fluids including vaginal fluid. The Barberio crystal test is based on the identification of spermine phosphate or picate crystals when the stain extract is treated with the appropriate anion. Another classical crystal test, the Florence test, relies on the identification of choline periodide crystals when the extract is treated with a solution of iodine in potassium iodide.

Following presumptive testing for semen, most commonly by the Florence test for choline and testing for seminal acid phosphatase, confirmatory testing is carried out. Acid phosphatase has been used as a confirmatory test for semen because the activity of this enzyme in semen is 500 to 1,000 times greater than in any other body fluid. Since vaginal secretions also contain acid phosphatase, any confirmatory testing must be quantitative. However, the standard confirmatory test for semen is either the microscopic identification of spermatozoa or the presence of the semen-specific protein p30.

Prostate specific antigen (PSA) or p30 (so called because it has a molecular weight around 30,000 Daltons) is a glycoprotein derived from the epithelial cells of the prostate gland and found in the semen of both vasectomised and non-vasectomised men. Prostate specific antigen is utilized in clinical testing for prostate malignancy. A variety of immunological tests use commercially produced antibodies to PSA which is present in semen at an average concentration of
1,200µg/ml (range 300-4,000µg/ml). Post ejaculate urine and urine from adult males may give a weak false-positive reaction because of the low level of PSA present (mean 250ng/ml). PSA, despite its name, is not prostate specific and occasional positive test results may be obtained from semen-free vaginal swabs, particularly around the time of menstruation. After intercourse with ejaculation the vaginal level of p30 declines to become undetectable on average within 24 hours (range about 12 to 48 hours). Another semen-specific marker which may in the future find forensic use is MHS-5 which is produced by the seminal vesicles and is not present in any other body fluid.

Post-coital vaginal deposits of semen show differential stability of the various elements with significant loss of p30 by 24 hours, of SAP by 48 hours and of spermatozoa by 72 hours. However, in rape-homicide victims spermatozoa and p30 may be detected on vaginal swabs several weeks after death, depending upon the specific conditions. The likely explanation is that in the rape victim who was immediately murdered there is no mechanical elimination of semen by natural drainage or hygiene activities and no biological elimination or physiological dilution by the now dead body of the victim. Dried seminal stains on fabric will test positively for the various semen factors for months if not years after deposition; in dried semen stains p30 may be detectable for up to 10 years.

The individualisation of semen using forensic serology was limited to ABO and Lewis blood groupings, phosphoglucomutase (PGM) and peptidase A (PepA). About 80% of the population are secretors, secreting the ABO antigens into body fluids including saliva, semen and vaginal secretions. The secretor status of an individual can be checked by comparing the ABO blood type with the presence or absence of the same antigens in a saliva sample. Alternatively the Lewis antigens in blood provide another indicator of secretor status. Individuals whose red blood cells are Le (a- b+) are secretors, Le (a+ b-) individuals are non-secretors, and the rarer Le (a- b-) type provides no information on secretor status. The secretor status of both the victim and the alleged assailant are important in the interpretation of any laboratory results. Blood groups may be detected in semen samples recovered from the vagina up to 20 hours after deposition, but are rarely recovered from the mouth or anus and rectum. Since traditional grouping is cheap, fast and universally available it is still valuable despite the introduction of DNA testing. If the semen is from an azoospermic male then ABO blood typing may be superior to DNA analysis. The enzymes PGM and PepA are found in semen and vaginal secretions regardless of secretor status. However, PGM and PepA levels decline rapidly in the vagina following intercourse and become non-detectable by 6 hours for the former and by 3 hours for the latter.

Other body fluids

In addition to blood and semen, the body fluids requiring identification in forensic practice are saliva, urine, faecal material and vaginal secretions.

Saliva is secreted into the mouth from the salivary glands and contains high concentrations of the enzyme alpha-amylase, the detection of which is the most commonly used test, and if positive is a strong indicator for saliva. The various testing methods make use of the hydrolysis of starch by alpha-amylase.

Urine, as well as containing a variety of inorganic ions, contains amines such as urea and creatinine, the detection of which is used as a presumptive test. Urea is detected by the addition of the enzyme urease which causes the production of ammonia. Creatinine is detected by the Jaffe reaction of a bright red colour on the addition of picric acid and a weak base.

Faecal material is identified by a combination of microscopy which discloses the presence of undigested food residues and bacteria, and testing for urobilin, which gives to faeces their characteristic colour. In the Edelman test any urobilinogen present is first oxidised to urobilin by alcoholic mercuric chloride. The addition of alcoholic zinc chloride results in a green fluorescence due to the formation of a stable zinc-urobilin complex.

There is no definitive test for vaginal secretions despite the fact that they are commonly encountered in forensic practice.
DEATH

Death is the extinction or cessation of life, but since life itself is difficult to define there is a reciprocal problem in defining death. The precise definition of death will always be a subject of controversy because it has social and religious aspects and is not a solely scientific issue. There are profound social and legal repercussions to the diagnosis. An added difficulty is that in nearly all circumstances human death is a process rather than an event. Within this process of dying there are points of no return, the identification of which is the medical diagnostic challenge.

Diagnosis of death

There is no legal definition of death. The diagnosis of clinical death, or somatic death, is traditionally made using the triad of Bichat which states that death is ‘the failure of the body as an integrated system associated with the irreversible loss of circulation, respiration and innervation’. Thus the diagnosis of death is made by excluding possible signs of life. The irreversible cessation of the circulation has been considered for centuries a point of no return. It still provides a practical and valid criterion for the irreversible loss of function of the human organism as a whole.

To ensure that opportunities for resuscitation are not missed, care must be taken in making this final diagnosis in order to avoid mistaking apparent death for actual death. In the overwhelming majority of deaths the diagnosis can be made by traditional clinical methods. The same criteria are applicable whether the death was expected or unexpected. Firstly, there is a need to make a rapid assessment, based upon a history and clinical observations, as to whether resuscitation attempts should be initiated. Cessation of the circulation results in a deathly pallor (pallor mortis) particularly of the face and lips, and primary muscular flaccidity leads to drooping of the lower jaw and sometimes open staring eyes. A complete physical examination should exclude the presence of a circulation or breathing. The absence of a pulse should be determined through the palpation of the carotid, radial and femoral arteries. The absence of heart and lung sounds should be determined by auscultation continually for one minute and repeated intermittently over not less than five minutes. Normal heart sounds may be indistinct in obese individuals or conditions such as pericardial effusion. At the same time observation should be made for respiration. Inspection of the eyes should disclose pupils which are non-reactive to a bright light. Indisputable signs of death develop later with the formation of livor mortis and rigor mortis. Some situations, most notably hypothermia, produce death-like states. Other conditions that can induce a death-like coma include drug overdose (particularly with barbiturates, alcohol, tricyclic antidepressants and anaesthetic agents), and metabolic states including myxoedema coma, uraemia, hypoglycaemia, hyperosmolar coma, and hepatic encephalopathy. Situations in which vigorous attempts at resuscitation may be successful include drowning, airways obstruction, electric shock, and a lightning strike.

Brain-stem death

The cessation of the circulation is only lethal if it lasts long enough to cause critical centres in the brain-stem to die. This is so because the brain-stem is irreplaceable in a way in which the pumping function of the heart is not. Viewing death in terms of brain-stem death rather than the cessation of the circulation is a modern view of the previously recognised facts of the dying process. This reappraisal has been precipitated by modern technology in medicine. Today it is possible to have a body whose brain is irreversibly dead but whose ventilation is maintained by a respirator, cardiac function by various drugs or pumping devices, feeding by the intravenous route and the elimination of waste products by dialysis.

The diagnosis of brain-stem death only arises in a hospital setting in which a patient has suffered irreversible damage to the brain but breathing is being maintained by a ventilator and the heart is continuing to function. The brain-stem is that part of the brain at its base which includes the mesencephalon or midbrain, the pons and the medulla. Within the brain-stem are the respiratory and vasomotor centres which are responsible respectively for breathing and the maintenance of blood pressure. It also contains the ascending reticular activating system, which maintains alertness or the capacity for consciousness. This capacity for consciousness, which is a function of the upper brain-stem, is not the same as the content of consciousness, which is a function of the cerebral hemispheres, but rather it is an essential pre-condition for the latter. Without the function of the brain-stem there can be no cognitive or affective life and no social interaction with the environment. The capacity to breathe is a brain-stem function and cessation of breathing, apnoea, is a critical manifestation of a non-functioning lower brain-stem. Once spontaneous breathing ceases the heart cannot continue to function for long and the circulation then ceases. Following judicial hangings which fracture the neck some cardiac activity can be maintained for up to a maximum of 20 minutes.

In 1967 the first heart transplantations were performed in which the hearts were harvested from beating-heart brain-dead donors. At that time there were no guidelines for the diagnosis of death in these heart donors. The following year Harvard medical school published criteria for the recognition of the ‘brain-death syndrome’, and in 1981 a model statute, called the Uniform Determination of Death Act, was published in
the United States. This defined death as either irreversible cessation of circulatory and respiratory functions or irreversible cessation of all functions of the entire brain. This concept of ‘whole brain death’, if taken literally, would mean that the detection of any activity by any means in any part of the brain would preclude a diagnosis of death. This difficulty was avoided in the United Kingdom when in 1976 brain death was defined as the complete and irreversible loss of function of the brain-stem. This definition is simple, reliable and robust. Internationally, medical opinion and practice has moved in a similar direction in accepting the concept of brain-stem death.

In hospital practice the diagnosis of brain-stem death is not technically difficult and can be made on purely clinical grounds. The purpose of the examination is to establish irreversible loss of brain-stem function. Mindful of the need to protect the right to life, most countries insist that the diagnosis is made by senior physicians in appropriate specialities such as neurology, anaesthetics or intensive care medicine, who are not in any way associated with the potential use of the patient’s organs for transplantation. The diagnosis of brain-stem death is made in three phases. The first step is to ascertain the cause of the coma and to establish that there is irremediable structural brain damage. The brain damage is judged irremediable based upon its context, the passage of time and the failure of all attempts to remedy it. The second step is to exclude all possible causes of reversible brain-stem dys-function, such as hypothermia, drug intoxication or severe metabolic disturbances. The third and final step is to demonstrate the absence of all brain-stem reflexes and the fact that the patient cannot breathe however strong the stimulus. The first two steps may take up to 48 hours but the third step, the testing of brain-stem function, takes less than half an hour.

The normal brain-stem reflex responses tested for are (1) constriction of the pupils in response to light (2) blinking in response to stimulation of the cornea (3) grimacing in response to firm pressure applied to just above the eye-socket (4) movement of the eyes in response to the ears being flushed with ice water and (5) coughing or gagging in response to a suction catheter being passed down the airway. The ability of the patient to breathe is assessed by ensuring full oxygenation through breathing 100% oxygen for several minutes and then disconnecting the ventilator while maintaining diffusion oxygenation into the trachea via a catheter. This test allows the carbon dioxide concentration in the blood to rise to levels more than sufficient to stimulate inspiratory effort. At the same time the patient is protected against serious oxygen deprivation while disconnected from the ventilator. Both apnoea and the absence of brain-stem reflexes must be confirmed twice.

Organ transplantation

The ability to transplant organs from one human being to another is one of the great achievements of modern medicine. The first transplant was of a kidney from a living donor into her identical twin sister in 1954. The first liver transplant was in 1963, pancreas in 1966, and heart in 1967. In the 1960’s, many organs were harvested from non-heartbeating corpses whose deaths had been certified on classical cardiopulmonary criteria. However, transplant of organs from these cadavers was less successful than using organs from live donors. This was because, with non-heartbeating cadavers, ischaemic injury to the organs began with the cessation of the circulation, so the organs suffered a relatively long ischaemic time before transplantation. As a consequence living-related and living-unrelated organ donation became common but gave rise to ethical concerns about the possibility of coercion of the donors and the potential for the sale of organs. With the development of the concept of brainstem death these heartbeating cadavers became the most common source of donor organs from the early 1970’s onwards. Such organs have the benefit of being clinically equivalent to organs obtained from living donors, having equivalent ischaemic times, but do not bring the same ethical concerns of possible coercion or commercialisation.

Nowadays the problem which has arisen is that demand for organs has far outstripped the supply and a quarter of all patients awaiting organ donation may die before receiving a transplant. Maximising the donation rate from all potential brainstem dead donors would still not meet the demand for organs. As a result there is now a return to the use of organ donation from individuals pronounced dead using classical cardiopulmonary criteria, and of whom there is a potentially large pool, such as those dead on arrival at hospital or where in-hospital resuscitation is unsuccessful. There has been also a resurgence of living-related and living-unrelated donations of kidneys, liver and lung lobe for transplantation.

The forensic interest in organ transplantation arises because of the use of organs from donors whose death requires a medico-legal investigation, such as victims of road traffic accidents, industrial accidents, assaults and sudden unexpected deaths. In such cases the consent of the investigating authorities is necessary prior to organ donation. That consent is usually given since it is self-evident that any organs suitable for transplantation must be functioning normally, and that any subsequent medico-legal autopsy on the donor will not be compromised by an inability to dissect and examine the donated organs.
Permanent vegetative state

A permanent vegetative state is a clinical condition of unawareness of self and environment in which the patient breathes spontaneously, has a stable circulation, and shows cycles of eye closure and opening which may simulate sleep and wakening. A vegetative state can be regarded as permanent after 3 months without non-traumatic brain damage or 12 months after traumatic injury. In this condition the brain stem is mostly spared, whereas the grey and white matter of both cerebral hemispheres is widely and severely damaged.

Consciousness has two main components, namely arousal or wakefulness, and awareness of the environment and of the self. Arousal is a brain stem function while awareness is dependent upon the functional integrity of the cerebral cortex and its subcortical connections. The vegetative state is a state of wakeful unawareness. Consequently the diagnosis of permanent vegetative state depends on providing evidence of a negative, a lack of awareness. The diagnosis is made when the patient shows no behavioural evidence of awareness of self or environment; there is brain damage, usually of a known cause consistent with the diagnosis; there are no reversible causes present; and at least six months, and usually 12 months, have passed since onset of the condition.

The three major sensory systems (auditory, visual and somatic) and the motor system are assessed to establish that some sensory stimuli can enter the central nervous system and that the motor pathway out is functioning but that there is no evidence of (1) any spontaneous meaningful motor activity, including voice (2) language comprehension or expression and (3) sustained, reproducible, purposeful, or voluntary behavioural responses to normal or noxious visual, auditory, or tactile stimuli.

Patients with the diagnosis of permanent vegetative state breathe spontaneously, because they have preserved brain stem function, and may live for many years if artificial nutrition and hydration is maintained. Stopping food and water inevitably leads to death within 14 days from dehydration, but doing so typically requires the consent of the court. The leading English case is that of Tony Bland, a young man diagnosed with permanent vegetative state as a result of crush asphyxiation in the Hillsborough (Liverpool) football stadium disaster of 1991. In 1993 the High Court approved withdrawal of artificial feeding and hydration and he died a week or so later. Since that landmark case there have been approximately 20 similar cases in England.

Patients in the permanent vegetative state raise ethical issues concerning the nature of consciousness, quality of life and the value society attributes to life. The diagnosis is difficult because there is no definitive test for awareness and the biology of consciousness is not understood. This is in marked contrast with the concept of brain stem death where both the anatomy and the physiology are well understood.

Cellular death

Clinical death represents somatic death, that is to say the death of a person as a whole. However, not all the cells of the body die at the same time. For some hours after death the pupils will still respond to pilocarpine drops by contracting, and electrical stimulation of muscles will cause contraction. The cornea of the eye may still be suitable for transplant up to 24 hours after death. Viable skin grafts can be obtained for up to 24 hours, bone grafts for up to 48 hours and arterial grafts for up to 72 hours after the circulation has stopped.

Post mortem the cells of the body are destroyed by the process of autolysis (literally ‘self-destruction’), with waves of cell death following somatic death. Destructive enzymes released from lysosomes within the cell initiate the process of autolysis. The process is more rapid in some organs, for example in the pancreas which also contains a large number of digestive enzymes normally secreted into the gut. At a microscopic level autolysis is evidenced by a homogenous staining of the cytoplasm of the cell and similar loss of characteristic staining and detail within the nucleus.

This post mortem change occurring in all the cells of the body is similar to the change which occurs in damaged cells in a living body. Within a living person individual cells or large areas of tissue, comprising groups of adjacent cells, may die without affecting the viability of the whole organism. This pathological cell death, or necrosis, is an abnormal change initiated by some insult to the tissues, such as hypoxia or physical or chemical trauma. Within a few hours of being irreversibly damaged the cells show the microscopic changes characteristic of autolysis. However, unlike post mortem autolysis, this type of cell death, necrosis, incites an inflammatory reaction from the surrounding living tissue. It is the presence of this inflammatory reaction, which can be identified microscopically, which distinguishes tissue necrosis which has occurred in life from post mortem autolysis. Unfortunately the inflammatory reaction only develops to a level at which it can be identified microscopically between ½ hours and 2 hours after injury. Consequently injuries which are inflicted very shortly before death, like tissue damage inflicted after death, show no vital inflammatory reaction. However, the degree of bruising of the tissues associated with the injury may give an indication as to whether or not there was a functioning circulation.
In the living body the cells of all tissues turnover with loss of some cells and their replacement by more cells created by mitotic division. Apoptosis (meaning dropping out or falling away) describes the energy dependant process by which individual cells are lost. The cell contracts and the nucleus fragments producing an apoptotic body, pieces of which are removed by scavenger cells, macrophages. Apoptosis is a normal process and does not stimulate an inflammatory reaction from the adjacent tissues. It is important in the natural turnover of many tissues such as the endometrium during the menstrual cycle. Unlike necrosis and autolysis, it has no forensic importance.
Changes after death

The three principal post mortem changes which occur within the first day after death are body cooling (algor mortis, literally: 'the chill of death'), livor mortis (literally: 'the darkening of death'), and rigor mortis (literally: 'the stiffening of death'). Putrefaction of the body and its variants are later post mortem changes. These changes which develop in a corpse well after death has occurred are of interest for several reasons. They are indisputable signs of death and indicate that any attempts at resuscitation would be futile. As they evolve these post mortem changes produce confusing artefacts and putrefaction destroys evidence of identity, injuries, and natural disease. However, each has its own specific forensic uses. Since they all evolve over time they all have been used also to estimate the time since death. The importance of body cooling lies solely in its value for the estimation of the time since death, and therefore it is discussed in that context in the next chapter.

Rigor mortis

Death is followed immediately by total muscular relaxation, primary muscular flaccidity, so that the body collapses into a position dictated by gravity and surrounding objects. Flaccidity is succeeded in turn by generalised muscular stiffening, rigor mortis, which fixes the body in that posture. It follows that rigor cannot freeze a body in a position which defies gravity, and any such appearance indicates that the body has been moved after rigor developed. If the body is supine then the large joints of the limbs become slightly flexed during the development of rigor. The joints of the fingers and toes are often markedly flexed due to the shortening of the muscles of the forearms and legs. After a variable period of time, as a result of putrefaction, rigor mortis passes off to be followed by secondary muscular flaccidity. There is great variation in the rate of onset and the duration of rigor mortis; the two main influencing factors are the environmental temperature and the degree of muscular activity before death. Onset of rigor is accelerated and its duration shortened when the environmental temperature is high and after prolonged muscular activity, e.g. following convulsions. Conversely, a late onset of rigor in many sudden deaths can be explained by the lack of muscular activity immediately prior to death.

Classically, rigor is said to develop sequentially, but this is not constant or symmetrical. Typically rigor is apparent first in the small muscles of the eyelids, lower jaw and neck, followed by the limbs. It involves first the small distal joints of the hands and feet, and then the larger proximal joints of the elbows and knees, and then the shoulders and hips. Ante-mortem exertion usually causes rigor to develop first in the muscles used in the activity. Generally rigor passes off in the same order in which it develops. Gently attempted flexion of the different joints will indicate the location of rigor and its degree (complete, partial, or absent joint fixation), providing no artefact has been introduced by previous manipulation of the body by others, such as during the removal of clothing. The forcible bending of a joint against the fixation of rigor results in tearing of the muscles and the rigor is said to have been 'broken'. Provided the rigor had been fully established, it will not reappear once broken down by force. The intensity or strength of rigor mortis depends upon the decedent's muscular development, and should not be confused with its degree of development, that is the extent of joint fixation.

Rigor involves voluntary and involuntary muscles. Rigor of the myocardium should not be mistaken for myocardial hypertrophy. Likewise secondary muscular flaccidity of the ventricles should not be mistaken for ante-mortem dilatation or evidence of myocardial dysfunction. Involvement of the iris muscles means that the state of the pupils after death is not a reliable indication of their ante-mortem appearance. Different degrees of rigor can cause irregularity and inequality of the pupils. Contraction of the arrectores pilorum muscles during rigor causes 'goose-flesh' (cutis anserina), a phenomenon commonly seen in bodies recovered from water. Involvement of the walls of the seminal vesicles by rigor may lead to discharge of seminal fluid at the glans penis.

The biochemical basis of rigor mortis is not fully understood. Post-mortem loss of integrity of the muscle cell sarcoplasmic reticulum allows calcium ions to flood the contractile units (sarcomeres) initiating the binding of actin and myosin molecules and mimicking the normal contraction process. Normal relaxation in life is achieved by energy-dependent (ATP-driven) pumping of calcium back across the membrane of the sarcoplasmic reticulum but this fails post-mortem because of membrane disruption and lack of ATP. The actin-myosin complex is trapped in a state of contraction until it is physically disrupted by the autolysis which heralds the onset of putrefaction. This process is characterised by proteolytic detachment of actin molecules from the ends of the sarcomeres, and consequent loss of the structural integrity of the contractile units. Although the biochemical basis of rigor mimics that of muscle contraction in life, it does not cause any significant movement of the body in death, a point of forensic importance.

Cadaveric spasm

Cadaveric spasm (synonyms: instantaneous rigor, instantaneous rigidity, cataleptic rigidity) is a form of muscular stiffening which occurs at the moment of death and which persists into the period of rigor mortis. Its cause is unknown but it is usually associated with
violent deaths in circumstances of intense emotion. It has medico-legal significance because it records the last act of life. Cadaveric spasm involving all the muscles of the body is exceedingly rare and most often described in battle situations.

Most commonly cadaveric spasm involves groups of muscles only, such as the muscles of the forearms and hands. Should an object be held in the hand of a corpse, then cadaveric spasm should only be diagnosed if the object is firmly held and considerable force is required to break the grip. This is seen in a small proportion of suicidal deaths from firearms, incised wounds, and stab wounds, when the weapon is firmly grasped in the hand at the moment of death. In such circumstances the gripping of the weapon creates a presumption of self-infliction of the injuries. This state cannot be reproduced after death by placing a weapon in the hands. It is also seen in cases of drowning when grass, weeds, or other materials are clutched by the deceased. Similarly, in mountain falls, branches of shrubs or trees may be seized. In some homicides, hair or clothing of the assailant can be found gripped in the hands of the deceased.

Livor mortis

Lividity is a dark purple discoloration of the skin resulting from the gravitational pooling of blood in the veins and capillary beds of the dependent parts of the corpse. Synonyms include livor mortis, hypostasis, post-mortem lividity, and, in the older literature, post-mortem suggillations. Lividity is able to develop post mortem under the influence of gravity because the blood remains liquid rather than coagulating throughout the vascular system as a consequence of stasis. Within about 30 to 60 minutes of death the blood in most corpses becomes permanently incoagulable. This is due to the release of fibrinolysins, especially from capillaries and from serous surfaces, e.g. the pleura. The fluidity and incoagulability of the blood is a commonplace observation at autopsy and is not characteristic of any special cause or mechanism of death.

Hypostasis begins to form immediately after death, but it may not be visible for some time. Ordinarily its earliest appearance, as dull red patches, is 20 to 30 minutes after death, but this may be delayed for some hours. Faint lividity may appear shortly before death in individuals with terminal circulatory failure. Conversely, the development of lividity may be delayed in persons with chronic anaemia or massive terminal haemorrhage. The patches of livor then increase in intensity and become confluent to reach a maximum extent and intensity on average within about 12 hours, although there is very great variation. Pressure of even a mild degree prevents the formation of lividity in that area of skin, so that a supine body shows contact flattening associated with contact pallor (pressure pallor) over the shoulder blades, elbows, buttocks, thighs and calves. Similarly tight areas of clothing or jewellery, as well as skin folds, leave marks of contact pallor. The distribution of lividity with its associated contact pallor helps distinguish lividity from bruising, and any doubts are resolved by incising the skin which reveals lividity as congested vessels and bruising as haemorrhage infiltrating tissues.

Lividity is present in all corpses, although it may be inconspicuous in some, such as following death from exsanguination. Lividity is usually well marked in the earlobes and in the fingernail beds. In a supine corpse there may be isolated areas of lividity over the front and sides of the neck resulting from incomplete emptying of superficial veins. Other isolated patches of hypostasis may be due to blood in the deeper veins being squeezed, against gravity, towards the skin surface by the action of muscles developing rigor mortis. Lividity is often associated with post-mortem haemorrhagic spots, punctate haemorrhages, (given the specific name ‘vibices’ in the German literature) which resemble the petechial haemorrhages associated with asphyxial deaths, and from which they must be distinguished. Easily recognised, occurring only in areas of lividity and sparing adjacent areas of contact pallor, they develop in the hours immediately following death as lividity intensifies.

Lividity occurs in the viscera as well as the skin and this provides some confirmation of the external observations. In the myocardium lividity may be mistaken for an acute myocardial infarction, and in the lungs may be misdiagnosed as pneumonia. Livid coils of intestine may falsely suggest haemorrhagic infarction. Lividity developing in the viscera of a body lying prone and resulting in a purplish congestion of organs usually found pale at autopsy can be disconcerting to those unaccustomed to these changes.

The importance of lividity lies in its distribution, as an indicator of body position and contact with objects, and in its colour, as an indicator of cause of death. The usual purple colour of lividity reflects the presence of deoxyhaemoglobin but it does not have the same diagnostic significance as cyanosis produced during life. In the corpse, oxygen dissociation from oxyhaemoglobin continues after death and there may be reflux of deoxygenated venous blood into the capillaries. For these reasons, the blood of a cadaver becomes purplish-blue, but this is not a reflection of a pathophysiological change which occurred in life. Bodies refrigerated very soon after death have a pink lividity due to retained oxyhaemoglobin. Death from hypothermia or cyanide poisoning also imparts the pink hue of oxyhaemoglobin, carbon-monoxide poisoning produces the cherry red of carboxyhaemoglobin, and poisoning from sodium chloride, nitrates and aniline derivatives impart the gray to brown colour of
methaemoglobin. Infection by Clostridium perfringens causing gas gangrene is said to give a bronze lividity.

After about 12 hours lividity becomes ‘fixed’ and repositioning the body, e.g. from the prone to the supine position, will result in a dual pattern of lividity since the primary distribution will not fade completely but a secondary distribution will develop in the newly dependent parts. The blanching of livor by thumb pressure is a simple indicator that lividity is not fixed. Fixation of lividity is a relative, not an absolute, phenomenon. Well-developed lividity fades very slowly and only incompletely. Fading of the primary pattern and development of a secondary pattern of lividity will be quicker and more complete if the body is moved early during the first day. However, even after a post-mortem interval of 24 hours, moving the body may result in a secondary pattern of lividity developing. Duality of the distribution of lividity is important because it shows that the body has been moved after death. However, it is not possible to estimate with any precision, from the dual pattern of livor, when it was that the corpse was moved. If a prone body is moved some hours after death but before lividity is fixed then the primary lividity will fade and may leave behind on the face any lividity-associated punctuate haemorrhages, or ‘vibices’, creating possible confusion with the petechiae of asphyxia.

Areas of lividity are overtaken early in the putrefactive process becoming green at first and later black. The red cells are haemolysed and the haemoglobin stains the intima of large blood vessels and diffuses into the surrounding tissues, highlighting the superficial veins of the skin as a purple-brown network of arborescent markings, an appearance referred to as ‘marbling’.

**Putrefaction**

Putrefaction is the post-mortem destruction of the soft tissues of the body by the action of bacteria and endogenous enzymes and is entirely capable of skeletonising a body. Refrigeration of a corpse delays the onset of putrefaction, freezing the body halts putrefaction, and chemical embalming prevents it. The main changes recognisable in tissues undergoing putrefaction are the evolution of gases, changes in colour and liquefaction. These same changes seen on the surface of the body occur simultaneously in the internal organs. Bacteria are essential to putrefaction and commensal bacteria, mainly from the large bowel, soon invade the tissues after death. Typically, the first visible sign of putrefaction is a greenish discoloration of the skin of the anterior abdominal wall due to sulph-haemoglobin formation. This most commonly begins in the right iliac fossa, i.e. over the area of the caecum. Any ante-mortem bacterial infection of the body, particularly scepticaemia, will hasten putrefaction. Injuries to the body surface promote putrefaction by providing portals of entry for bacteria. Putrefaction is delayed in deaths from exsanguination because it is blood which usually provides a channel for the spread of putrefactive organisms within the body.

Environmental temperature has a very great influence on the rate of development of putrefaction, so that rapid cooling of the body following a sudden death will markedly delay its onset. In a temperate climate the degree of putrefaction reached after 24 hours in the height of summer may require 10 to 14 days in the depth of winter. Putrefaction is optimal at temperatures ranging between 21 and 38°C (70 and 100°F), and is retarded when the temperature falls below 10°C (50°F) or when it exceeds 38°C (100°F). Heavy clothing and other coverings, by retaining body heat, will speed up putrefaction. The rate of putrefaction is influenced by body build because this affects body cooling. Obese individuals putrefy more rapidly than those who are lean.

Gases produced by putrefaction include methane, hydrogen, hydrogen sulphide and carbon dioxide. The sulphur-containing amino acids, cysteine, cystine and methionine yield hydrogen sulphide, which combines with haemoglobin and ferrous iron to produce green sulph-haemoglobin and black ferrous sulphide respectively. De-carboxylation of the amino acids ornithine and lysine yields carbon dioxide and the foul smelling ptomaines, putrescine (1,4-butenediamine) and cadaverine (1,5-pentanediamine) respectively. These ptomaines are detectable by the cadaver dogs used to locate clandestine graves. Deamination of L-phenylanaline yields ammonia, and phenylpyruvic acid which forms a green complex with ferric ion. Bacterial and fungal fermentation yield ethyl alcohol (ethanol), confounding the interpretation of post-mortem alcohol concentrations.

Early putrefaction is heralded by the waning of rigor, green abdominal discolouration, a doughy consistency to the tissues and haemolytic staining of vessels. Localised drying of the lips, tip of the nose and fingers may be seen. The face swells and discoulorns and the swollen lips are everted, making facial recognition unreliable. The skin, which now has a glistening, dusky, reddish-green to purple-black appearance, displays slippage of large sheets of epidermis after any light contact with the body, e.g. during its removal from the scene of death. Beneath the shed epidermis is a shiny, moist, pink base which dries, if environmental conditions permit, to give a yellow parchmented appearance. This putrefactive skin-slip superficially resembles ante-mortem abrasions and scalds. Body hair and nails are loosened and the skin of the hands comes away like gloves taking with it fingerprint evidence of identity. The remaining dermis has a much shallower reverse print which is technically more difficult to document.
Distention of the abdominal cavity by putrefactive gasses characterises the bloating stage of decomposition. In males gas is forced from the peritoneal space down the inguinal canals and into the scrotum, resulting in massive scrotal swelling. Gaseous pressure expels dark malodorous fluid, purge fluid, from the nose and mouth, mimicking ante-mortem haemorrhage or injury. Similar fluid flows from the vagina and anus, the rectum is emptied of faeces and prolapse of the rectum and uterus may occur.

The doughy consistency of the tissues of early putrefaction is replaced by the crepitant effect resulting from gaseous infiltration beneath the skin and in deeper tissues. Large sub-epidermal bullae fill with gas, sanguinous fluid or clear fluid. Gas bubbles appear within solid organs such as liver and brain giving a ‘Swiss-cheese’ appearance, and the blood vessels and heart are filled with gas. These putrefactive changes are relatively rapid when contrasted with the terminal decay of the body. The more dense fibro-muscular organs such as the prostate and uterus remain recognisable until late in the process, thus aiding in the identification of sex. When the putrefactive juices have drained away and the soft tissues have shrunk, the speed of decay is appreciably reduced.

The progression of putrefaction may be modified by vertebrate or invertebrate animal activity. Wild animals, domestic pets, livestock, fish and crustaceans may be involved but most commonly it is insects, particularly fly larvae (maggots). In a hot humid environment with heavy insect activity a corpse may be skeletonised in as little as 3 days. All soft tissues are generally lost before the skeleton becomes disarticulated, typically from the head downward, with the mandible separating from the skull and the head from the vertebral column, and from central to peripheral, i.e. from vertebral column to limbs.

Mummification

Mummification is a modification of putrefaction characterised by the dehydration or dessication of the tissues. The body shrivels and is converted into a leathery or parchment-like mass of skin and tendons surrounding the bone. Skin shrinkage may produce large artefactual splits mimicking injuries, particularly in the groins, neck, and armpits. Mummification develops in conditions of dry heat, especially when there are air currents, e.g. in a desert. Mummification of bodies in temperate climates is unusual unless associated with forced hot-air heating in buildings or other man-made favourable conditions. The importance of mummification lies in its preservation of tissues which aids in personal identification and the recognition of injuries. However, mummified tissues may be attacked by rodents and insects, particularly the omnivorous larvae of the brown house moth (Hofmannophila pseudospretella) which is found in many countries worldwide.

Adipocere

Adipocere formation, or saponification (literally: ‘making soap’), is a modification of putrefaction characterised by the transformation of fatty tissues into a yellowish-white, greasy, wax-like substance which is friable when dry. During the early stages of its production it has a very persistent ammoniacal smell but once its formation is complete it has a sweetish rancid odour. Adipocere, also known as ‘grave wax’ or ‘corpse wax’, develops as the result of hydrolysis of fat with the release of fatty acids which, being acidic, inhibit putrefactive bacteria. Fatty acids combine with sodium or potassium to form hard soap (‘sapo durus’) or soft soap (‘sapo domesticus’) respectively. Calcium gives an insoluble soap which contributes a more brittle quality to the adipocere. However, fat and water alone do not produce adipocere. Putrefactive organisms, of which Clostridium welchii is most active, are important, and adipocere formation is facilitated by post-mortem invasion of the tissues by commensal bacteria. A warm, moist, anaerobic environment favours adipocere formation. Adipocere develops first in the subcutaneous tissues, most commonly involving the cheeks, breasts and buttocks. Rarely, it may involve the viscera such as the liver. The adipocere is admixed with the mumified remains of muscles, fibrous tissues and nerves. Putrefaction, adipocere and mummification may coexist in the same corpse or in adjacent corpses within mass graves as a consequence of differing micro-environments. The importance of adipocere lies in its preservation of the body, which aids in personal identification and the recognition of injuries.

Maceration

Maceration is the aseptic autolysis of a foetus, which has died in-utero and remained enclosed within the amniotic sac. Bacterial putrefaction plays no role in the process. The changes of maceration are only seen when a still-born foetus has been dead for several days before delivery. Examination of the body needs to be prompt since bacterial putrefaction will begin following delivery. The body is extremely flaccid with a flattened head and undue mobility of the skull. The limbs may be readily separated from the body. There are large moist skin bullae, which rupture to disclose a reddish-brown surface denuded of epidermis. Skin slip discloses similar underlying discoloration. The body has a rancid odour but there is no gas formation. Establishing maceration of the foetus provides proof of a post-mortem interval in-utero, and therefore proof of stillbirth and conclusive evidence against infanticide.
TIME SINCE DEATH

All death certificates require an estimate of the time of death as well as a statement of the underlying cause of death. If the death was witnessed then providing a time of death presents no difficulty, but in an un-witnessed death it can be problematic. Establishing the time of death is of assistance in any police investigation of a death, whether it was from natural or un-natural causes. Establishing the time of an assault and the time of death is critical in criminal proceedings in which there are legal issues of alibi and opportunity to commit the crime. If an accused can prove that he was at some other place when the injury and death of the victim occurred then his innocence is implicit. However, the time of injury, or indeed the time of onset of an acute illness, may be separated from the time of death by a significant survival period.

Evidence of the time elapsed since death, the post-mortem interval, may come from the body of the deceased, from the environment in the vicinity of the body, and from information on the deceased’s habits, movements, and day-to-day activities. All three sources of evidence - corporal, environmental and anamnestic - should be explored and assessed before offering an opinion on when death occurred. The longer the post-mortem interval then the less accurate is the estimate of it based upon corporal changes. As a consequence, the longer the post-mortem interval then the more likely it is that anamnestic or environmental evidence will provide the most reliable estimates of the time elapsed.

Many physico-chemical changes begin to take place in the body immediately or shortly after death and progress in a fairly orderly fashion until the body disintegrates. Each change progresses at its own rate which, unfortunately, is strongly influenced by largely unpredictable endogenous and environmental factors. Consequently, using the evolution of post-mortem changes to estimate the post-mortem interval is invariably difficult, and always of limited accuracy.

Body Cooling

Body cooling is the most useful single indicator of the post-mortem interval during the first 24 hours after death. The use of this method is only possible in cool and temperate climates, because in tropical regions there may be a minimal fall in body temperature post-mortem, and in some extreme climates, such as desert regions, the body temperature may even rise after death.

Since body heat production ceases soon after death but loss of heat continues, the body cools. The fall in body temperature after death is mainly the result of radiation and convection. Evaporation may be a significant factor if the body or clothing is wet, and heat loss by conduction may be considerable if the body is lying on a cold surface. Newton's law of cooling states that the rate of cooling of an object is determined by the difference between the temperature of the object and the temperature of its environment, so that a graphical plot of temperature against time gives an exponential curve.

However, Newton's law applies to small inorganic objects and does not accurately describe the cooling of a corpse which has a large mass, an irregular shape, and is composed of tissues of different physical properties. The cooling of a human body is best represented by a sigmoid curve when temperature is plotted against time. Thus, there is an initial maintenance of body temperature which may last for some hours - the so-called ‘temperature plateau’ - followed by a relatively linear rate of cooling, which subsequently slows rapidly as the body approaches the environmental temperature. The post-mortem temperature plateau is physically determined and is not a special feature of the dead human body. Any inert body with a low thermal conductivity has such a plateau during its early cooling phase. The post-mortem temperature plateau generally lasts between a half and one hour, but may persist for as long as three hours, and some authorities claim that it may persist for as long as five hours.

It is usually assumed that the body temperature at the time of death was normal i.e. 37°C. However, in individual cases the body temperature at death may be sub-normal or markedly raised. As well as in deaths from hypothermia, the body temperature at death may be sub-normal in cases of congestive cardiac failure, massive haemorrhage, and shock. The body temperature may be raised at the time of death following an intense struggle, in heat stroke, in some infections, and in cases of haemorrhagic stroke involving the pons. Where there is a fulminating infection, e.g. septicaemia, the body temperature may continue to rise for some hours after death.

Thus the two important unknowns in assessing time of death from body temperature are the actual body temperature at the time of death, and the actual length of the post-mortem temperature plateau. For this reason assessment of time of death from body temperature cannot be accurate in the first four to five hours after death when these two unknown factors have a dominant influence. Similarly, body temperature cannot be a useful guide to time of death when the cadaveric temperature approaches that of the environment. However, in the intervening period, over the linear part of the sigmoid cooling curve, any formula which involves an averaging of the temperature decline per hour may well give a reasonably reliable approximation of the time elapsed since death. It is in this limited way that the cadaveric temperature may assist in estimating the time of death in the early post mortem period.
Unfortunately the linear rate of post-mortem cooling is affected by environmental factors other than the environmental temperature and by cadaveric factors other than the body temperature at the time of death. The most important of these factors are body size, body clothing or coverings, air movement and humidity, and wetting or immersion in water. Body size is a factor because the greater the surface area of the body relative to its mass, the more rapid will be its cooling. Consequently, the heavier the physique and the greater the obesity of the body, the slower will be the heat loss. Children lose heat more quickly because their surface area to mass ratio is much greater than for adults. The exposed surface area of the body radiating heat to the environment will vary with the body position. If the body is supine and extended, only 80% of the total surface area effectively loses heat, and in the foetal position the proportion is only 60%. Clothing and coverings insulate the body from the environment and therefore slow body cooling. The effect of clothing has a greater impact on corpses of low body weight. A bedspread covering may at least halve the rate of cooling. For practical purposes, only the clothing or covering of the lower trunk is relevant.

Air movement accelerates cooling by promoting convection, and even the slightest sustained air movement is significant if the body is naked, thinly clothed or wet. Cooling is more rapid in a humid rather than a dry atmosphere because moist air is a better conductor of heat. In addition the humidity of the atmosphere will affect cooling by evaporation where the body or its clothing is wet. A cadaver cools more rapidly in water than in air because water is a far better conductor of heat. For a given environmental temperature, cooling in still water is about twice as fast as in air, and in flowing water, about three times as fast.

Simple formulae for estimating the time of death from body temperature are now regarded as naive. The best tested and most sophisticated current method for estimating the post-mortem interval from body temperature is that of the German researcher Henssge. Even so, it is acknowledged that the method may produce occasional anomalous results. It uses a nomogram based upon a complex formula, which approximates the sigmoid-shaped cooling curve. To make the estimate of post-mortem interval, using this method requires (a) the body weight, (b) the average environmental temperature since death and (c) the core body temperature measured at a known time, and assumes a normal body temperature at death of 37.2°C. Empiric corrective factors allow for the effect of important variables such as clothing, wetting and air movement. At its most accurate this sophisticated methodology provides an estimate of the time of death within a time span of 5.6 hours with 95% probability. Gathering the data necessary to use this method for estimating time of death means that the body temperature should be recorded as early as conveniently possible at the scene of death. The prevailing environmental temperature should also be recorded at the same time, and a note made of the environmental conditions at the time the body was first discovered, and any subsequent variation in those conditions. Measuring the body core temperature requires a direct measurement of the intra-abdominal temperature. Oral and axillary temperatures of a corpse do not reflect the core temperature and cannot be used. Either the temperature is measured rectally, or the intra-hepatic or sub-hepatic temperature is measured through an abdominal wall stab. An ordinary clinical thermometer is useless because its range is too small and the thermometer is too short. A chemical thermometer 10 to 12 inches (25 to 30 cm) long with a range from 0 to 50°C is ideal. Alternatively a thermocouple probe may be used and this has the advantage of a digital readout or a printed record.

Whether the temperature is measured via an abdominal stab or per rectum is a matter of professional judgement in each case. If there is easy access to the rectum without the need to seriously disturb the position of the body and if there is no reason to suspect sexual assault, then the temperature can be measured per rectum. It may be necessary to make small slits in the clothing to gain access to the rectum, if the body is clothed and the garments cannot be pushed to one side. The chemical thermometer must be inserted about 4 inches (10 cm) into the rectum and read in situ. The alternative is to make an abdominal stab wound after displacing or slitting any overlying clothing. The stab is made over the right lower ribs and the thermometer inserted within the substance of the liver, or alternatively a right subcostal stab will allow insertion of the thermometer onto the undersurface of the liver.

These temperature readings from the body represent data, which if not collected at the scene of death is irretrievably lost. Therefore the decision not to take such readings is always a considered one. If sequential measurements of body temperature are taken then the thermometer should be left in situ during this time period. Taking sequential readings is much easier with a thermo-couple and an attached print-out device.

**Supravital reactivity**

The fact that cellular death occurs in waves within the body tissues following somatic death is evidenced not only by the possibility of organ and tissue transplantation, but also by the persisting excitability of muscle after death, supravital reactivity. Skeletal muscle may be induced to contract in a corpse using mechanical stimulation or electrical stimulation. Mechanical excitation of a variety of muscles in the limbs and face can be achieved by striking them in the immediate post mortem period but the times at which this excitability is lost is not sufficiently well documented to be of forensic use in the determination
of the time of death. The testing of electrical excitability of skeletal muscle requires specific electrical apparatus and the insertion of needles into the muscle. Using this technique on the facial muscles some reaction may be obtained up to 22 hours after death.

In practice two tests for the mechanical excitability of skeletal muscle in a corpse are of forensic value. Striking the lower third of the quadriceps femoris muscle about 4 inches (10cm) above the patella causes an upward movement of the patella because of a contraction of the whole muscle. If present this reaction indicates death within 2½ hours. It is described as Xsako’s phenomenon after the person who first described it. Similarly, striking the biceps brachii muscle and producing a muscular bulge at the point of impact, due to local contraction of the muscle, indicates that death had occurred within 13 hours. The absence of muscle contraction in either test provides no useful information.

In the post mortem period the smooth muscle of the iris is reactive to electrical and chemical stimulation for a longer time than skeletal muscle. The early death of the cells of the nervous system effectively denervates the smooth muscle of the iris, which becomes supersensitive to chemicals which act at the neuromuscular junction. A change in the size of the pupil of the eye of a corpse can be produced by chemical stimulation of the iris following subconjunctival injection of solutions of acetylcholine, noradrenaline, and atropine. The strongest and longest surviving post mortem chemical stimulation is by acetylcholine and noradrenaline, the former producing miosis (papillary contraction) and the latter mydriasis (papillary enlargement). Reactivity to these two chemical neurotransmitters is lost at the earliest 14 hours after death and persists at the latest until 46 hours after death. Atropine produces mydriasis; the reactivity is lost by 3 hours post mortem at the earliest and is present until 10 hours post mortem at the latest.

**Biochemical methods**

A wide range of biochemical tests have been explored in an attempt to find one of use in estimating time of death, but without any success. This is not surprising since all post-mortem biochemical changes will be temperature dependent and therefore less reliable than the use of body temperature itself in time of death estimation.

The biochemical method most frequently referred to is the measurement of potassium in the vitreous humour of the eye. There are sampling problems because the potassium concentration may differ significantly between the left and right eye at the same moment in time. The confounding effect of possible ante-mortem electrolyte disturbances can be excluded by eliminating all cases with a vitreous urea above an arbitrary level of 100 mg/dl, since high urea values in vitreous humour always reflect ante-mortem retention and are not due to post mortem changes. Having eliminated cases with possible ante-mortem electrolyte imbalance, there is a linear relationship between potassium concentration and time after death up to 120 hours, but the 95% confidence limits are ± 22 hours, so the method is too imprecise to have practical value.

**Rigor mortis**

There is great variation in the rate of onset and the duration of rigor mortis, so that using the state of rigor mortis to estimate the post-mortem interval is of very little value. In general, if the body has cooled to the environmental temperature and rigor is well developed, then death occurred more than 1 day previously and less than the time anticipated for the onset of putrefaction, which is about 3 to 4 days in a temperate climate. Gently attempting flexion of the different joints will indicate the degree and location of rigor. Typically slight rigor can be detected within a minimum of one half hour after death but may be delayed for up to 7 hours. The average time of first appearance is 3 hours. It reaches a maximum, i.e. complete development, after an average 8 hours, but sometimes as early as 2 hours post-mortem or as late as 20 hours. As a general rule when the onset of rigor is rapid, then its duration is relatively short. The two main factors which influence the onset and duration of rigor are the environmental temperature and the degree of muscular activity before death. Onset of rigor is accelerated and its duration shortened when the environmental temperature is high, so that putrefaction may completely displace rigor within 9 to 12 hours of death.

The forcible bending of a joint against the fixation of rigor results in tearing of the muscles and the rigor is said to have been ‘broken’. Provided the rigor had been fully established, it will not reappear once broken down by force. Re-establishment of rigor, albeit of lesser degree, after breaking it suggests that death occurred less than about 8 hours before rigor was broken.

**Livor Mortis**

The development of livor is too variable to serve as a useful indicator of the post-mortem interval. Lividity begins to form immediately after death, but it may not be visible for some time. Ordinarily its earliest appearance, as dull red patches, is 20 to 30 minutes after death, but this may be delayed for up to 2, or rarely 3 hours. The patches of livor then deepen, increase in intensity, and become confluent within 1 to 4 hours post-mortem, to reach a maximum extent and intensity within about 6 to 10 hours, but sometimes as early as 3 hours or as late as 16 hours. Faint lividity may appear shortly before death in individuals with
terminal circulatory failure. Conversely, the development of lividity may be delayed in persons with chronic anaemia or massive terminal haemorrhage.

Putrefaction

There is considerable variation in the time of onset and the rate of progression of putrefaction. As a result, the time taken to reach a given state of putrefaction cannot be judged with accuracy. An observer should not assert too readily that the decomposed state of a body is inconsistent with a time interval alleged. As a general rule, when the onset of putrefaction is rapid then the progress is accelerated. Under average conditions in a temperate climate the earliest putrefactive changes involving the anterior abdominal wall occur between about 36 hours and 3 days after death. Progression to gas formation, and bloating of the body, occurs after about one week. The temperature of the body after death is the most important factor determining the rate of putrefaction. If it is maintained above 26°C (80°F) or so then the putrefactive changes become obvious within 24 hours and gas formation is seen in about 2 to 3 days.

The progression of putrefaction may be modified by vertebrate or invertebrate animal activity. Wild animals, domestic pets, livestock, fish and crustaceans may be involved but most commonly it is insects, particularly fly larvae (maggots). In a hot humid environment with heavy insect activity a corpse can be skeletonised in as little as 3 days. All soft tissues are generally lost before the skeleton begins to disarticulate, typically from the head downward, with the mandible separating from the skull and the head from the vertebral column, and from central to peripheral, that is from vertebral column to limbs. Remnants of ligaments and tendons commonly survive about one year, and an odour of decomposition for a few years.

Skeletal remains are of forensic interest only if the time since death is less than a human lifespan, about 75 years, because any perpetrator of a crime may still be alive. Dating skeletons is difficult but is aided by associated artefacts, such as personal effects, and evidence from the grave and its environment. Usefully the bones of individuals who died after the 1940s contain high levels of strontium-90 acquired in life from the atmospheric contamination caused by nuclear explosions.

The presence of any adipocere indicates that the post-mortem interval is at least weeks and probably several months. Under ideal warm, damp conditions, adipocere may be apparent to the naked eye after 3-4 weeks. Ordinarily, this requires some months and extensive adipocere is usually not seen before 5 or 6 months after death. Extensive changes may require not less than a year after submersion, or upwards of three years after burial. Once formed, adipocere will ordinarily remain unchanged for years.

Mummification develops in conditions of dry heat, especially when there are air currents. The time required for complete mummification of a body cannot be precisely stated, but in ideal conditions mummification may be well advanced by the end of a few weeks.

Gastric contents

If the last known meal is still present in the stomach of a corpse and the time of that meal is known, then it can give some general indication of the interval between the meal and death. In general if all or almost all of the last meal is present within the stomach then, in the absence of any unusual factors, there is a reasonable medical certainty that death occurred within 3 to 4 hours of eating. Similarly if half of the meal is present then it is reasonably certain that death occurred not less than one hour and not more than 10 hours after eating. However, these are broad generalisations and difficulties arise in individual cases because the biology of gastric emptying is complex and influenced by a wide variety of factors including the size and type of meal, drugs, stress and natural disease.

Remarkably liquids, digestible solids and non-digestible solids ingested together in the same meal will leave the stomach at different rates. The emptying of low-calorie liquids is volume-dependant (monexponential) resulting from the motor activity of the proximal stomach. By contrast digestible solids empty more slowly, in an approximately linear pattern after an initial lag period, primarily as a result of the motor activity of the distal stomach. Non-digestible solids which cannot be ground up by the stomach into smaller particles are emptied after the liquid and digestible solids, during the so called inter-digestive period, as a result of a specific wave of motor activity in the stomach. In general meals of a higher osmotic and caloric content are emptied more slowly.

However, there is a substantial variation in gastric emptying rates in normal people. Individuals who suffer severe injuries resulting in coma and survive several days in hospital may still have their last meal within the stomach at autopsy. These are extreme examples of delayed gastric emptying but serve to illustrate the point that the stomach is a poor forensic time-keeper.

There have been several cases of alleged miscarriages of justice in which medical experts have wrongly used the stomach contents at autopsy to provide estimates of time of death to an accuracy of half an hour whereas the degree of accuracy possible is at best within a range of 3 or 4 hours.
Entomology

Insects will colonise a corpse if given the opportunity. The most important flies whose larvae (maggots) feed on corpses belong to the groups Calliphoridae or blow-flies and Sarcophagidae or flesh-flies. The blow-flies are the bright metallic blue and green ‘bottle flies’ commonly found around refuse. Each part of the world has its own indigenous species of these flies and, as a consequence of the movement of human populations, some old world species have been introduced into North America and Australasia. While fly larvae feed on the corpse, beetles feed on the larvae, although some beetle groups will feed directly on the corpse. Beetles appear on the corpse later than flies and are some of the last insects to colonise fragments of soft tissue remaining on skeletonised bodies.

Fly eggs are laid on the moist body parts such as the eyes, nares, mouth, perineum and wounds. Head hair, folds in clothing and the crevice between the body and the ground are sites of oviposition also. Early maggot colonisation of parts of the body not usually colonised suggests that there was a breach in the skin, a wound, at that site to attract oviposition, e.g. on the palms of the hands. After the adult female fly has laid its eggs they hatch within a few hours, depending on species and the ambient temperature, giving rise to the first of three stages (instars) of larvae. They are very small, usually less than 2mm in length, and difficult to see. Flesh-flies, unlike blow-flies deposit first instar living larvae rather than eggs on the corpse. First instars moult, shedding their exoskeleton, to produce second instar larvae which grow to a length of up to 4-6mm. The second instars moult to yield third instar larvae, the largest maggot stage, and that most commonly observed; they are voracious feeders on the corpse. When present in large masses they generate considerable heat and a strong odour of ammonia, their main excretory product. Post feeding larvae, prepupae, migrate from the corpse, wandering off to find a protected place for pupation. The exoskeleton of the third stage larvae hardens and browns forming the puparium. This pupal stage of development is similar to the chrysalis of butterflies where metamorphosis to the adult form of the species occurs. In due course an adult fly will emerge from the pupa.

For each species this life-cycle follows a known temperature-dependent time course. Consequently, maggots of a known stage of development and species found on a corpse give an indication, from the time required for their development, of the minimum period since death. Even in bodies long dead the remnants of insects such as pupa cases and the exoskeletons of beetles may provide useful information.

Moving a body or burying it some days after death interrupts the normal succession of insects, from which it can be deduced that an event occurred to disturb the normal chain of entomological events. Blow-flies of certain species are found in either an urban or a rural habitat. Finding urban blow-fly larvae on a corpse in a rural setting would suggest death and blow fly oviposition in an urban environment followed by dumping of the body in the rural environment.

The larve feeding on a corpse may contain any drugs present in the corpse, and are often easier to analyse than body tissue because the corpse contains large numbers of masking chemicals produced by decomposition. Many years after the death, drugs may still be identified in the remnants of pupal cases associated with skeletonised remains.

Botany

Plants and parts of plants may provide evidence of time since death if a plant is in contact with the body or buried with human remains. Ideally a botanist should attend the scene, otherwise colour photographs must be taken and the plant material preserved by drying it between sheets of newspaper. Perennial plants, such as trees, often have seasonal or annual growth rings which can provide a minimum age for human remains where the plant has grown through them or has been damaged by their deposition. Roots can be useful in a similar way.

Annual plants give an indication of time because they complete their life cycles in known time periods in specific seasons, so that disturbances which can be related to a point in the life cycle can be dated. Bodies lying over green plants shade and kill the chlorophyll, and new shoots may develop from damaged stems, changes upon which a time frame can be placed.
EXAMINATION OF THE DEAD

Every post-mortem examination begins with a review of the available investigative information, an evaluation of the medico-legal issues arising, and the development of a strategy for the conduct of the examination. This strategy draws upon a repertoire of techniques, and structures the conduct of the examination to obtain the maximum amount of evidence relevant to the specific issues of the case. Such a strategy is inevitably flexible since the on-going examination may bring new information to light.

The law, regulations and local administrative practices governing medico-legal post-mortem examinations vary from place to place. Whatever the jurisdiction, prior to the start of any examination there must be legal authority to proceed. The doctor receiving the authority should permanently record how it was received, from whom and when. The next requirement is a formal identification of the decedent by a method meeting the necessary legal standards. In a case in which no court proceedings are anticipated, simply noting the details contained on the mortuary body tag and any accompanying documentation may suffice. In other circumstances visual identification of the decedent by next of kin, possibly in the presence of the doctor, or the use of a scientific method may be required. Whatever method is used, it should be recorded in the report.

In many deaths it is appropriate to limit the post-mortem examination to an external examination without internal dissection. This is a judgement to be made by the doctor, and any law officer responsible for the death investigation, in light of the available information concerning the death, the legal requirements, public health concerns, potential for criminal prosecution or civil litigation, resource constraints, and the religious and personal views of the next of kin. The external examination embraces everything on and upon the body including clothing, physical evidence, and medical paraphernalia.

Who, when, where, and why are some of the questions which the examination assists in answering. Who has died is not usually at issue, but establishing identity can be a problem when there is no circumstantial evidence of identity, or visual identification is impossible because of decomposition, fire damage, physical disruption or mutilation. The commonest methods of scientific identification are fingerprints, dentistry, radiology, and DNA analysis (see the two chapters on identification).

When the person died is usually best established from the circumstantial evidence, and is not commonly an important issue (see the chapter on time of death). Where a person died is usually where the body was found but not always. Homicide victims may be dumped, and bodies may travel long distances in rivers and the sea prior to their recovery. The autopsy may provide evidence that the body has been moved after death, as well as trace evidence originating from the place of death.

Establishing why the person died, that is the disease or injury initiating the sequence of events, short or prolonged, leading to death, is one of the most important functions of the examination. However, the cause of death may be readily apparent from the circumstances and the condition of the body, such as a passenger in an air crash, and in these cases an autopsy may be required for other investigative reasons, such as victim identification and accident reconstruction.

Clothing

Examination of the undisturbed clothing is a valuable part of the examination. In some jurisdictions the body is stripped naked by police officers, or mortuary staff, prior to examination, but this is an unwise practice. The clothing can provide a wealth of useful information on the lifestyle of the decedent, events surrounding the death, and the cause of death. If the examining doctor does not document the clothing then it is often not documented at all. Each article of clothing should be described in appropriate detail and, when the body is unidentified, details of the labels and laundry marks also. Description of the clothing should include general descriptions of any disarrangement, damage and stains. Recovery of trace evidence from clothing may be undertaken either at the scene of death or in the mortuary depending upon local practice and the nature of the case (see the chapter on scene of death). Trace evidence might include hairs, fibres, paint chips, glass fragments, vegetation and insects. The collection and storage of this trace evidence must meet the legal requirements for the chain of custody.

The appropriateness of the clothing should be assessed against the scene of death and anamnestic information, particularly in potential hypothermic deaths. Stains, scuff-marks and tears to clothing may assist in traffic accident reconstruction or in clarifying events surrounding a death. Gunshot holes and stab wounds to clothing provide useful information in themselves, but more so when correlated with the underlying injuries to the body. Bloodstain patterns to clothing may illuminate the events following trauma and the activities of the victim prior to collapse. Jewellery may provide evidence of identification, medical bracelets and necklaces may indicate a chronic disease, pockets may contain medication or drugs of abuse, and personal papers may give information on identity, medical history, and lifestyle.

Stains

Prior to removing clothing and personal effects, and
cleaning the body of any stains, it may be necessary to make a permanent record through photography, particularly in a case of homicide. After removal of the clothing a head to toe detailed examination of the naked body is made. In the first instance this external examination should document stains and soiling, general and specific identifying characteristics, evidence of medical intervention, and post-mortem changes.

Stains to the exposed body surfaces may be described at the time of the examination of the clothing and supplemented with a description of stains to the whole body. The location, extent and type of staining or soiling can provide useful information. Blood flow patterns from wounds reflect body position after wounding; high velocity impact blood splatter and gunshot residue stains on the hands may indicate suicide; 'coffee grounds vomitus' around the mouth and melaena staining of the buttocks suggest death from massive gastro-intestinal haemorrhage; vomitus containing tablet debris raises the possibility of suicidal overdose.

**General descriptors**

The general external description should include height, weight, build, sex, race, head and body hair, eyes, dentition, scars, tattoos and body piercings as well as evidence of natural disease, particularly ankle oedema, varicose veins and trophic changes from peripheral vascular disease, which raise the possibility of a cardiovascular death. The back, anus and perineum, palms and soles must always be examined but are particularly important in custody deaths where allegations of physical abuse may arise. A specific search for petechial haemorrhages in the eyelids, conjunctivae, inner lips, face and neck is mandatory because they are easily overlooked but are of considerable importance in the diagnosis of asphyxia. It is best to roll the upper eyelids inside out to look for petechiae on the tarsal plates.

The recording of post-mortem changes to the body does not usually include body temperature unless there is a specific concern about the time of death. Core body temperature obtained per rectum, or by a liver stab, is required and should be taken at the scene of death rather than in the mortuary. The presence or absence, and the pattern of post-mortem lividity, rigor mortis and putrefaction are routinely observed and recorded.

Post-mortem lividity (livor mortis or hypostasis) reflects gravitational pooling of blood after death, and thus body position. Areas of contact pallor produced by pressure from clothing or adjacent objects should correlate with the scene of death findings. Unusual patterns of lividity should be noted. A pink lividity, rather than the usual purple-red, raises the possibility of death from carbon monoxide poisoning, cyanide or hypothermia. Rigor mortis, which develops some hours after death, fixes the body in the position in which it came to rest and should also correlate with the scene of death findings. The assessment of rigor needs to be made before it is disturbed by the undressing of the body. However, neither lividity nor rigor is of any substantial value in estimating time of death.

Post mortem injuries produced during the recovery of the body or by the feeding of insects, birds, animals, or water life are recorded separately from injuries produced in life in order to avoid confusion and their description is best included with other post-mortem changes.

**Medical intervention**

In many deaths there is evidence of attempts at lifesaving medical intervention. Where disposable medical equipment is attached to the body this should not be removed at the scene of death but transported with the body to the mortuary to be recorded by the examining doctor. The most common items are airways and solutions for intravenous infusion. All of these require description and an assessment of their correct placement together with recording of any associated bleeding, bruising or other tissue damage. Emergency medical treatment is rarely documented in detail at the time it is given because of the urgency of the circumstances, and consequently the autopsy record is often the most complete and reliable record. It may be important in any civil litigation for malpractice. Injuries produced by medical intervention, particularly those in the neck, may be misinterpreted as assaultive if not viewed in context. Recording intravenous lines eliminates the possibility that the associated needle puncture marks may give rise to a false suspicion of intravenous drug abuse.

**Injuries**

The final stage of the external examination is the documentation of injuries. These are described systematically either by grouping them according to injury type and anatomical location, or by numbering them, without implying an order of infliction or ranking of severity. Each injury is characterised by its type, for example bruise, abrasion, laceration, incised wound, stab wound, gunshot wound, burn, and its general anatomical location. The precise anatomical site of an injury is recorded in cases of homicide or if it is of particular significance for the reconstruction of the circumstances, for example a single suicidal gunshot wound or an imprint injury from a vehicle striking a pedestrian. Precisely locating a wound is analogous to giving a latitude and longitude with respect to fixed landmarks, which may be the midline of the chest, the heel or top of the head, or any fixed bony prominence. The size, shape and other relevant features of the injury, depending on its type, are observed and...
recorded. For this purpose photography is of particular value in documenting a wealth of detail.

Special procedures

Special procedures utilised during the external examination include photography for the purposes of identification and documentation. Infra-red and ultra-violet photography will enhance tattoos, bruises and patterned injuries. High-contrast black-and-white photography or computer-assisted image enhancement can be used to enhance patterned injuries. Trace material can be identified with ultra-violet, laser or alternative light sources. Fingerprinting may be required for identification purposes, and is routine for all homicide victims. Where sexual assault is suspected, the collection of physical evidence includes what would normally be collected in a living victim (see the chapter on sex and sexual assaults). Collecting insect specimens, such as fly maggots, may be useful for estimating time of death or for toxicological studies. Gunshot residues can be collected from the skin surface. Radiological examinations assist in identification, locating foreign objects such as projectiles, and documenting old and recent bony injury. The latter is of particular importance in suspected child abuse when full body X-rays are required. Angiography and more sophisticated techniques such as CT and NMR scanning may be useful in special circumstances.

Internal examination

For many deaths the information obtained from the history, scene of death and external examination is sufficient and no dissection of the body is required. The post-mortem dissection of a corpse for medical or medico-legal purposes is an autopsy, which literally means to see for oneself. In Britain it is sometimes called a necropsy.

The purpose of continuing the examination with a dissection is to obtain additional information not otherwise available, but necessary for the investigation. This internal examination almost always requires the opening of all three body cavities, namely the head, chest and abdomen, for the purpose of completely removing all internal organs and dissecting them in a systematic manner. In some jurisdictions the opening of all three body cavities is mandatory under regulations governing the conduct of medico-legal autopsies. Even where no such rules exist, it is usually unwise to omit the examination of any of the three cavities. Such an incomplete autopsy may foster lingering doubts, brings into question the competence and judgement of the doctor and may unnecessarily precipitate a reopening of the investigation. It is a particularly undesirable practice where the body is to be shipped overseas for disposal, when distance and the differences in language, culture and legal processes combine to compound misunderstandings. However, when the purpose of the dissection is solely to establish a natural cause of death, and a non-survivable natural event is found, such as a haemopericardium from a ruptured myocardial infarction, then the autopsy may be curtailed in order to minimise mutilation of the body.

All penetrating wounds, such as gunshot and stab wounds, must be traced from their entry point through the body to their termination or exit point. All non-penetrating injuries need to be associated with damage to the underlying tissues such as bony fractures, lacerated blood vessels and resultant haemorrhage. Individual external injuries are described in continuity with any associated internal injuries in the final autopsy report.

As well as recording evidence of trauma and natural disease, important negative observations, such as the absence of coronary artery disease, pulmonary thrombo-emboli and bony fractures, are also recorded. This serves to provide valuable exclusionary information, and to document the completeness of the internal examination. The quality of documentation required of both the external and the internal examinations is that sufficient for another doctor to reach a reliable independent interpretation of the findings.

Special techniques

Some types of trauma and complications of trauma require special autopsy techniques. In cases of suspected assault the muscles of the anterior shoulders, the anterior abdominal wall and the back are examined. If these muscles are not exposed and dissected then bruises within them may escape detection, particularly since there is often no visible injury on the skin surface. Venous air embolism is a potentially lethal condition and can cause sudden death in association with abortion, labour or penetrating injuries to the neck, such as stab wounds. The diagnosis may be suspected from the circumstances of the death but ultimately rests upon the observation of air in the right side of the heart and great veins at the time of autopsy. This air may be seen on a chest or abdominal X-ray of the decedent. Careful exposure of the inferior vena cava and the opening of the heart under water at the very beginning of the dissection of the body are essential if it is to be visualised. If not considered at the outset, air embolism is easily missed and the evidence destroyed by the routine dissection procedure.

Deaths related to abortion, labour or violent sexual assaults may require the removal of the internal genitalia in continuity with the external genitalia and perineum.

The most important special dissection technique used in forensic pathology is the dissection of the neck.
Pathological findings in the neck are of crucial importance in deaths from hanging, ligature strangulation, manual strangulation, and impacts to the head and neck. In these cases the physical evidence of injury may be very little when contrasted with the fatal outcome. The forensic dissection technique allows for examination of the neck structures in situ, layer by layer, in a bloodless field, i.e. after draining the neck of blood by removing the brain and the chest organs. In this way the creation of false haemorrhages as a result of the dissection technique is avoided, and even tiny areas of true haemorrhage can be identified. Failure to use this technique may result in the production of false haemorrhages, which are then erroneously interpreted as evidence of trauma in life.

Ancillary investigations

Ancillary investigations, which support the medico-legal autopsy, include a potentially large range of hospital laboratory tests and forensic laboratory examinations. In practice, the most important of these is toxicology because of the prevalence of prescribed and illicit drug usage, which may represent a cause or contributory factor in a death. Approximately one-third of all un-natural deaths in the UK have evidence of recent alcohol ingestion and consequently alcohol analysis is the commonest toxicological investigation. Specimens for toxicological analysis should include femoral venous blood (rather than blood from the torso), vitreous fluid, urine and liver, as well as stomach contents where there is suspicion of recent ingestion. The original volume of the gastric contents should be recorded so that the drug concentration found in a sample on toxicological analysis can be used to calculate the mass of residual unabsorbed drug in the stomach. Stomach contents may be relevant also to estimation of time of death. Biochemical studies can be performed on blood, urine, cerebrospinal fluid and vitreous fluid, although potential testing is more limited than in the living because of interfering post-mortem biochemical changes.

Samples from all major organs should be preserved in formaldehyde for possible histological examination, the extent of which is at the later discretion of the pathologist. Histological examination may identify disease states not apparent during autopsy dissection and assist in ageing injuries and natural diseases, such as myocardial infarction. Samples for micro-biological analysis are taken in accord with hospital autopsy practice and are of particular importance in the forensic setting of a sudden infant death, when occult infection is a common cause.

Blood, skeletal muscle or spleen are appropriate samples for DNA profiling for purposes of identification, archiving, or the deletion of decedents from DNA criminal databases.

Autopsy report and certification

The medico-legal examination of a corpse requires both the accurate observation and the accurate documentation of the findings. The end-product of the autopsy is the written and signed report submitted to the legal authority instructing the examination. The pathologist should retain a duplicate signed original. The report should contain all relevant administrative information such as the time, date and place of examination, the authorising legal authority, and method of identification of the body, as well as the examination findings, results of ancillary investigations, and chain-of-custody details. The report should always include a section offering an opinion on the interpretation of the autopsy findings in the light of the other available investigative information. Only the pathologist is in a position to advise the legal authorities on the significance of the autopsy findings, and failing to do so through a permanent written record defeats the purpose of the autopsy.

There are many causes of death, both natural and unnatural, which cannot be established with certainty by autopsy alone. Examples include epilepsy, asthma and drowning. In such deaths the autopsy assists by excluding other potential causes of death from trauma, drugs or natural disease. In a forensic autopsy, the demonstration of the absence of injury or disease may be as important as the demonstration of unequivocal or occult injury. Some natural diseases, particularly coronary artery disease, are chronic and can kill at any time, so that their identification at autopsy represents a potential cause of death but not necessarily the cause of death. Similarly, drugs such as alcohol, morphine and other opiates, to which individuals develop tolerance, may be found at autopsy in concentrations in blood sufficient to account for death but not necessarily lethal. Very minor injuries may be consistent with a subtle form of homicidal trauma, such as soft smothering, and sufficient to arouse suspicion but not provide conclusive proof. Only in a minority of deaths are there autopsy findings of a trauma, poisoning, or natural disease incompatible with life, so that the cause of death is established with certainty by the autopsy alone. For the great majority of death investigations, establishing why the person died requires the integrated analysis of the autopsy findings, scene of death and anamnestic data. In a small number of deaths a thorough investigation, including an autopsy with toxicological testing, may fail to establish the cause of death. Such a case is characterised as a ‘negative autopsy’ in the English speaking world and as a ‘white autopsy’ in many other jurisdictions.

In addition to the autopsy report, the medical certificate of cause of death provides a documentary record of the death (see the chapter on death certification). The person who signs the certificate records not only the cause of death but also the identity of the person and
the time and place of death. In some death investigative systems this responsibility rests with the legal authority, such as the coroner, but in other jurisdictions may lie with the autopsy pathologist. Formulation of the cause of death on the certificate is in accordance with international rules approved by the World Health Organisation. These rules were established primarily to allow for the classification and coding of deaths with a view to acquiring community-based statistics for health monitoring and planning purposes. A cause of death formulated according to these rules cannot express the complexities which may be of interest in legal proceedings, so that, in the courtroom, the certified cause of death represents only the starting point for comment.