

CHAPTER 1

BLUNT FORCE INJURIES

A wound is a disruption of the continuity of tissues produced by external mechanical force. The term injury is used synonymously with wound, but can have a wider meaning and encompass not only damage produced by physical force, but also damage produced by heat, cold, chemicals, electricity and radiation. The English word 'injury' derives from the Latin word 'injuria' which literally means not lawful. The term lesion originally meant an injury but has now come to be more widely applied to include any area of injury, disease, or local degeneration in a tissue causing a change in its function or structure. Therefore use of the terms injury or wound imply damage from unnatural causes, while the use of the term lesion is non-committal on whether the cause of the damage was natural disease or not.

Wounds, or injuries, are generally classified according to their cause as blunt force injuries, sharp force injuries, gunshot injuries, and a miscellany of others, including for example burns. This is the classification followed in this text, and each group of injuries is discussed in successive chapters.

A blunt force injury is any bodily damage resulting from forceful contact between the body and a blunt object. The forceful contact most commonly involves movement and impact, with the resulting transfer of kinetic energy. Either the moving object strikes the body as in a blow, or the moving body strikes an unyielding object as in a fall. Less often the physical force is applied more slowly by the pressure of crushing, squeezing, or pinching. Crushing or scraping of the skin produces abrasions (grazes). Bruises occur when the elastic limit, the tolerance, of subcutaneous blood vessels is exceeded so that they tear and bleed. Greater forces are needed to tear the skin to produce lacerations. These three blunt force injuries – abrasions, bruises, and lacerations – may occur singly or together in any combination.

Abrasions

An abrasion is an area of crushing or loss of skin, or mucous membrane, resulting from contact with a blunt object. Abrasions are typically superficial, trivial injuries which may be overlooked easily but provide useful forensic information. They bleed only slightly, heal quickly and leave no scar. Tangential impact between an object and the skin causes a typical graze in which the superficial skin layers are scraped off and piled up as skin tags at the far end of the injury. The location of the skin tags indicates the relative direction of movement between the object and the body.

Broad patches of abrasion are sometimes described as brush abrasions, the frictional element of which gave rise to the term 'brush burns', as in for example 'carpet burns'. They are seen in their most florid form as 'road rash', following a motor vehicle collision in which the victim, typically a motorcyclist, slides along the road surface.

Trace evidence from the impacting surface may be present within a graze and can be recovered for identification and matching with the impacting object, for example paint from a vehicle in a hit and run death of a pedestrian. Similarly, fragments of skin transferred to the impacting object may be matched to the victim using serology or DNA techniques. Examination of a graze for the presence of skin tags and trace evidence is easier using a hand-held magnifying glass.

A scratch is a linear abrasion produced by drawing a sharp point over the surface of the skin or mucous membrane. Plant thorns and barbed wire are common causes of scratches. The presence of skin tags at one end indicates the direction of the scratch. Human fingernails and animal claws are other important causes of scratch abrasions. The spacing and convergence of several scratch marks may allow a distinction between human and animal and between adult and child. Small quantities of skin can be recovered from beneath fingernails which have been used for scratching, and linked to the person who was scratched by serology or DNA techniques. Tissue from beneath the fingernails is recovered by clipping the nails with clean scissors, scraping beneath the nails using wooden 'orange sticks', or cleaning beneath the nails using pointed cotton swabs.

Whereas tangential impact between an object and the skin will produce a graze or scratch, direct impact at approximately right angles (90°) to the skin surface will produce a crush abrasion, which imprints the pattern of the causative object upon the skin surface. These imprint or patterned abrasions may result from forceful impact, as in a pedestrian struck by a vehicle, or alternatively may result from a more slowly applied crushing force, such as in strangulation by ligature. The importance of patterned abrasions lies in the fact that their shape can be matched to the causative object, and for this reason they should be documented by photography with a scale, or by an accurate tracing. Common examples of imprint abrasions are the sole patterns of footwear in kicking assaults, and motor vehicle tyre tread marks in run-over pedestrian deaths. In deaths from hanging the ligature typically leaves a distinct imprint abrasion which is accentuated by the continued suspension of the body after death. Fingernails, as well as producing scratch abrasions, can produce semi-lunar and linear imprint abrasions. The hilt of a knife may leave an imprint abrasion adjacent to a stab wound, suggesting the forceful penetration of the entire length of the blade. A diagonal abrasion across the chest of a motor vehicle collision victim suggests that a three-point seatbelt was being worn at the time of impact.

In the most superficial abrasions there is crushing and loss of only the epidermis so that there is no bleeding and often no inflammatory reaction. Slightly deeper abrasions involve the superficial dermis, appear red-based, may bleed slightly, and produce an exudate which dries within a day to form a yellow-brown scab. Abrasions produced after death, or immediately prior to death, have a dried red, orange or yellow base and are described as parchmented abrasions because they resemble parchment. As the post-mortem interval increases these parchmented abrasions dry out and harden further, becoming a

dark brown or black colour. Superficial abrasions on a wet or damp corpse may escape notice until they dry out and darken.

Overall, abrasions provide useful forensic information because their location reflects precisely the point of impact with the causative object, grazes indicate direction, imprint abrasions reflect the shape of the causative object, trace evidence from the object may be found in the graze, and skin fragments from the victim may be recovered from the causative object.

Bruises

A bruise is a haemorrhage into tissues. Synonyms are contusion and ecchymosis (plural: ecchymoses). Very small bruises, ranging in size from a pinpoint to a pinhead, may be described as petechiae, or petechial haemorrhages, or alternatively as punctate haemorrhages. Haemorrhage or bleeding is the process which produces a bruise in tissues, but the term haemorrhage also encompasses bleeding which may not be associated with bruising, e.g. a bleeding nose, or a bleeding stomach ulcer.

Any tissue may bruise, but bruises confined to deeper tissues, such as skeletal muscle, are not visible on the skin surface. Bruises of the deep tissues, even when fatal, may not be evidenced by any injury to the skin surface. For example, a fatal head injury, such as a sub-dural haematoma, may be encountered without recognisable superficial bruising; fatal strangulation with extensive bruising of the muscles of the neck may be accomplished without obvious bruising of the skin; and blows to the abdomen, although producing bruising and ruptures of internal organs, may not produce any abdominal wall bruising.

Only bruising of the skin and subcutaneous tissues, and the mucosa of the mouth, vagina and anus, is visible at a clinical examination. A simple bruise is a discolouration resulting from haemorrhage beneath the skin or mucosa, without any associated breach in the surface. The blood vessels ruptured are typically the capillaries and small veins rather than arteries. Skin and mucosal bruises may be accompanied by abrasions and lacerations, but they are not usually associated with cuts and stabs, where there is a free flow of blood from the cut vessels rather than infiltration of blood into the tissues. Bruises are accompanied by swelling from the haemorrhage itself and the resulting inflammation. If the extravasated blood collects as a discreet tumour-like pool, the lesion is referred to as a haematoma. Bruises are usually painful and tender to palpation as a result of damage to local nerve endings and the inflammation. Focal necrosis of subcutaneous fat may occur at the site of a bruise, and a secondary aseptic inflammation in response to the irritant effect of fat liberated from the ruptured cells produces a hard chronic lesion. This is more of clinical than forensic importance since a common site is the breast where it may be mistaken for a carcinoma. Bruises of the skin if numerous and large enough can be life-threatening as a result of blood loss but this is an uncommon occurrence.

Bruises are produced typically by a blunt force impact, such as a blow or a fall, but can result also from crushing, squeezing or pinching. Bruising, with or without abrasion, to the bony

prominences of the back (i.e. the shoulder blades, sacrum and pelvis) may be caused by force applied to the front of a supine body with resultant counter-pressure between an underlying firm surface and the back, as in forceful restraint on the ground during a rape, or a stamping assault. Similar 'counter-pressure bruising' may be seen on the bony prominences of the front of the pelvis (the anterior superior iliac crests) in attempted anal rape. So called 'love-bites' ('hickeys' in American slang) are superficial bruises produced by the negative pressure of mouth suction. They are commonly found on the side of the neck and occasionally the breast and inner thigh. They are sometimes self-inflicted on accessible parts of the arms to simulate evidence of an assault. Natural diseases in which there is an abnormality of the clotting mechanism of the blood, such as leukaemia, scurvy (vitamin C deficiency), and liver disease, cause so-called 'spontaneous bruising' which is thought to result from unrecalled trivial trauma. Florid spontaneous bruising (purpura) may be seen in children with fulminating meningococcal infection.

The size of a bruise is an unreliable indicator of the degree of force which caused it, because several other factors, such as anatomical site, gender, age and the presence of natural disease, all influence bruise size. Bruising occurs more readily where there is a lot of subcutaneous fat, such as on the buttocks and thighs, and therefore more readily in the obese, in women and in infants. Bruising occurs readily in loose tissues, such as around the eyes and genitals, and less readily where the skin is strongly supported by fibrous tissue, such as the palms, soles and scalp. The elderly bruise more easily because of degenerative changes in the blood vessels and the supportive tissues of the skin and subcutaneous fat. Senile purpura, characterised by sharply-defined geographic areas of 'spontaneous' bruising to the backs of the hands and forearms is found in the very old or frail. Natural diseases which affect blood clotting, degenerative diseases of small blood vessels, and high blood pressure make some individuals more susceptible to bruising. Skin colour does not change the extent of bruising but does significantly influence its appearance because the bruise is viewed through the semi-translucent skin. Bruising is most easily seen in very pale-skinned individuals, particularly blondes and redheads, and can be completely masked by the natural skin colour of blacks. Examination under ultraviolet light helps reveal bruises which are otherwise difficult to see. Black and white photography, which is more sensitive to ultraviolet light than colour photography, may allow the bruise to stand out more clearly in a photograph.

Bruises tend not to reflect accurately the shape of the object which produced them, and they change shape with time. Exceptionally the surface detail of the striking object may be imprinted as a patterned bruise on the skin, often associated with a patterned abrasion. Such patterned or imprint bruises typically occur following a heavy impact, such as from a shod foot or motor vehicle, or from the muzzle or foresight of a gun in a contact gunshot wound, with death occurring soon after injury, and so limiting the diffusion of blood and the obscuring of the imprinted pattern. Patterned bruises commonly have a bright red intra-dermal component whose diffusion is limited also by the dense collagenous dermal tissue. Occasionally

clothing or jewellery may leave a patterned bruise on a body when it is crushed into the skin surface by an impacting object, e.g. a motor vehicle striking a pedestrian, or a kick through clothing.

Sometimes bruises give a more general impression of the causative object, for example a doughnut-shaped bruise is produced by a hard object with a rounded contour, such as a cricket ball or a baseball. A similar mechanism produces two parallel linear bruises, so-called 'tram-line' or 'rail-track' bruises, as the result of a blow from a long object which has a circular cross-sectional shape, such as a police baton, or an electric flex. If the flex has been looped then this may be apparent as curved rail-track bruising. When a blow with a rod is struck against the buttocks, - a particularly pliable, curved, soft surface - the tissues are compressed and flattened under the impact and the resulting rail-track bruise will follow the curved contour of the buttocks. A pliable weapon such as a strap or electric flex may produce a similar appearance because it can wrap around the body on impact. Bruises produced by finger-pads as a result of gripping are usually larger than the finger-pads themselves, but their number, pattern and location on the victim suggests the mechanism of causation. Finger-pad bruising is seen on the neck in throttling, on the upper arms in restraint, on the thighs in rape, and on the chest and face in child abuse.

Bruises change colour as they age before finally fading away. A fresh bruise is dark red, the colour of venous blood, turning soon to a dusky purple. Thereafter the colour changes progressively from the periphery of the bruise towards the centre through brown, green, yellow and a pale straw colour before disappearing. These colour changes reflect the breakdown of haemoglobin into coloured products as part of the inflammatory process. The time frame of the colour change is extremely variable depending upon bruise size, depth, location and the general health of the individual, but most bruises disappear within one to four weeks. 'Love bites', which are small and superficial, typically complete this sequence in seven days.

In general, bruises which have a green or yellow margin are three or more days old and those which appear entirely dark red or dusky purple are fresh having occurred within a day or so. The accurate estimation of the age of a single bruise is not possible but fresh bruises are easily distinguished from bruises several days old. Establishing that bruises are of different ages, and therefore inflicted at different times, is important in the assessment of allegations of repeated assaults, such as in child abuse and spousal abuse. Chronic alcoholics commonly have multiple bruises of different ages over their legs and arms as a result of repeated falls when drunk, often with the extent of bruising made worse by disturbances of blood clotting secondary to alcoholic liver disease.

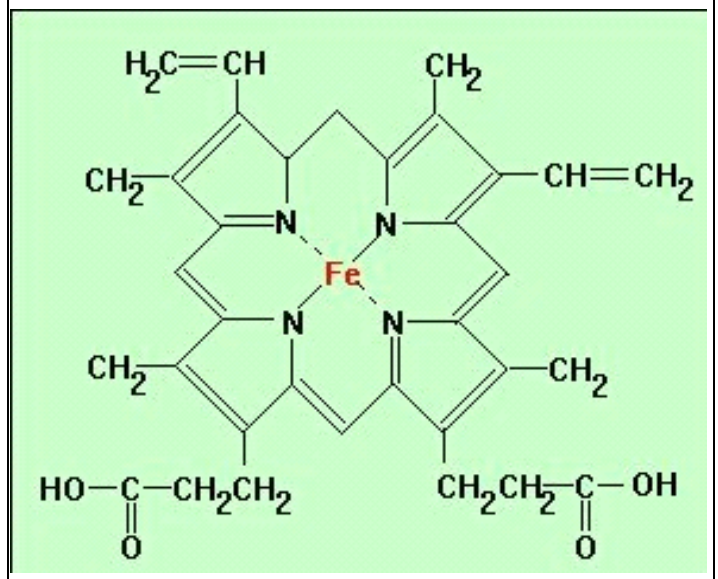
The colour of bruises does not change after death, but they may become more evident against the now pale skin, or alternatively be obscured by the post-mortem skin colour changes of lividity and decomposition. It is not possible to distinguish a bruise sustained at the time of death from one which occurred up to a few hours earlier, and such bruises are

best described as having occurred 'at or about the time of death'. If the microscopic examination of a bruise shows an inflammatory reaction then it was likely inflicted more than a few hours before death, and almost certainly more than half an hour before.

Haemoglobin and bilins

Haemoglobin, the oxygen-carrier of red blood cells, is a conjugated protein in which globin, the protein component, is combined with haemin, a ferric porphyrin component (see structural formula below). The intense colour of haemoglobin is not caused by iron but by porphyrin, a complex cyclic structure of four pyrrole rings (each pyrrole ring is a 5-membered closed structure containing 1 nitrogen and 4 carbon atoms) linked to each other by methine groups (-CH=). The iron atom is kept in the centre of the porphyrin ring by interaction with the four nitrogen atoms.

In oxyhaemoglobin, which is bright red, the ferrous ion is bound to oxygen. Deoxyhaemo-globin, or reduced haemoglobin, is oxyhaemo-globin minus oxygen and is purple in colour. The metabolic products of the haeme portion of haemoglobin include a series of yellow, green, red, and brown non-metallic compounds arranged as linear chain structures of pyrrole rings rather than in the cyclic configuration of porphyrins. These are the bilins, or bilichromes. One of these bilins is the blue-green compound, biliverdin (C₃₃ H₃₄ O₆ N₄), and another, bilirubin (C₃₃ H₃₆ O₆ N₄), is a yellow-red product formed from biliverdin by the addition of two hydrogen atoms. It is these and other biological pigments, or biochromes, formed as catabolic products of the porphyrin of haeme which are responsible for the colour changes in bruises.



Bruises to the deep tissues can be present without any evident skin surface injury, particularly if the force applied is by a smooth object over a wide area. Such deep bruises may spread under the influence of gravity and body movement, following the path of least resistance along natural or traumatic planes of cleavage of the tissues. This shifting of

deep bruises explains their delayed appearance at the skin surface some days after infliction, often at sites distant from the points of impact. The delayed appearance of bruising around the eyes follows a blow to the forehead, bruising behind the knee follows a blow to the lateral thigh or a fractured neck of femur, and bruising to the neck follows a fractured jaw. A second examination of a victim of an assault after an interval of a few days may show visible bruising where previously there had been only the swelling or tenderness of deep bruising. Such second examinations are recommended as best practice.

Bruising is essentially a vital phenomenon in which the infiltration of blood into the tissues occurs under the pressure of the circulating blood. After death, the lack of blood pressure means that it requires considerable force to produce a bruise in a corpse. Such post mortem bruises are disproportionately small relative to the force applied, which may be evident from associated fractures, and the resultant bruises are usually only a few centimetres in diameter. In assessing the possibility that bruising may be post mortem, the findings and circumstances as a whole should be considered, and against this background quantitative differences between ante mortem and post mortem bruises are usually so great that confusion is unlikely. It is seldom difficult to distinguish between injuries with vital bruising resulting from a vehicle running over a live body, and the tearing and crushing of dead tissues. However, finger-pad bruises to the insides of the upper arms may be produced by simply lifting a corpse, particularly in those elderly women who have abundant loose upper-arm fat, which is often congested due to post mortem lividity. Any livid dependent areas of a corpse bruise more readily post-mortem as a result of the vascular congestion.

Post-mortem lividity, which is gravitational pooling of blood within the blood vessels after death, may be confused with bruising. However, the pattern and distribution of lividity usually makes the distinction straightforward. In doubtful cases, incision of the skin discloses blood oozing from the cut ends of vessels and washing the cut surface removes the blood, whereas the blood infiltrating the tissues in bruises cannot be washed away. If needed the distinction may be confirmed by microscopic examination. In a fair-skinned corpse the congested muscles of the base of the thumb (thenar eminence) and dorsum of the foot may be visible through the thin overlying skin and superficially resemble bruising. Post mortem decomposition with its initial green discolouration of the anterior abdominal wall is readily distinguished from bruising by its colour and location. Putrefactive haemolysis of blood within blood vessels and decompositional breakdown of vessel walls results in extravasation and diffusion of haemolysed blood into adjacent tissues, and this haemolytic staining of tissue may entirely mask small ante mortem bruises, e.g. in the neck muscles in cases of throttling.

Lacerations

A laceration is a tear in tissue. Any tissue may be lacerated, and for example a fracture of bone or a rupture of an Achilles tendon is a laceration. Only lacerations of the skin are discussed here, and lacerations of deeper tissues are discussed

in later chapters. Split-like lacerations of the skin are the result of direct crushing, typically between an unyielding object on the one side and underlying bone on the other. Skin splits occur most commonly where the skin lies close to the underlying bones, such as the scalp, eyebrows, cheeks, elbows, knuckles, knees, and shins.

The splitting or tearing of the skin which produces a laceration characteristically leaves intact some strands of stronger subcutaneous tissue, such as small arteries, nerves and fibrous bands, which straddle the depths of the wound. These tissue bridges are of diagnostic importance in identifying the injury as a laceration and therefore as a result of blunt force, and not a cut wound resulting from a sharp-edged object. Lacerations are ragged-edged and commonly associated with bruising and abrasion, the latter indicating the point of impact. However, some lacerations produced by impact against a thin object, or edge of an object, can have sharply defined margins with no apparent bruising or abrasion, so that they may be confused with cut wounds. Inspection of the wound margins using a magnifying glass often allows the distinction to be made between a laceration and a cut, and recognising tissue bridges within the depth of the wound establishes conclusively that the wound is a laceration.

Lacerations do not generally reflect the shape or size of the object which produced them; they may be smaller than the impact area or may extend beyond the impact area. A stellate laceration radiating from a contact area follows a blow from an object such as a poker end. A linear weapon, such as a stick, commonly causes a linear laceration which may bifurcate at one end giving it a Y-shape, or bifurcate at both ends. If the impact is tangential and raises a skin flap then the direction of the flap is the direction in which the force was applied. A hammer, or weapon with a similar circular striking face, produces circular or crescentic lacerations. Lacerations to the insides of the lips result from crushing of the lips against the teeth following a blow to the mouth. Sometimes these mucosal lacerations can be matched to the edges of the teeth, particularly teeth which are mal-aligned and protruding or sharp-edged from caries.

Lacerations are produced also when the skin is stretched and the forces exceed the elastic limit, or tolerance, of the skin. These tears or stretch lacerations are seen in the groins of pedestrians struck from behind by a vehicle, or of persons who have fallen from a great height and landed with the legs bent back on themselves. Over-stretching injuries also occur in falls from aeroplanes or sky-scrapers as a result of flailing of the limbs, a phenomenon known as 'wind-milling'. Extreme tangential shearing forces, such as those produced by motor vehicle tyres when a pedestrian is over-run, produce flaying (or 'degloving') injuries with the raising of very large areas of skin from the underlying soft tissues.

Lacerations penetrate the full thickness of the skin to involve the underlying tissues and will bleed, but less so than a comparable cut wound. The disrupted blood vessels and tissue in a laceration are partly crushed and initiate thrombosis by the extrinsic pathway so reducing blood loss. The depths of a laceration often contain foreign material, a source of wound contamination to be cleaned away in clinical practice, but a

source of trace evidence to be collected in forensic practice. Trace evidence recovered from a laceration may link it to the impacting object, while blood and tissue recovered from the suspect object may be linked to the injured victim, through DNA techniques or less sophisticated blood and tissue typing.

CHAPTER 2

SHARP FORCE INJURIES

Sharp force injuries caused by objects which are keen-edged or sharp-pointed are the second major class of injuries. There are two types: incised wounds (cuts) and puncture wounds (stabs).

An incised wound is a breach of the skin resulting from contact with a keen edge. Penetration of at least the full thickness of the skin is usual, but shallow, partial-thickness skin cuts can occur, such as the common 'paper cut'. In clinical practice it sometimes happens that incised wounds and true lacerations are both loosely referred to as lacerations. This may create confusion when clinical medical records are used in legal proceedings because the diagnosis of an incised wound implies contact with a sharp-edged object or weapon, whereas the diagnosis of a laceration implies contact with a blunt object or weapon as the cause of the injury.

A puncture wound or stab wound is a penetrating injury with a depth greater than the wound length on the skin surface. Puncture wounds are caused by objects which are long, thin, and usually sharp-pointed, such as a knife. Both incised wounds and stab wounds typically retain no trace evidence from the weapon, but they are associated with blood staining of the weapon, clothing and scene.

Incised wounds

Most incised wounds are knife wounds, but this is not a conclusion that can be safely drawn from the examination of the wound alone. Similar wounds are produced by shards of glass, sharp edges of machinery and other sharp-edged objects.

The key features of incised wounds reflect the fact that they are produced by keen-edged objects rather than blunt objects. Incised wounds typically have well defined margins with no associated abrasion or bruising of the adjacent skin and there is always complete severance of all tissues within the depth of the wound, so that there are never any of the tissue bridges which characterise lacerations.

Occasionally incised wounds have irregular or ragged margins because either the causative object had an irregular edge, such as broken glass, or the cut was made across skin folds, such as on the neck or palm, so that the wound assumes a different shape when the folds flatten out. A single incised wound may be discontinuous if it involves surface ridges but misses troughs of soft tissue, for example across the helix of the ear, or across the tip of the nose and adjacent cheek. Similar discontinuous injuries can occur across joints so that the degree of joint flexion at the time of injury can be established by lining up the wounds, as with a cut across the anterior forearm and upper arm sparing the cubital fossa.

Incised wounds inflicted with heavy-bladed instruments, such as an axe, adze, sharpened spade, machete, meat-cleaver, hatchet, sabre, and boat propeller characteristically show abrasion, and sometimes bruising, of the wound margin. This

results when the edges of the first-formed incised wound are crushed by the entry of the following thick blade. The prominence of the abrasion is highly variable and usually less prominent if the blade is very sharp and not very thick, such as a sabre. However, the large size of the wounds and the usually associated cuts and fractures to underlying bone are more obvious pointers that the weapon used was heavy-bladed. An associated blow with the blunt poll at the back of an axe blade, or similar weapon may leave an imprint bruise-abrasion.

The shattering of the tempered glass side windows of vehicles during collisions produces a shower of small dice-like fragments which strike the vehicle occupant producing a characteristic pattern of scattered, small, irregular incised wounds associated with small irregular abrasions, the wound shapes reflecting the different angles of impact of the glass fragments. The incised wounds result from the impact of the sharp edges and corners of the dice-like fragments, and the abrasions result from the impact of the flat sides of the fragments. These dicing injuries occur on the side of the face adjacent to the shattered window, so that it is possible to determine whether the individual was seated on the left or right hand side of the vehicle at the time of impact, distinguishing driver from front-seat passenger.

Cuts produced by a serrated knife blade may give an indication of the nature of the blade if the cut is tangential to the skin surface so that the serrations leave shark-fin skin tags at the wound edge, or parallel scratch abrasions on the adjacent skin. Light contact with the skin by serrated blades can produce a line of punctate abrasions or parallel scratches without an associated incised wound. Blade serrations will leave corresponding striations on the cut surfaces of the cartilage of the ribs or larynx.

Accidentally inflicted incised wounds are common. They may result from the use of knives in the home or workplace, broken window glass in burglaries, broken drinks glasses, and sharp-edged machinery or parts of vehicles.

The ready availability of knives and the relative painlessness of incised wounds has encouraged their use in suicide since antiquity. The Romans were said to have favoured opening the veins of the arms whilst lying in a warm bath. The Japanese ritual suicide of seppuku involves both self-stabbing and self-incision. Self-inflicted incised wounds are directed towards parts of the body where large blood vessels are close to the skin surface, such as the neck, wrists and, less commonly, the elbows, knees and ankles. Suicidal incised wounds are commonly, but not invariably, accompanied by parallel, shallow, tentative wounds, reflecting a testing of the weapon as well as the indecision so often present in suicidal acts. These tentative wounds, also referred to as hesitation wounds, show a wide range of appearance between cases. Having produced one deep cut, the person intent on suicide may then produce repeated cuts in the depth of the wound, and these can be seen as multiple trailing cuts arising from the ends and sides of the principal wound.

The stereotypical suicidal cut throat is an incision starting on the side of the neck opposite the dominant cutting hand, and

passing slightly obliquely downwards across the midline. Any repeated cuts in the depth of the wound then produce sharply angled tags of skin at the wound ends and edges. The cuts may be so deep that the vertebrae are scored.

Self infliction of shallow cuts as a form of self harm falling short of attempted suicide is seen as parallel, shallowly incised wounds to the fronts of the wrists and forearms which heal leaving multiple, fine, horizontal, linear white scars. Often they are most prominent on the non-dominant arm. When seen in clinical practice, the scars raise the possibility of a personality or psychiatric disorder, with an attendant risk of suicide in custody. If seen at autopsy, they raise the index of suspicion for suicide, alcohol and drug abuse, and high risk behaviour leading to accidental death.

Occasionally individuals self-inflict incised wounds in order to falsely allege that they have been assaulted. These factitious injuries have a pattern which reflects handedness, easy accessibility of the injured parts, infliction by pressing the sharp edge against the skin and then running it across the skin surface rather than slashing movements, and the avoidance of sensitive or critical areas such as nipples, lips and eyes. Although usually superficial, these factitious injuries can be extensive and mutilating. Some individuals have self-inflicted such injuries in the pattern of the stigmata of Christ, claiming that they appeared spontaneously.

Individuals defending themselves against a knife attack commonly sustain defensive injuries to the hands and forearms, which range from subtle to devastating. Defensive wounds to the legs may be present in a victim who was attacked when on the ground. Cuts to the palms and fingers result from attempts to grab or deflect the weapon, while slash and stab wounds to the backs of the hands and the forearms result from shielding movements. By contrast with the pattern seen in defensive wounds, if the knife slips within the hand of the assailant then the blade typically cuts either the base of the little finger or the web of the thumb of the assailant. Defensive injuries to the hands and forearms although typical of knife attacks may be absent in sudden, overwhelming attacks (so called 'blitz' attacks), or if the victim is unable to offer a defence as a result of the effects of alcohol and drugs, unconsciousness, bindings, or other physical and emotional circumstances. Since defensive wounds reflect anticipation of injury and an attempt to ward off the harm, they may be seen in accidents as well as assaults. For example, multiple cuts to the palms of the hands can be produced in a fall onto a glass-strewn floor, as well as during an attack with a broken bottle.

Assaultive incised wounds to the face may be inflicted with the intention to disfigure. In a prison setting the classical improvised weapon for this purpose is a plastic tooth-brush handle into which is embedded two closely placed parallel razor blades. The Stanley knife, a work knife which has a very sharp retractable blade, is another weapon commonly used.

Cleanly severed blood vessels within an incised wound bleed profusely, and if large vessels are cut then the haemorrhage may be sufficient to kill. Such extensive haemorrhage

commonly results in blood soaking of clothing together with blood staining and spattering of surroundings, opening up the possibility of reconstruction of the events through blood spatter analysis. When large veins are severed, particularly in the neck, air may be drawn into the circulation, obstructing the flow of blood through the heart and killing by air embolism

Stab wounds

Stab wounds, like incised wounds, typically yield no trace evidence, are associated with blood and tissue staining of the weapon, as well as blood staining and damage to clothing, and offer the opportunity for reconstruction of the events from interpretation of blood spatter at the scene.

Stab wounds are penetrating injuries produced by a long, thin object, which is typically pointed. Most commonly the instrument is flat with a sharp point, such as a knife, a shard of glass or a length of metal or wood. Other weapons may be long and thin with a sharp point, such as a skewer, hypodermic needle, ice pick, or old-fashioned hatpin. With sufficient force even long, rigid objects which are blunt-ended will produce puncture wounds, e.g. a screwdriver, wooden stake, or protruding parts of machinery or motor vehicles. The appearance and dimensions of the resulting wound often provides useful information about the object which produced it.

The skin surface appearance of a stab wound is influenced both by the nature of the weapon and characteristics of the skin. The skin contains a large amount of elastic tissue and will both stretch and recoil. This elastic tissue is not randomly distributed but is aligned so as to produce natural lines of tension (Langer's lines) which can be mapped out on the skin surface. In survivors of knife assaults, the extent of wound scarring will be influenced by the alignment of the wounds relative to Langer's lines. Wounds which have a long axis parallel with Langer's lines gape only slightly, a fact made use of by surgeons who align their incisions in this way to promote healing and reduce scarring. Wounds aligned at right angles to Langer's lines tend to gape widely, and scar prominently, because the natural lines of tension of the skin pull the wound open. Wound gaping is also influenced by the extent of damage to the underlying supporting fascia and muscles.

When a stab wound in a corpse is gaping then the wound edges must be re-approximated to reconstruct the original shape of the wound, something that is easily achieved with transparent tape. It is the dimensions of the reconstructed wound rather than the original gaping wound that are of value in predicting the dimensions of the blade. One purpose in examining stab wounds is to establish whether potential weapons could have or could not have produced the wounds.

The dimensions of a stab wound give some indication of the dimensions of the blade of the weapon. If a stabbing with a knife is straight in and out then the length of the stab wound on the skin surface will reflect the width of the knife blade. However, there are important qualifications which apply. The skin wound length may be marginally (a few millimetres)

shorter than the blade width as a result of the elastic recoil of the skin. If the knife blade has a marked taper and the entire length of the blade did not enter the body then the skin wound length may not represent the maximum width of the blade. If the blade did not pass straight in and out but instead there was some rocking of the blade, or if it was withdrawn at a different angle from the original thrust, then the skin wound will be longer than the inserted blade width. Consequently, the most reliable assessment of blade width is made from the deepest wound with the shortest skin surface length, because this wound represents the greatest blade penetration with least lateral movement. A single weapon can produce a series of wounds encompassing a wide range of skin-surface lengths and wound depths. This is often seen in a multiple stabbing fatality and is consistent with the use of only one weapon. However, it is rarely possible to exclude any speculative suggestion of more than one weapon, and, by inference, more than one assailant.

The depth of the wound gives an indication of the length of the weapon. Clearly the wound track length may be less than the blade length if the entire blade did not enter the body. Less obvious is the fact that the wound track length may be greater than the blade length. This may occur if the knife thrust is forceful and the tissues are compressed, so that when the weapon is withdrawn the track length in the now decompressed tissues is greater than the blade length. This tissue compression effect is most marked in wounds to the anterior chest and abdomen, since the entire chest or abdominal wall can be driven backwards by the blow. A small pocket-knife, with a blade of about 2 inches or 5 cm, can cause, in a slim person, a fatal stab wound to the heart or one which penetrates the abdomen to transfix the aorta. An added difficulty in measuring the wound track length at autopsy is that the corpse is supine with the viscera in a slightly different relative position to a living person standing or sitting, and with the tissues dissected and displaced. For all of these reasons the wound track depth should be used with caution in predicting the blade length of the weapon. If by chance some fixed bone, such as a vertebra, is damaged at the end of the wound track, then the assessment of depth of penetration is easier, but still subject to inaccuracy.

As well as providing an indication of blade width and blade length, a stab wound may provide other useful information about the weapon. Wound breadth on the skin surface is a reflection of blade thickness and a typical small kitchen knife with a blade thickness between 1 and 2mm produces a very narrow wound. The use of a thicker-bladed weapon may be readily apparent from the measured wound breadth on the skin surface.

Most knives have a single-edged blade, with one keen edge and one blunt edge to the blade. The resultant wound reflects the cross-sectional shape of the blade and, with the wound gaping, often appears boat-like with a pointed prow and a blunted stern. Skin elasticity may distort the blunted stern shape into a double-pronged fishtail. The thicker the blade of the weapon, the more obvious is the blunting of one end of the wound when contrasted with the other pointed end. Knives with double-edged blades (daggers) are specifically designed

for use as weapons and produce a wound that is pointed at both ends, but an identical double-pointed wound can be produced by a thin, single-edged blade.

If the blade penetrates to its full length then the wound may show features from the hilt and adjacent part of the blade. A knife with a folding blade has a notch at the hilt end of the sharp edge of the blade. This notch, or 'kick of the tang' (the tang being the rectangular portion of metal from which the blade was forged), leaves a rectangular or square abrasion at the end of the wound if the blade penetrates to its full length. A hunting-type knife, having a rectangular metal plate between the blade and the handle, leaves rectangular abrasions, which may be subtle, at both ends of the wound. Any imprint from the hilt of a weapon is usually only partial because the knife entry is most often at an angle. Dove-tail abrasions originating from either side of the stab wound are typical of a wooden-handled blade. A semi-lunar abrasion at one side of a stab wound is typical of a pen-knife handle. Other protrusions from the base of the blade or hilt may leave abrasions characteristic of the weapon. Serrated blades or blades with a saw-type component, as is seen in some hunting knives, can leave parallel linear abrasions or incised wounds on the skin in association with the stab wounds. Serrated blades leave corresponding striations on cartilage and occasionally the dermis. Mottled bruising associated with the wound may be produced by the knuckles of the assailant's hand, and an imprint bruise-abrasion from the clothing reflects a similar mechanism of forceful impact from the hand.

The cross-sectional shape of the weapon, particularly the back of the blade, may be accurately reproduced if it passes through bone, eg skull, pelvis, sternum or ribs. At the same time, passage through bone may cause trace material from the blade, eg. paint, to be scraped off and deposited in the wound. Stab wounds in solid organs such as the liver retain the profile of the weapon, and this can be visualised by filling the wound track with a radio-opaque contrast material and taking an x-ray, or by making a cast of the wound using modern dental casting material.

Weapons other than knives may produce characteristic stab wounds. Bayonets, which have a ridge along the back of the blade and a groove along either side, to lighten the blade and facilitate withdrawal of the weapon, produce wounds like an elongated letter "T". A pointed metal bar which is square in cross-section typically produces a cruciate wound, whereas one which is circular in cross-section, e.g. a pitchfork, produces an elliptical wound. Similarly a triangular file will produce a three-cornered wound. If the cross-sectional shape of the weapon varies along its length, eg a screwdriver, then the depth of penetration will affect the appearance of the wound. A forked instrument produces paired stab holes at different distances depending upon the angle of penetration. A scissors leaves paired wounds, off-set and pointed towards each other. A screwdriver or arrow wound may be indistinguishable from a gunshot wound because all leave a central defect with a marginal abrasion produced by the same mechanism of an inward pushing with grazing of the skin margin. Post-mortem drying turns the marginal abrasion dark brown or black.

Relatively blunt instruments such as pokers, closed scissors and files, tend to bruise and abrade the wound margins, a feature not otherwise seen in stab wounds. The blunter the object and the thicker its shaft then the more likely will the skin surface wound become a ragged, often cruciate, split. In cases where the wound appearance is unusual, it is helpful to conduct experiments with a duplicate suspect weapon and pig skin, in order to see whether the wound appearances can be reproduced.

Stab wounds inflicted during a struggle, with knife thrusts at awkward angles and with movement of both victim and assailant during the attack, may show characteristics reflecting this. Even so, it is rarely if ever possible to reconstruct the positions of victim and assailant from the location and direction of the wounds. A notch on the otherwise cleanly cut edge of the wound is a result of withdrawal of the blade at a different angle from the entry thrust. Exaggeration of this effect leads to a V-shaped or even cruciate wound when there is marked twisting of the blade or twisting of the body of the victim. By contrast, clothing tends to twist with the blade so that a linear stab hole in clothing may overlie a V-shaped stab in skin. A linear abrasion (scratch) extending from one end of the wound results from the withdrawn blade tip running across the skin. A single stab hole on the skin surface may be associated with more than one wound track through the tissues, reflecting a knife thrust followed by continuing struggle or repeated thrusts of the weapon without complete withdrawal.

A commonly asked question in the courts is the amount of force required to produce a specific stab wound. This is usually a difficult if not impossible question to answer. The sharpness of the point of the weapon is the most important factor in determining the degree of force required to produce a stab wound. In general, relatively little force is required to produce a deeply penetrating stab wound using a sharply pointed weapon, and the amount of force is easily over-estimated. The greatest resistance to penetration is provided by the skin and once this resistance is overcome then the blade enters the tissues with greater ease. In this respect an analogy can be made with the stabbing of a ripe melon. The important implication is that the depth of the wound is not a measure of the degree of force applied. However, penetration of any bone implies a significant degree of force; all the more so if the tip of the blade has broken off and remains embedded in the bone, something which is best identified by X-ray. Similarly a significant degree of force may be inferred from the presence of the hilt mark of the weapon on the skin surface, an uncommon finding, or a wound track significantly longer than blade length, suggesting forceful tissue compression during the stabbing. Even so, the stabbing force may have been a combination of both the thrust of the weapon and also any forward movement of the victim, such as in a fall. This latter proposition is commonly raised by the defence, and is rarely possible to discount, in deaths from single stabs.

CHAPTER 3

GUNSHOT INJURIES

An understanding of gunshot wounds requires some knowledge of the construction and mechanism of guns and the ammunition which they fire. Rifles and handguns, but not shotguns, have rifled barrels. The rifling consists of spiral grooves cut the length of the interior or bore of the barrel. When a rifled weapon is discharged, the rifling causes the bullet to spin around its long axis, like a spinning-top, and this lends gyroscopic stability to the bullet in flight, increasing the accuracy of the weapon. Shotguns by contrast have a smooth bore with no rifling and are designed to fire multiple pellets, although they can fire a single slug. Rifled weapons, their ammunition and the injuries they produce are discussed first. Smooth bore weapons, or shotguns, their ammunition and the injuries which they produce are discussed later.

Rifled weapons

Handguns and rifles are the two most frequently encountered rifled weapons. Handguns, as the name implies, are designed to be fired from the hand and may be revolvers or auto-loading pistols, so called 'automatics'. Revolvers have a revolving cylinder containing several chambers each of which holds one cartridge, whereas auto-loading pistols have a removable magazine storing cartridges with a mechanism for auto-loading after each firing. Revolvers do not have manual safety catches. Rifles are firearms with a rifled barrel designed to be fired from the shoulder, and there are many designs around this theme. Other rifled weapons used by the military include submachine guns, which are designed to be fired from the shoulder or the hip and are capable of fully automatic fire, and machine guns which are heavier weapons also capable of fully automatic fire.

The ammunition or cartridges, for these rifled weapons consists of a cartridge case within the base of which is a chemical primer, with a chemical propellant above and the bullet clamped on top. The word bullet comes from the French word boulette which means a small ball, reflecting the fact that original projectiles were small lead spheres, the musket ball. Rifles are either rim-fire, having the primer located in the rim of the cartridge case, or are centre-fire, having the primer located at the centre of the cartridge case. Rim-fire rifles produce wounds similar to those of low velocity handgun wounds. Rim-fire cartridges are used mostly in small-bore, low velocity weapons, both handguns and rifles, of .22 calibre.

When the trigger of the gun is pulled, the firing pin strikes the base of the cartridge case where the primer is located, causing the primer to explode and to ignite the propellant. The propellant burns to produce large volumes of gasses under pressure. The original propellant, or gunpowder, used in firearms was black powder but this is now obsolete and smokeless powder is used instead. The heat generated by the burning propellant causes the cartridge case, which is typically made of brass, to expand and seal the chamber against any rearward escape of gasses. The large volume of hot gasses forces the bullet out of the cartridge case and down the rifled

barrel to exit the muzzle of the weapon. Bullets come in a variety of shapes. The simplest are made from lead alloys and are traditionally fired from revolvers and .22 inch calibre rifles. Other bullets have a lead core encased by a copper alloy metal jacket. In military ammunition this metal jacket completely encases the lead core – full metal jacketing. In hunting rifle and semi-automatic pistol ammunition the lead core is partly exposed at the tip of the bullet – partial metal jacketing.

Wound ballistics

Ballistics is the science of the motion of projectiles and wound ballistics is the study of the projectile penetration of tissues. A moving projectile, or bullet, has kinetic energy proportional to its weight and its velocity squared ($KE \propto WV^2$). The wounding potential of a bullet is directly related to its kinetic energy and since kinetic energy increases in proportion to increases in the velocity squared, it is high velocity projectiles which have the greatest wounding potential. The transfer of kinetic energy from the bullet to the tissues produces the wounding effect. If the bullet does not exit the body then all its kinetic energy will be transferred to the tissues, but if it exits the body then only some of its kinetic energy will have been transferred. Wobbling and tumbling of the bullet as it passes through the body, bullet deformation and break-up, long wound tracks and passage through denser tissues are all factors which influence the loss of kinetic energy and increase the wounding effect.

Bullets disrupt tissues by two principal mechanisms. Direct laceration of the tissues occurs as the bullet penetrates just as with any penetrating object. This is the main mechanism of tissue damage in low velocity gunshot wounds from such weapons as pistols, and the permanent cavity visible in the body accurately reflects the tissue damage produced by the bullet. The second mechanism of tissue damage is temporary cavity formation and occurs only in high velocity rifle wounds. The transfer of kinetic energy from these bullets is so great that they create a temporary cavity up to 30 times the diameter of the bullet at a pressure of 100 to 200 atmospheres over a time span of 5-10 milliseconds. The result is a small permanent cavity surrounded by a wide zone of haemorrhage and an even wider zone of tissue damage which may not be obvious on examination. Other mechanisms of tissue damage include shock-waves, travelling at the speed of sound, which may cause the rupture of gas-filled organs such as the bowel; the creation of secondary projectiles such as shattered bone fragments in gunshot wounds to the head; and the effects of discharge gases which enter the wound track and produce tissue disruption if there is hard contact between the muzzle of the weapon and the body at the time of firing.

Rifled gunshot wounds

A gunshot wound is a penetrating wound which leaves a skin defect where the projectile passes through the skin. As a general rule a gunshot exit wound is larger and more irregular than a gunshot entry wound due to the effect of bullet tumbling and bullet deformation. Characteristic of entry wounds, particularly from handguns, is an abraded margin

which surrounds the skin defect and is a result of the forward motion of the bullet indenting the skin and grazing it at the time of entry. The abrasion ring appears reddish-brown but can darken to almost black with post mortem drying. An abraded margin is typically absent from exit wounds, aiding their distinction from entry wounds. However, an abraded margin may be seen occasionally around an exit wound when the skin has been 'shored up' by a firm surface, such as a wall or a belt, so that the everted skin margins of the exit wound are impacted against the supporting surface. High velocity rifle bullets tend not to produce an abraded margin but rather micro-tears, which are fine radiating lacerations around the edge of the skin defect, best seen with a magnifying glass.

Lubricant and debris on the bullet surface is wiped off onto the wound edge and appears as a grey ring of bullet wipe, but this is often obscured by blood and is more frequently seen on overlying clothing. The presence of bullet wipe is an indication that the wound is an entry and not an exit wound. As well as the bullet, other products of discharge of the weapon exit the muzzle and may deposit on the clothing, the skin or enter the wound track. These products of discharge include soot, unburned, partially burnt and burning grains of propellant, and hot gasses including flame, which is incandescent gas. Together the effects of these are sometimes termed 'powder burns', but the more precise terms which refer to the effects of each element are preferable. Deposition of soot is described as smudging; propellant grains abrading and embedding themselves into the skin is described as tattooing; burning of the skin and its hairs is described as singeing.

If the muzzle of the weapon is in contact with the skin at the time of discharge then it may leave a muzzle impression, which is an imprint bruise-abrasion surrounding the skin defect of the gunshot wound. The mechanism producing a muzzle impression is the passage of discharge gasses into the wound track with resultant billowing out of the skin against the muzzle. In these circumstances the stretched skin may lacerate leaving an irregular stellate entry wound. Gunshot wounds through the skull, ribs, sternum and pelvic bones may leave coned defects with a smaller hole on the entry side of the bony plate and a larger bevelled defect on the other side.

The appearance of a gunshot entry wound will depend upon the range of fire, and determining the range of fire from the wound appearance is of forensic importance. If the shot is from a distance then only the bullet will strike the victim leaving a skin defect, typically with an abraded margin or with micro-tears in a high velocity wound, and possibly a grey ring of bullet wipe. At close range, which is within a muzzle to target distance of about arms length or 3 feet (1 metre), the defining characteristic is propellant tattooing on the skin surface. This feature may be blocked by head hair or overlying clothing, although the propellant grains commonly penetrate one or two layers of thin clothing to embed in the skin beneath. Individual grains of gunpowder should be retained for laboratory analysis since they may indicate the type of ammunition and therefore the type of weapon used.

In contact wounds, when the muzzle of the weapon is in contact with the body or only a few centimetres from the body,

all of the elements which exit the muzzle on discharge produce their effects on the wound. The edges of the contact wound are seared by gasses and blackened by soot and propellant baked into the skin, and this may form a broader seared blackened zone around the skin defect. Soot may be deposited in a wide zone around the wound. This soot deposit can be easily wiped off if the wound is washed. Washing blood from the wound carefully using a spray of hot water without scrubbing, should wash off the blood but leave behind any soot deposit. In suicides, the hand steadying the muzzle of the weapon against the skin may show soot deposition on the radial margin of the forefinger and the adjacent surface of the thumb. If the weapon is a revolver then soot deposits may arise also from the cylinder gap (the gap between the revolving cylinder holding the ammunition and the muzzle) when products of discharge emerge in a fan-like pattern at right angles to the long axis of the barrel. Soot, propellant grains, gunshot residues from the primer and gasses including carbon monoxide, as well as fragments of overlying clothing, are forced into and along the wound track.

Shotguns

Shotguns have a smooth bore and are designed to fire multiple pellets, although they can fire a single slug. The calibre or internal diameter of the bore of a shotgun is measured by an archaic system of gauge. The gauge of a shotgun is the number of lead balls of the given bore diameter required to make up one pound weight. The most popular shotgun gauge is 12, which represents a bore diameter of 0.729 inches (18.2mm). Confusingly a shotgun with a bore diameter of 0.410 inches (10.2mm) is not described by its appropriate gauge but as a 'four-ten'. At the muzzle end of most shotgun barrels is a partial constriction of the bore known as a choke, whose purpose is to restrict the spread of the fired pellets.

Shotgun ammunition, or shells, differ in some design aspects from cartridges for rifled weapons. The cartridge case has a brass lower part, or head, containing the primer mechanism and overlying propellant, and above this is a tube, traditionally made of compressed paper but nowadays of plastic. This paper or plastic tube is closed at the top to retain the lead shot within. Lead shot comes in a variety of sizes which broadly fall into two categories, namely bird-shot, which is used for birds and small game, and buck-shot which is used for large game. The standard pellet sizes are designated by a numbering system, and an individual cartridge bears the appropriate number to indicate the size of shot within. Bird shot pellets range in size from 0.05 inches to 0.18 inches in diameter. Buckshot pellets range in size from 0.24 inches (number 4) to 0.36 inches in diameter (number 000). By recovering a representative sample of the lead shot, determining the average weight and comparing this with a standard table, the size of the lead shot, a characteristic of the ammunition used, can be determined.

Ammunition in the form of single lead slugs is available and an improvised 'home made slug' can be produced by partially incising the shotgun shells circumferentially just above the brass head so that this line of weakness breaks on firing and the lead shot exits the barrel still within the otherwise intact

cartridge. Although single round lead bullets fired from smooth bore weapons are generally obsolete, they are still used in 0.410 inch muskets for riot control in India.

Within the shotgun shell the propellant and pellets are separated by plastic or cardboard wadding. When the shotgun is fired, these wads exit the muzzle and travel a short distance, sometimes up to 20 feet. At ranges up to about 8 feet the wadding may enter the wound but at greater ranges it impacts the skin adjacent to the wound to leave a wad abrasion. Modern plastic wadding forms a tube around the pellets which opens like the petals of a flower on exiting the muzzle and leaves distinctive petal mark abrasions around the shotgun wound or on the adjacent skin surface. The diameter of cardboard or plastic wadding recovered from a wound or crime scene gives an indication of the gauge of the shotgun used, and may give some indication of the ammunition manufacturer.

Shotgun wounds

In shotgun injuries the pellets rarely exit and therefore the wounding effect is the full wounding potential. Within increasing range, fewer pellets strike the target due to pellet dispersion, and the velocity of the pellets falls off rapidly due to poor pellet aerodynamics. As a result, the range of fire has a critical effect on injury severity in shotgun shootings. At ranges of 20 to 50 metres and beyond there is a uniform peppering of shot which is rarely lethal.

Shotgun wounds of entry have a skin defect and abraded margin and, depending upon range of fire, may show soot smudging, propellant tattooing, singeing from hot gasses and a muzzle impression as with gunshot wounds from rifled weapons. Shotgun wounds display other features reflecting the fact that they are produced by multiple pellets rather than a single bullet, and sometimes show wad marks from the cardboard or plastic wadding of the shotgun shell. Contact shotgun wounds to the torso appear externally innocuous with a circular defect approximating the weapon bore. A muzzle imprint is commonly seen and if there is loose contact or clothing between the muzzle and the skin then there is surrounding soot smudging. The large amount of carbon monoxide forced into the wound produces a faint cherry-red discolouration of the underlying muscle due to the formation of carboxyhaemoglobin.

Contact shotgun wounds to the head are extremely mutilating due to the large amount of energy dissipated by the shot and the large volume of gas which enters the wound. The great majority of wads and pellets will exit and typically the cerebral hemispheres are eviscerated. The shotgun entrance wound to the head is easily identified by the large amount of surrounding soot, the seared and blackened wound edges and the radiating large lacerations. By contrast the exit wound is often not identifiable because of the disruption of the head and fragmentation of the skull. When the entrance wound is intra-oral there are stretch lacerations in the naso-labial folds and at the margins of the mouth.

Propellant tattooing indicating a close range shotgun wound begins at a muzzle to body distance of about 1 to 2cm and disappears at a range beyond about 1 metre. Beyond this range the appearance of the wound reflects the spread of pellets and any impact of wadding. As the range increases the round skin defect develops scalloped margins (hence its description as a 'rat hole' wound in the USA), and then a few adjacent satellite pellet holes before a definite cuff of satellite pellets appears at a range of about 2 metres. At greater ranges there is considerable variation in the pellet pattern depending upon the ammunition used, the degree of choke of the shotgun and the actual weapon. The pattern of spread of the pellets is documented by photography, and measurements of the diameter of the spread are made ignoring aberrant outlying pellets, so called 'fliers'. The peeled back petals of plastic wadding may produce a patterned abrasion, in the shape of a Maltese cross, around the wound with the wadding entering the wound at ranges up to 2 or 3 metres. At greater ranges the trajectory of the wadding deviates from that of the pellets so that it impacts the skin adjacent to the wound leaving an abrasion. Precise estimates of range of fire in shotgun injuries require a comparison of the wound pattern on the skin and clothing with test patterns made by firing the same shotgun with the same ammunition. If the pellets strike an intermediate target, such as a pane of glass or wire door-screen, before striking the victim then this will increase the dispersion of pellets making range assessment liable to error. The dispersion of pellets within a body as indicated by X-ray is always greater than that seen on the skin surface as a result of the 'billiard ball effect' produced by the pellets bouncing off each other as they bunch up on entering the body. Shotgun exit wounds are usual where the entry is to the head or neck but exceptional where the entry is to the trunk, unless the trajectory is tangential through the body or there is a contact wound in a thin person or with buckshot ammunition.

X-rays

X-rays of gunshot victims, both living and dead, provide valuable information and X-rays should always be performed in autopsies on gunshot fatalities if the facilities are available. X-rays identify any projectiles present and their location, or may identify projectile fragments if the bullet has exited. The path of the projectile through the body can be determined and often the type of ammunition or weapon used. Projectiles do not necessarily follow a straight line path in the body but may ricochet off bones, most commonly the inner table of the skull, or enter the cardiovascular system and embolise. If the bullet is intact then the exact calibre cannot be determined due to the magnification effects of the X-ray. The presence of pellets generally indicates the use of a shotgun, although novel shot-containing ammunition is available for some rifled weapons. A 'lead snow-storm' comprising scores of lead fragments along the wound track as a result of fragmentation of a partial jacketed bullet is a diagnostic but not ubiquitous feature of high velocity rifle hunting ammunition. Military bullets are specifically designed not to break up in this way within the body but they may do so under some circumstances. With partial metal jacketed bullets, the jacket and core can separate in the body and the lead core exit leaving behind the jacket, with its important rifling marks. Large shotgun slugs are

distinctive with designs specific to the manufacturer. The components of some projectiles, such as aluminium jackets, plastic tips and plastic shot shell wadding, are poorly radiopaque and difficult to identify on routine X-rays.

Bullet embolisation is uncommon but may occur within the arterial or the venous system. Arterial embolisation occurs when the bullet enters the heart or aorta and typically embolises to the legs. Venous embolisation occurs when the bullet enters a large vein such as the vena cava or a cerebral sinus and then embolises to the right heart or pulmonary arteries. Small calibre, low velocity projectiles such as a .22 calibre bullet or birdshot pellets are most likely to embolise.

Ballistics

Rifles and handguns, but not shotguns, have rifled barrels. The rifling comprises spiral grooves cut the length of the interior or bore of the barrel. The raised metal between the grooves is the lands, and the calibre of a weapon is measured from land to land. When a rifled weapon is discharged, the rifling produces marks on the bullet as the bullet passes down the barrel. Some rifling marks have class characteristics indicative of the make and model of the firearm. Other markings have individual characteristics which reflect imperfections peculiar to a particular firearm, and may allow the identification of that specific firearm.

The class characteristics of bullet rifling marks include (1) the number of lands and grooves, which are usually between 4 and 6 but range from 2 to 22. (2) the diameter of the lands and the grooves. (3) the width of the lands and the grooves. (4) the depth of the grooves. (5) the degree of twist of the rifling, that is the number of inches or centimetres of bore required for one complete rifling spiral. (6) the direction of the rifling twist, which is commonly a right or clockwise twist and less commonly a left or counter-clockwise twist.

The individual characteristics of bullet rifling marks include (1) imperfections of the grooves, which are most readily seen in lead bullets as a result of the soft lead being pressed into the grooves. (2) imperfections of the lands, which are most readily seen in jacketed bullets because the copper alloy jacketing is harder.

In a shooting in which a jacketed bullet has been used and the jacket has separated from the lead core, then it is the jacket rather than the lead core which must be recovered for ballistic comparison. Linking a fired bullet to the suspect firearm requires the comparison of the recovered bullet with a test bullet. The test bullet is obtained by taking similar ammunition and firing it from the suspect weapon. The rifling markings on the test bullet and the crime bullet are then compared using a comparison microscope. This type of microscope has a split field in which the crime bullet and test bullet rifling markings can be viewed simultaneously and the bullets rotated to align the markings. If the usually 26 to 28 inch (66 to 71cm) long barrel of a shotgun is sawn off to aid concealment of the weapon for criminal purposes, then the rough edges around the newly fashioned muzzle may leave marks with individual characteristics on the exiting plastic

wadding, permitting a ballistics comparison similar to rifling marks.

Cartridge cases also have markings with class and individual characteristics which include: (1) the type of breach block marking. (2) the size, shape and location of extractor marks (3) the size, shape and location of ejector marks. (4) the size, shape and location of firing pin marks.

Fingerprints are rarely recovered from firearms but may be obtained from cartridge cases, which are necessarily gripped between the fingers when the weapon is loaded. Cartridges too small to retain useful fingerprints may have recoverable DNA.

Testing for gunshot discharge residues

The primer, which explodes on compression and ignites the propellant, is commonly a mixture of lead styphnate, barium nitrate, and antimony sulphide. The metals lead, barium and antimony are vaporised and exit the muzzle, to condense and settle on surrounding objects including the hands of the shooter. Bullet metals also may be included in these gunshot residue particles. Cotton tipped swabs moistened with either 10% hydrochloric acid or 5% nitric acid are used to recover residues from the hands of suspects. When taking the hand-wipings plastic gloves must be worn. The 4 areas swabbed are the backs of both hands and the palms of both hands, and each area is swabbed twice. On the back of the hand, the radial aspect of the forefinger, the dorsal aspect of the thumb and the skin web between is swabbed. On the palm of the hand the palmar aspect of the forefinger and thumb together with the base of the forefinger and thumb and the skin web between is swabbed, or alternatively the entire palm is swabbed. The swabs are then analysed for traces of the elements lead, barium and antimony which characterise gunshot discharge residues.

Flameless atomic absorption spectrometry (FAAS) is used to detect all three metals, and neutron activation analysis (NAA) can be used to detect antimony and barium only. Both NAA and FAAS are quantitative elemental analytical methods which do not distinguish the source of the metals, so that false positives are theoretically possible. Barium is a common element found in soil and lead is a common environmental pollutant from motor vehicle exhaust fumes. An alternative analytical method is to recover the residues using adhesive material and examine them using scanning electron microscopy (SEM) and elemental dispersive X-ray analysis (EDAX), which can identify gunshot residue by its characteristic appearance as well as elemental content.

In practice gunshot residues are detectable in about 90% of people who have fired handguns, but only about 50% of people who have fired rifles or shotguns. It is not only the presence and quantity of the elements, but also their distribution on the hands which is evaluated. Residues on the palms of the hands only may indicate a defence gesture, or alternatively the handling of a previously fired weapon. In cases of suicide, residues are often detected on the non-firing hand used to steady the muzzle against the body. Recently lead-free primer has been developed because of health and

safety concerns about the accumulation of lead in the atmosphere in firing ranges. This novel primer contains titanium and zinc. Firing different ammunition in succession from the same weapon produces residues with a mixture of elements from the different primers and bullets used.

Gunpowder

Gunpowder is a generic term for any one of several low-explosive mixtures used as propellant in guns. The first gunpowder was black powder which is a mixture of saltpetre (potassium nitrate), sulphur, and charcoal (carbon) in the approximate proportions 75:15:10. The saltpetre was originally extracted from compost piles and animal wastes.

The consensus is that black powder originated in China, and was being used in fireworks and signals by the 10th Century. By about 1300 the Arabs had developed the first real gun, a bamboo tube reinforced with iron. Whether knowledge of black powder entered Europe by contact with the Mongols or the Arabs is unclear but the English medieval scholar Roger Bacon recorded its recipe in 1242 in a coded form of Latin.

Black powder is relatively insensitive to shock and friction and has to be ignited by flame or heat. Its burning is a surface phenomenon so that the fineness of the granulation influences the explosive effect. The product of burning is 40% gaseous and 60% solid, the latter accounting for the large amount of whitish smoke produced. Beginning in the middle of the 19th Century black powder was supplanted by nitrocellulose-based propellants. Nitrocellulose is manufactured by nitrating cellulose fibres from cotton or wood pulp using nitric and sulphuric acids. It is inherently unstable and burns by decomposing rapidly, forming hot gasses. By contrast with black powder, it produces almost all gas upon combustion, from which it obtained the name smokeless powder.

Today most forms of gunpowder are either single-based, that is nitrocellulose alone, or double-based, comprising a combination of nitrocellulose and nitroglycerine. Glyceryltrinitrate, or nitroglycerine, is a detonating or high explosive characterised by extremely rapid decomposition and the development of high pressure. It is an important ingredient of most forms of dynamite but is employed also in medicine as a vasodilator in the treatment of angina pectoris. Nitroglycerine is 18.5% nitrogen and contains sufficient oxygen to oxidise the carbon and hydrogen atoms in its molecular structure while at the same time liberating nitrogen. Nitroglycerine was first prepared in 1846 by the Italian chemist Ascanio Sobrero, but it was the Swedish scientist Alfred Nobel who invented dynamite in 1867. Nobel died in 1896 leaving the bulk of his fortune in trust to establish the Nobel Prizes.

CHAPTER 4

BURNS AND ELECTRICAL INJURIES

A burn is an injury caused by heat or by a chemical or physical agent having an effect similar to heat. Most burns are produced by dry heat, and result from contact with flame or a heated solid object, or from exposure to radiant heat. Burns caused by moist heat are described as scalds. Chemical burns are produced by acids and alkalis, or by vesicants developed for chemical warfare. Microwaves and electricity also produce burns.

Circumstances

The majority of burn deaths are related to accidental domestic fires, in which children and the elderly are particularly at risk, as a result of carelessness coupled with an inability to effectively combat or escape the fire. Alcoholics and drug addicts are a third at risk group. Suicide by burning is rare, likely because of the pain involved. It is more common in eastern cultures than in the west, and the ritual suicide of a Hindu widow on the funeral pyre of her husband, Suttee, is now prohibited in India. Rarely a homicide victim may be doused in flammable liquid and then set alight. Necklacing is a method of homicidal burning developed in South African townships; it involves placing a vehicle tyre around the neck of the victim (hence 'necklacing') and setting it alight. Petrol bombs, or 'Molotov cocktails', are improvised incendiary devices which have become popular in urban disturbances, but do not usually result in death.

Burns resulting from flaming clothing have a pattern reflecting both the nature of the clothing and the position and movements of the victim. The area and depth of burn tends to be greater with the faster burning cotton fabrics than with polyester, while higher fabric weight reduces burn severity. Flash burns, which are typically partial thickness, result from sudden deflagrations burning exposed skin surfaces which are not protected by clothing. Burns produced as a result of contact with a hot solid object often leave a brand-mark in the shape of the object, for example the triangular base of an iron. Burns from cigarettes are of the expected size, round and punched out. To produce cigarette burns requires firm contact for some seconds, and implies deliberate infliction, more obviously so when multiple.

Burn severity

The severity of a burn caused by dry heat is assessed by two parameters, firstly the depth of the burn injury, and secondly the extent of injury, that is the size of the burn relative to the total body surface area. In practice it is a common error to underestimate the depth and to overestimate the extent of burns. Other factors determining mortality are the location of the burn, the age of the victim and the presence of other injuries or natural disease. Burns involving more than 50% of the body surface carry a poor prognosis. However, age is a major factor for children under 2 years and adults over 60 years, so that in these victims more than 20% surface area involvement carries the same poor prognosis. In the first 2

days after burning the major threats to life are hypovolaemic shock and shock-induced organ failure, primarily renal failure.

The traditional classification of burn depth is into three degrees. A first degree burn involves only the epidermis and is characterised by erythema, oedema, and pain. First degree burns are produced by prolonged exposure to low intensity heat or very brief exposure to high intensity heat. Healing, associated with skin peeling, is usually uneventful and completed in five to ten days with no residual scarring. A second degree burn involves both the epidermis and a variable depth of the underlying dermis. The most superficial second degree burns implicate only the upper third of the dermis and are characterised by blister formation. They are extremely painful but heal in 7 to 14 days with minimal scarring. A deep second degree burn extends beyond the upper third of the dermis but not beyond the dermis itself. Paradoxically these deeper burns are less painful as a result of destruction of nerve endings in the dermis. Healing is extremely slow, sometimes requiring months and usually leading to dense scarring, if not treated by skin grafting. A third degree burn destroys the full thickness of the epidermis and dermis. Heat coagulation of dermal blood vessels leaves the tissue avascular with a characteristic waxy white appearance. If there is prolonged contact between subcutaneous fat and flame then the burn has a leathery brown, or black, charred appearance. There is characteristic lack of pain, due to heat destruction of all nerve endings. Spontaneous regeneration of skin (primary re-epithelialisation) will not occur and such burns require skin grafting. Burns can also be classified, according to the modern classification, as partial thickness or full thickness, depending upon the depth of skin involved. Beneath any burned skin there is usually a zone of marginally viable tissue which is readily converted to non-viable tissue by physiological stressors. In this way a second degree burn frequently converts to a third degree burn.

The second parameter of burn severity is the extent of injury. This is expressed as the percentage of the total body surface area which is burnt. It can be estimated using the 'rule of nines' which divides the body into areas representing 9% or multiples of 9% of the total body surface. Thus the head and neck are 9%, each arm 9%, each leg 18%, the anterior trunk 18%, the posterior trunk 18%, and the genitalia and perineum 1%. In making the assessment a very rough guide is that the victim's palm is approximately 1% of the total body surface area. In children under 15 years body proportions differ and the estimates must be age-adjusted.

Scalds

Scalds are produced by moist heat which may be steam or any hot liquid, such as water, oil or even molten metal. They are typically less severe than burns produced by dry heat. The scalded area appears erythematous with desquamation and blistering of the usually sharply demarcated area of injury. Unless the liquid is super-heated, such as oil, there is no singeing of hairs or charring and carbonisation of tissues. Scalds generally occur on exposed skin. In clothed areas the influence of the clothing on the severity of the injury is variable. Fabrics which are poorly permeable protect the skin,

whereas absorbent fabrics may hold the hot liquid against the skin, reduce natural cooling, and make the scald more severe.

The pattern of the scald can give an indication as to how it occurred. Major areas of scalding are sharply demarcated with trickle marks reflecting the flow of hot liquid under the influence of gravity. There may be splash marks. Dipping injuries of the limbs appear as well-demarcated glove and stocking scalds. Distinguishing accidental from deliberately inflicted scalds, by evaluating the pattern of injury against the alleged circumstances, is of particular importance in childhood injuries, both fatal and non-fatal. Industrial accidents involving super-heated steam produces severe scalds in a pattern similar to flash burns.

Chemical burns

Chemical burns are produced by corrosive acids and alkalis. As with scalds, the pattern of chemical burns can give an indication as to how they occurred. Chemical burns are commonly accidental; homicide by this method is rare. Throwing liquid corrosives, such as acid, over a victim (vitriol-throwing or vitriolage) is more often intended to produce facial disfigurement than death. Suicide by the ingestion of strong acid or alkali has now become rare in the developed world but is still seen in poorer countries. Immediate symptoms are pain, vomiting and difficulty in breathing and swallowing, followed by shock in serious cases. Typically there is staining of the lips, and often the cheeks, chin and neck, as well as chemical burns of the mucosa from lips to stomach, sometimes extending into the small bowel. Oesophageal and gastric perforations are most common with sulphuric and hydrochloric acids, and can be precipitated by attempts to pass a tube for gastric lavage. Contamination of the airways may lead to bronchopneumonia in survivors, and long-term complications include oesophageal and other stenoses.

The amount of tissue damage caused will depend upon the strength of the chemical, its concentration, the quantity applied to the skin or mucosal surface, the duration of contact, and the extent to which it penetrates the tissues. Chemical burns continue to develop for as long as the causative agent is not neutralised by another chemical, or inactivated as a result of reaction with the body tissues. While the pattern of injury on the skin surface resembles that of scalds caused by liquids, the injuries differ in physical appearance, reflecting different mechanisms of tissue damage. Acids with a pH less than 2 precipitate proteins causing coagulation necrosis. The resultant burns are clearly demarcated, dry and with a leathery scab, the colour of which depends upon the acid. Nitric acid gives a yellow-brown scab, sulphuric (vitriol) a black-brown scab, hydrochloric acid (spirit of salt) a white to grey scab, and carbolic acid (phenol or Lysol) gives a light grey to light brown scab. Alkalis with a pH above 11.5 cause more tissue damage than acids because they induce liquefactive necrosis, which facilitates ever deeper penetration of the alkali. The caustic alkalis, such as sodium hydroxide (caustic soda or lye) and ammonium hydroxide, leave a grey-white mucoid burn.

Chemical warfare agents

One group of chemical warfare agents produce skin burns. The best known of these vesicants is mustard gas, or more correctly sulphur mustard, which was named because its smell is like garlic or mustard. First used during World War I, it is mainly an incapacitating agent with a mortality rate of 2-3%. Death within 24 hours of exposure is very rare, while later deaths are the result of respiratory effects or bone marrow depression with associated infections.

The vapour of sulphur mustard rapidly penetrates clothing to damage the underlying skin. Blisters are characteristic of mustard gas, by contrast with acids and alkalis which do not produce blistering when they burn the skin. Following exposure, there is a latent, symptom-free and sign-free period of some hours. If the exposure is severe, the first sign is striking erythema, reminiscent of scarlet fever. There is mild skin oedema, and itching is common. As the erythema fades, areas of hyperpigmentation are left behind, as occurs with sunburn. By 18 hours typical vesicles appear. These blisters are uncomfortable but not painful, although they are delicate and easily rubbed off, leaving the now painful blister base. Crops of new blisters may appear as late as the second week post-exposure. Deep burning, leading to full thickness skin loss, is followed by eschar formation. The effects on the eye mirror those in the skin and late onset blindness is a distressing effect of exposure.

Lewisite is another chemical warfare agent which, like mustard, is a vesicant. It is non-persistent, making it possible for attacks to be launched on previously contaminated ground. However, lewisite is rapidly hydrolyzed when mixed with water, making it ineffective as a weapon under wet conditions. It has not been used in warfare but deaths have resulted from accidental exposure. A related arsenical vesicant, phenyldichlorarsine, was used on a large scale during World War I because it was capable of penetrating the respirators then available.

Electrical burns

The principal bodily barrier to an electrical current is the skin, and once beyond the dermis the current passes easily through the electrolyte-rich fluids. The current, the flow of electrons, tends to take the shortest route between entry and easiest exit to earth (ground). Alternating current (AC) is more dangerous than direct current (DC), so that DC injuries are uncommon but examples include encounters with lightning, car batteries, electro-plating, some public transportation systems, and some industrial systems. The effects of AC depend on the magnitude, frequency and duration of the current, whereas the voltage is of importance only because it is a factor in determining the current, in accordance with Ohm's law which states that $\text{current} = \text{voltage} \div \text{resistance}$.

The mechanism of death in electrocutions is most commonly a cardiac dysrhythmia, usually ventricular fibrillation, less commonly paralysis of the respiratory muscles, and rarely a direct effect on the brain stem as a result of passage of the current through the head and neck. Hand-to-hand passage of a

high-voltage current has a reported immediate mortality of 60% as a result of cardiac arrhythmia. Generally, for those who survive an electric shock, the prognosis is good and the majority make a complete recovery, so that delayed deaths from electrocution are uncommon.

Skin burns are a common form of electrical injury and a pathognomonic marker for death by electrocution. However, in some fatalities there may be no visible injury. When present, the typical skin lesion is a thermal burn resulting from the heating of the tissues by the passage of the electric current. Tissue damage from this heating effect may be insufficient to produce a visible injury if the surface contact area is broad and the conductivity of the skin is high because of dampness. When they occur, electrical burns may be of firm contact or spark (arc) type. Both types can occur in the same victim as a result of the irregular shape or movement of the conductor, or movement of the victim during electrocution.

A firm contact electrical burn of entry typically leaves a central collapsed blister, which may reproduce the shape of the conductor, with a surrounding areola of pallor. The blister is created by steam, and when the current ceases, the blister cools and collapses to leave a crater with a raised rim. Should the blister burst during its formation, as a result of its large size or the continued passage of current, then the epidermis may peel off leaving a red base. With the passage of an electric current, metallic ions from the metal conductor combine with tissue anions to form metallic salts which are deposited, and may be demonstrated by chemical, histochemical and spectrographic techniques. The histological appearance of electrical skin marks is closely similar to thermal injuries with cellular eosinophilia and nuclear streaming.

Contact electrical burns at exit points are not seen often but should be searched for. When present, in low voltage deaths, they are similar to, but less severe than, the corresponding entry mark. In high-voltage (more than 1,000 volts) electrical burns the contact injury of exit often appears as a blow-out type of wound.

A spark (arc) burn occurs when there is an air gap between the conductor and the skin so that the current arcs across the gap as a spark. The distance which the spark can jump is proportional to the voltage, so that 1,000 volts can jump a few millimetres, 5,000 volts can jump 1 cm, and 100,000 volts can jump 35 cm. The extremely high temperature of sparks causes the epidermal keratin to melt and, after cooling, this leaves a raised brown or yellow nodule of fused keratin surrounded by an areola of pale skin. A brief arc transmits only enough energy to cause a superficial skin burn which is most commonly seen on the hands. High voltage spark burns may cause large areas of skin damage resulting in an appearance of 'crocodile skin'. Spark burns involving the clothing can cause the clothing to ignite, so that the victim suffers superimposed flame burns.

The severity of the electrical injury to the deep tissues depends upon the amperage, i.e. the actual amount of current passing through the tissues. Although it is impossible to know the

amperage, it can be inferred from the voltage of the source as either high or low. A low-voltage household source is capable of causing death if a sufficient current passes through the body, and 60 milli-amperes (mA) will produce cardiac fibrillation. However, no deep tissue damage is evident at autopsy because the current pathway is too diffuse to cause thermal damage. Consequently, there are no characteristic internal findings in fatal electrocutions, although skeletal fractures and joint dislocations may occur as a result of tetanic contractions. Skeletal muscle damage leads to release of myoglobin, and muscle-specific intra-cellular enzymes, with resultant myoglobinaemia and myoglobinuria in survivors.

A high-tension source producing a current of 5,000 mA or more is usually required to produce severe widespread tissue necrosis, which is a result of not just heat but also short-term effects of electric fields. Even very brief exposures to high amperage will produce massive deep tissue damage. These types of electrical injuries are more akin to crush injuries than to thermal burns in that the damage below the skin is usually far greater than the outward appearance would indicate. If, after death, the electric current continues to flow then there may be severe damage to the body with peeling and blistering of the skin, charring, and cooking of the underlying tissues.

Most electrocutions are accidental and the bathroom is a place of particular danger because of the wet conditions. An unusual and characteristic finding in electrocution in the bath is that the subsequent development of hypostasis (post-mortem lividity) is limited by the water-line, resulting in a stark and unusual demarcation. Currents of less than 0.2 mA will not cause a skin injury or death by electrocution but are sufficient to evoke a startle reaction and may precipitate a lethal accident, such as a fall from a height. Currents of 30 mA cause muscular spasm, so that the hand of a victim will continue involuntarily to hold-on to a live conductor which had been accidentally gripped. Suicidal electrocution is uncommon but increasing, and may be difficult to distinguish from an accident. Homicidal electrocutions are also rare, except in a judicial setting; the first execution by electricity was in New York in 1890.

Lightning

Each year, lightning causes hundreds of deaths world-wide, especially in the tropics. A lightning bolt is produced when the charged undersurface of a thunder-cloud discharges its electricity to the ground. Very large electrical forces are involved with currents up to 270 kA. The lightning may directly strike the victim, strike a nearby object and then jump from the object to the victim (a side flash), or strike an object which conducts the lightning to a victim in contact with the object e.g. a worker in contact with a metal crane. The lightning current may spread over the body surface, pass through the body or follow both routes. The resulting injuries may be electrical, burns, or blast from the wave of heated air created by the lightning strike. The pathological findings range from a completely unmarked body to bizarre and extreme trauma. An unmarked dead body found in the open should raise the possibility of a lightning strike. When injuries are present, the clothing may be torn and the shoes burst, with the

body partly stripped and the clothing scattered. The hair may be seared. Patterned skin burns result from the heating of metal objects such as zippers, which are left magnetised or melted. Severe burns, with blisters and charring, rupture of the tympanic membranes, fractures and lacerations may be found. Fern-like or aborescent patterns on the skin are pathognomonic of a lightning strike but are rarely seen. They appear as irregular red marks, several inches long, which tend to follow skin creases and the long axis of the body.

CHAPTER 5

HEAD INJURIES

In developed countries head injury is a leading cause of death in young adults and of disability in survivors. About one third of all trauma deaths and half of road traffic deaths are attributable to head injury. The head is particularly vulnerable to acceleration/deceleration and rotational forces because it is heavy in relation to its size (average 4.5 kg or 10 lb), is freely mobile in three dimensions and occupies a relatively unstable position on the neck. Blunt force head injuries are associated with acceleration/deceleration of the head which causes movement of the brain within the skull. Head injuries of this type have mechanisms and pathological features distinct from gunshot injuries. Gunshot injuries in general do not produce significant acceleration-deceleration and are not characterised by diffuse brain damage, the exceptions being high velocity gunshot wounds and some contact gunshot wounds. Gunshot injuries to the head are discussed in the chapter on gunshot injuries.

Following a blunt force head injury there may be loss of consciousness and, in general, the longer the period of unconsciousness and the deeper the coma then the more likely it is that irrecoverable brain damage has occurred. Cerebral concussion, or stunning, following head injury is characterised by temporary loss of consciousness due to a disturbance of brain function without any identifiable pathological changes in the brain, should the individual die from an unrelated cause. Therefore in an individual who has suffered a minor head injury, for example from punches, and subsequently died from another cause, for example a gunshot wound to the chest, the absence of injury to the brain does not necessarily indicate that there was no loss of consciousness from the punches. Concussion may be followed in survivors by retrograde amnesia, which is loss of memory extending backwards in time from the moment of impact. In clinical practice the severity of a head injury is assessed using the Glasgow Coma Scale (GCS) which produces a numerical score from 3, the worse, to 15 (normal), based upon the assessment of ocular, verbal and motor responses to testing.

When there is brain damage following head injury it may be the immediate result of the physical force, so called primary trauma, or a secondary patho-physiological effect initiated by the primary trauma. The term 'primary trauma' encompasses blunt force injuries to the scalp, skull fracture, contusions and lacerations of the brain, various intracranial haemorrhages, and traumatic axonal injury (TAI). The secondary effects include hypoxia/ischaemia, brain swelling and resulting raised intracranial pressure, and infection as a late complication.

Infection is usually the consequence of a skull fracture. Fractures of the cranial vault and the skull base allow bacteria to enter the subarachnoid space from the air sinuses, resulting in meningitis. An open head injury, with a scalp laceration exposing an underlying skull fracture, predisposes to the development of a brain abscess.

Primary trauma

The scalp consists of several distinct layers: hairy skin, the subcutaneous fat and connective tissue layer, the galea or aponeurosis (a thin fibrous layer to which the flat epicranial muscles are attached), a thin layer of connective tissue and finally, the periosteum of the skull. Blunt force injuries to the scalp are discussed in the chapter on blunt force injuries. Importantly these scalp injuries, particularly abrasions, are indicators of the point of impact. A notable exception is bruising of the scalp over the mastoid area behind the ear, 'Battle's sign', which develops as a consequence of a basal fracture of the skull involving the middle cranial fossa, and is not therefore an indicator of an impact at this site.

Bruises of the scalp are associated with prominent oedema and tissue swelling, if the survival time is sufficiently long for this to develop. Discolouration from the bruise may not be obvious from the scalp surface because of hair, the thickness of the skin of the scalp, and the tendency for the bruising to develop not within the scalp itself but beneath the thick fibrous membrane, the galea aponeurotica, which lies between the scalp and the skull. At autopsy an incision is made from ear to ear and the scalp peeled forwards and back to disclose its undersurface when any bruising present is revealed. Since the scalp is very vascular, any lacerations bleed profusely leaving blood spatter evidence at the scene. Head injuries may passively ooze a considerable amount of blood after death, so that the head comes to lie in a large pool of blood. Any blunt object, weapon or otherwise, producing a scalp injury may have trace evidence in the form of blood, tissue and hair present upon it. For comparison purposes, a control hair sample, and a DNA sample, either an oral swab or blood in the living or skeletal muscle in a corpse, are routinely taken from the victim.

Skull fracture

A skull fracture is an indicator of the application of a significant degree of force to the head with the associated risk of intracranial haemorrhage and cerebral injuries. It is the associated intracranial trauma rather than the skull fracture which is life-threatening. The cranial vault, the calvarium, is a structure of bony plates with thickened buttresses and an irregular base. When blunt force is applied to the skull some distortion in shape may occur and forces are transmitted along the bony buttresses towards the skull base until the structure yields and a fracture is initiated at the point of maximum stress, which is commonly distant from the point of impact on the head. The force required to cause a fracture is very variable but may result from merely walking into a fixed obstruction (70 Newtons or 5 foot-pounds of force), from the 4.5 kg adult head falling from a height of 1 metre onto a hard surface (500 N), the head falling from a standing position (870 N), running into a obstruction (1000 N), or from a 100g stone thrown with moderate force against the thin bone of the temple.

The different types and patterns of skull fracture give an indication of the mechanism of causation. In making such an

interpretation the evidence provided by the scalp injuries must be considered also. A bone bruise, comprising haemorrhage into the bone and multiple micro-fractures, resulting in discoloration of the skull surface but no visible fracture line may follow a blunt impact. The simplest fracture is a linear or fissured fracture of the cranial vault which may extend along the bony buttresses to the base of the skull. A fall onto a broad flat surface or a blow produces a fissured fracture. The cranial vault comprises two bony layers, referred to as tables, with a looser spongy scaffolding of bone, the diploë, sandwiched between, and a fissured fracture may involve both the outer table and inner table of the skull or either. If two separate impacts to the skull produce linear fractures, then the sequence is indicated by the fact that the second linear fracture will stop at, and not cross, the pre-existing fracture line.

Where a fracture line extends across the base of the skull then the alignment of that fracture indicates the direction of application of force. So that a basal skull fracture from ear to ear indicates a force applied in the coronal plane, while a fracture from front to back indicates a force applied in the sagittal plane. When severe these basal fractures may bisect the cranium resulting in a 'hinge fracture'. Fractures of the base of the skull predispose to infection and cause the leakage of cerebro-spinal fluid (CSF) through the ear and nose. In one third of patients with leakage of CSF there is associated trapping of air in the cranial cavity.

A ring fracture of the skull base is a circular fissured fracture more or less centred on the junction between the spinal column and the skull base. It is an indicator of the application of very severe force, with the spine being driven into the skull, as in a fall from a height onto the feet or buttocks or alternatively the head being driven down onto the spine following an impact on the vertex of the skull. An impact beneath the occiput or beneath the chin can also produce a ring fracture. Basal skull ring fractures were produced by blows to the occiput with an iron bar in executions by the Khmer Rouge government in Cambodia during the mass killings of the 'killing fields' in the late 1970s.

A localised depressed fracture, in which the skull is displaced inwards, results from a blow from a heavy weapon with a small striking surface, such as a hammer, or alternatively a fall upon a point such as the corner of a stone. The breaking of the bone into two or more fragments, comminution, is a common complication of depressed fractures and typically has a stellate pattern. A comminuted fracture may be produced also in a vehicular collision, a fall from a height, or a blow from a heavy weapon. Depressed skull fractures may tear the underlying dura and the associated blood vessels resulting in intracranial haemorrhage, which in survivors carries an increased risk of post-traumatic epilepsy. A pond fracture, which is similar to a dent in a ping-pong ball, is a type of depressed fracture particular to children as a consequence of the elasticity of the child's skull.

Fractures may occur also at a distance from the point of impact and not as a direct extension of the fracture lines. These distant fractures typically involve the thin bones of the roofs of the nose and the eye sockets, the orbital plates, following an impact to the back of the head, which is usually

the result of a fall. Since these fractures occur on the opposite side of the head to the point of impact they are known by the French term *contre-coup* fractures (literally: opposite the blow fractures).

Sharp force and gunshot injuries to the skull have their own characteristics. A low-velocity gunshot wound to the cranial vault produces a skull defect which is bevelled internally so that the hole on the inner table is larger than the hole on the outer table of the skull. Conversely, the exit wound shows external bevelling with the hole on the inner table smaller than the hole on the outer table. A bullet striking at a very low angle, almost parallel with the skull surface, may barely penetrate, leaving a combined entry-exit skull defect, inwardly bevelled in the entry half and externally bevelled in the exit half, a so-called 'keyhole' fracture. In high-velocity gunshot wounds the creation of a temporary cavity within the skull vault produces an egg-shell shattering of the skull and facial bones, a devastating exit wound, and multiple scalp and facial lacerations from protruding bone. A stab wound to the skull will retain the cross sectional shape of the weapon. Chop wounds to the skull from a weapon such as an axe may raise a plate of bone, producing an elevated fracture, if the impact is tangential, but otherwise produce large cuts in the bone with extending fracture lines.

Intracranial haemorrhage

Intracranial haemorrhage, bleeding in and around the brain, is a common consequence of a head injury. If the haemorrhage forms a distinct mass it is referred to as a haematoma. The haemorrhages are categorised by their anatomical location so that an extradural (epidural) haematoma lies between the inner surface of the skull and the thick membrane, the dura mater (or simply *dura* for short), which normally lies in close apposition. A sub-dural haematoma lies beneath the dura and separated from the brain by a delicate thin transparent membrane, the arachnoid membrane. Sub-arachnoid haemorrhage lies beneath the arachnoid membrane over the surface of the brain.

An extra-dural haematoma is associated with a skull fracture in 90% of cases since it typically results from laceration of meningeal blood vessels which lie in grooves on the inner table of the skull, normally well protected from trauma unless there is a fracture. The most commonly damaged vessel is the middle meningeal artery which lies on the inner surface of the thinnest part of the skull vault at the temple, the squamous temporal bone. An extra-dural haematoma may occur without an associated skull fracture in children. The haematoma is localised and biconvex because it must forcibly strip the dura off the inner table of the skull in order to extend itself. In the great majority of cases the bleeding is arterial in origin but in a minority is venous, arising from the sagittal or lateral venous sinuses, and the haematoma accumulates more slowly.

The classical clinical picture of extra-dural haematoma is of a head impact with initial loss of consciousness followed by a lucid interval lasting hours to days, which is overtaken by a coma due to the pressure effects of the accumulating haematoma. The lucid interval is the state of consciousness between the two episodes of unconsciousness. Other causes of

a lucid interval following head injury include a subdural haematoma, diffuse brain swelling, fat embolism and infection. In practice an extradural haematoma is seen most commonly not in isolation but in association with severe head injuries with skull fracture, and there is stupor progressing to deep coma. If the pressure effects of an extradural haematoma are relieved the prognosis is good and there are no long term sequelae, but an untreated haematoma of 75-100 ml is fatal.

Subdural haemorrhage results from laceration of communicating veins which cross the subdural space between the dura and the brain. These bridging veins pass between the venous sinuses, which lie within the dura, and the cortical veins which lie on the surface of the brain. Communicating veins are most numerous over the vertex where they drain into the superior sagittal sinus, so that the para-sagittal area is a common site for sub-dural haemorrhage. This haemorrhage from the bridging veins then spreads freely within the subdural space over the surface of the brain. Lacerations of the communicating veins result from over-stretching as the inertia of the brain causes it to move at a different rate from the skull when acceleration/deceleration forces are applied. This type of injury may arise without direct impact to the head, although head impact is usual. The mechanism of haemorrhage does not require a skull fracture, by contrast with extradural haematoma, although a skull fracture is often present in association with sub-dural haemorrhage because both are common results of head trauma. Sometimes there is no clear history of trauma, suggesting that these haemorrhages can result from relatively minor impacts, particularly in the elderly in whom the bridging veins straddle a widened space as a result of cerebral atrophy. Alcoholics are particularly vulnerable to subdural haemorrhage as a consequence of repeated falls and altercations, cerebral atrophy and disorders of blood clotting associated with chronic liver disease. Individuals with bleeding disorders and those on anticoagulant therapy are susceptible to sub-dural haemorrhages also.

An acute sub-dural haematoma consists of red clotted blood but is absorbed and changes colour over time with yellowing from haemosiderin deposition. Some haematomas become chronic and are surrounded by a membrane of developing scar tissue, granulation tissue, from which repeated bleeds can arise. An acute sub-dural haematoma may possibly become life-threatening, as a result of pressure effects, when its volume reaches 50ml, and definitely life threatening when its volume reaches 100ml.

Haemorrhage into the subarachnoid space may originate from cerebral contusions and lacerations, or from fractures of the skull base which can tear the large blood vessels of the brain, the cerebral arteries and cerebral veins, which lie there. Occasionally intense sub-arachnoid haemorrhage at the base of the brain may be the only intra-cranial evidence of trauma, and is categorised as 'traumatic basal sub-arachnoid haemorrhage'. This is a specific pathological entity which is invariably fatal and therefore described in forensic practice but not in clinical practice. The victim is typically drunk with associated poor co-ordination and lack of control of head movements. There is a head impact, most commonly a punch, sometimes a kick, and occasionally a fall following a punch or

push, which results in immediate unconsciousness and death, typically within minutes but sometimes within hours, without any recovery of consciousness. The fatal outcome is commonly a surprise to the perpetrator and eyewitnesses alike because the force of any blows, although heavy, were not unusually so. The point of impact to the head may be anywhere on its circumference at approximately the level of the base of the skull, that is to say the level of the ears, or sometimes on the lower jaw. Occasionally there is an eyewitness account of the blow but no evident skin injury from it.

The dramatic autopsy finding is very prominent sub-arachnoid haemorrhage at the base of the brain. This haemorrhage originates from over-stretching lacerations of the cerebral arteries produced by the jerking rotation from the head impact. Often the precise site of bleeding cannot be identified but common origins are the posterior inferior cerebellar artery (PICA), the vertebral arteries just after they have passed through the dura, and the vertebral arteries within the neck where they course within bony foraminae in the lateral processes of the upper cervical vertebrae. In occasional cases there is impact to the neck and fracture of a cervical transverse process.

The diagnosis of traumatic basal sub-arachnoid haemorrhage may be suspected from the history and confirmed at autopsy prior to opening the head by injecting the vertebral arteries with a suitable radio-opaque liquid, and X-raying the skull to disclose leakage into the sub-arachnoid space. Observing the under-surface of the brain in situ and injecting water into the vertebral arteries may permit identification of the bleeding site. If the bleeding originates from the extra-cranial vertebral artery then the cervical vertebrae are removed, dissected, X-rayed individually for evidence of a fracture of a transverse process, particularly of C1, the arteries dissected out of the bony foraminae and examined microscopically for evidence of laceration. Since the principal differential diagnosis is with sub-arachnoid haemorrhage from a developmental aneurysm, a berry (saccular) aneurysm, a careful search of the intracranial cerebral arteries for remnants of such an aneurysm must be undertaken.

Cerebral injuries

Injuries to the brain may be focal, occurring just deep to the area of head impact, or diffuse as a consequence of acceleration/deceleration forces applied linearly or rotationally. Focal lesions of the brain comprise bruises, conventionally referred to as contusions when occurring on the brain or any other internal organ, and also lacerations. Contusions may occur in the area of impact, in association with fracture lines and in a stereotypical distribution resulting from impact with bony irregularities of the skull present in the anterior and middle cranial fossae. The areas of brain most frequently involved are the frontal poles and the contiguous under-surfaces of the frontal lobes, the temporal poles and the contiguous lateral and inferior surfaces of the temporal lobes, and the occipital poles, specifically the cortex adjacent the Sylvian fissures.

When trauma deaths are near-instantaneous, the brain may show few if any contusions, even in the presence of severe skull fractures, because the instantaneous cessation of the circulation limits their development. In survivors, healed contusions leave yellow or orange indentations on the crests of the gyral folds of the brain ('plaques jaunes'). Old ischaemic damage is distinguishable from old trauma because it is most severe within the depths of the sulci rather than on the crests of the gyri. The colouration is due to haemoglobin derived pigments, particularly haemosiderin. Lacerations reflect more severe trauma and are commonly associated with skull fractures. An area of confluent contusions and lacerations with associated subdural haemorrhage is referred to as a 'burst lobe'. In severe head injuries with hyper-extension, the head being thrown back in a whiplash fashion, as occurs in high-speed vehicular collisions, instantaneous death may result from a laceration of the brain stem, a ponto-medullary rent.

When the pattern of contusions and lacerations to the surface of the brain is related to the point of impact, as indicated by the scalp trauma, and to a lesser extent the skull fracture pattern, it may be possible to determine whether the impact was the result of a fall or a blow. When a blow is struck to the head, particularly if the head is fixed, as with a person lying on the ground, the damage to the brain is more severe in the area beneath the point of impact (the coup injury) than on the side of the brain opposite the impact (the contre-coup injury). The relative severity of the coup and contre-coup injuries is reversed when there is a fall and the moving head is arrested by impact with an unyielding object. The classical example of the latter is a person falling backwards when the resultant contusions to the occipital lobes of the brain, the coup injury, are less pronounced than the injuries to the frontal lobes, the contre-coup injuries. The distinction between coup and contra-coup injuries is not so obvious in a fall onto the forehead and the distinction may be more difficult with impacts to the side of the head. With some side impacts the contre-coup contusions are not on the opposite side of the brain, but rather on the opposite side of that cerebral hemisphere as a consequence of impact with the falx, the semi-lunar sagittal extension of the dura which passes downwards from the skull vertex and separates the uppermost parts of the right and left cerebral hemispheres from each other.

Haemorrhages within the substance of the brain may be related to surface contusions or to the phenomenon of traumatic axonal injury (TAI), which is axonal damage directly attributable to trauma and usually the result of acceleration/deceleration effects. There is a wide spectrum of severity, with diffuse TAI, originally termed diffuse axonal injury (DAI), being the most severe. Individuals with diffuse TAI are typically unconscious from the time of impact and remain unconscious until death. With lesser degrees of TAI there may be recovery of consciousness with or without persisting disability. Diffuse TAI is a microscopic diagnosis but its presence can be inferred from a constellation of macroscopic appearances including small haemorrhages in the corpus callosum, the intraventricular septum with associated intra-ventricular haemorrhage, the dorso-lateral quadrants of the rostral brain stem, the superior cerebellar peduncles, and the para-sagittal white matter in the superior part of the

cerebral hemispheres. Recognisable microscopic changes develop in TAI if there is survival for more than about 2 to 4 hours. Focal accumulation of beta-amyloid precursor protein (APP), identifiable by immuno-histochemical staining, is the earliest finding. With survival for 12 to 24 hours, thickening of the axons, axonal varicosities, are seen on silver stains. After survival for one day to two months, axonal swellings or 'retraction balls' can be identified without the use of special staining techniques.

In some individuals who die within minutes of a head injury the only autopsy findings within the brain are pin-point haemorrhages in the white matter of the frontal and temporal lobes, the thalamus and the brain stem, attributable to diffuse vascular damage. A similar appearance is seen in cerebral fat embolism which commonly occurs in individuals with multiple long-bone fractures and pulmonary fat embolism manifesting as dyspnoea, hypoxia and confusion two to three days after injury.

Secondary effects

The secondary effects of head injury include brain swelling which occurs commonly and is a result of congestion of the brain, damage to blood vessels with resultant vasogenic oedema, and an increase in the water content of the neurones, that is cytotoxic cerebral oedema. The swelling of the brain tissue may be localised around contusions or diffusely involve both cerebral hemispheres. Children and adolescents in particular are susceptible to diffuse brain swelling after relatively minor head injury. Swelling of the brain causes distortion of the surface convolutions with flattening of the gyri and obliteration of the sulci.

Since the cranial cavity is a rigid box-like compartment partly subdivided by partitions of the dura (the falx cerebri and the tentorium cerebelli), differences in pressure between compartments cause herniation of brain tissue from one compartment into another. The three common sites of internal herniation of the brain are subfalcine herniation of the medial temporal lobe (the uncus or parahippocampal gyrus), and trans-foraminal herniation of the cerebellar tonsils often called 'coning'. Internal herniation also stretches vulnerable blood vessels and nerves with resulting haemorrhage in the midbrain and upper pons (Duret haemorrhages), infarction in the area of supply of the posterior cerebral artery, compression of the oculomotor nerves, and compression of the cerebral peduncles.

Widespread hypoxic-ischaemic brain damage is common also in cases of head trauma as a consequence of generalised hypoxia, hypotension, and raised intracranial pressure. Ischaemic changes may be found in the watershed areas between major arterial territories of the cerebral cortex, and in the basal ganglia, hippocampus and brainstem.

CHAPTER 6

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 dysfunction, 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 waking. A vegetative state can be regarded as permanent, three months after 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 sub cortical 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.

CHAPTER 7

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 suffusions. 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 chlorate, 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 punctate 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 septicæmia, 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-butanediamine) and cadaverine (1,5-pentanediamine) respectively. These ptomaines are detectable by the cadaver dogs used to locate clandestine graves. Deamination of L-phenylalanine yields ammonia, and phenylpyruvic acid which forms a green complex with ferric iron. 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 discolours 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 mummified 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.

CHAPTER 8

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 subnormal 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 super-sensitive 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 sub-conjunctival 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-dependant 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.

The pattern of corpse colonisation by successive waves of insects provides a source of further 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 larvae 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.

CHAPTER 9

INJURY AND DEATH IN WATER

The investigation of presumed drownings is challenging because the mechanism of death in drowning is neither simple nor uniform, and the specific circumstances of a drowning introduce more variables. Furthermore, not every corpse recovered from water is a victim of drowning.

Pathophysiology

Immersion in water up to the neck increases the work of breathing against the hydrostatic pressure of the water on the chest and abdomen, but this causes no difficulty in a healthy individual. By contrast, sudden immersion in cold water causes a sudden fall in skin temperature and initiates cardio-respiratory reflexes known as the 'cold-shock' response. The respiratory part of the response is an initial gasp followed by uncontrollable hyperventilation and a sensation of breathlessness which makes swimming difficult, and is likely a major reason why victims fail to swim effectively in cold water. The maximum breath holding time is also reduced to less than 20 seconds, increasing the risk of aspirating water. The cardiovascular side of the response is an increase in heart rate and cardiac output which may cause a cardiac arrhythmia in persons with heart disease. The 'cold-shock' response explains why drownings can occur even amongst good swimmers at short distances from safe refuge. Immersion of the face in cold water initiates a diving response characterised by apnoea, peripheral vasoconstriction and bradycardia. The competing effects of the 'cold-shock' and the diving response reflexes cause a variety of cardiac arrhythmias.

Prolonged immersion in cold water causes hypothermia, defined as a deep body temperature less than 35°C. For an average adult wearing outdoor clothing the body temperature reaches 35°C after about one hour in water at 5°C, after two hours at 10°C and after three to six hours at 15°C. When the core temperature falls below 34°C consciousness becomes impaired and aspiration of water is likely. At temperatures less than 28°C ventricular fibrillation and cardiac arrest may occur, and at 24-26°C there is asystole.

Drowning was originally defined as suffocation due to the mechanical obstruction of the airways by any liquid, and drowning is often classified with the asphyxial deaths. However, as well as mechanical obstruction of the airways there are also fluid and electrolyte shifts between the aspirated liquid and the blood, but the relative importance of these different factors in causing death is unclear.

Fresh water is hypotonic and hyponatraemic relative to blood with the result that the inhalation of fresh water leads to movement of water from the alveoli into the blood and movement of sodium from the blood into the alveoli. The result is haemodilution, hypervolaemia, hyponatraemia, and haemolysis with associated hyperkalaemia, leading to fatal ventricular fibrillation. By contrast seawater, which is very hypertonic relative to blood, results in water movement from blood into the alveoli and movement of sodium, chloride and

magnesium from the alveoli into the blood. Consequently there is haemoconcentration, hypovolaemia and hypernatraemia. Both freshwater and saltwater damage alveoli, destroy surfactant and induce pulmonary oedema. There is decreased lung compliance and ventilation-perfusion mismatch so that blood flows through under-ventilated portions of the lung. The result is hypoxia with secondary metabolic acidosis. It is these general effects of aspiration of water, rather than fluid and electrolyte shifts, which appear to dominate the pathophysiology of human drowning.

Bodies recovered from water

The investigation of a death when a body is recovered from water requires answers to a series of questions. First, did death occur prior to or after entry into the water? That is to say, was the victim alive or dead at the time of entry into the water? Second, is the cause of death drowning? If not drowning, then what is the cause of death? Third, why did the victim enter the water? Fourth, why was the victim unable to survive in the water? Answering these questions requires correlation of information about the circumstances preceding the death, including the past medical history, the circumstances of recovery of the body, and the autopsy and laboratory analyses. Unfortunately there are no autopsy findings which are pathognomonic of drowning. Therefore obtaining proof that the victim was alive on entering the water and excluding natural, traumatic and toxicological causes of death are critical. Some autopsy findings are characteristic of drowning, but the diagnosis is largely one of exclusion. The overall investigation of the drowning episode must assess both environmental and human factors, such as inexperience, poor judgement, and intoxication.

The majority of drowning victims are young adults and children who die accidentally. It is possible to drown in quite shallow water so long as it is sufficiently deep to cover the nose and mouth. Toddlers may drown accidentally in a garden pond, pool, the bath and even in a bucket of water. Among adults, males predominate and there is a strong association with alcohol consumption. Persons diving into shallow water and striking the forehead against the bottom sustain hyperextension injuries of the neck with loss of consciousness or death. Homicidal drowning is uncommon because it is physically difficult to achieve. Disposal of a corpse in water may occur when the victim has already been killed by another means. Suicide by drowning is common in some cultures and in these cases clothing may be left in a neat pile close to the water, weights may be tied to the body or the pockets filled with stones. A person intent on suicide may tie together the hands or the feet and an examination of the ligatures should aim to establish whether they could have been tied by the deceased. Persons jumping from a height into water can suffer severe injuries from impact with the water and sustain fractures of the ribs, sternum and thoracic spine as well as lacerations of the heart and lungs.

Drowning in a domestic bath may occur following an epileptic seizure, cardiac arrhythmia, intoxication from alcohol or drugs or a minor head injury from a slip and a fall. Suicidal drowning in the bath is uncommon, and the victim is often

found face down and partly or fully clothed. Homicidal drowning in the bath is rare because it is difficult to achieve. Lack of accurate information on the position of the body and level of the water often makes the investigation of bathtub deaths more difficult. Intra-venous drug abusers may place an addict who collapses into the bath in the belief that it will assist recovery.

Skin diving is essentially an extension of swimming using fins and a mask, and presents similar hazards. Amongst snorkelers, unconsciousness due to breath-holding following hyperventilation, sometimes loosely termed 'shallow water black-out', is a common cause of drowning. Recreational SCUBA divers die most commonly from drowning and barotrauma. SCUBA is an acronym for Self-Contained Underwater Breathing Apparatus, which contains compressed breathable gasses allowing the diver to reach greater depths not usually achieved by skin divers. If a SCUBA diver makes an uncontrolled ascent without exhaling, the volume of lung gas expands as the ambient pressure falls, and if the diver does not exhale, air is forced from the alveoli into the pulmonary circulation, to the heart and thence into the cerebral circulation. This phenomenon of pulmonary barotrauma, with acute pulmonary emphysema and pneumothorax, and cerebral air embolism is also known as extra-alveolar air syndrome. The characteristic history is that the diver came to the surface rapidly, cried out and then lost consciousness within minutes.

Immersion artefacts

All bodies recovered from water show a spectrum of post mortem artefacts resulting from immersion. These changes occur in any corpse immersed in water, irrespective of the cause of death. Although they are typically the most striking features in a body recovered from water, they do not contribute to proof of death by drowning. The most common immersion artefact is maceration of the skin which becomes blanched, swollen and wrinkled, as a result of increased hydration of the epidermis. Maceration is first apparent in the skin of the finger-pads and then appears on the palms, backs of the fingers and back of the hand in that order. The hydration changes are similar to those seen when the hands of the living are immersed in water for a long time, hence the alternative name of 'washer-woman's hands'. In warm water the early post-mortem changes can be seen within an hour but the process may be delayed for several days in very cold water. With the development of putrefaction, the epidermis, including the nails, peels off like a glove or a stocking. Fingerprints may be easily prepared from the glove of epidermis. The remaining exposed dermis will yield a reverse fingerprint. Occasionally chromogenic bacteria (*Bacillus prodigiosus* and *Bacillus violaceum*) invade the dermis after one to two weeks immersion, and produce patterns of pigmentation giving the impression of tattoos. Anserina cutis or goose-skin is another common immersion artefact seen in freshly recovered bodies. This appearance is a roughening or pimping of the skin as a result of rigor mortis of the erector pilae muscles associated with the fine hairs of the skin. It is of no diagnostic significance and no importance should be attached to it.

A body in water will usually sink. Some bodies float because the specific gravity of a corpse is very close to that of water and small variations, such as from air trapped in clothing, have a considerable effect on buoyancy. Having once sunk to the bottom, the body will remain there until putrefactive gas formation decreases the specific gravity of the body sufficiently to create the buoyancy to allow it to rise to the surface and float. Heavy clothing and weights attached to the body may delay but will not usually prevent the body from rising. The principal determinant of the rate of putrefaction is the temperature of the water, so that in deep, very cold water the body may never resurface because there is no appreciable putrefaction. In the water a body normally floats face down with the head, arms and legs lower than the torso. Consequently livor mortis (hypostasis) is most prominent in the head, neck and anterior chest. Putrefactive changes, when they develop, are most prominent within these areas of lividity. Bodies which sink to the bottom of very cold, deep water, such as the North American Great Lakes or North Sea, may float on the bottom face-up because of the change in buoyancy which occurs when water replaces all air in the lungs and the cold inhibits putrefactive gas formation. Bodies immersed in water for some months may develop adipocere, a soap-like transformation of subcutaneous fat (see changes after death).

Having sunk to the bottom, a body, drifting along the water bed or being washed ashore, will sustain a pattern of post mortem injuries reflecting its usual face-down floating position. Post mortem abrasions are typically found over the forehead, the prominent points of the face, the anterior trunk, the backs of the hands and the fronts of the lower legs, and also the dorsum of the feet if there is no footwear. A wide range of other injuries may be produced by the battering of the body against rocks or by passing watercraft. The body may be attacked by sharks, small fish and other fauna. The soft parts of the face are particularly vulnerable to fish and crustaceans. Post mortem injuries may be inadvertently inflicted during the recovery of the body when using grappling irons, hooks and ropes. Post mortem injuries in areas of dependant lividity, such as the face, ooze blood mimicking antemortem wounds. The movement of the body in water also disturbs the clothing so that loose garments, such as a jacket, may be turned inside out and partly removed, giving the impression of partial undressing.

Pathology of drowning

Some autopsy findings are suggestive of drowning but are non-specific and are not universally present. None are pathognomonic of drowning. The concurrent observation of foam around the nose and mouth, frothy fluid in the airways, and 'emphysema aquosum' with overlap of the medial edges of the two lungs is very strongly supportive of a diagnosis of drowning, but may be found in only 10% or so of cases. The fine white froth or foam may be seen exuding from the mouth and nostrils, and is found in the trachea and main bronchi. Sometimes it is tinged with blood, imparting a pink colour. The foam is a mixture of air, mucus, protein rich pulmonary oedema fluid, and to a lesser extent inhaled water, all whipped up by respiratory efforts. Thus it is a vital phenomenon.

However, it is not specific to drowning and is found in other instances of severe pulmonary oedema such as acute heroin overdose, congestive cardiac failure and neurogenic pulmonary oedema. In a body in water the foam persists until it is destroyed by putrefaction, which produces in turn pseudo-foam of reddish-brown malodorous fluid containing bubbles of putrefactive gas, a finding of no diagnostic significance.

Emphysema aquosum is the second autopsy finding suggestive, but not pathognomonic, of drowning. Inhalation of the drowning medium as well as reactive pulmonary oedema and the struggling breaths of the victim results in over-inflation of the lungs and air trapping in the alveoli by fluid in the bronchial tree. The lungs appear ballooned, voluminous and bulky. As a result, overlap of the medial edges of the two lungs is seen on removal of the breastplate at autopsy. The lungs feel doughy, pit on pressure and on sectioning there is a flow of watery fluid. The pleural surface has a marbled appearance with grey-blue to dark red areas interspersed with pink and yellow-grey zones of more aerated tissue. Although subpleural petechiae are rare, larger ecchymoses are sometimes seen, most often on the interlobar surfaces of the lower lobes. These haemorrhages are a consequence of rupture of the alveolar walls, which is also the cause of blood tingeing of any foam in the airways. All of these findings in the lungs, although characteristic of drowning may be seen also in cases of severe acute pulmonary oedema from any cause. The characteristic microscopic appearance is of over-distension of the alveoli, thinning of the alveolar septae and compression with narrowing of the capillary network.

Contrary to expectations lung weights in freshwater drowning are not statistically different from lung weights in saltwater drowning. In a minority of drowning deaths, perhaps amounting to 10-15%, the lungs are not heavy and waterlogged but rather are 'dry'. In the past these cases were often characterised as 'dry drowning', a confusing and misleading terminology which is not recommended. In the immediate post mortem period following drowning there is transudation of fluid from the lungs into the pleural cavity so that there is a time-dependant decrease in the lung weight and a reciprocal increase in the pleural effusion volume.

Material such as sand, silt, seashells and weed may be found in the airways, lungs, stomach and duodenum of bodies recovered from water. Some foreign material may enter the pharynx, trachea and larger airways during submersion postmortem, and small quantities may enter the oesophagus and stomach. However, the finding of abundant foreign material generally distributed within the alveoli provides strong evidence of immersion during life, as long as the body is recovered early, that is within twenty-four hours. Finding large quantities of sand in the upper airways raises the possibility of inhalation of the thick suspension of sand in seawater produced by heavy surf; death is very rapid in these circumstances. The presence of large quantities of water and contaminating debris within the stomach strongly suggests immersion during life because it indicates the swallowing of water in an attempt to breath-hold for as long as possible. Rarely, weeds, branches and other material may be found

fixed in the hand of the victim by cadaveric spasm (instantaneous rigor). This observation provides good evidence that the victim was alive and conscious at the time of submersion.

Victims struggling violently to survive in the water may bruise or rupture muscles, particularly those of the shoulder girdle, neck and chest. These muscle haemorrhages, which are found in a minority of drowning deaths, are strong indicators that the victim was alive in the water. Middle ear and mastoid air cell haemorrhages are occasionally seen in bodies recovered from water. These haemorrhages produce a blue-purple discolouration of the bone of the roof of the mastoid air cells and middle ear, visible after stripping off the dura mater following removal of the brain. The pathogenesis is unresolved and may be the result of barotrauma, the irritant effects of aspiration of fluid into the eustachian tubes, or extreme congestion. Identical haemorrhages are found in cases of head trauma, electrocution and mechanical asphyxiation so that their presence does not constitute evidence of death by drowning.

The absence of a pathognomonic autopsy sign of drowning has led to a search for a diagnostic laboratory test. Awareness of the fluid and electrolyte shifts which may occur in drowning suggested measurement of the specific gravity of blood, plasma chloride and plasma magnesium as possible diagnostic tests. However, these testing methods are no longer considered reliable because unpredictable changes in blood electrolytes always occur after death, post-mortem fluid and electrolyte shifts occur between the drowning medium, the lungs and the heart blood, and the relative role of fluid and electrolyte shifts in the mechanism of human drowning is unclear. Currently the only diagnostic laboratory test for drowning to have gained widespread acceptance is the diatom test. Diatoms or bacillariophyceae are a class of microscopic unicellular algae of which about 15,000 species are known, approximately half living in freshwater and the other half in sea or brackish water. The cell structure of diatoms is unique in that they secrete a hard siliceous outer box-like skeleton called a frustule which is chemically inert and almost indestructible, being resistant to strong acids. The classification of diatoms is based upon the structure of their siliceous valves. During drowning, smaller diatoms present in the drowning medium enter the systemic circulation, having passed through the filter of the lungs, and become lodged in tissues such as the bone marrow, where they can be demonstrated following acid digestion of the tissues. Lung tissue is not used for the tests since it can be readily contaminated post-mortem by diatoms.

The diatom test for drowning relies not only upon the identification of diatoms in the bone marrow, but also upon the identification of the same species of diatoms as found in a sample of water obtained from the place of recovery of the body. Given adequate precautions to prevent contamination, the demonstration of diatoms of the appropriate species in organs such as bone marrow is strong corroborative evidence of death by drowning. This is true for decomposed bodies as well as fresh bodies, provided there is no gross mutilation of the corpse.

CHAPTER 10

INJURY AND DEATH IN FIRE

People are injured or killed by fire as a result of smoke inhalation, burns, heat shock, and blunt force trauma from falls or collapsing structures, or any combination of these factors. Burns and blunt force trauma have been discussed in detail in previous chapters. Here the focus is on smoke inhalation and the investigation of fire-related deaths.

Smoke inhalation

The majority of fire fatalities are the result of smoke inhalation and, in building fires, evidence of smoke inhalation is found in 90% or more of victims. Smoke inhalation is a general term embracing the inhalation of particulate matter and gases produced in the fire by combustion or by pyrolysis (decomposition by heat without ignition). Developed countries have seen an increase in the number of fatal and non-fatal fire casualties, with a significant increase in the proportion overcome by smoke inhalation. Some fire-fighters believe the fire atmosphere has become increasingly smoky in recent times, possibly due to the widespread use of synthetic polymers in construction, furnishings and decoration. An increased quantity and optical density of smoke obscures vision and exacerbates the problems of escape and rescue, and an increase in the toxicity of smoke and fire gases results in the more rapid incapacitation of victims.

Most deaths from smoke inhalation result from hypoxia caused by a combination of carbon monoxide (CO) intoxication, a low inspired-oxygen tension, and ventilation-perfusion mis-matching in the smoke-damaged lungs. Of building fire fatalities, 85% have some evidence of CO inhalation and 50% have evidence of CO poisoning sufficient to cause death. A low inspired-oxygen tension can contribute to hypoxemia because in a burning room the oxygen level falls from a normal 21% to between 10% and 15%. However, air containing 12% oxygen is necessary to maintain flaming combustion and oxygen levels below 10% are unusual in room fires. A fall in oxygen to 8% is necessary, by itself, to cause collapse, and the level must fall to 6% for about 8 minutes to cause death. In general, if there is sufficient oxygen to maintain a fire, then there is sufficient oxygen to maintain life. Nevertheless, a low inspired-oxygen tension can contribute to death by its combined effect with other factors. Ventilation-perfusion mis-matching as a result of airways injury can also contribute to hypoxemia.

There are four pathophysiological mechanisms by which smoke inhalation produces respiratory damage leading to hypoxaemia: (a) thermal injury to the respiratory tract, (b) carbon monoxide poisoning, (c) direct particulate injury, and (d) smoke poisoning. Direct particulate injury and smoke poisoning can be grouped together as 'smoke toxicity'.

In smoke inhalation, thermal injury of the face and upper airways occurs frequently, but is usually limited to the mouth, the nasal passages, the glottis and epiglottis, the pharynx and

the larynx. The heat energy contained in hot dry air is so low and the heat-exchanging efficiency of the respiratory tract is so high that even super-heated air is cooled before it gets below the larynx. However, with air temperatures of 150° C and above, the laryngeal thermal trauma may cause spasm sufficient to produce suffocation, and the development of laryngeal oedema can have the same effect. Breathing very hot air may cause a reflex cardiac death (vagal inhibition of the heart). Moist air with its increased heat capacity is more likely than dry air to produce burns within the lung.

Microscopically burns of the tracheobronchial tree show swelling and superficial coagulation necrosis of the epithelium, elongation and pallisading of the epithelial nuclei, evaginations of the mucosal glands, fragmentation and clumping of erythrocytes in mucosal vessels, oedema of the submucosa and general mucosal hyperaemia. Taken together these findings are strong indicators of the inhalation of hot air during life. With survival for up to 24 hours there is increased oedema, sloughing of the mucosa, and focal collapse and haemorrhagic consolidation of the lung. After 24 hours, infection supervenes with broncho-pneumonia and necrotising tracheobronchitis.

Carbon monoxide poisoning

Carbon monoxide (CO) poisoning (see non-medicinal poisons) is a pathophysiologically distinct cause of inhalation injury producing its effects by tissue hypoxia. CO is a colourless, odourless gas formed by the incomplete combustion of carbon compounds and normally found in the atmosphere in levels well below 1 part per million (ppm). CO at levels of 1000 ppm can kill in about half an hour, and at 5000 ppm is rapidly fatal. The affinity of haemoglobin for CO is 200 times greater than for oxygen so that the carboxy-haemoglobin (COHb) concentration of blood is high even when the CO concentration in the inhaled gas is low. As well as diminishing the oxygen-carrying capacity of the blood, COHb also alters the dissociation characteristics of the remaining oxyhaemoglobin, making less oxygen available to the tissues.

The toxicity of CO depends upon (a) the rate of inhalation of the gas i.e. the concentration of gas in the inspired air and time of exposure, (b) any physical activity, which influences oxygen requirements, and (c) individual variations in susceptibility.

COHb is estimated by spectroscopic methods making use of the fact that oxyhaemoglobin and COHb have different absorption spectra. The result is expressed as the percentage saturation and is the ratio of COHb to total haemoglobin x 100. The effects of COHb at different percentage saturation levels are generally taken as follows: 0-10% no immediate ill effects; 10-20% may produce dizziness and shortness of breath on exertion; 20-30% dizziness, headache, nausea and fatigue; 30-40% impaired judgement, unconsciousness may occur; 40-60% unconsciousness and death likely.

COHb levels up to 10%, or more, may be found in cigarette smokers or from atmospheric pollution. Motor vehicle engines

are responsible for about 70% of carbon monoxide liberated into the atmosphere. Occasionally COHb levels up to 15% are found in heavy smokers, since tobacco smoke may contain up to 5% by volume of CO. COHb levels above 30% produce confusion and may explain failure to escape a fire. The normally accepted fatal COHb level is 50% but children, the very old and persons with cardio-respiratory disease may succumb to levels as low as 30%.

Carbon monoxide and other noxious gases which are inhaled may have an additive effect and account for some deaths with COHb levels below 50%. With the exception of cyanide, laboratory tests for the presence of these noxious gases is rarely, if ever, performed on the bodies of fire victims. CO and cyanide produced in fires have an additive effect. Cyanide is a powerful and rapidly active poison which is commonly produced in fires as a result of the burning of materials such as wool, silk, horsehair, polyurethane and polyacrylonitrile. Normal blood cyanide levels are less than 8 $\mu\text{mol/L}$, non-fatal toxic effects begin at about 50 $\mu\text{mol/L}$, and the fatal threshold is above 100 $\mu\text{mol/L}$. Alcohol (ethanol), a central nervous system depressant, might be expected to have an additive or synergistic effect with CO but in practice this has not been demonstrated.

85% of building fire victims show evidence of CO inhalation and approximately 50% have a COHb level sufficiently high to account for death. Of those with evidence of the inhalation of CO there is associated evidence of soot inhalation in approximately 95%. The presence of an elevated COHb level, and/or evidence of soot inhalation, provides proof that the victim was alive, but not necessarily conscious, at the time of the fire. However, a COHb level below 10% is not proof that a victim was dead prior to the start of the fire. Possible explanations for an absence of COHb include little or no CO production in the fire due to abundant oxygen allowing complete combustion, e.g. a forest fire, or a rapid death in a flash fire.

Smoke poisoning and toxicity

Smoke poisoning describes the effects of the various noxious gases other than carbon monoxide which are produced by the thermal degradation of both natural and synthetic materials. Filter masks do not protect against the inhalation of these fumes; for this purpose the mask must have a self-contained air supply. There are hundreds of such products of combustion. Whereas modern synthetic materials, such as PVC, may have increased this problem, natural materials, including wood, wool, and silk, also generate similar gases.

These noxious gases rarely reach lethal levels by themselves, but they may cause incapacitation or death in combination with other factors. Some gases have an additive or a synergistic interaction so that non-fatal levels of each in combination may kill. The concentration of the noxious gas within the smoke is of importance. Hydrogen cyanide, produced from wool, wood or polyurethane, is rapidly fatal at 3,000 parts per million (ppm). Nitrogen dioxide from acrylonitrile is rapidly fatal at 2,000 ppm. Hydrogen chloride

from PVC is rapidly fatal at 2,000 ppm. Hydrogen sulphide from rubber or wool has a toxic effect comparable to hydrogen cyanide and is rapidly fatal at 1,000 ppm. Acrolein, an aldehyde product of combustion from wood and paper, produces pulmonary oedema after a few seconds exposure at 10 ppm.

The pathological effects of these noxious gases are difficult to separate from direct particulate injury, and the two are together described as smoke toxicity. Soot and particulate matter produce direct injury to the respiratory mucosa because they are super-heated and contain some of the toxic agents responsible for smoke poisoning. Injury to the deeper parts of the respiratory tract is likely to be caused by smoke poisoning rather than by thermal injury from hot air. The depth to which soot particles penetrate within the respiratory tract depends on their effective aerodynamic diameter because of the resultant filtration effect. Large particles (greater than 10 μm) tend to be deposited in the nares, while the optimal diameters for deposition in the tracheobronchial tree and alveolar spaces are 5-7 μm and 1-3 μm respectively. The filtration effect is almost independent of the air flow so that rapid deep breathing increases the amount of material deposited but not the site of deposition.

In building fires evidence of soot inhalation is very common (approximately 90% of cases) but it is uncommon to find any soot deposition beyond the primary bronchioles. Deposition of soot on the tongue, in the nares, the oropharynx and nasopharynx, cannot be taken to imply life during the fire since this contamination could occur in an open-mouthed corpse. Deposition of soot below the level of the larynx indicates that the victim was breathing and thus alive at the time of the fire. This soot which coats the mucosa of the tracheobronchial tree can be identified through a window cut in the trachea prior to removal of the neck and thoracic structures at autopsy. The presence of soot in the oesophagus and stomach implies that it has been swallowed and also indicates life at the time of the fire.

Bodies from fire

The majority of fire related deaths are accidental and there is typically abundant collateral evidence, from police and fire brigade investigations, to exclude suicide or homicide. The young and the elderly comprise the overwhelming majority of victims of accidental fires. The deaths are usually the result of carelessness, in allowing clothing to brush against fires, in manipulating matches or other lighted objects such as cigarettes, maintaining faulty electrical and heating appliances, as well as being unable to effectively combat or escape a fire. Where clothing has caught alight, the pattern of burns may corroborate the circumstances, e.g. sparing of the lower legs and inner thighs with a burning nightdress. Occasionally a natural disease such as epilepsy or ischaemic heart disease, may cause the victim to collapse onto a heater, starting a fire; the same natural disease may explain failure to escape the fire. Alcoholics and other individuals under the influence of drink or drugs represent another at risk group. Slow, smouldering fires, such as bedclothes ignited by a dropped cigarette, often

result in death by smoke inhalation. Mass accidental fire deaths occur when large buildings, such as hotels, catch fire, or following transportation accidents, particularly aircraft crashes but also vehicular collisions. Deliberately set fires may inadvertently result in the death of the fire-setter.

Suicide by burning is uncommon. The victim typically pours a flammable liquid, such as petrol, over the clothing and then sets light to it, sometimes after swallowing some. These suicides may occur outdoors, in a building, or in a vehicle. The distribution of burns and damage to clothing can indicate whether the person was standing, sitting or laying at the time the fire was set. In India most victims of self-immolation are young Hindu women with dowry problems who douse themselves with kerosene, which is available as domestic fuel. Fire, as the god Agni, plays an important role in Hindu religious rituals.

Homicide by fire is rare and fire is more often used to conceal evidence of a homicide committed by other means. The use of petrol bombs, or arson directed against a private residence, can result in deaths.

Bodies recovered from fires present similar problems of investigation to bodies recovered from water. In both instances the integration of information obtained from the examination of the scene, the examination of the body, and the history of the decedent, is particularly important. Several investigators with different areas of expertise are typically involved. The key questions to be resolved are: (1) Did death occur prior to or after the start the fire? i.e. was the victim alive or dead during the fire? (2) Is the cause of death fire related? If so, is the cause of death smoke inhalation, burns, heat shock, or trauma? If not, what is the cause of death? (3) Why was the victim in the fire? (4) Why was the victim unable to escape the fire?

To resolve these issues the following information must be correlated: (1) circumstances preceding the death (2) evidence of the origin, development and nature of the fire and (3) autopsy and laboratory analyses. A full investigation of the circumstances preceding the death requires the identification of the victim which therefore becomes a priority. If the identity of a charred corpse is suspected, then identity can usually be proved scientifically. Where specific identification (individualisation) is not possible, then circumstantial evidence of identity is usually available. In fire related deaths there is always a fire investigator, usually an officer in the local fire brigade, who can provide valuable information on the origin, development, and nature of the fire.

An elevated blood COHb level or soot in the airways provides proof of life at the time of the fire, but the absence of these findings does not necessarily indicate death prior to the fire. Carbon monoxide poisoning may explain failure to escape the fire as well as the death. Soot in the airways is sufficient to account for death by smoke inhalation in the absence of a fatal COHb level in blood. Natural disease together with carbon monoxide poisoning may explain some deaths with a low COHb level. A careful search for injuries must be made, and

this is greatly facilitated by X-raying any badly burnt body. Although fire-setting to conceal a homicide is uncommon, the possibility needs to be kept in mind. Toxicological studies for alcohol and drugs may be yield results which provide an explanation for an accidental fire, the failure to escape, or a contributory factor in the cause of death. Even in very badly burnt bodies some tissue such as liver or skeletal muscle can be recovered for analysis.

The distinction between burns inflicted during life and burns inflicted on an already dead body can be difficult, if not impossible, at autopsy. Modern written authorities do not agree on the criteria for making the distinction. A broad band, 1-2 cm wide, of inflammatory reddening at the burn margins is a clear indication of an ante-mortem burn but in practice is rarely seen. The ante-mortem nature of some burns may be obscured by the continued burning of the body after death. A thin band, 1-2 mm wide, of congestive reddening at the burn margin is a common post-mortem artefact, attributed to heat-induced tissue contraction, and is not an indicator of ante-mortem burning. These diagnostic difficulties combined with the social pressure to attribute death to the less painful effects of smoke inhalation, results in few fire deaths being attributed to burns.

Fire artefacts

In most circumstances the effects of a fire on a body continue beyond death so that the final state of the body of a person killed in a fire is largely the result of post mortem damage. This fire damage will occur in any corpse exposed to a fire, including that of a person already dead when the fire started. A burnt body may have only minor local superficial burns or be reduced to calcined skeletal fragments without any remaining soft tissue. In general the effect of heat on body tissues is to burn, dehydrate, shrink and fix by protein denaturation. Direct contact with flame results in a greater loss of tissue mass because the organic matter of the body acts as a fuel.

The post mortem burning of skin can produce a thin red margin and skin blisters mimicking the appearance of a burn sustained in life. Exposure to the heat of a smouldering fire leaves the skin leathery, firm and brown whereas high temperatures and contact with flame produces charring. Hair becomes frizzy and brittle, assuming a fox-red, dark brown to black colour at 240°C as a result of melting of the hair keratins. The friction ridge skin of the palms of the hands and the soles of the feet when affected by a second degree post-mortem burn has a whitish discolouration associated with swelling, wrinkling and vesicular detachment, which may extend to glove-like peeling of the entire skin of the palm. The appearances superficially resemble the 'washerwoman's skin' changes found in bodies recovered from water. Tight clothing and contact with a non-flammable object protects the skin. However, burning clothing can act as a wick and the body fat as a fuel to sustain a low-heat localised fire which causes deep destruction of one part of the body leaving the remainder undamaged. This bizarre pattern of post mortem burning was the origin of the myth of spontaneous human combustion.

The heat of a fire causes tissue to shrink as a result of loss of water. The consequence is tightening and splitting of the skin, protrusion of the tongue from the open mouth, and a pugilistic (boxer's) posture to the body. The skin of the face is shrunken, leathery and hard and the mouth usually open with shrunken lips, whilst the eyes are closed and the shrunken eyelids can be opened only with difficulty. Prolonged heat exposure leads to splitting of the skin which superficially resembles a cut or laceration. These heat splits often involve the arms, thighs or abdomen. They are typically linear with sharp edges and extend into the subcutaneous fat, and sometimes muscle, but this exposed tissue is unburned, suggesting that the splits may result from body cooling or body handling. Shrinkage of the tissues of the neck causes protrusion of the tongue, in the same manner that ligature pressure on the neck in a hanging causes protrusion of the tongue. Shrinkage of the peri-anal skin causes the anus to gape and this may be misinterpreted as evidence of an anal assault.

The typical posture of a charred body is a pugilistic or boxer's attitude, with the arms abducted at the shoulder joints and flexed at the elbow joints, and with the legs abducted and flexed at the hip joints and flexed at the knee joints. The cause of this posture is heat shrinkage of muscles which is more marked in the bulkier flexor muscles. The same process produces clenching of the hands and contraction of the feet. The development of a pugilistic posture is most marked when the body lies prone and may be limited by overlying and surrounding objects. Heat causes fixation and shrinkage of the internal organs which become firm, shrunken so-called 'puppet organs'. Red blood cell fragmentation and haemolysis causes an intense red discolouration of the intima of blood vessels similar to that which occurs during normal putrefaction.

Burning of the body causes loss of soft tissue, exposure of the body cavities, amputation of the extremities, and finally, consumption of the internal organs. The skeleton may be exposed but is never completely consumed in a typical building fire. In a car fire the extent of destruction of the body is usually greater because of the higher temperatures produced. Above 700°C there is complete combustion of the organic matter of bone and re-crystallisation of the inorganic matter, a process called 'calcination'. The bones can shrink by up to 10% and they fracture easily in patterns typically unlike those seen following blunt force trauma in life.

Heat fractures of long bones may appear arc-shaped on the surface or occasionally as 'street-and-avenue fractures' composed of jagged lines of fracture at right angles to each other. Heat fractures occur only in charred or calcined bones so that heat fractures of the cranial vault occur when the bone is exposed by the direct effects of flame. An associated artefact is a heat haematoma in the epidural (subdural) space resulting from a shift of bloody fluid from the diploë of the skull and the venous sinuses. It appears brick-red, dry and crumbly but if burnt further becomes a delicate black charred honeycomb. By contrast, a haematoma in the subdural space is an indication of ante-mortem trauma. Heat shrinkage of the dura mater and associated heat splitting causes a mushroom-like extrusion of the brain tissue into the epidural space. Loss

of facial soft tissues exposes the teeth so that the heated tooth enamel fragments and amalgam fillings vaporise, making dental identification more difficult.

Toxic Gases and Vapours Produced from Burning Materials

Gas	Source
Carbon monoxide, Carbon dioxide	All combustibles containing carbon
Nitrogen dioxide	Cellulose, polyurethanes, acrylonitrile
Hydrogen chloride	Chlorinated polymers, e.g. polyvinylchloride
Hydrogen cyanide	Wool, silk, nylons, N-containing plastics, polyurethanes
Aldehydes	Wool, cotton, paper, plasters, wood, nylon, phenol-formaldehyde, polyester resin
Benzene	Petroleum, plastics, polystyrene
Ammonia	Melamine, nylon, urea-formaldehyde
Sulphur dioxide	Rubber, thiokols
Phenol	Phenol-formaldehyde
Acrolein	Wood, paper

CHAPTER 11

INJURY AND DEATH ON THE ROAD

Road traffic incidents are a leading cause of morbidity and mortality worldwide. However, the epidemiological pattern differs between the industrialised and the developing world. Although two-thirds of all motorised vehicles are in North America or Western Europe, about 85% of traffic-related fatalities occur in developing countries. In the industrialised countries the vehicular occupants represent the majority of fatalities, followed by pedestrians and bicyclists. By contrast, in developing countries non-motorised occupants and pedestrians together with motorcyclists and bicyclists account for the majority of those killed and injured. Preventative measures, such as road improvement, vehicular design, drink-driving legislation, helmets for motorcyclists and safety belts and air-bags for car occupants, have reduced casualties in most industrial countries.

Accident investigation

Investigation of vehicular incidents provides important information for criminal and civil court proceedings as well as for the development of preventative measures. The overall purpose of the investigation is to reconstruct the sequence of events leading to a crash through evaluation of the vehicles, the roadway, the environment and human factors. The documentation and evaluation of the first three elements is usually carried out by a police road traffic investigator. The evaluation of human factors includes the interpretation of alcohol and drug levels, as well as patterns of injury.

Pedestrians

In frontal impacts of a vehicle on a pedestrian, five patterns of pedestrian trajectory are recognised. In the 'wrap' pattern the pedestrian is struck by the bumper, injuring the lower leg, and by the front edge of the bonnet (the hood) injuring the upper leg or pelvis, then the body is rotated so that the head, shoulders and chest strike the bonnet, windscreen or pillars. If the car is breaking it slows faster than the victim who comes off the front and slides across the road surface. In the wrap pattern of trajectory the distance between the initial point of impact and the point on the road surface where the victim strikes the ground is known as the throw distance. This distance loosely correlates with the speed of the vehicle provided that the vehicle was breaking hard at or immediately after the impact.

The second pattern, called 'forward projection', occurs with high-fronted vehicles or when a small child is struck by a car, so that following the primary impact the victim is thrown forward onto the road surface with the risk of over-running. The third pattern, known as 'wing-top', occurs when the pedestrian is struck by the front wing, carried over the wing and falls to the ground at the side. The fourth or 'roof-top' pattern occurs at high speeds or if the vehicle accelerates after impact, so that the victim slides up the front of the vehicle and over the roof to fall onto the roadway behind. The fifth and least common is the 'somersault' pattern which occurs in a

high speed impact, when there is sufficient force at the primary impact to somersault the victim into the air, so that there is no secondary impact with the vehicle but instead the victim impacts with the road.

The typical pedestrian struck by a motor vehicle is an upright adult struck by a car, with the primary impact occurring between the vehicle bumper (fender) and the legs. Damage to the bumper, lights and front of the vehicle reflect the point of primary impact. Abrasions and patterned bruise/abrasions imprinting the shape of the component of the vehicle, or clothing crushed between the vehicle and the skin, indicate the point of impact. However, primary impact injuries to the leg may be represented by bruising of the deeper soft tissue without bony fracture or externally apparent injuries, emphasising the importance of dissection of the legs in pedestrian fatalities. If the pedestrian is struck side-on then this may be obvious from the injury to the side of one leg.

The level of impact above the heel, measured with the victim still wearing footwear, can be compared with the ground to bumper height of the vehicle as well as the injury pattern. The height of the bumper varies between vehicles but in European cars averages 16-20 inches (40-50cm) above the ground, corresponding to the level of the upper tibia of a pedestrian. Vehicle breaking lowers the impact point by pitching the car forward. If the pedestrian was walking then the impact point on the weight bearing leg in contact with the ground will be at a higher level than on the raised non-weight bearing leg.

Primary impact fractures of the lower legs in pedestrians result either from angulation, as a consequence of the direct translational force, or from rotation, as a consequence of the body twisting around its vertical axis or from both. An angulation fracture is either transverse or oblique and occurs at the level of impact. It is wedge-shaped with the base of the triangular wedge on the side of impact and the point of the wedge indicating the relative direction of travel of the vehicle. Rotational, twisting forces produce spiral fractures at the weakest point of the bone rather than at the point of impact, so that they may occur at different levels in the adjacent tibia and fibula. They imply that the foot was fixed on the ground at the moment of impact. In a child the primary impact fracture is likely to be in the femur.

Since the primary impact is below the centre of gravity of an adult's body the pedestrian is thrown up onto the front of the vehicle to strike the bonnet (hood), windshield or pillars with resultant secondary impact injuries. Severe head injuries may occur at this time with associated damage to the vehicle and trace evidence from the vehicle present in the wounds. At the same time blood, tissue and hair are deposited on the vehicle. In a pedestrian struck from behind, the backward arching of the body may cause multiple stretch lacerations in the groin and lower abdomen, in appearance similar to the abdominal striae of pregnancy. A pedestrian will be over-run rather than thrown onto the vehicle bonnet if the vehicle is high fronted such as a bus, truck or van, or if the pedestrian is a small adult or child who is struck above their centre of gravity, or if the vehicle is travelling relatively slowly (less than 20-25 kph) so that the victim is pushed over onto the roadway.

In the typical situation with a pedestrian receiving primary impact injuries from the bumper and secondary impact injuries from being thrown onto the vehicle, the victim then strikes the ground to sustain tertiary impact injuries, commonly to the head and torso. Abrasions associated with these injuries commonly have traces of the roadway surface embedded in them. In broad grazed abrasions, the multiple lines of accentuation indicate the direction of movement. One clearly delineated end to such an abrasion indicates the start, the initial contact with the roadway, and the other poorly delineated feathery end indicates the finish of the moving contact. Severe tertiary impact injuries may show minimal haemorrhage when contrasted with the primary and secondary impact injuries if death, and the cessation of the circulation, was near instantaneous following primary impact. The overall severity of pedestrian injury increases in proportion to the weight and speed of the vehicle, and for any given impact the elderly will sustain more numerous and severe injuries.

Over-running injuries may be associated with oil staining from the undersurface of the vehicle, the impacting parts of which may retain tissue and blood stains. The turning vehicle wheels can leave prominent imprint bruise/abrasions of the tread-pattern, and commonly create shearing forces which lacerate the subcutaneous fat and peel off the skin as de-gloving or flaying type injuries. Occasionally the skin is intact with an underlying pocket of subcutaneous haematoma and pulped fat. Crushing thoracic and abdominal injuries are common. When the over-running is by a second vehicle following after the car which first struck the pedestrian, then the over-running injuries, although severe, may show minimal haemorrhage because the victim was already dead and without a circulation at the time of over-running. In some run-overs the victim was lying in the roadway drunk prior to impact. The intention of the victim in these circumstances is difficult to gauge, and the possibility that they were a victim of a physical assault needs to be explored but may be impossible to resolve.

Vehicular occupants

The pattern and severity of injuries sustained by vehicle occupants during a collision is influenced by the force of the impact and its direction, as well as any intrusion into the passenger compartment. The location of the person within the vehicle, the use of seatbelts and the deployment of airbags are other factors. The most important factor is the rate of deceleration of the vehicle. Spreading the deceleration over a longer time period through the 'crush design' of the vehicle to facilitate a steady deformation of its frame during a collision is an important safety feature.

Injuries to vehicular occupants are produced by impact with the interior of the vehicle, distortion of the vehicle frame with intrusion into the compartment, and partial or complete ejection of the occupant. Wearing a seatbelt substantially reduces the risk of ejection but does not eliminate it. Intrusion into the passenger compartment is more likely in high speed collisions and in vehicle rollovers, increasing the risk of severe and fatal injuries. If vehicle occupants are unrestrained then rollover crashes are commonly fatal as a result of partial or complete ejection from the vehicle. Side impacts can produce

severe injuries because the vehicle side provides poor protection against intrusion. Side impacts generally produce more severe injuries than frontal impacts even in restrained occupants because the seatbelts are less effective. Seatbelts and airbags may protect against impact with the interior of the vehicle but cannot protect from the severe deceleration forces of high-speed collisions. Additionally post-crash fire, fall over a precipice or submersion in water may be critical factors in individuals incidents. Level crossings where roadways cross railways are the site of train/vehicle collisions, and together with trespassing on the railway line, account for the overwhelming majority of railroad deaths. Railroad deaths of any type are characterised by severe injuries, which often include traumatic amputations of the limbs and severe disruption of the torso.

The classically described injuries of vehicular occupants are those occurring in a head-on collision without the use of safety restraints. At impact forward movement of the driver and front-seat passenger produces knee impact against the fascia with soft tissue injuries which may be associated with fractures. Bracing for impact, by pressing the feet against the floor, results in lower leg injuries to the passenger, while hard braking may cause a similar injury to one leg of the driver. The shoe soles of the driver should be examined for imprint marks which, if present, should be compared with the rubber covers of the accelerator and break pedals. Continued upward and forward movement of the occupants produces head impact against the roof interior, windshield and pillars and chest impact against the steering wheel or, in the case of the front seat passenger, the dashboard. Unrestrained rear seat passengers similarly sustain lower limb injuries and head and torso injuries from striking the back of the front seat or being thrown over the front seat and striking the vehicle interior.

Chest injuries, almost invariably including rib fractures, are common. Contusions of the lungs are the commonest visceral injuries, and occur most often in the paravertebral areas. Movement of the heart within the chest during rapid deceleration may lacerate the arch of the aorta, usually where the vessel becomes fixed to the vertebral column at the junction between the arch and the descending aorta. A skull fracture, typically a transverse hinge fracture of the skull base, is seen in about half of all occupant fatalities following a frontal collision with impact speed greater than 30 miles per hour (50 kph). Road traffic accidents are the commonest cause of traumatic axonal injury of the brain. Cervical spine injuries, most commonly an atlanto-occipital disruption with associated instantaneous death, occur in a significant minority.

Seatbelts and airbags

A seatbelt prevents ejection from the vehicle and restrains the body during deceleration, protecting the head by preventing impact with the vehicle interior. Seatbelts are designed to stretch and allow for some small movement during deceleration so as to extend the time of deceleration and reduce the rate of application of force. At the same time the belt spreads the deceleration forces over the entire contact area thereby reducing the force per unit area. Seatbelts produce their own pattern of internal injuries. A 2-point lap belt

compresses abdominal organs against the vertebral column and allows hyper-flexion of the body around the seatbelt. Lumbar spine and lower abdominal vascular damage, lacerations of the mesentery and rupture of the diaphragm result. The three-point seatbelt with its diagonal strap allows hyper-flexion or hyperextension of the neck with resultant cervical and upper thoracic spine fractures and trauma to the soft tissues of the neck, heart, lung and aorta. Improperly placed and adjusted seatbelts increase the risk of such trauma. Overall, the use of seatbelts, while reducing the severity of injuries, has influenced the pattern of injuries with an increase in facial and skull fractures in drivers and an increase in rib and sternal fractures and abdominal bruising in all vehicle occupants.

Airbags offer added protection in a collision but are associated with some risks. Airbags function by sensing the deceleration of a vehicle following impact and detonating an explosive, commonly of sodium azide, which produces a large volume of gas to fill and deploy the bag. The explosion produces a large amount of white smoke which may be mistaken for evidence of a vehicle fire. When the sodium azide explosive propellant is ignited, the deploying airbag explodes towards the vehicle occupant at a speed of up to 200 miles per hour (320 kph). Injuries can result from contact with either the airbag module cover or the canvas-covered airbag itself.

The airbag module covers are located in the steering wheel and in the dashboard panel on the passenger side. Hand and arm injuries occur if these body parts are in contact with the airbag module at the moment of deployment. A driver holding the steering wheel may have an arm across the module or may be attempting to blow the horn, the button of which is commonly located within the module cover, while a front seat passenger may be bracing for impact with hands over the dashboard module. The resultant injuries to the hands and arms include fractures, amputation and degloving-type soft tissue loss. If the module cover makes contact with the occupant's face, head or neck, then severe injuries can result. All of these serious injuries occur during the punch out phase of the bag deployment. At the mid-stage, the catapult stage of deployment, the rapidly inflating bag forces the head backwards and may cause hyperextension type neck injuries, including atlanto-occipital dislocation and cervical fractures. The final stage of deployment is appropriately called 'bag slap' because the canvas bag fabric slaps the occupant's face, most commonly producing abrasions to the forehead, nose and both cheeks. Both the airbag module cover and the airbag may have trace evidence of contact with an occupant, such as blood, tissue and also makeup, for example lipstick, rouge and mascara.

Children and adults of short stature may sustain fatal airbag injuries in low speed collisions. Rear-facing child seats placed in the front passenger seat may result in fatal neck injuries to the child when the passenger-side airbag deploys.

Identifying the driver

Occasionally when there is a dispute about who was driving and who was a passenger, the injury pattern may help to

distinguish. The rapid deceleration of a vehicle crash causes the vehicle occupants, whether they are restrained or not, to move initially towards the primary area of impact. This occupant movement or occupant kinematics predicts the direction of movement of each occupant and therefore what part of the vehicle interior they will strike and the type, as well as probability, of injury. Matching the pattern of injuries of survivors and decedents with the interior surfaces and components of the vehicle will help identify an occupant's position and establish who was driving.

When a three-point seatbelt has been worn then the direction of the resultant oblique bruise/abrasion over the anterior chest and shoulder provides a clear indication on which side of the vehicle the person was seated. A line of laceration and pulping of subcutaneous fat corresponding with the position of the seatbelt may be better defined at autopsy than overlying bruises and abrasions. The heat-tempered side window glass of a vehicle commonly shatters on collision raining the interior with small dice-like fragments of glass, which produce a characteristic pattern of dicing injuries, most prominent on that half of the face nearest the side window. The glass and plastic laminated windshield glass, by contrast, fractures into shards which are held intact and do not produce this injury pattern but rather larger irregular impact injuries.

The classical injury seen in an unrestrained driver is an imprint bruise/abrasion on the chest, with underlying fracture of the sternum and ribs, from impact with the steering wheel. However, restrained drivers in frontal impacts typically sustain head injuries due to contact with the steering wheel. Often imprint abrasions resulting from forceful contact against protruding interior fittings of the vehicle can be matched with the causative object and so assist in locating the individual within the vehicle at the moment of impact. Often drivers have multiple injuries of this type over the anterior inner thighs from contact with the fascia on either side of the steering column. Imprint injuries to one outer thigh commonly represents impact with door fittings. Co-transference of trace evidence from the vehicle into wounds and from clothing and wounds onto vehicle components provides evidence of what part of whose body impacted with what portion of the vehicle interior. Hair, tissue and blood deposited on the windshield interior, steering-wheel, roof and pillars can, through hair comparison, serology or DNA profiling, be matched with a specific vehicle occupant.

Retention of clothing will allow comparison with clothing fibres recovered from parts of the vehicle interior and any imprinted fabric pattern on the airbag module cover. The clothing should be examined for evidence of marks from the seatbelt, and occasionally the forces involved are so great that synthetic clothing fabric is melted. Similarly the seat-belt may have traces of adherent fabric. The soles of leather shoes may reveal the imprint of the accelerator or brake pedal or the imprint of the floor mat.

Motorcyclists and bicyclists

Motorcyclists commonly sustain leg injuries from the primary impact or as a result of entanglement in the motorcycle but

these injuries are rarely fatal. Head and neck injuries resulting from being thrown forward are a common cause of death. The wearing of a helmet will protect against scalp injuries and fractures of the skull vault but not against acceleration/deceleration brain injuries. The crash helmet should always be examined for evidence of external damage since this is the evidence which would otherwise have been apparent on the skin surface. A motorcyclist thrown forward and striking the top of the head on the roadway or other fixed object may sustain a ring fracture of the base of the skull when the head is compacted onto the torso. Being thrown at speed off the motorcycle and sliding along the roadway produces broad patches of prominent grazing abrasions contaminated with road surface debris (so-called 'road rash'), if there is insufficient protection from clothing.

Bicyclists are most commonly killed and injured when struck by a motor vehicle, or sometimes a protruding part of a vehicle during overtaking. Damage to the bicycle rather than to the bicyclist may reflect the primary impact and have trace evidence from the striking vehicle. Bicyclists struck from behind often show primary impact imprint-type bruise/abrasions to the area of the buttock and back of the thigh. Secondary impact injuries may result from either being thrown up onto the vehicle or from being knocked to one side onto the roadway. As with pedestrians, bicyclists may be over-run by succeeding vehicles following the initial collision.

Manner of death

Not all deaths on the road are accidents, some are the result of natural disease and others are suicides or homicides. Natural disease causing sudden death in the driver of a motor vehicle is typically not so rapid in its onset that the driver cannot exercise some control, slowing the vehicle and veering off the road. When witnessed the unusual nature of the incident raises the suspicion of natural disease, and typically the vehicle driver has minimal non-lethal injuries which are insufficient to account for death. Ischaemic heart disease is the main cause of natural death at the wheel, and occasionally the cause is a disease of the brain.

Suicide by motor vehicle represents a very small percent of driver fatalities in industrialised countries. Typically the single occupant car veers at high speed, for no apparent reason, into oncoming traffic, often a heavy goods vehicle. Establishing that the death was a suicide may be difficult and indeed, masking the suicide as an accident may be the intent. Parking at a railway level crossing may be used also as a suicide method but more common methods of suicide by train are lying on the railway track, jumping in front of a train, or jumping from the platform at an underground (metro) station.

A driver may use a vehicle as a weapon, deliberately striking a pedestrian, bicyclist or other road user. Occasionally a simulated vehicle crash and post-crash fire is used in an attempt to conceal a homicide, in the same way that a building fire can be used for the same purpose.

CHAPTER 12

ALCOHOLS

Alcohol is the common name for ethyl alcohol (ethanol, $\text{CH}_3\text{CH}_2\text{OH}$), one of a chemical family of alcohols, all characterised by a hydrocarbon chain and a hydroxyl group (OH). Methanol (methyl alcohol, CH_3OH) and isopropanol (isopropyl alcohol, 2-propanol, $\text{CH}_3\text{CHOHCH}_3$) are other members of this same chemical family. A related chemical family of compounds possessing two adjacent hydroxyl groups are the glycols (diols), one member of which, ethylene glycol (an antifreeze) is also of forensic interest.

Alcohol (Ethyl Alcohol)

Ethanol is produced by the natural fermentation of plant sugars by micro-organisms, particularly yeasts. Alcoholic beverages produced in this way have been consumed by humans for at least 6,000 years. Natural fermentation ceases when the alcohol concentration reaches between 10 and 15%, so that distillation is then required to produce beverages, such as whisky, having a much higher alcohol concentration. The aroma, flavour and colour of different alcoholic beverages are largely a result of congeners, which are higher molecular weight alcohols and aldehydes.

The typical alcohol concentrations of common beverages are beer 2-6%, table wines 8-12%, fortified wines (sherry, port, vermouth) 14-22%, liqueurs 15-40%, and spirits (whisky, vodka, rum, brandy, gin) 35-45%. This alcohol content, stated on bottle labels, is the percentage alcohol by volume (%v/v), i.e. the number of millilitres of alcohol per 100 millilitres of solution. Ethanol has a specific gravity of 0.79 so that 1ml of ethanol weighs 0.79gm, and consequently alcohol content expresses as %weight/weight, %weight/volume and %volume/weight are not the same. Proof spirit contains 57% alcohol by volume, so that this concentration represents '100% proof'. Thus British proof value is 1.75 times the percentage alcohol concentration by volume e.g. whisky 40% v/v is 70% proof. However, US proof value is twice the alcohol percentage by volume e.g. US whiskey 40% alcohol v/v is 80% US proof. Various standards of volume for the sale of alcoholic beverages are used in different countries; one pint is 570mls or 20 fluid ounces or 4 gills. The medical literature discusses alcohol consumption in terms of units of alcohol, where 1 unit is 10ml or 8gms. As an approximate guide, one unit of alcohol is present in half a pint of beer, 100ml of table wine, or 25ml of spirits.

Absorption of alcohol

After the consumption of a simple alcoholic drink the combined effects of absorption, metabolism and excretion produce a characteristic blood alcohol/time curve. Initially the blood alcohol concentration (BAC) rises steeply to a distinct peak BAC during the absorption phase; then follows an irregularly curved fall as tissue levels reach equilibrium; thereafter the BAC falls progressively in a linear fashion during the elimination phase.

Alcohol is absorbed from the stomach, but mostly from the proximal small intestine. Absorption is rapid with the peak BAC occurring about 30 minutes after ingestion, although this may be delayed until two hours if a large amount of alcohol is consumed or if gastric emptying is delayed. The rate of gastric emptying is important because it governs the rate of access of ethanol to the main absorptive surfaces in the small bowel. A meal, particularly one rich in carbohydrates, delays gastric emptying thereby reducing the rate of ethanol absorption and reducing the peak BAC, which may be half that achieved with the same dose of ethanol on an empty stomach. Alcohol absorption, which is by passive diffusion, is most rapid with alcohol concentrations around 10-20%. The higher alcohol concentrations in neat spirits irritate the gastric mucosa, induce mucus secretion, and delay gastric emptying, and so reduce the rate of absorption. Absorption of any alcohol consumed is generally complete within 1-3 hours.

Once absorbed, ethanol distributes uniformly throughout the body water, so that its concentration in any tissue is directly proportional to the water content of that tissue. Muscle, a tissue rich in water, will contain more alcohol than adipose tissue. The total amount of body water available for alcohol to distribute within will depend upon total body weight and body build, i.e. leanness or obesity. Since women are on average smaller than men and have a higher proportion of body fat, they have a lesser volume of body water into which alcohol can distribute, and commonly achieve a higher BAC than men after drinking the same amount of alcohol. The consumption of one unit of alcohol will typically produce a peak BAC of 15mg% in the average man and 20mg% in the average woman (mg% is milligrams per 100 millilitres, mg/100mL or mg/dL).

The Widmark equation gives an approximation of the peak BAC expected following ingestion of a known quantity of alcohol. According to this equation peak BAC (in mg%) equals alcohol ingested (in grams) x 100, divided by corrected body weight (in kgs x Widmark factor); where the Widmark factor is an estimate of body water content and averages 0.7 for men and 0.65 for women.

A very small percentage of absorbed alcohol is excreted in the breath and urine, while 95% or more is metabolised in the liver. The hepatic enzyme alcohol dehydrogenase converts ethanol to acetaldehyde, which in turn is metabolised by aldehyde dehydrogenase to acetate, and eventually to carbon dioxide. A hepatic microsomal P450 enzyme, CYP2E1, or simply microsomal oxidising system, also metabolises ethanol and may play a larger role in chronic drinkers as a consequence of enzyme induction by regular ethanol consumption. Another enzyme, UDP-glucuronyltransferase, forms the minor metabolite ethyl glucuronide, which can serve as a marker for heavy alcohol consumption.

The rate of clearance of alcohol from the blood is generally taken as 15mg% per hour, which is the equivalent of one unit of alcohol per hour. However, the range of clearance varies from 10 to 30mg% per hour, and even up to 60mg% per hour in heavy drinkers. There are genetic polymorphisms which affect the enzymes alcohol dehydrogenase and aldehyde dehydrogenase, and so influence ethanol metabolism. A

mutation in some Asians produces an inactive aldehyde dehydrogenase enzyme so that high blood acetaldehyde levels occur, causing unpleasant symptoms including facial flushing. Individuals with this mutation are forced to avoid alcohol. The drug disulfiram (antabuse), used to treat alcohol dependence, replicates the effect of the mutation by inhibiting aldehyde dehydrogenase, resulting in an alcohol-flush reaction which has the effect of dissuading the user from further alcohol consumption. Dimethyl- dithiocarbamate pesticides are chemically similar to disulfiram, and although not toxic in themselves will induce a similar reaction. Through a similar mechanism the orally active anti-diabetic drugs chlorpropamide and tolbutamide, the antibacterial drugs metronidazole and tinidazole, and the cephalosporin antibiotics cephalexin and latamoxef cause an alcohol-flush reaction.

Effects of alcohol

Alcohol is a central nervous system depressant and its actions resemble those of the volatile anaesthetics like chloroform. As well as causing a general non-specific depression of nerve and cellular function, ethanol has specific psycho-active actions. Like the benzodiazepines and the barbiturates, ethanol enhances GABA-mediated inhibition, which explains why these drugs enhance the actions of ethanol. Other varied effects on excitatory and inhibitory nerve processes account for the complex pharmacology of ethanol.

Ethanol first affects the higher functions of the brain and only when in high concentrations affects the vital respiratory and vasomotor centres of the brain stem. Depression of cortical functions causes disinhibition and more extrovert behaviour mimicking a stimulant effect; depression of the limbic system causes memory loss, confusion and disorientation; depression of the cerebellum causes inco-ordination and slurring of speech; depression of the reticular formation in the upper brain stem causes stupor and coma; depression of the respiratory and vasomotor centres in the lower brain stem may cause death.

Traditional tabulations of the level of BAC against specific effects are unreliable because tolerance develops in chronic alcohol use so that the effect of a given BAC on an occasional social drinker may be very different from that in a chronic alcoholic. The first detectable deterioration in driving skills begins at 30mg%. Up to about 100mg% there is a stage of excitement with talkativeness, and loss of emotional restraint and judgement. Between 100 and 200mg% is the stage of confusion with inco-ordination, slurred speech, and emotional lability. Beyond 200mg% is the stage of stupor with the danger of inhaling vomit and the possibility of coma. For a young inexperienced social drinker a BAC above 200mg% may kill, and for a typical social drinker a level above 300mg% may kill. By contrast a chronic alcohol may appear little affected and be conscious and engaged in conversation at a BAC of 500mg%. Death from acute alcohol intoxication is commonly associated with airways obstruction either as a result of inhalation of vomit or positional (postural) asphyxiation where the stuporous victim passes out slumped in an awkward position, partially obstructing the upper airways.

Excessive alcohol intake over a prolonged period can produce a wide range of physical, psychological and social complications. Among the more common are multiple bruises of various ages due to frequent drunken falls, oesophagitis, gastritis, peptic ulcer, pancreatitis, alcoholic liver disease, hypertension and cardiomyopathy, alcoholic dementia, male impotence, anaemia, pneumonia, anxiety, depression, and a high suicide risk, domestic violence and problems at work. Chronic alcoholics are at high risk of accidental deaths of all types. There is a risk of infection particularly bronchopneumonia and lobar pneumonia, fatal haemorrhage from ruptured oesophageal varices, or hepatic failure secondary to cirrhosis or alcoholic hepatitis. Hypothermia may follow severe intoxication or injury. A minority of chronic alcoholics died from obscure causes not readily demonstrable at autopsy such as a cardiac arrhythmia (associated with a prolonged QT interval) or a metabolic disturbance e.g. alcoholic ketoacidosis.

Measuring alcohol

Fast and reliable methods of alcohol analysis are needed in clinical and emergency medicine whenever a patient presents with unconsciousness smelling of alcohol, in order to decide whether gross intoxication or head trauma or both are the cause of the unconsciousness. Also ethanol intoxication needs to be quickly distinguished from poisoning by more dangerous alcohols such as methanol, isopropanol and ethylene glycol. In clinical laboratories a variety of analytical methods are available, all based upon use of the enzyme alcohol dehydrogenase. In forensic practice the method used is gas chromatographic headspace analysis. The sample is placed in an air-tight vial, allowed to equilibrate at a controlled temperature and a portion of the vapour in equilibrium with the sample in the vial is removed by a syringe and analysed by gas chromatography. The sensitivity and specificity of this method make it the gold standard in forensic practice.

When alcohol is measured in hospital laboratories the sample is usually plasma or serum and the units of measurement are in the SI system of moles per litre. By contrast forensic laboratories test whole blood samples and report results in terms of mass per unit volume (mg/dL). Since alcohol distributes uniformly in body water and the water content of plasma, or serum, is higher than whole blood, alcohol concentrations in plasma are about 14% higher than in whole blood. An alcohol concentration of 100mg/dL in blood (0.100g% w/v or 21.7mmol/L) is equivalent to a plasma concentration of 114mg/dL (24.7mmol/L).

Electrochemistry and infrared methods are used for analysing breath alcohol. A small amount of the alcohol a person drinks is expelled unchanged in the breath and breath-alcohol measurements provide a fast and non-invasive way to monitor alcohol in the body. The main application of breath-alcohol analysis is in traffic law enforcement for testing drunk drivers, and also for workplace alcohol testing. Most of the hand-held screening devices used for roadside testing incorporate electro-chemical fuel-cell sensors that oxidise ethanol to acetaldehyde and in the process produce free electrons. The

electric current generated is directly proportional to the amount of ethanol consumed by the cells. Acetone, which is the most abundant endogenous volatile exhaled in breath, and reaches high levels during prolonged fasting or in diabetic ketoacidosis, does not give false-positive results. However, methanol and isopropanol do interfere with the testing. Larger evidential breath-testing instruments identify and measure the concentration of alcohol by its absorption of infrared energy.

The role of alcohol intoxication in causing traffic accidents is well recognised. Most countries have alcohol concentration limits for blood, breath and urine defined by statute, and known as 'per se alcohol limits'. A statutory limit for breath alcohol when driving avoids the need to convert a breath alcohol into an equivalent blood alcohol reading. In most jurisdictions there is a statutory conversion factor assumed of 2100:1 (i.e. 100mg/dL blood is equated with 100mg/210 litres breath), although a more scientifically accurate ratio would be 2300:1 for blood, and 2600:1 for plasma or serum.

In the USA testing for alcohol in the workplace is also regulated by statute. In some Islamic countries there is an absolute prohibition on the sale and consumption of alcohol.

Alcohol post-mortem

Blood analysis for alcohol is the commonest request in post-mortem toxicology and is positive in around one third of all unnatural deaths. Although the technical aspects of measuring ethanol in body fluids are much the same in the living and the dead, the interpretation of results obtained from autopsy samples is confounded by several problems. The two most important are microbial alcohol production and alcohol diffusion from gastric residue or airways contaminated by vomit.

Distinguishing between alcohol ingestion in life and microbial production after death is a common problem. Within a few hours of death gut bacteria penetrate the portal venous system and, after about six hours, contaminate the systemic vessels. In the blood, glucose and lactate provide the substrates for microbial ethanol production by a pathway opposite to that of its catabolism in the living body. High environmental temperatures after death, terminal hyperglycaemia, terminal septicaemia, abdominal trauma, and severe trauma with wound contamination all provide particularly fertile conditions for ethanol synthesis. At room temperature, a blood ethanol of 150mg% (33 mmol/l) can be reached in a few days, although more typically values are below 70mg% (15 mmol/l). Disruption of the body of a severity commonly seen in aircraft accidents carries a high risk of post mortem alcohol production. Collecting the blood sample into a tube containing fluoride will inhibit further alcohol production by micro-organisms but will not undo the damage already done.

Given the seriousness of the problem and the potential legal importance of the analytical result, it is important that ethanol measurements in post mortem blood are corroborated by analyses of other body fluids. Vitreous humour, from the eye, and bladder urine are helpful here. Vitreous, which is easily

obtained, is valuable because it is well protected from bacterial infiltration after death. Similarly, urine is useful because it normally contains little or no substrate for bacterial conversion to ethanol, the exception being diabetics with glycosuria. Consequently, the presence of ethanol in vitreous and urine is a good indicator of alcohol consumption and its absence an indicator of post-mortem artefact in the matching blood sample.

Post mortem diffusion of alcohol from stomach contents, or from airways contaminated with gastric material, is another confounding factor. Individuals dying soon after drinking may have significant amounts of unabsorbed alcohol in the stomach at the moment of death. Passive diffusion of alcohol from the stomach and small bowel, which is the mechanism of absorption in life, continues after death, raising blood ethanol concentrations in the heart and great vessels. Consequently, alcohol concentrations in blood from the heart and torso vessels may be significantly higher than in blood from peripheral vessels. These differences between sampling sites can exceed 400%. For this reason, autopsy samples should be obtained from a peripheral vessel, such as the femoral vein, never from the heart or great vessels and particularly not from blood allowed to pool in the pericardial sac, chest cavity, or abdominal cavity.

Methanol

Methanol (CH_3OH , methyl alcohol or wood alcohol) is used as antifreeze, photocopier developer, a paint remover, and a solvent in varnishes. Together with pyridine and a violet dye, it is added as a denaturant to ethanol, specifically to rectified spirit (95% ethanol), to give methylated spirit or 'meths'. Destitute alcoholics may resort to drinking meths.

Acute methanol poisoning produces a distinct clinical picture with a latent period of several hours to days between consumption and the first symptoms. The majority of victims have blurred vision with abdominal pain and vomiting and in severe cases there are visual disturbances, pancreatitis, metabolic acidosis, encephalopathy and death. Survivors may be left blind. Methanol poisoning is characterised by a metabolic acidosis resulting from formic acid produced by methanol catabolism and lactic acid resulting from disturbed cellular metabolism. The severity of poisoning correlates with the degree of metabolic acidosis rather than the blood concentration of methanol. Both methanol and ethanol are substrates for hepatic alcohol dehydrogenase but the enzyme has a much higher affinity for ethanol. Therefore the catabolism of methanol to toxic formic acid can be blocked by giving ethanol as a competitive inhibitor.

Some methanol is present as a congener in alcoholic beverages, with highest concentrations in brandy (200-300mg/L) and whisky (80-200mg/L) and lowest concentrations in beer (1-10mg/L). Chronic heavy alcohol consumption causes a build-up of methanol over time because the continuing ethanol consumption competitively inhibits methanol metabolism. When the alcoholic stops drinking the elimination of methanol lags behind ethanol by 12 to 24 hours

and follows approximately the same time course as ethanol withdrawal symptoms. This has led to speculation that methanol or its metabolites have a role in alcohol withdrawal and hangover symptoms. Trace amounts of methanol (less than 1mg/L) are produced in the body normally, but in alcoholics the concentration in blood may reach 20mg/L. Blood levels of methanol higher than this suggests methanol poisoning rather than simply chronic alcoholism.

Isopropanol

Isopropanol ($\text{CH}_3\text{CHOHCH}_3$, isopropyl alcohol) is used as a substitute for alcohol in many industrial processes and in home-cleaning products, antifreeze and skin lotions. A 70% solution is sold as 'rubbing alcohol' to be applied to the skin and then allowed to evaporate as a means of reducing body temperature in a person with fever. It has a characteristic odour and a slightly bitter taste. Deaths may follow accidental ingestion or in alcoholics using it as a substitute for ethanol. It is metabolised by liver alcohol dehydrogenase to acetone which produces a sweet ketotic odour on the breath. High blood levels of acetone with ketotic breath are found also in diabetic keto-acidosis and starvation ketosis. Death may occur rapidly as a result of central nervous system depression or may be delayed and follow shock with hypotension.

Ethylene Glycol

Ethylene glycol (1,2-ethanediol, $\text{HOCH}_2\text{CH}_2\text{OH}$) is often referred to as 'glycol' because it is the simplest and commonest member of this family of alcohols which have two hydroxyl (OH) groups attached to a carbon chain. It is widely used as an anti-freeze in vehicular cooling systems, in brake fluid, and in the manufacture of man-made fibres. A colourless, oily liquid with a sweet taste and mild odour, ethylene glycol sometimes causes accidental poisoning in humans, and the careless disposal of vehicular anti-freeze results in the accidental poisoning of cats and dogs also. The fatal dose is less than 200ml. Propylene glycol (1,2-propanediol), another member of the same family of glycols, is not toxic and is used extensively in foods, cosmetics and oral hygiene products as a solvent. Ethylene glycol is metabolised by the same liver enzymes which metabolise ethanol and methanol. As ethanol is metabolised to acetaldehyde and then to acetic acid, and methanol is metabolised to formaldehyde and formic acid, so also ethylene glycol is metabolised to oxaldehyde and oxalic acid. The treatment for ethylene glycol poisoning, as with methanol poisoning, is the intravenous infusion of dilute ethanol which acts as a competitive inhibitor of the liver enzymes and allows the kidneys time to excrete the ethylene glycol before it is oxidised to toxic oxalate. The symptoms of ethylene glycol poisoning initially resemble mild alcohol intoxication and may cause death through heart failure within 24 hours or, if there is survival for a few days, death from acute renal failure as a consequence of crystallisation of oxalate within the renal tubules. The identification of the fan-shaped crystals of calcium oxalate on microscopic examination of the kidney tissue is diagnostic. Oxalate poisoning may also occur as a result of consumption of oxalic acid, which is the toxic agent in rhubarb leaves and many other plants.

CHAPTER 13

NON-MEDICINAL POISONS

Carbon monoxide

Carbon monoxide is known as the silent killer because it is a colourless odourless gas. It is formed by the incomplete combustion of organic carbon-containing material, so that the relative lack of oxygen produces carbon monoxide (CO) rather than carbon dioxide (CO₂). Carbon monoxide poisoning is a common cause of death in building fires and this is discussed in the chapter on injury and death in fire.

CO has an affinity for haemoglobin some 200 to 250 times that of oxygen, so that CO displaces oxygen to form carboxy-haemoglobin (COHb). Small environmental concentrations of CO thus cause toxic effects by reducing the oxygen carrying capacity of blood. CO also interferes with the dissociation of oxyhaemoglobin and produces a direct cytotoxic effect by inactivating some mitochondrial respiratory enzymes in the cytochrome oxidase complex. In addition, binding to cardiac myoglobin causes myocardial depression, hypotension and arrhythmias. The relative importance of these different toxic mechanisms is uncertain.

Historically carbon monoxide poisoning was a common method of suicide in the mid 20th century when many homes were supplied with 'coal gas' for cooking and heating purposes and this gas contained up to 20% carbon monoxide. Coal gas is a mixture of mainly hydrogen, methane and carbon monoxide formed by the destructive distillation (heating in the absence of air) of bituminous coal. Coal gas was replaced by natural gas, consisting primarily of methane and ethane, found associated with natural oil fields. The elimination of the supply of coal gas prevented suicide by this method and caused a dramatic reduction in the suicide rate in some countries, providing the classical example of how a reduction in the availability of a suicidal poison can reduce the suicide rate because there is not a complete transference to alternative methods.

Suicide by carbon monoxide continues as a method of suicide using motor vehicle exhaust fumes which contain up to 8% of the gas. Either the victim runs the vehicle engine in a closed garage or attaches piping, such as vacuum-cleaner hosing from the vehicle exhaust to the interior of the vehicle. Occasionally persons are found dead lying in the open air adjacent to the exhaust outlet, having been exposed to a locally high concentration of CO. The more elaborate the arrangements the clearer is the suicidal intent. Modern vehicles are fitted with catalytic converters to reduce the amount of nitrogen oxides, carbon monoxide and hydrocarbons in the vehicle emissions and so reduce air pollution. When effective, such a catalytic converter may reduce the carbon monoxide content of motor vehicle exhaust to such an extent that suicide is impossible with it, but this is not invariably the case. Leakage of exhaust fumes into a car may cause an accident through the effects of carbon monoxide on the driver, or in a stationary vehicle with the engine running to maintain heating, it may kill the occupants.

Any heating device which burns a carbon fuel may produce carbon monoxide if there is incomplete combustion through restriction of the airflow. This can occur when the exhaust flue is blocked or the heater is allowed to operate in a room without ventilation. Gas water heaters in a bathroom and charcoal-burners in a dwelling are examples from different countries of the potential hazards which may cause accidental carbon monoxide poisoning. The onset of symptoms is insidious so that victims may be overcome before they become aware of the danger. Burning charcoal in a sealed room is an increasingly used method of suicide in Japan and Hong Kong.

Methylene chloride (dichloromethane) is a clear, colourless liquid with a mild, sweet odour whose primary metabolites are CO and CO₂. It is widely used in industry as a solvent in paint and varnish thinners as well as in aerosol propellants in hair sprays, anti-perspirants, and spray paints. Occupational exposure occurs in industry, and non-occupational exposure by inhalation occurs mainly in association with household paint-stripping and use of aerosols, which may contain up to 50% methylene chloride. The liver is the primary site of metabolism to CO, CO₂, formaldehyde and formic acid. Production of CO may continue for hours after exposure and produce levels of blood carboxy-haemoglobin up to 40%.

CO is formed endogenously almost exclusively from the metabolism of haeme by haeme oxidase. This is sufficient to produce a carboxy-haemoglobinaemia of 0.4 to 0.7% only. In industrialised countries a level of 1 to 2% is found in urban non-smokers as a result of industrial pollution, and a level of 5 to 6% is typical in cigarette smokers, although it may be as high as 10% and occasionally up to 15%. For practical purposes a COHb level over 10% is usually regarded as evidence of CO exposure additional to smoking and pollution. The bacteria of putrefaction, such as α -haemolytic Streptococci, Proteus and Bacillus cereus, can produce CO from haemoglobin and this accounts for COHb levels of up to 80% sometimes found in putrefactive body cavity fluids, particularly in bodies from water. Blood samples submitted for CO analysis should fill a sealed tube leaving little empty dead-space above because CO is lost from blood on prolonged exposure to air. An analytical result of 50% COHb or more is sufficient to explain death in an otherwise healthy adult, and lower levels of 30 to 40% may kill infants and the infirm.

Cyanide

Hydrogen cyanide (hydrocyanic acid or prussic acid, HCN) is a highly volatile, colourless compound found naturally in many fruits, but particularly in bitter almonds to which it imparts the characteristic odour. Hydrogen cyanide is a gas and hydrocyanic acid is a solution of the gas in water. It is extremely poisonous and death can be expected following the ingestion 300mg of hydrogen cyanide. Exposure to a concentration of 200 to 500ppm in air for 30 minutes is fatal. The toxic effect is produced by the inhibition of the cytochrome oxidase system within all cells, so blocking oxygen utilisation and producing anoxia at a cellular level. After ingesting the acid, death is almost immediate, but after ingesting the salts may be delayed for 10 or 20 minutes while the hydrochloric acid of the gastric juice liberates hydrocyanic

acid from the potassium or sodium cyanide. Death may be preceded by agonising dyspnoea and convulsions. The body shows a pink lividity as a consequence of the blocking of utilisation of oxygen at a cellular level so that, even after death, the blood retains the bright red colour of oxyhaemoglobin. Other than a possible irritant haemorrhagic gastritis, the only distinctive autopsy finding is the odour of bitter almonds, most readily appreciated when the body cavities and stomach are first opened. However, approximately 10% of the population are unable to smell the distinct bitter almond odour of cyanide. The amount of cyanide liberated in the stomach may represent a hazard for the prosecutor, producing the toxic symptoms of headache, giddiness and dyspnoea.

Cyanide poisoning may be accidental or suicidal but is rarely homicidal. Accidental poisonings occur with the use of cyanide for fumigation and in industry, particularly electroplating. Its use in suicide is typically by chemists or those having access to it in their workplace. If death is near instantaneous then the container used for the poison is likely to be obvious near the body, but in death delayed by some minutes the suicide may be able to dispose of the container. The action of cyanide is so rapid that knowledge of the antidote is of theoretical rather than practical value. The antidotes amyl nitrite and sodium nitrite act by converting haemoglobin into methaemoglobin which in turn combines with cyanide to produce non-toxic cyanmethaemoglobin. The antidote sodium thiosulphate converts cyanide to relatively non-toxic thiocyanate.

Cyanide poisoning has been used as a method of judicial execution in the United States, with the condemned person strapped into a chair within a sealed gas-chamber and the cyanide released as a gas by dropping pellets of cyanide salts into strong acid. Cyanide was used in a mass rite of murder-suicide by members of the 'peoples temple' religious community led by James ('Jim') Jones in Jonestown, Guyana on November 18th 1978 resulting in 913 deaths. Cyanide is the suicidal poison used by spies both in novel and reality because of its near instant effect. Members of the Tamil tigers (liberation tigers of Tamil Eelam), an insurgent group active in northern Sri Lanka, if faced with unavoidable capture may commit suicide by swallowing cyanide capsules they wear around their necks. Michele Sindona, an Italian financier associated with the Vatican, who was convicted of fraud and murder died in his prison cell from cyanide poisoning two days after being sentenced to life imprisonment in 1986.

Arsenic

The use of the colourless, tasteless compound arsenious oxide as a homicidal poison was common until chemical methods to detect arsenic were developed in the 19th century. Arsenic is a chemical element in the nitrogen family (group Va) of the periodic table, and is best classified as a non-metal, although some forms of arsenic are metal-like. Arsenious oxide (As_2O_3) or white arsenic is used as a pesticide, a decolouriser in the manufacture of glass, and as a preservative for hides. Arsenic pentoxide (As_2O_5) is a major ingredient of insecticides, herbicides and metal adhesives. Arsine (AsH_3) or arsenic

hydride is a colourless poisonous gas which has been used in chemical warfare. Arsenic acid (H_3AsO_4) and its salts lead and calcium arsenate are used for sterilising soils and controlling pests. Numerous organic compounds of arsenic were once employed in the medical treatment of a variety of diseases.

Arsenic poisoning is most commonly the result of the ingestion or inhalation of insecticides. It exerts its toxic effect by combining with sulphhydryle groups within enzymes and so interfering with cellular metabolism. Dimercaprol, or British anti-lewisite (BAL), so called because it was developed as an antidote against the chemical warfare agent Lewisite, was found to be an antidote against poisons such as arsenic, gold, lead and mercury, which act on cellular sulphhydryle groups. The antidote is given by an intramuscular injection of the oily solution and acts by virtue of its sulphhydryle groups which competitively bind arsenic. Arsenic is deposited in skin, hair and nails because the keratin found there is rich in sulphhydryle groups. Since occipital head hair grows at an average rate of 1cm per month, analysis of hair cut into 1cm lengths gives an indication of the time-frame of the poisoning and is a technique which can be applied to the living, the freshly dead and exhumed bodies.

The fatal oral dose in an adult is 200mg or less, and symptoms of an intense gastro-enteritis develop within 15 to 30 minutes. First there is a burning upper abdominal pain, nausea and persistent vomiting which becomes bloody. Diarrhoea, the dominant effect, soon follows and the watery blood-tinged stools resemble the 'rice-water stool' of cholera. Intense thirst is a constant feature but drinking water causes vomiting. Dehydration results in oliguria and muscle cramps and collapse, and sometimes convulsions or coma preceding death. With the development of coma, death may occur within 2 to 3 hours of ingestion of the poison but from the gastrointestinal symptoms death may not occur for up to 2 days. Those who survive the acute attack may die a week or so later from sub-acute poisoning or heart failure, or many weeks later from chronic poisoning with liver and kidney failure. In acute deaths the dominant autopsy findings are those of dehydration and a gastro-enteritis. The gastric mucosa is oedematous red and covered with tenacious blood-tinged mucus giving an appearance classically described as red velvet. The mucosa of both the small and large intestines, particularly the proximal small intestine and rectum, appear inflamed also.

Individual susceptibility to arsenic poisoning varies and tolerance may develop in individuals exposed to repeated small doses. Chronic poisoning which may follow an acute attack or represent repeated small doses mimics a systemic natural disease. The earliest signs are loss of appetite, loss of weight, nausea and commonly intermittent attacks of vomiting and diarrhoea. This may be followed by a catarrhal stage with symptoms similar to a common cold, to be followed in turn by the third stage of skin rashes which may be vesicular and mimic a nettle rash. Long exposure produces characteristic skin changes with a generalised pigmentation having a scattering of small pale areas, described as a rain-drop appearance. Hyperkeratosis of the palms and soles follows and at this point there may be loss of hair and brittleness of the nails. Horizontal white bands across the nails of the fingers

and toes, Mee's lines, indicate periods of arrested growth. The fourth and last stage is that of a peripheral neuritis, similar to that seen in chronic alcoholism, with tingling and numbness of the hands and feet and tenderness of the muscles.

At autopsy the findings are non-specific and reflect the symptomatology. Liver, the organ which most concentrates arsenic, should be submitted for analysis together with head hair and nails. Arsenic is excreted mainly in the urine and after a single dose appears in the urine within half an hour and continues to be excreted for about 2 weeks or longer. Since arsenic is ubiquitous in the soil, control samples of soil from around and below a coffin must be analysed together with material from an exhumed body.

Mercury

Mercury, or quicksilver is a liquid metal of Group 11b, or the zinc group, of the periodic table. Mercury has been found in an Egyptian tomb of about 1500BC and was also known to the ancient Chinese and Hindus. Its name originated in 6th century alchemy, in which the symbol of the planet Mercury was used to represent the metal. The common name for mercury is quicksilver, and the chemical symbol Hg derives from the Latin 'hydrargyrum', literally 'liquid silver'. Metallic mercury is not poisonous if swallowed because it is not absorbed, and so the danger from a broken glass-mercury thermometer in the mouth comes from the glass and not the mercury.

Mercuric nitrate was used for making felt for hats and the expression 'as mad as a hatter' is a reference to the effects of chronic poisoning in this industry. The tremor of mercury poisoning is known as hatter's shake. Chronic mercury poisoning may result from occupational inhalation of mercury vapour or volatile organic mercurials, or from absorption through the skin of various mercury salts. The clinical hallmarks are a metallic taste and excessive salivation, gingivitis, loosening of the teeth, peripheral neuritis, loss of weight and personality changes.

Mercury exerts its toxic effects by combining with the sulphhydryle groups present on intracellular enzymes, in a similar manner to arsenic. Acute mercury poisoning following the ingestion of soluble mercury salts, resembles arsenic poisoning with a severe gastro-enteritis followed by collapse, convulsions and death. If death is delayed for a few days then the nephro-toxic effects of mercury manifest as acute renal failure. Occasionally in acute poisoning there is a blue-black line on the gums as with lead poisoning. Deposition of mercury in the capsule of the lens of the eye, mercuria lentis, is an early finding observable through a slit lamp.

Poisoning with organic mercurial compounds produces central nervous system symptoms. Progressive muscle weakness, loss of vision, impairment of cerebral function, eventual paralysis and sometimes coma and death result. This type of mercury poisoning is known as 'Minamata disease' because of a dramatic outbreak occurring in Minamata, Japan in the early 1950's when methyl mercurials in factory effluent poisoned fish and shellfish which were harvested and eaten by fishermen and their families, as well as sea birds and

household cats. Seed grain for planting, pre-treated with organic mercurials, has caused similar outbreaks of poisoning when the grain has been eaten.

Thallium

Thallium (Tl) is a metal of the main Group 11a, or boron group of the periodic table. Thallium metal has no commercial use, and thallium compounds have no major commercial application, although thallium sulphate was used in the past as a rodenticide and insecticide. Thallium compounds have a few limited uses in the production of special glass and in photo-electric cells. The salts of thallium, which are soluble in water, colourless and nearly tasteless, are highly toxic. Thallium has been used as a homicidal poison in Europe and Australia. Symptoms of acute poisoning appear within 12 to 36 hours and mimic a gastro-enteritis with abdominal pain, vomiting and diarrhoea. Neurological symptoms may dominate and these include peripheral neuritis, ataxia, impaired vision and convulsions. Loss of hair, which is a characteristic effect of thallium poisoning, occurs one or two weeks after ingestion. Hair loss involves the head, lateral two thirds of the eyebrows and axillae. As with arsenic poisoning, transverse lines on the fingernails, Mee's lines, are seen. The triad of gastro-enteritis, poly-neuritis and hair loss is a strong indicator of thallium poisoning. A fatal dose is about 1gm of a soluble salt and death follows within two days or two weeks depending upon the dose. The autopsy findings are non-specific and reflect the symptoms. An abdominal X-ray will disclose thallium as a high-density opacity within the intestine and the liver, where it is concentrated. Thallium can be detected in exhumed bodies as well as in the ashes of cremated bodies.

Mineral acids and caustic alkalis

The mineral acids (sulphuric, nitric, and hydrochloric) and the strong alkalis (of sodium, potassium and ammonium) are corrosive poisons, although in very dilute solutions they act as irritants. Many household bleaches and cleaners are corrosive also. Their corrosive action produces similar symptoms and signs resulting from local chemical burns (see chapter 4 burns) and systemic shock. Following swallowing of the corrosive there is a burning sensation in the mouth, throat and upper gut, followed by intense thirst, difficulty in swallowing and continuous retching and vomiting. If the corrosive enters the airways then breathing difficulties occur. Death is typically the result of systemic shock with consciousness retained until close to death. The external appearances reflect the corrosive damage to the lips, mouths and surrounding skin of the face as well as dribble and trickle marks running from the mouth onto the chin, neck and chest. Internally there are chemical burns to the mucosa of the upper gastrointestinal tract. Coughing and spluttering may leave spatters of the corrosive on the hands, clothing and surrounding objects.