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Lecture Notes

Bodies from Fires

SUMMARY

1. The key questions to be resolved are: Was the victim alive or dead at the time the fire started? Is the cause of death fire related? If so, is the cause of death smoke inhalation, burns, or heat shock? If not, what is the cause of death?

2. To resolve the above questions the following information must be correlated: (a) the circumstances preceding the death, (b) evidence of the origin, development, and nature of the fire, and (c) the autopsy findings.

3. Fire artefacts occur in any corpse exposed to a fire, irrespective whether death was a result of the fire or the person was already dead when the fire started. Characteristically the body assumes a pugilistic posture with the limbs flexed; tissue desiccation and fractures produce body shortening and the limbs may be partly burned away; desiccation produces up to 60% weight loss. The facial features are distorted by blackening and skin contraction.

4. Heat contraction of the skin of a corpse often produces splits involving the arms, thighs and abdomen. These split artefacts may be misinterpreted as tears and cuts inflicted during life. The heat of the fire may fracture bones or make them so brittle that they are fractured during recovery of the body. Artefactual fractures are particularly common in the skull where distinction from ante-mortem injury can be difficult. Artefactual accumulation of blood beneath the skull (extradural haematoma) may also be mistaken for an ante-mortem injury.

5. 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. The ante-mortem nature of burns may be obscured by the continued burning of the body after death.

6. People killed by fire die as a result of smoke inhalation, or burns, or heat shock or any combination of these. More than half of all fire fatalities are the result of smoke inhalation.

7. Smoke inhalation is the inhalation of soot and gases produced in the fire. The hot air may burn the face and upper air passages which may cause reflex cardiac death (vagal inhibition). Fires generate a wide variety of noxious gases which, when inhaled, may cause incapacitiation or death in combination with other factors; these noxious gases rarely reach lethal levels by themselves. The exception is carbon monoxide (see below). Inhalation of soot particles damages

the airways because they are super-heated and contain toxic agents.

8. In building fires evidence of soot inhalation is very common (90% or more of cases). Autopsy evidence of soot in the airways below the level of the vocal cords is proof that the victim was alive at the time of the fire. Soot in the gullet (oesophagus) and stomach implies the swallowing of soot and has similar significance.

9. In building fires evidence of the inhalation of carbon monoxide is very common and is usually associated with evidence of soot inhalation (approximately 95%). Blood taken at autopsy is analysed for carbon monoxide content and the result expressed as a percentage of the blood pigment (haemoglobin) which is saturated with carbon monoxide. Levels of up to 10% carboxyhaemoglobin may be found in the normal population as a result of cigarette smoking or atmospheric pollution; levels above 10% indicate inhalation of carbon monoxide and therefore life at the time of the fire.

10. A carboxyhaemoglobin level over 50% is generally accepted as sufficient in itself to account for death. Persons with pre-existing natural disease of the heart or lungs may succumb to levels as low as 30%. In building fires approximately half of all fire victims have a carboxyhaemoglobin level sufficiently high to account for death.

11. Carboxyhaemoglobin levels of 20-30% produce dizziness, headache, nausea and fatigue; levels of 30-40% produce impaired judgement and unconsciousness may occur. In a fire situation, levels above 30% may explain failure to escape.

12. The presence of an elevated carboxyhaemoglobin level and/or soot in the airways beyond the vocal cords provides proof that the victim was alive, but not necessarily conscious, at the time of the fire.

13. Carbon monoxide and other noxious gases which are inhaled may have an additive effect and account for deaths with carboxyhaemoglobin 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.

14. Cyanide is commonly produced in fires and results from the burning of materials such as wool, silk, horsehair, polyurethane and polyacrylonitrile. Cyanide is a powerful and rapidly active poison. Normal blood cyanide levels are less than 8 μmol/L, non-fatal toxic effects begin at about 50 μmol/L, and the fatal threshold is above 100 μmol/L.

THE INVESTIGATION

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 decedant, is particularly important. Several investigators with different areas of expertise are typically involved. 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.

The questions to be resolved by the investigation are:

1. Did death occur prior to or after the commencement of the fire? (i.e. was the victim alive or dead at the time the fire started?).

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.

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.

CIRCUMSTANCES AND MANNER OF DEATH

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 are the common victims of accidental fires. In Britain, about one-third of accidental fire victims are under 14 years, and about one-half are over 65 years of age. 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. Alcoholics and other individuals under the influence of drink or drugs represent a third at risk group. Occasionally a natural disease such as epilepsy or a myocardial infarction, may cause the victim to collapse onto a heater, starting a fire; the same natural disease may explain failure to escape the 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. Slow, smouldering fires, such as bedclothes ignited by a dropped cigarette, often result in death by smoke inhalation; the majority of body burns are post-mortem and there is sparing of the back and the back of the legs.

Mass accidental fire deaths may occur when large buildings, such as hotels, catch fire, or following transportation accidents, particularly aircraft crashes but also vehicular collisions.

In Britain, suicide by burning is rare probably because of the awareness of the pain involved. The victim commonly pours inflammable liquid, e.g. petrol over the clothing and then sets light to it. The victim may swallow some accelerant. This may occur outdoors, in a building, or in a vehicle. The distribution of burns may indicate whether the person was standing, sitting or lying at the time the fire was set.

In some Eastern cultures there is a tradition of suicide by self-immolation. The ritual suicide of a Hindu widow on the death of her husband (Suttee) has been prohibited in India. Self-immolation as a political protest by Buddist monks in Vietnam in the late 60's and early 70's increased the awareness in Western countries of this method of suicide.

Homicide by fire is rare, but a fire may sometimes be set in an attempt to conceal evidence of a homicide. This possibility should always be kept in mind in investigating fire deaths. The use of

petrol bombs, or arson directed against a private residence, may result in a homicidal fire death. Deliberately set fires may inadvertently result in a death.

SMOKE INHALATION

The lethal effects of smoke inhalation were known in the first century AD when Pliny reported that the Romans executed prisoners by placing them over the smoke of greenwood fires. In the UK over the past 30 years there has been a general rise in the total number of fatal and non-fatal fire casualties with a three-fold increase in the proportion of all casualties overcome by smoke inhalation. The cause of this trend is disputed, but firemen believe the fire atmosphere has become increasingly smokey in recent decades, possibly due to the widespread introduction of synthetic polymers in construction, furnishings and decoration. An increased quantity and/or optical density of smoke obscures vision and exacerbates the problems of escape and rescue; an increase in the toxicity of smoke and fire gases results in more rapid incapacitation of the victim.

(Reference: R.A. Anderson, et. al., Fire Deaths in the Glasgow Area, Medicine, Science and the Law, 1981, Volume 21, pp. 175-183).

"Smoke inhalation" is a general term embracing the inhalation of particulate matter and gases produced in the fire by combustion or pyrolysis (decomposition by heat without sufficient oxygen to cause ignition). Most deaths from smoke inhalation result from hypoxia caused by a combination of

carbon monoxide intoxication, a low inspired-oxygen tension, and ventilation-perfusion mis-matching. 85% of building fire victims show evidence of CO inhalation and 50% of victims have evidence of CO poisoning sufficient to cause death. A low inspired-oxygen tension may contribute to hypoxemia and 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, in 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 may contribute to death by its combined effect with other factors. Ventilation-perfusion mis-matching as a result of airways obstruction or lower airway injury may also contribute to hypoxemia.

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

(a) Thermal Injury/Burns to the Respiratory Tract

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 result in spasm sufficient to cause suffocation. Laryngeal oedema may have the same effect. Breathing very hot air may cause reflex cardiac death (vagal inhibition).

Moist air with its increased heat capacity is more likely that dry air to produce burns in the lung.

Histologically 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 hyperemia. 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, atelectasis and haemorrhagic consolidation of the lung. After 24 hours infection supervenes with bronchopneumonia and necrotising tracheobronchitis.

(b) Carbon Monoxide Poisoning

See below.

(c) Direct Particulate Injury

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 with 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 naropharynx, cannot be taken to imply life during the fire. Deposition of soot below the level of the larynx indicates that the victim was alive at the start 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 and is most easily seen if the mucus is spread on a white sheet of paper. The presence of soot in the oesophagus and stomach implies that it has been swallowed and also indicates life at the start of the fire.

Histologically the soot particles within the tracheobronchial tree lie loosely on the mucosal surface or embedded in the mucus. The particles are not incorporated into the tissues and are readily distinguished from anthracotic pigment.

(d) Smoke Poisoning

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 manmade materials. There are hundreds of such products of combustion. Whereas modern synthetic materials, e.g. PVC, may have increased this problem, natural materials, e.g. wood, wool, and silk, produce similar gases (see the findings in 496 fire deaths in the 1942 Cocoanut Grove fire in Annals of Surgery, Volume 117, 1943). The pathological effects of these noxious gases are difficult to separate from direct particulate injury. With the exception of carbon monoxide and hydrogen cyanide, post mortem analyses for toxic gases are rarely, if ever, performed.

Filter masks do not protect against the inhalation of fumes; for this purpose the mask must have a self-contained air supply.

These noxious gases rarely reach lethal levels by themselves, but they may cause incapacitation or death in combination with other factors. Some gases have a synergistic interaction, e.g. carbon monoxide and hydrogen cyanide, so that non-fatal levels of each in combination may cause death.

The concentration of the noxious gas is of importance. Hydrogen cyanide produced from wool, wood or polyurothane is rapidly fatal at 3,000 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 toxicity comparable to hydrogen cyanide and is rapidly fatal at 1,000 ppm. Acrolein is an aldehyde product of combustion of wood and paper and produces pulmonary oedema after a few seconds exposure at 10 ppm.

Toxic Gases and Vapours Produced from Burning Materials

(Reference: D H Napier, Hazardous Materials and the Gases they Produce, Medicine, Science and the Law, 1977, Volume 17, pp. 83-90).

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, polyurethanes, N-containing plastics
Aldehydes	Wool, cotton, paper, plasters, phenol-formaldehyde, wood, nylon, polyester resin
Benzene	Petroleum, plastics, polystyrene
Ammonia	Melamine, nylon, urea-formaldehyde
Sulphur dioxide	Rubber, thiokols
Phenol	Phenol-formaldehyde
Acrolein	Wood, paper

Summary of the Toxicological Findings in Victims of the Apollo Spacecraft Accident, January 27th 1967

(Reference: Apollo Accident, Report of Accident to Administrator of the National Aeronautics and Space Administration, Government Printing Office, Washington D.C., 1967)

COHb%	45	38	48
Blood CN ug%	33	67	100

Lung gases (mol%)	**RC**	**EW**	**VG**
Acetylene	40	22	39
HCN	32	16	38
fluoromethanes	14	5	10
fluoroethanes	2	1	2
butane	7	2	7
methane	5	54	13

CARBON MONOXIDE

Carbon monoxide is a colourless, odourless gas formed by the incomplete combustion of carbon compounds and normally found in the atmosphere in levels well below 1 ppm. Motor vehicle engines are responsible for about 70% of carbon monoxide liberated into the atmosphere.

CO poisoning is a pathophysiologically distinct cause of inhalation injury producing its effects by tissue hypoxia. The affinity of haemoglobin for CO is 200 times greater than for oxygen so that carboxyhaemoglobin concentration is great even when the CO concentration is less than 5% in the inhaled gas. As well as diminishing the oxygen-carrying capacity of the blood, carboxyhaemoglobin 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. concentration of gas in the inspired air and time of exposure), (b) physical activity which influences oxygen requirements, and (c) individual variations in susceptibility.

Carboxyhaemoglobin is estimated by spectroscopic methods making use of the fact that oxyhaemoglobin and carboxyhaemoglobin have different absorbtion spectra. The result is expressed as the percentage saturation and is the ratio of carboxyhaemoglobin to total haemoglobin x 100. The CO-oximeter is an automated instrument using a spectrophotometric method. An alternative gas chromatographic method is both specific and very sensitive. Carbon monoxide liberated from a known volume of blood is estimated on a gas partitioner and the haemoglobin content estimated spectroscopically.

The effects of carboxyhaemoglobin at different percentage saturation levels are generally taken as follows:

0-10%	No immediate ill effects; found in cigarette smokers or from industrial/motor vehicle pollution.
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.

Interpretation of results

- CO at levels of 1000 ppm can kill in about half an hour, and at 5000 ppm is rapidly fatal.

- COHb levels up to 10% may be found in cigarette smokers or from atmospheric pollution. Occasionally levels up to 15% are found in heavy smokers. (Tobacco smoke may contain up to 5% volume of CO).

- 85% of building fire victims show evidence of CO inhalation and approximately 50% of victims have a COHb level sufficiently high to account for death.

- 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%.

- COHb levels above 30% produce confusion and may explain failure to escape the fire.

- 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.

- An elevated COHb level is usually but not universally (approximately 95%) associated with evidence of soot in the respiratory tract.

- The presence of an elevated COHb level and/or with soot in the airways distal to the vocal cords provides proof that the victim was alive, but not necessarily conscious, at the time of the fire.

- A COHb level below 10% is not proof that a victim was dead prior to the commencement of the fire. Possible explanations may be (a) little or no CO production in the fire due to abundant oxygen allowing complete combustion, e.g. a forest fire, (b) rapid death in a flash fire.

- CO and cyanide produced in fires have an additive effect.

- Sanguinous fluid from the thoracic cavity in decomposed bodies may have COHb levels as high as 80% resulting from the post mortem production of CO.

(Reference: Post Mortem Formation of Carbon Monoxide, by T. Kojima, et. al, Forensic Science International, 1982, Volume 19, pp. 243-14).

- Methylene chloride, found in paint strippers and hairsprays is metabolised in vivo to CO and may produce COHb levels up to 40%.

HYDROGEN CYANIDE

Hydrogen cyanide is a product of the thermal degradation of many materials which contain nitrogen, e.g. wool, silk, horse hair, polyurethane, polyacrylonitrile. HCN is a potent toxin with a very rapid action. It has a half life in blood in the order of 1 hour being metabolised to thiocyanate. HCN in blood is contain principally in the red blood cells and disappears rapidly from plasma with a half life of only 15 minutes.

Normal blood cyanide levels are less than 8 umol/1, non-fatal toxic effects begin at 50 umol/1 and the fatal threshold is about 100 umol/1. Artefactual formation and metabolism of cyanide can occur in post mortem samples of blood and tissue. Artefactual formation of cyanide in post mortem blood is more likely in refrigerated samples than at room temperature.

(Reference: Fire Deaths in the Glasgow Area, R.A. Anderson, et. al, Medicine Science and the Law, 1981, Volume 21, pp. 288-294 (CO), and 1982, Volume 22, pp. 35-40 (HCN)).

BURNS

The morbidity and mortality associated with burns is related to the depth of burn injury and the extent of injury, i.e. the size of the burn relative to that of total body surface (TBS) area.

Burn Depth

The traditional classification of depth of burn injury is in degrees, i.e. first degree, second degree and third degree.

A first degree burn involves only the epidermis; it is characterised by erythema and mild pain, the latter resolving in 48 to 72 hours. Healing is usually uneventful and completed in five to ten days with no residual scarring. Sunburn is the most common first degree burn.

Second degree burns involve the epidermis and a variable portion of the dermis. 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 seven to fourteen days with minimal scarring. A deep second degree burn extends beyond the upper third of the dermis, but not beyond the dermis itself. These deeper burns are less painful than superficial second degree burns; healing is extremely slow, sometimes requiring months and usually leading to dense scarring (if the wound is allowed to heal primarily, rather than skin grafted). The fluid loss and metabolic effects of deep second degree burns are essentially the same as those seen with third degree burns.

A third degreee burn, or full thickness burn, implicates the entire epidermis and dermis. Primary re-epithelialisation will not occur and the wound will require skin grafting. Heat coagulation of dermal blood vessels leaves the tissue avascular with a characteristic waxy white colour. Prolonged contact of subcutaneous fat with a flame source produces a leathery brown, or black, charred appearance. There is characteristic lack of pain, due to heat destruction of all nerve endings.

Burns may also be classified as either partial thickness or full thickness. Partial thickness wounds contain viable epithelial elements capable of spontaneously re-epithelialising the wound (first

degree, superficial and deep second degree burns). Full thickness burns have no viable epithelial elements and always require cutaneous autografting. Partial thickness burns are usually caused by a brief exposure to heat or contact with hot liquids and appear pink to mottled red, wet, covered with vesicles and bullae, and are painful. Full thickness burns result from contact with flame, electricity or chemicals. They are often dry and charred, may be translucent, and may have thrombosed superficial veins. They are insensate.

Beneath the dead burned tissue there is usually a zone of ischaemia of marginally viable tissue which is readily converted to non-viable tissue (eschar) by any further insult, such as hypoxia, decreased blood flow or infection. By this process a deep second degree burn frequently converts to a third degree burn. Prevention of wound conversion of this type is of major importance in the resuscitation period.

Burn Extent

A determination of the burn surface area is obtained using the "rule of nines", each arm being 9%, each leg 18%, the anterior trunk 18%, the posterior trunk 18%, and the head 9%, of total body surface area. In assessing irregular areas of burn, it is useful to remember that the surface area of the person's palm is roughly equivalent to 1% of the TBS. In children under 15 years of age the relative body surface area of the head, upper leg and lower leg, differs from an adult; this may lead to inaccuracy in the estimation of burn injury; use of the Lund and Browder Burn Diagram provides age-adjusted data accurate for both adults and children.

It should be remembered that the extent of burn is often over-estimated, and the depth of burn under-estimated.

Morbidity and Mortality

In addition to burn depth and extent of injury, other factors determining morbidity and mortality are the location of the injury, the age of the victim, and the presence of injuries or natural disease. Age is a major factor in survival for children under two years, and adults over 60 years.

The dermis acts as a barrier preventing loss of body fluid by evaporation and the loss of excess body heat. Consequently, loss of water through burned skin and loss of heat play a major role in the pathophysiological changes see post-burn. The skin is also the primary protective barrier against invasive infection, so that wound infection is a major cause of mortality and morbidity in the late post-burn period. Within the superficial dermis are the nerve endings that mediate pain. Consequently, partial thickness injuries which expose these nerves will be extremely painful whereas full thickness burns, which destroy the nerves, are usually anaesthetic.

The local and systemic responses to burns follow a time course continuum, but for convenience can be divided into three periods: (1) up to 48 hours, (2) two to six days, and (3) seven days to wound closure.

(1) In the first 48 hours following severe burns, hypovolaemic shock and shock induced organ failure, primarily renal failure, are the major threats to life. Hypovolaemia can also lead to wound conversion. Although the exact pathophysiology of the post-burn vascular changes and volume shifts remain to be determined, two processes are involved: an increase in microvascular fluid flux into the interstitium, both local to the burn and generalised (remote from the burn wound), and a generalised impairment in cell membrane function, resulting in cell swelling.

With modern therapy, adequate initial volume restoration is achieved in more than 95% of burns cases. Correction of hypovolaemia by the intravenous infusion of fluids is complicated by generalised burn oedema formation which may result in (a) further ischaemic insult to already damaged cells, (b) chest wall oedema with resultant increase in the work of breathing, and (c) upper

airway oedema. The latter two complications may lead to rapidly fatal respiratory failure.

Burned skin loses its elasticity. It is less compliant and unable to stretch to accommodate an increase in interstitial oedema. If burns are circumferential, particularly around the distal extremities, a tourniquet effect is produced. Initially this impedes venous return, resulting in an increase in capillary pressure and further oedema. Impairment of arterial blood flow may follow with the potential for distal tissue ischaemic necrosis. Tissue pressure can be decreased by making an incision through the burned tissue, i.e. an escharotomy. A chest wall escharotomy may be required to ease respiratory difficulties; incisions are made in the bilateral anterior axillary lines from the clavicles to the costal margins; a transverse escharotomy at the level of the costal margins connecting the two vertical escharotomies is helpful if the abdominal wall is burned to a significant degree. Escharotomy can be a bedside procedure and does not require anaesthesia because full thickness burns are insensate. If the deeper viable tissues are not incised then bleeding should be minimal.

Third degree or full thickness burns often require excision prior to cutaneous autografting. The two commonly employed techniques of excision are tangential excision (sequential shaving of non-viable tissue until a uniformly viable wound bed is obtained), and excision to fascia. The disadvantage of tangential excision is primarily that of massive blood loss.