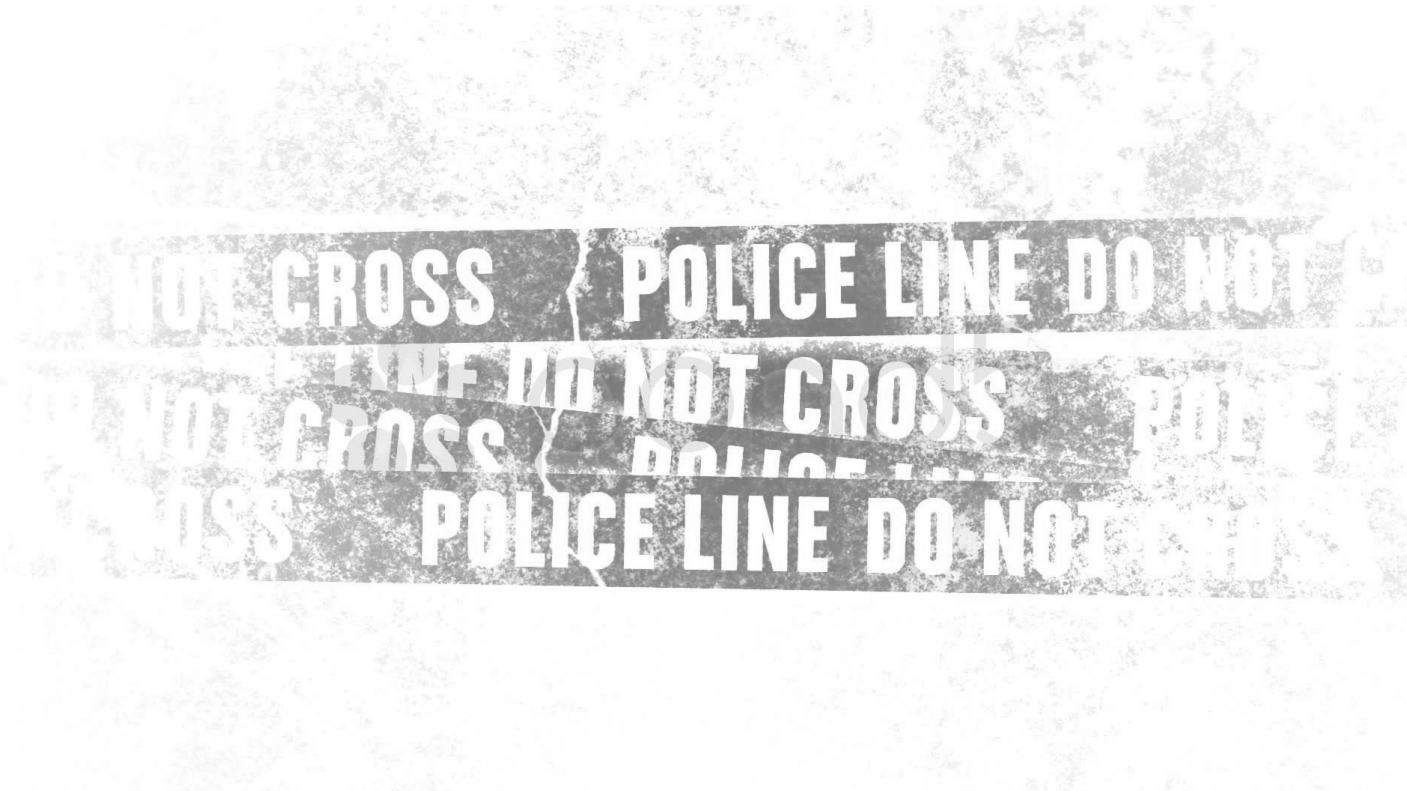

Physical Agents

Chapters 15-17 & appendix 3

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Correction file

Chapter 15

Asphyxia

'Asphyxia' means 'absence or lack of pulsation'(lack of oxygen whether it is partial (hypoxia) or complete (anoxia)

In forensic medicine, asphyxia describes a situation where there has been a physical obstruction between the mouth and nose to the alveoli.

Simple classification of 'asphyxial mechanisms' is described below:

Classification of Asphyxia:

Mechanical	• Hanging (pressure applied to the neck by means of a ligature combined with the weight of the body) • Compression asphyxia (pressure applied to the chest or abdomen, resulting in a physical interference with the ability to breathe effectively) • Choking (physical obstruction within the airways) • Smothering (physical obstruction of the mouth/ nose preventing effective breathing
Non-mechanical	• Carbon monoxide poisoning (chemical interference with respiration at a cellular level) • Cyanide poisoning.
Miscellaneous	• Drowning

Asphyxial insults are not necessarily fatal, the outcome dependent on:

1- the nature of the insult. 2- its degree. 3- and its length of time.

- Long-term health effects, depends on the length of time inadequate oxygenation was experienced.
- In the living, appropriate examination, documentation and investigations will optimize the recovery.Where the insult has resulted in death, examination by a forensic pathologist is recommended in order to identify potentially subtle evidence of assault.

Phases and signs of 'asphyxia'	• Dyspnoea phase – expiratory dyspnoea with raised respiratory rate, cyanosis and tachycardia (may last for a minute or more). • Convulsive phase – loss of consciousness, reduced respiratory movements, facial congestion, bradycardia, hypertension, fits (may last for a couple of minutes). • Pre-terminal respiratory phase – no respiratory action, failure of respiratory and circulatory centres, tachycardia, hypertension (may last a couple of minutes). • Gasping for breath – respiratory reflexes. • Terminal – loss of movement, areflexial, pupillary dilatation.
Traditionally, the 'classic signs of asphyxia' were described as:	· Petechial haemorrhages in the skin of the face and in the lining of the eyelids; · Congestion and oedema of the face; · Cyanosis (blue discoloration) of the skin of the face; · Right heart congestion and abnormal fluidity of the blood. Of these 'classic signs', the finding of petechiae in the face/neck is of most importance to the forensic pathologist.
in survivors of an 'asphyxial episode':	• pain and tenderness around the neck and structures within the neck. • damage to the larynx and associated cartilages. • damage to the hyoid bone. • dried saliva around the mouth. • cyanosis (particularly if the survivor is found immediately after the attack). • congestion and oedema of the structures above any level of compression. • petechiae above the level of the compressive force that has caused the asphyxia. • haemorrhage from the mouth, nose and ears. • elevation of the larynx and tongue, closing the airway at the level of the pharynx.

Types of mechanical asphyxial mechanisms:

- ***Pressure to the neck:*** Three most important forms in forensic medicine:

1. Manual strangulation 2. Ligature strangulation 3. Hanging

it is not possible to predict how rapidly death will occur; in some cases, death will be relatively 'slow', allowing time for the development of the 'classic signs of asphyxia, while in other cases such signs will be entirely absent.

The application of pressure to the neck may lead to any of the following:

- obstruction of the jugular veins, causing impaired venous return of blood from the head to the heart (leading to cyanosis, congestion, petechiae);
- obstruction of the carotid arteries which, if severe, causes cerebral hypoxia;
- stimulation of carotid sinus baroreceptors at the bifurcation of the common carotid arteries resulting in a neurologically mediated cardiac arrest;
- elevation of the larynx and tongue, closing the airway at the level of the pharynx (unless extreme pressure is applied to the neck, the cartilaginous trachea is more resistant to compression)

loss of consciousness following hanging was thought to occur within 10 seconds. However filmed hanging analysis suggests:

lack of recognizable respiratory movements after 2 minutes.

lack of muscle movements after 7.5 minutes.

Potential for their survival up to 14 minutes following obstructive asphyxiation.

- ***'Vagal inhibition' or reflex cardiac arrest***

Mechanical stimulation of the carotid sinus baroreceptors in the neck can result in an unpredictable, and sometimes fatal outcome. Death may supervene at any time following the application of pressure to the neck, and it is thought that such 'vagal inhibition' may explain why so many individuals found hanging show none of the classic signs of asphyxia.

- ***Hanging:*** describes suspension of the body by the neck. Any material capable of forming a ligature can be used.

The pressure is produced by the weight of the body; it is not necessary for the body to be completely suspended

The mechanism of death in hanging is incompletely understood.

Hanging by judicial execution involves a 'drop'; result in cervical spinal cord injury and fracture-dislocation of the cervical spine, but without decapitation.

Non-judicial hanging is mostly a suicidal act of males. Post-mortem toxicological analysis should be performed in order to determine whether the individual was capable of self-suspension.

In survivors of accidental or suicidal hanging there may be no adverse sequelae. For some there may be residual hypoxic brain, or neuropsychological assessment. Others have motor and/sensory loss as a result of brain damage.

- ***Strangulation:*** Application of pressure to the neck using the hands.

homicide.

The external signs include bruises and abrasions on the front and sides of the neck.When pressure to the neck is sustained, additional features of manual strangulation can include the 'classic asphyxialsigns',includingf acialpetechiae.

- In the living victim, clinical evaluation may reveal pain on swallowing, hoarseness, stridor, neck, head or back pain.
- Ligature strangulation may be homicidal, suicidal or accidental and involves the application of pressure to the neck by an item capable of constricting the neck, (like holding a belt around someone's neck until he dies).
- There is frequently a clear demarcation of congestion, cyanosis and petechiae above the level of the constricting ligature, and there is usually a 'ligature mark' on the neck at the site of constriction (so the belt well leave a mark on the neck).
- Soft and broad-surface ligatures, however, may leave very little evidence of compression on the skin of the neck, or even injury to underlying structures.
- Distinguishing ligature strangulation from hanging, in which the individual's body weight against a ligature leads to pressure being exerted on the neck (Strangulation vs hanging).
- The 'strap muscles' in the neck and injury to the superior horns of the thyroid cartilage, are particularly vulnerable to compressive injury. Calcification and ossification of the hyoid bone and thyroid cartilage occurs with increasing age, and such change is associated with less flexible structures that are more prone to injury following neck compression.

- ***Choking:*** Accidental ingestion of objects or food can cause choking. There are risks associated with individuals who are sedated or anaesthetized; Obstruction commonly leads to respiratory distress with congestion and cyanosis of the head and face.

• ***Café coronary:*** One of the commonest causes of choking is the entry of food into the air passages, If food enters the larynx during swallowing, it usually causes gross choking symptoms of coughing, distress and cyanosis, which can be fatal unless the obstruction is cleared by coughing or some rapid treatment is offered.

• However, if the piece of food is large enough to occlude the larynx completely, it will prevent not only breathing but also speech and coughing.

• The individual may die silently and quickly, the cause of death remaining hidden until the autopsy.

- ***Compressional and positional asphyxia:*** Pressure on the trunk (chest and/or abdomen) result in an inability to breathe and death.

Occasionally, individuals are crushed by the weight of many other people fleeing danger, such as during a fire in a sports stadium (like in hajj) or they may, for example, attempt to squeeze through small gaps in railings, or small open windows, and become wedged preventing expansion of the chest.

- ***Suffocation and smothering:***

used to describe a fatal reduction of the concentration of oxygen in the respired atmosphere. Mechanical obstruction of the upper airways can lead to suffocation.

Autoerotic asphyxia: used to describe those fatalities occurring during some form of solitary sexual activity.

The presence of the following features should be considered when 'diagnosing' autoerotic asphyxiation:

- Evidence of solo sexual activity;
- Private or secure location;
- Evidence of previous similar activity in the past;
- No apparent suicidal intent;
- Unusual props including ligatures, clothing, and pornography;
- Failure of a device or set-up integral to the activity causing death, the presence of classic asphyxial signs is variable. The presence of gags or other means of occluding the airways.

Chapter Images:

Figure 15.10 Suicidal hanging: the rope rises to a point, leaving a gap in the ligature mark – the suspension point – on the neck.

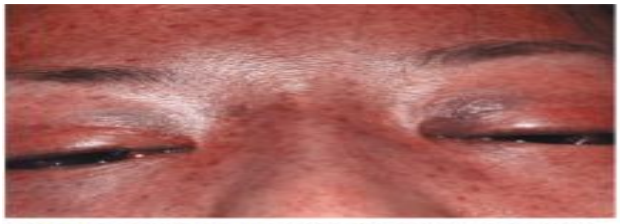


Figure 15.2 Petechial (and more confluent) haemorrhages in the facial skin and conjunctivae following manual strangulation.

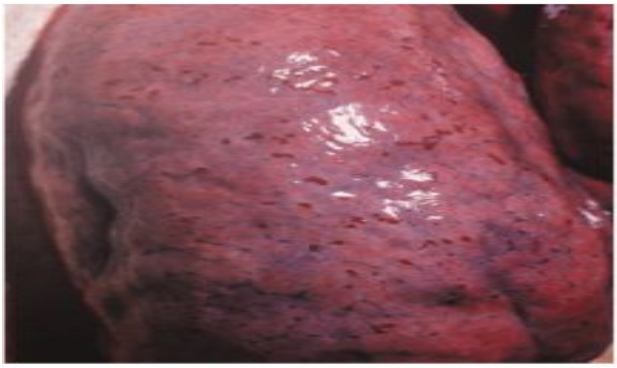


Figure 15.3 Tardieu spots (subpleural petechial haemorrhages) following manual strangulation. These are no longer considered to be specific for asphyxia.

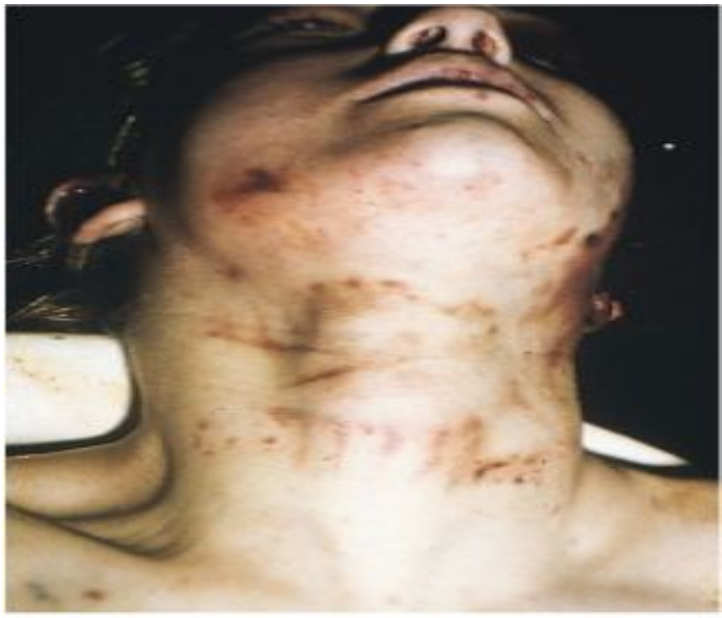


Figure 15.6 Surface injuries on the neck and jaw in manual strangulation. Note multiple bruises and abrasions; some of these injuries are caused by the victim trying to release the grip of the assailant.

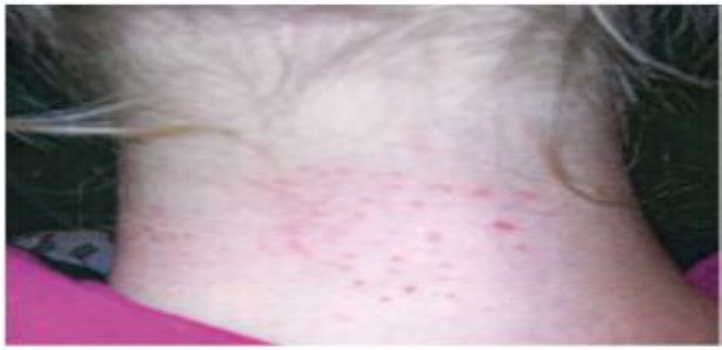


Figure 15.7 Ligature mark from a nylon scarf. Although the fabric was broad, tight stretching of the fabric resulted in a well-defined linear "band" that could be mistaken for that made by a cord or wire.

Chapter 16

Immersion and Drowning

- Most bodies found in water are there as a result of accident or suicide.
- Drowning can occur in only a few inches of water.
- Jumping or diving into water may result in limb or head injuriesthat render a person incapable of swimming.
- Bodies' retriever from water requires the expertise of marine recovery units.
- **The pathologist** must attempt to address all of these potential explanations for death and determine if there is any pathological evidence capable of supporting a diagnosis of drowning.

Examples of reasons for death in a body recovered from water:

- Died of **natural causes before** entering the water (eg. a myocardial infarction).
- Died of **natural causes while** in the water, having entered the water either voluntarily or accidentally (e.g. micturating into a canal and losing balance).
- Died from exposure and **hypothermia** in the water.
- Died of **injuries before** entering the water.
- Died of **injuries after** entering the water.
- Died from **submersion**, but not drowning
- Died from true drowning as a result of **aspiration** of water.

Evidence of immersion

A number of changes appear on the skin and body surface after a body has been in water for enough time. Appear at very variable times. Depending on:

1-whether the water is salt or fresh.

2-whether the water source is tidal or non-tidal.

3-the presence of possible predators,

4-water temperature.

5-clothing worn on the body and type of surface at the base of the water.

- If left in water for long enough, the skin will become wrinkled and macerated. The fingertips become opaque and wrinkled ('washerwoman's fingers')
- as immersion time increases, macerated skin begins to separate, leading to skin peeling and 'degloving' of the skin of the hands and feet.
- Loss of pigment layers may be apparent, causing colour change in skin.
- Estimating the post-mortem interval from signs of immersion, and decomposition, in a body recovered from water is completely unreliable and temperature dependent.
- 'Rule of thumb' recognizing that decomposition in water in temperate climates occurs at roughly half the rate of a body left in air.
- Bloating of the body (face, abdomen and genitals) owing to gas formation in soft tissues and body cavities is often evident after approximately a few days' immersion in temperate conditions, after which skin and hair loosening leads to their detachment. However, the skin and hair can remain *in situ* for weeks at a time.
- Gaseous decomposition and bloating often causes the body to 'float to the surface' of the water in which it is submerged, leading to its discovery. If the body has a lot of fat it may sink for only a short period, even in the absence of bloating.

Post-mortem artifact and immersion

- Bodies moved by the flow of water may come into contact with sand/silt, rocks, piers and other underwater obstructions.
- can injure the skin and deeper structures.
- Contact of a body with propeller blades classically leads to deep ‘chop’ wounds and/ or lacerations. Post-mortem injuries produced in such circumstances must be differentiated from ante-mortem injuries suggestive of assault.
- Other artefactual injuries characterized by immersion include damage to the body by marine life (for example, fish, crustaceans, molluscs and larger animals) and as the post-mortem interval increases, fragments/limbs may become detached and lost.

Pathological diagnosis of drowning

Pathophysiology of drowning

- the process of experiencing respiratory impairment from submersion in a liquid – may result in pulmonary surfactant insufficiency/damage, pulmonary oedema, alveolitis, hypoxaemia and metabolic acidosis.
- in cold water, As hypothermia develops, cognitive function becomes impaired increasing the risks of (1) wrong decisions and (2) aspiration of water.
- Fresh water is hypotonic compared with plasma and, when inhaled, is rapidly absorbed into the bloodstream, causing transient (but clinically irrelevant) electrolyte dilution and hypervolaemia. It causes alveolar collapse.
- Sea water is hypertonic compared with plasma and, when inhaled, results in fluid shifts into alveoli, plasma electrolyte hyperconcentration and hypovolaemia causes surfactant loss.
- Aspiration of fresh or seawater leads to systemic hypoxaemia causing myocardial depression, reflex pulmonary vasoconstriction and altered pulmonary capillary permeability.
- There is an inverse relationship between survival and the volume of aspirated fluid (sea water being twice as ‘lethal’ as fresh water).

Signs of drowning

Autopsy findings ascribed to drowning reflect the pathophysiology of submersion and aspiration of the drowning medium. so the autopsy diagnosis of drowning is one that can cause considerable difficulty.

Alternative mechanisms of death in immersion

- **Dry drowning** if there are no autopsy signs of aspiration of water like because of trauma.
- **Diving response** Stimulation of trigeminal nerve receptors by immersing the face in water which is augmented by anxiety/fear.
- Water temperature of less than 20°C and possibly alcohol, increasing the likelihood of the development of a fatal arrhythmia.
- Cardiac arrest has also been documented following entry of water into the nose.
- **The cold shock response** initiated by peripheral subcutaneous receptors, causes respiratory and cardiovascular effects Co-stimulation of both diving and cold shock responses may precipitate supraventricular arrhythmias.

The role of alcohol in drowning

Alcohol is frequently found in the blood of adult drowning victims but a causative role has not been proven. Vasodilation from alcohol intake may hasten hypothermia.

Other investigations in bodies recovered from water:

- Post-mortem blood chloride and Specific gravity analyses used to differentiate fresh and seawater drowning; such tests are of no utility in the diagnosis of drowning.
- **Blood strontium analysis has been proposed as a marker of drowning, but this test has not found widespread acceptance.**
- Diatoms are microscopic organisms present in sea and fresh water. The use of diatomology in the forensic diagnosis of drowning must be used with caution.

Chapter 17

Heat, Cold and Electrical Trauma

Injury Caused by Heat

The heat source may be dry or wet; where the heat is dry, the resultant injury is called a ‘burn’, whereas with moist heat from hot water, steam and other hot liquids it is generally known as ‘scalding’.

Complete epidermal necrosis can occur **at 44°C if exposed for 6 hours,** while such necrosis occurs within 5 seconds at 60°C and less than 1 second at 70°C.

Burns (dry burns)

Severity of Burn

The most widely used historically is:

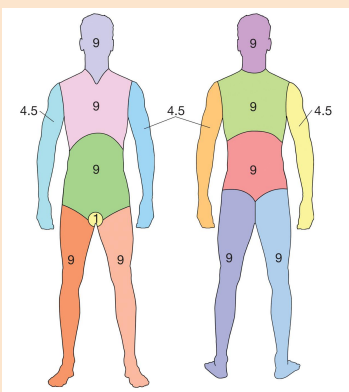
- **First degree**
- **Second degree**
- **Third degree**

Another classification has been used to reflect potential treatment options: (box17.1)

- **Full thickness**
- **Partial thickness:**
  - **Superficial**
  - **deep**

Extent of Burn

The rule of nine “Oh we already know that, thanks to surgery :)”



The size of the area of burning may be more important in the **assessment of the dangers** of the burn than the depth.

Scalds (wet burns)

*The pattern of scalding will depend upon the way in which the body has been exposed to the fluid: immersion into hot liquid resukts in an upper **‘fluid level’**.

*Splashed or scattered droplets of liquid result in scattered **punctate areas of scalding.**

*These runs or dribbles will generally flow under the influence of gravity and this can provide a marker to the orientation or position of the victim at the time the fluid was moving (Figure 17.4).

Children pulling saucepans with hot liquid off a cooker can result in this type of injury.

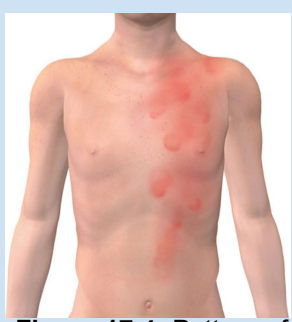


Figure 17.4: Pattern of scalding from running water.

Burns (dry burns)

Severity of Burn

Box 17.1 A classification of burns related to extent of tissue damage

- Very superficial burns – for example those caused by sunburn – may simply cause reddening with mild blistering that may occur after 12–18 hours. After 5–10 days the damaged layers of cells peel off without residual scarring
- Partial-thickness burns destroy the whole of the epidermis and possibly part of the next cellular layer – the dermis
- Superficial partial-thickness burns result in fluid production which lifts off the dead epidermis forming blisters and subsequently scabs. Sensory nerves are damaged and the burn is very painful. New epithelium grows quickly and the burn heals in 10–14 days with little or no scarring
- Deep partial-thickness burns are often less painful as nerve endings are destroyed and scarring is likely to be marked if the wound is allowed to heal spontaneously without skin grafting
- Full-thickness burns destroy all skin elements and may require substantial reconstructive surgery because of the potential for incapacitating scarring (Figure 17.1)

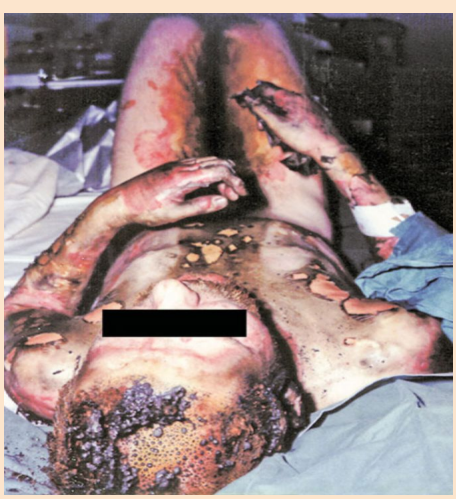


Figure 17.1: The extensiveness of burns on a body recovered from a fire may be varied. This individual had second and third degree burns after dousing himself with petrol before setting himself on fire (self-immolation). Note the molten and singed hair.

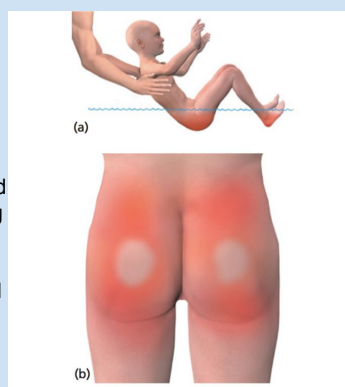
Scalds (wet burns)

Accidental VS Intentional Scalds
when a child is placed in a tub or bath of hot water

1-Accidental: rom hot beverages/liquids being pulled of a table top, etc.) are predominantly 'spill' injuries from 'flowing liquid', characterized by scalds with irreg- ular margins and burn depth, and lacking a 'glove and stocking' distribution.

2- Intentional are predominantly those caused by forced immersion in hot water, giving rise to symmetrical 'glove and stocking' injuries to the limbs, sparing skin folds (and buttocks in those forced to sit in hot water), which are of uniform depth (Figure 17.5).
Other injuries or marks suggestive of non-accidental injury may accompany intentional scalds (see Chapter 13, p. 137).

Figure 17.5 Pattern of scalding from forced immersion in a hot bath. Note the clear demarcation between scalded and uninjured skin representing the fluid level of the bath. Sparing of skin on the buttocks reflects firm contact between those parts and the base of the bath.



Where else it could be seen?

1. Industrial accidents where steam pipes or boilers burst.
2. Children who pull kettles and cooking pots down upon themselves.
3. Child physical abuse and are the most common intentional thermal injury in children.

• Pathophysiological Consequences of Thermal Injury

- ✓ Burned/scalded tissue > Acute inflammatory response > Increased capillary permeability > Tissue fluid loss > Dehydration, electrolyte disturbance.
- ✓ If burn area exceeds 20% > Release of systemic inflammatory mediators > Acute lung injury or multiple organ dysfunction/failure.
- ✓ Burned skin provides no protection against infection, increasing the risk of sepsis in survivors.

• Exposure to Heat /Hyperthermia

Hyperthermia: a condition where the core body temperature is greater than 40°C (100°F), Its development is more likely in those who:

- Have taken substances (e.g. drugs, including cocaine and amphetamine) that elevate metabolic rate/heat production.
- Reduce sweating.
- Those with medical conditions (e.g. hyperthyroidism).

Autopsy Findings in 'Heat illness including '*heat stroke*', are non-specific but can include *pulmonary and/or cerebral oedema*, v*isceral surface petechiae* and features in keeping with *'shock'* and *multiple organ failure* in those who survive for a short period.

• Pathological investigation of bodies recovered from fires

The fire scene must be examined by specialist investigators with expertise in the interpretation:

*Causes and 'point of origin' or 'seat' of fires
*Use of accelerants, such as petrol .

Attendance at the scene by a pathologist is important and assists subsequent interpretation of post-mortem findings.

The position of the body when discovered is important because sometimes, when flames or smoke are advancing, the victim will retreat into a corner, a cupboard or other hiding place, or they may simply move to a place furthest away from the fire or to a doorway or window, all of which may indicate that the victim was probably still alive and capable of movement for some time after the start of the fire.

Figure 17.7 The finding of a body in a burnt-out car should always be treated with suspicion. Carboxyhaemoglobin levels may be low in rapid flash petrol fires leading to difficulties in assessing vitality at the time of the fire.

• Pathological investigation of bodies recovered from fires

The pathological investigator of bodies recovered from fires should attempt to:

1-Confirm the identity of the deceased:

- By visual identification (if heat did not cause major distortion of facial features)
- Personal effects as unique medical features as scars or some factors as tattoos.
- Dental examination and comparison of the dentition with available ante-mortem records or DNA analysis.
- Post-mortem radiography it helps in identification and in excluding projectiles such as bullets and shrapnel.

Figure 17.6 Charred body at the scene of a fire showing the 'pugilist attitude' and post-mortem skin splits on the chest. Extreme care must be taken to preserve the teeth in such cases, in order to assist identification of the deceased.

2-Determine when was the deceased died (before or after the fire)

- Determination of vitality maybe possible by finding soot in the airways, esophagus or stomach.
- Blood sample to assess the level of carboxyhemoglobin (commonly elevated in fire deaths above 50%)

3-Determine the cause of death

4-Determine the manner of death(or give an opin...)

Note: Cigarette smokers can tolerate high levels of carboxyhemoglobin up to 20%. While individuals with chronic heart or lung disease may not tolerate even low levels before succumbing in a fire.

• Death Occurring during a fire

Majority of deaths occur not due to direct effect of burns, but from

1- exposure to smoke, carbon monoxide,cyanide and cocktai of toxic combustion by-products.

2-inhalation of hot air/gases,

Leading to many mechanisms that causes deaths such as:

- Hypovolemic Shock following fluid loss.
- Infection or inhalation of combustion products → leading to acute lung injury.
- Renal failure or Blood clotting abnormalities.

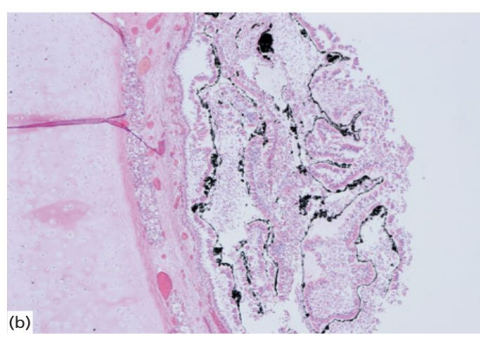


Figure 17.9 Pathological evidence of vitality at the time of the fire. Soot staining following inhalation of the combustion products of fire is clearly visible to the 'naked eye' in this trachea **(a)**, and such a finding can be confirmed under the microscope **(b)**.

Box 17.3 Examples of mechanisms of death in fires

- Interference with respiration (owing to a reduction in environmental oxygen and/or the production of carbon monoxide and other toxic substances)
- Inhalation heat injury leading to laryngospasm, bronchospasm and so-called 'vagal inhibition' and cardiac arrest
- Exposure to extreme heat and shock
- Trauma
- Exacerbation of pre-existing natural disease or burns

Manner of Death needs an appropriate medicolegal authority, an opinion from the forensic pathologist on the interpretation of artifacts related to exposure to fire:

- "Pugilist attitude" heat-related contraction of the muscle→ leading to flexion of the forearms,hands and thighs. *See the pic
- Post mortem splitting of fragile burn skin.
- Fire and heat related fractures
- Heat related extradural haemorrhage.

These artifacts may cause concern to police and may be misinterpreted as ante mortem violence.

How to differentiate it from trauma?

1- The lack of naked eye

2-Microscopic evidence of vitality such as erythema,blistering,tissue swelling,bruises or an acute inflammatory reaction.

Manner of death may be:

- Homicide
- Accident
- suicide

Cold Injury (Hypothermia)

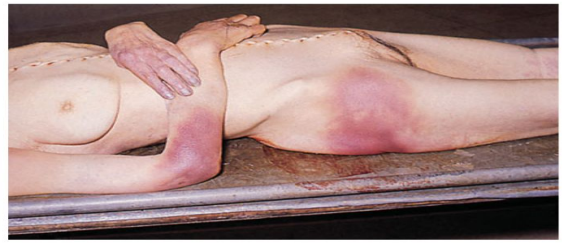


Figure 17.15 Pinkish discoloration over the large joints in fatal hypothermia.

- Hypothermia is common in healthy individuals due to cold environment, It's caused when a person's normal body temp. Drops to 35 C. It can be **triggered by:** Prolonged exposure to cold,Rain,wind, Sweat, inactivity or being in cold water.
- It can also be caused by a *Thyroid or pituitary dysfunction* or may be associated with *immobile* or *demented patients* or in conditions such as *pneumonia.* Which is characterized by depression and poor functioning of cardiovascular and nervous systems.
- Susceptible patients are generally elderly, children, traumatic.
- To prevent the any further heat loss the body will respond by processes including shivering (which keeps the major organs at normal temp.), restricted blood flow to the skin and releasing hormones to generate heat.
- Factors that might worsen hypothermia ***alcohol consumption*** by causing vasodilation and increased heat radiation and ***Cold water*** it causes cooling effects that is 20-30 times that of dry air, cold water survivors usually

accompanied by severe hypoxic brain injury.

- Hypothermia is diagnosed usually on basis of typical symptoms and environment can be divided depending on the core of temperature Into:

Mild cases (32-35)	Moderate (32-30)	Severe (<30)
- Shivering - Feeling cold - Lethargy - Cold,pale skin	- Violent, uncontrollable shivering - Cognitive impairment, confusion, loss of judgment - Loss of coordination - Memory loss drowsiness - Slurred speech,apathy - Slow , shallow pulse - Weak pulse	- Loss control of hands, feets and limbs - Shivering stops suddenly - Unconsciousness - Shallow or no breath,weak - Irregular or no pulse - Stiff muscle - Dilated pupils

Autopsy findings:

- In 50% of presumed hypothermia deaths, *Indistinct red or purple* skin discoloration over large joints and in areas of skin is found iit may be due to capillary damage and plasma leakage by microscope there will be no red blood cell extravasation which **distinguish it from bruises.**
- Hemorrhagic **gastric lesions** “wischnewsky spots” which represents mucosal necrosis with haematin formation due to disturbance of gastric circulation and exposure of hemoglobin to gastric acid. Usually those lesions are also found in deaths following sepsis, shock and alcohol misuse.
- Other gastrointestinal lesions including 1. haemorrhagic erosion and infarction in the small bowel due to RBC sludging and submucosal thrombosis. 2. Haemorrhagic pancreatitis with fat necrosis.
- **Frostbite** caused by severe cold injury to the extremities which varies in severity from erythema to infarction following microvascular injury and thrombosis.
- Lastly, hypothermia can cause behavioral abnormalities as paradoxical undressing or “hide and die syndrome”. In which they can be found in the scene of death.

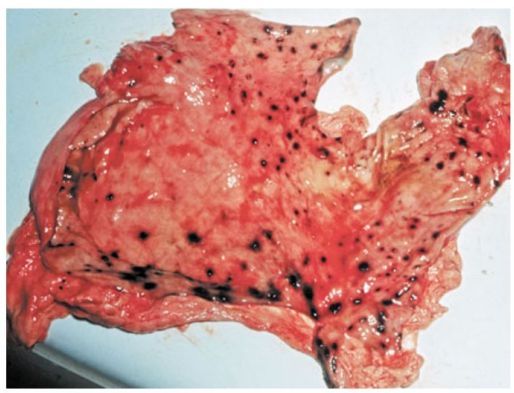


Figure 17.16 Numerous superficial haemorrhagic gastric erosions of the lining of the stomach in hypothermia. These are often called ‘Wischnewsky’ spots.

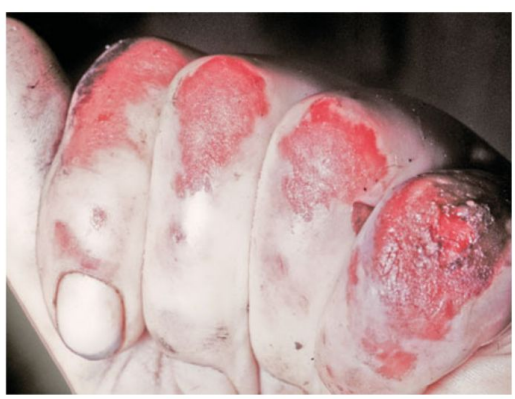


Figure 17.17 Frostbite of the knuckles.

Electrical Injury

Injury and death from the passage of an electric current through the body common in industrial and domestic circumstances. The essential factor in causing harm is an increase in the current (electron flow) and a drop in tissue resistance. Most of cases originate from the public power supply (110 V or 240V) rare death occur at less than 100V.

- The current enter through hand ,and exit via the other hand or the feet, crossing the thorax → causing shock because of the risks of cardiac arrest or respiratory paralysis. Even rarer the current has entered the head and caused primary brain-stem paralysis, which has resulted in failure of respiration.
- When a live meta conductor is gripped by the hand → causes pain and muscle twitching (10 mA) → Spasm of muscle which cannot voluntary releases the conductor,it occurs when the current is more than 30 mA → fatal ventricular fibrillation and cardiac arrhythmia when the current across the chest is 50mA or more.
- Thick dry skin → decrease the current due to an increase in tissue resistance while wet skin with decrease the resistance hence it will lead → markedly increased current.
- Victims will have different findings depending on the cause of death: Arrhythmia → look pale, peripheral respiratory paralysis→ usually looks cyanosed and in rare cases current may enter the head→ causing primary brain stem paralysis→ respiratory failure.
- The scene of a suspected electrical death should be reviewed to try and identify causative agents and ensure that no risk persists. Health and safety legislation may require that an electrical death (e.g.in the work setting) should be fully reviewed to prevent further electrical exposure.

Electrical lesion Findings:

1- Discrete multiple or single focal point of entry maybe obvious or inconspicuous usually it's a blister → which usually collapses after infliction → forming a raised rim with a concave center, the skin often peels off the blister leaving a red base.
2- Skin is pale or white(due to vasoconstriction) accompanied by a hyperaemic rim.
3-Sparks burn, where the is an air gap between metal and skin.A center nodule of fused keratin, brown or yellow surrounded by pale skin.
4- Both lesions blisters and sparks may be adjacent to each other.

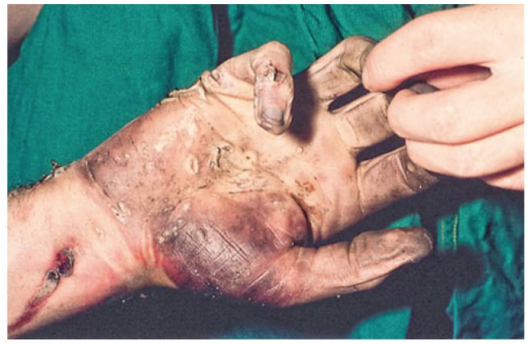


Figure 17.19 Multiple electrical marks/burns on the hand, associated with scorching and blistering.

5- In high voltage burns,multiple sparks may cause large areas of damage called→ “crocodile skin” .
6- Internally in fatal electrocution there is no characteristic finding and the gross pathological diagnosis relies upon the external appearance.

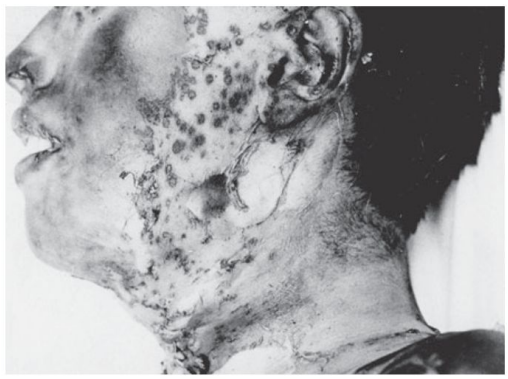


Figure 17.20 Multiple burns from high-voltage (multi-kilovolt) electrical supply lines. The ‘crocodile skin’ is caused by arcing of the current over a considerable distance.

Death from lightning

A Lightning strike from cloud to earth may produce million of amperes and phenomenal voltages if huge electrical forces is involved. Some lesions cause to those who struck directly or simply close to it, but others results from burns and yet others result from explosive effects of a compression wave of heated air leading to burst eardrums, pulmonary blast injury and muscle necrosis.

Autopsy findings:

- All kind of bizarre appearance can be found especially the partial or complete stripping of clothing.
- Severe burns, fractures and gross lacerations
- Magnetization or even fusion of metallic objects in the clothing.
- “Fern or branch-like” patterns on the skin called Lichtenberg figure.
- Red streaks following skin creases or seat-damped tracks.
- Some bodies are completely unmarked.

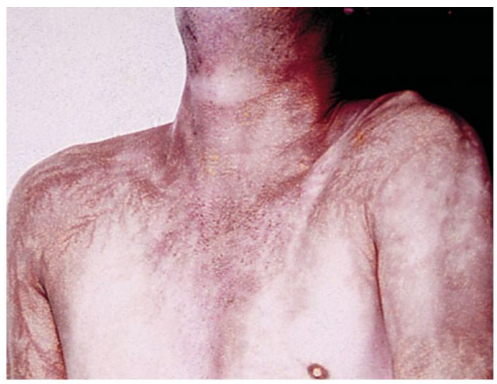


Figure 17.21 The ‘Lichtenberg figure’ and lightening fatalities. Note the fern-like branching pattern of skin discoloration on the chest.

Appendix 3

Diagnosing Poisoning Carbon Monoxide Health Protection Agency Guidelines

Diagnosing Poisoning-Carbon Monoxide.
Health Protection Agency Guidelines

Patient presenting with:
Headache, nausea/vomiting, drowsiness, dizziness, dyspnoea, chest pain
COULD THIS BE A CASE OF CO POISONING? 1

Ask the patient:
- Do you feel better away from your house or place of work?
- Is anybody else in your family or house experiencing the same symptoms as you?
- Have you recently had a heating or cooking appliance installed?
- Have all gas, coke/coal, wood or oil fired appliances, eg, cookers, fires, boilers at your home been serviced within the last year?
- Do you ever use your oven or gas stove for heating purposes as well as for cooking?
- Has there been any change in ventilation in your home recently, eg, fitting double glazing?
- Have you noticed any sooty stains around appliances or an increase in condensation?
- Does your work involve possible exposure to smoke, fumes or motor vehicle exhaust?
- Is your home detached, semi-detached, terraced, flat, bedsit or hostel? 2

You are suspicious: Could this be a case of CO poisoning?

You are confident: This is **NOT** a case of CO poisoning

Action to take: *GP - General Practice* ED - Emergency Department

1 **Test for CO**
GP - breath test for exhaled CO if device is available. (Note: Only indicates recent exposure; interpretation difficult in smokers. For interpretation of results see TOXBASE).
ED - heparinized venous blood sample for COHb estimation. For interpretation of results see TOXBASE and contact the National Poisons Information Service (NPIS).

2 **Management - Commence oxygen therapy**
GP - follow advice on TOXBASE; refer to ED if required.
ED - follow advice on TOXBASE. Contact NPIS for severe poisoning. (See CMO/CNO letter November 2008: www.dh.gov.uk/cmo).

3 **Protect your patient and others** - Contact your local Health Protection Unit (HPU). They will co-ordinate services for your patient and provide further CO guidance. Telephone gas, oil or solid fuel helpline (see Notes).

4 **DO NOT** allow patient home without a warning NOT to use the suspect appliances.

5 **Follow up**
GP - note that symptoms may persist or develop later.
ED - advise patient to see GP for follow-up. Note this advice in discharge letter. 3

If patient does not improve
- Contact NPIS for advice.
- Contact local HPU for advice.
- Reconsider diagnosis. 4

Box 1 Carbon monoxide is a mimic

Carbon monoxide poisoning is notorious for simulating other more common conditions, including flu-like illnesses, migraine, food-poisoning, tension headaches and depression.

Headache is the commonest symptom - think CO!

Box 2 Carbon monoxide sources are multiple

The source of CO may be in the home, in the car due to a leaking exhaust system, or in the workplace. Gas, oil, coal, coke and wood heating appliances are the commonest sources in the home.

Malfunctioning heating appliances may be indicated by there being yellow rather than blue flames (if it is not a 'decorative flame' fire) and by the deposition of soot on radiants or on the wall adjacent to the fire. There may be more than one source of carbon monoxide.

Poisoning is not limited to those from lower income groups. Carbon monoxide can leak into a semidetached or terraced house/flat from neighbouring premises. It is unlikely that a patient will know about servicing of appliances at his/her workplace, but it is worth asking about the sort of heating devices in use.

It is also worth asking: "Have you recently started to re-use heating appliances/boilers after the summer break/during an unexpected cold spell?"

Box 3 Stopping further exposure is essential

Preventing further exposure is the most important thing you can do. Breath tests and blood samples may prove inconclusive some hours after exposure has ended: CO levels in the blood decline with a half-life of about 6 hours. Note that a normal concentration of carboxyhaemoglobin (COHb) does not disprove CO poisoning unless the sample has been taken soon after exposure ended. A heparinized venous blood sample should however, always be taken and sent to the local Clinical Chemistry Laboratory for analysis. *For interpretation of results and detailed advice on CO poisoning see TOXBASE and call NPIS.*

If you strongly suspect CO poisoning do not wait for the result of the analysis before taking the other steps listed in Box 3. Contacting the gas (**0800 111999**), oil (**0845 6585080**) or solid fuel (**0845 6014406**) safety services is essential. Contacting your local HPU is essential as they will co-ordinate Environmental Health, Safety, Social and other services to protect your patient and others. Follow-up is important as further consequences of chronic exposure to CO may be delayed, or mild symptoms may persist, multiply or intensify. Recommend the purchase of an audible carbon monoxide alarm for installation in the home.