Ministry of Defence

Synopsis of Causation

Burns

Author: Dr Douglas Dingwall, Medical Author, Medical Text Edinburgh Validator: Mr Kenneth W Dunn, South Manchester University Hospital, Manchester

September 2008

Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

The Ministry of Defence accepts full responsibility for the contents of this synopsis, and for any claims for loss, damage or injury arising from the use of this synopsis by the Ministry of Defence.

1. Definition

- 1.1. Burning injury signifies damage to body tissues due to exposure to:
 - Heat
 - o scalds spill or immersion
 - o contact with hot objects or substances
 - o fire flash or flame
 - Electricity
 - Chemical agents
 - Radiation
 - Friction
 - Excessive cold
- 1.2. Most burn injuries are to the skin and may involve deeper tissues, but burns can also occur directly to the eyes and to respiratory and gastrointestinal tissues.

Classification

- 1.3. Skin burns are usually classified according to:
 - severity and depth of tissue damage
 - surface area of damage

Severity and depth of tissue damage

- 1.4. **Superficial burns** cause damage to the <u>epidermis</u>. It should be noted that <u>erythema</u> due to burn injury is not included in this definition as it is self limiting.
- 1.5. **Dermal burns** can be subdivided into two types:
 - superficial dermal burns that damage the epidermis and superficial (papillary) layer of the dermis
 - deep dermal burns that damage the epidermis and deep (reticular) layer of the dermis
- 1.6. **Full thickness burns** involve the full thickness of skin and destroy the epidermis and dermis including the <u>subdermal plexus</u> of vessels.
- 1.7. **Penetrating burns** cause full thickness destruction of the skin and <u>subcutaneous</u> tissue, and may involve the underlying <u>fascia</u>, muscle, bone, or other structures such as the brain.

1.8. In common usage, especially in North America, is a system of depth description that is not anatomical but describes the burn depth in terms of First, Second, Third and Fourth Degree. These loosely relate to the anatomical system described above, which is the internationally accepted system. The anatomical system avoids the confusion commonly seen with nondescriptive methods.

Surface area of damage

- 1.9. The extent of burn injury to the skin can be estimated using the "rule of nines". This allocates approximate percentages to the major anatomical areas relative to the total body surface area (TBSA). The adult head and neck is allotted 9% of TBSA, each upper limb 9%, each lower limb 18%, 18% each to the <u>anterior</u> and <u>posterior</u> surfaces of the trunk, and 1% to the <u>perineum</u> and urogenital structures. The area of an adult palm and fingers is approximately 1% of TBSA and the total of multiple scattered areas of burn injury can be estimated using the open hand size as a guide.
- 1.10. Another method of early estimation of the approximate proportion of the body surface that has been burned is serial halving.¹ Using this method, the assessor decides if the area burned is greater than half of TBSA, between a quarter and half, between an eighth and a quarter, or less than an eighth. This can be useful in decisions about initial management and does not depend on knowledge of the proportion of the major anatomical areas relative to the TBSA.
- 1.11. More accurate assessment can be made using a Lund and Browder chart that maps the percentage TBSA in a little more detail and includes some of the variations that occur with age, from birth to adulthood.
- 1.12. It can also be useful to subdivide the total burn injury area into partial thickness (superficial and superficial dermal) and full thickness (deep dermal and full thickness) percentages, as a differentiation of those areas that may heal spontaneously from those that will not.

2. Clinical features

The clinical features of burns due to heat are described in this section. Burns caused by electricity and friction usually have similar clinical features to those caused by heat, which is the main component of the damaging effect. However, variations in clinical features associated with these types of burns, or with burns due to chemical or radiation exposure or to cold injury, are described in the appropriate aetiology subsection below.

Burns of the skin

- 2.1. **Erythema** blanches on pressure, and can be associated with tenderness and pain. Blistering does not occur, and <u>two-point touch discrimination</u> is retained. Resolution of the inflammation occurs over several hours or days, with no scarring.
- 2.2. In **superficial burns** (first degree) the epidermis separates from the dermis to form thin walled blisters. There is copious fluid loss and extreme sensitivity. The wounds are red and blanch readily. Healing occurs usually in 7 to 10 days, with no scarring.
- 2.3. In dermal burns some of the skin tissue remains viable.
 - 2.3.1.Superficial dermal (second degree) burns appear pink moist and soft, and are exquisitely tender to touch. They often display some blisters but heal in approximately 2 to 3 weeks, usually with minimal scarring.
 - 2.3.2.Deep dermal (second degree) burns have a mixed red and white appearance. Blisters are thick-walled and often are ruptured. Two-point touch discrimination may be diminished, but pressure sensation is retained. Depending on the anatomical area and skin characteristics, these burns may heal in 3 to 6 weeks if no infection occurs, but scarring is common.
- 2.4. **Full thickness burns** have a white, black or deep red, leathery appearance with underlying clotted blood vessels, and the burned area is <u>anaesthetic</u>. Unless the burn wounds are small enough to heal by re-<u>epithelialisation</u> from the edges (in wounds less than 1 cm in diameter), skin grafting is usually necessary.
- 2.5. **Penetrating burns** involve destruction of all skin layers and damage to underlying tissues. Assessment of these requires considerable expertise.
- 2.6. Clinical assessment of superficial or full thickness burns is usually accurate, but experienced assessment of dermal burns has a 70% accuracy in predicting the need for skin grafting or not. New techniques, such as laser Doppler imaging,^{2,3} are being developed to help improve burn depth determination, particularly in this group.
- 2.7. Typically, about 24 hours after a third or fourth degree full thickness burn, three concentric areas of damage are thought to exist (Jackson's Model):
 - a central area of <u>coagulation</u> that consists of cells which are dead due to heat damage and absent blood flow
 - an intermediate area of stasis that will not blanch on pressure, suggesting stagnant blood

flow. However, over the next two days, blood flow may steadily cease totally, causing the area to become <u>necrotic</u>

- an outer area of <u>hyperaemia</u> that has increased blood flow. This usually heals within a week
- 2.8. In full thickness burns, desiccation and loss of skin elasticity over large areas of the chest or neck can cause restriction to breathing, and circumferential involvement of limbs can lead to serious <u>ischaemia</u>, due to a <u>tourniquet effect</u>.
- 2.9. If burns cause deep damage to skin, fibrosis in the healing areas may cause <u>contractures</u>. These can result in deformities, reduced range of movement and impaired function, depending on the areas affected.
- 2.10. Extensive burns can cause gastric and duodenal ulceration (Curling's ulcer).

Systemic effects

- 2.11. When skin is burned the stimulation of pain fibres and alteration of tissue proteins trigger a number of reactive inflammatory processes that cause water, solutes and proteins to escape from the circulatory system into the surrounding tissue areas. In slight burn injury this process remains localised, but if 10 to 15% TBSA is exceeded the loss of <u>intravascular</u> fluid can cause circulatory <u>shock</u>. This, in turn, can lead to renal failure.
- 2.12. The inflammatory processes may also reduce a patient's immunity to bacterial and fungal infections. This can be of particular significance as burn sites can readily become infected and essential treatment processes such as central venous catheterisation, urinary catheterisation and tracheostomy can expose the vulnerable patient to increased risk of infection.
- 2.13. Also potentially serious is the effect on gut <u>mucosa</u>, where <u>vascular</u> damage and ischaemic change can occur, leading to reduced motility, reduced absorption and alteration of gut bacteria that may then become an important source of infection. <u>Peritoneal oedema</u> can sometimes occur and may result in splinting of the diaphragm, with adverse effects on respiration.
- 2.14. Other possible systemic effects include haemolytic anaemia , weight loss and deep vein thrombosis.

Inhalation Burns

- 2.15. Inhalation of hot gases can damage the tissues of the nose, mouth, tongue, palate and larynx. Swelling may occur and restrict the airway.
- 2.16. Thermal damage to tissues below the larynx is rare, as tracheal and bronchial tissues can readily dissipate heat. However, steam is particularly dangerous and can damage the deeper airway structures, with loss of <u>ciliary</u> action and impaired air flow due to oedema.
- 2.17. Smoke particles may cause chemical <u>alveolitis</u> and noxious gases may cause systemic poisoning. Examples include carbon monoxide produced by incomplete combustion and hydrogen cyanide produced by burning plastics.

2.18. <u>Bronchoscopy</u> may sometimes be indicated to determine the extent of tissue damage within the respiratory tree.

Burns of the Eye

- 2.19. Most burns to the eye are due to chemical agents, with <u>thermal</u> burns occurring in less than a fifth of cases.
- 2.20. Chemicals often damage both eyes and there can be a serious threat to vision. Severe burns can cause:
 - glaucoma
 - corneal ulceration
 - scarring and perforation
 - cataract
 - damage to the conjunctiva and eyelids
- 2.21. Thermal burn damage to the eyelids is uncommon and contractures occur in some cases. Other complications include:
 - corneal ulceration
 - scarring and perforation
 - cataract
 - conjunctivitis

Gastrointestinal Burns

2.22. <u>Gastrointestinal</u> tissues dissipate heat very effectively, so thermal burns are rare. Most gastrointestinal burns are due to ingested corrosive chemicals.

3. Aetiology

As described in paragraph 1.1, burns may arise due to a number of different mechanisms, and these are discussed in this section.

Thermal burns

- 3.1. The amount of damage that an area of tissue can suffer as a result of a thermal burn is related to the rate of transfer of heat from the injuring agent. This depends on:
 - the agent's **heat capacity**, which is calculated from the <u>specific heat</u> of the material and the amount of the hot material. The specific heat of water (the most common cause of scalding burns) is higher than that of most gases, metals and solids. Copper heated to the same temperature as water will transfer about ten times less heat than the same amount of water
 - the agent's **temperature**. Water can reach temperatures of 100 degrees C, while cooking oils can be heated to 200 degrees C or more
 - the **duration of contact**. Immersion scalds are usually more damaging than water splash scalds, partly because of longer contact
 - the **transfer coefficient** of the agent and the tissue. This is defined as the amount of heat that passes through a unit area of contact between two materials, when the temperature difference between these materials is 1 degree C. Transfer of heat can occur by <u>conduction</u>, <u>convection</u> or radiation. Conduction is the usual method between solids and liquids and the skin, while gases transfer by convection. The transfer coefficient of steam is 30 times that for water, so steam causes a more severe thermal injury than heated water when the length of exposure is identical
 - the **conductivity** of the tissues in contact with the agent. Cornified layers on the soles and palms provide some insulation and areas with good blood flow can disseminate heat better than areas where the blood flow is poorer

Scalding

- 3.2. Hot liquids produce scalding by coming into contact with tissues either because of spillage, ingestion or inhalation, or because of immersion.
- 3.3. Spillage on clothed areas of the body can cause more damage than on exposed areas as clothing retains the fluid and thus the heat.

Water

3.4. Water has a high specific heat, can be heated to 100 degrees C and is a common cause of scalds.

- 3.5. Hot water causes much more damage from immersion than from spillage because of the higher heat capacity of the surrounding quantity of water and the usually longer duration of contact.
- 3.6. Spillage of water-based liquids such as soups or gravy can cause more severe damage, as the hot layer is thicker and may remain on the affected tissue for longer.

Steam

- 3.7. Steam can cause much more serious scald damage than water because of its much higher transfer coefficient.
- 3.8. In addition, steam under pressure can reach temperatures much higher than 100 degrees C and is thus extremely dangerous if released accidentally in boiler rooms, for example.⁴ In such circumstances, damage by direct contact and inhalation can be substantial.

Cooking oils

3.9. Oils used in cooking can be heated to 200 degrees C or more, are viscous and adhere to tissues readily. For these reasons scalds from cooking oil spillage can be very damaging.

Grease

3.10. Hot grease from engines can cause severe scalds for reasons similar to those for cooking oils.

Tar

- 3.11. Hot tar is used for road construction and for roofing. For road construction, tar has to be heated to 135 to 150 degrees C, while for roofing purposes, temperatures of 230 to 260 degrees C are needed. Hence, hot pitch or asphalt for roofing can cause substantially more scald injury than that used for road work.
- 3.12. Whatever the temperature, tar is very viscous and cools rapidly into a solid form that adheres firmly to scalded tissues and can be very difficult to remove.

Contact

- 3.13. Burns may result from contact with anything hot, including stoves, coals, cigarettes, burning plastic and materials heated in industrial processes. The severity of thermal damage depends mainly on the heat characteristics of the hot material and the duration of contact.
- 3.14. Phosphorus can cause both contact and chemical burns and is described under "Chemical burns" below.

Powder burns

- 3.15. The term "powder burns" has been used to describe marks on the skin in close proximity to gunshot wounds.
- 3.16. Some feel that the term is inaccurate as the marks are due to sooty deposits rather than to heat damage. Also, modern ammunition uses smokeless powder.

3.17. The term is occasionally used to describe burns sustained from exposure to ignited gunpowder, in fireworks for example.

Fire

3.18. In accidents involving fire, consumption of alcohol is not infrequently a contributory factor. Individuals at particular risk include those suffering from epilepsy and mental illness.

Flame burns

- 3.19. Flame burns result from house fires, accidental contact with burning flammable liquids, bonfires, automobile accidents and many other causes.
- 3.20. The severity of burns sustained from contact with flames varies according to the circumstances. In particular, if clothing or bedding has been alight, then full thickness burns are likely.

Flash burns

- 3.21. Flash burns are usually due to explosions of flammable gases or liquids and are characterised by intense heat lasting for a very short time.
- 3.22. The presence of clothing is often protective and exposed tissues facing the explosion are usually the most damaged.
- 3.23. Hot gases may rarely enter the airway and cause thermal damage to the upper areas of the respiratory tree.

Electrical burns

3.24. Electricity passing through body tissues generates heat in proportion to the voltage applied and the electrical resistance of the tissues. Electrical conduction can cause other substantial problems within the body, especially to the heart, brain and <u>peripheral nerves</u>.

Domestic electricity

3.25. Exposure to electric current from the relatively low-voltage domestic supply may cause localised burns at the entry or exit points. These can be deep and can involve nerves or tendons in the small area affected.

High-voltage electricity

- 3.26. Exposure to high-voltage electricity causes intense heat that can be dissipated more readily in the trunk than in the smaller-volume, <u>distal</u> areas of the limbs, so trunk damage may be less than damage to the fingers, hands, forearms, feet and lower legs. However, when the contact point is on the trunk itself, substantial internal organ damage can occur, even though external appearances may reveal little.
- 3.27. Full thickness injuries are often sustained if current arcs between tissues. This can occur between joints that are close to each other at the time of injury. Arcing may occur between the forearm and the arm if the elbow is flexed, and between the arm and the axilla if

the arm is <u>adducted</u>.

3.28. Severe electrical burns can cause disruption of muscle cells, releasing <u>myoglobin</u> into the circulation. This can lead to acute renal failure, so must be considered at an early stage of management.

Lightning

- 3.29. Lightning contact directly with the body is rare. Usually it strikes adjacent objects and causes burns when clothes are set alight, metal buckles, jewellery or coins are heated, or steam is formed by vapourisation of rainwater or sweat on the skin.
- 3.30. Rarely, lightning can cause distinctive burn patterns on the skin that are quite different from those caused by high-voltage, generated electricity.

Chemical burns

- 3.31. These usually result from industrial accidents with strong acids or alkalis, but can also occur in the home due to powerful cleaning agents or solvents.
- 3.32. The damaging action of the chemical tends to continue until it is removed, usually by flushing with water.

Acids

3.33. Acids tend to produce a thickened layer of damaged surface tissue that may reduce deeper trauma to some extent.

Alkalis

- 3.34. Alkalis interact with lipids and <u>saponify</u> tissues until they are neutralised or washed off.
- 3.35. The benign appearance of some chemical burns may belie their true severity and underestimation of the depth of damage can have serious consequences.

Acid or alkali burns to the eye

- 3.36. Acids damage the corneal epithelium and basement membrane but rarely damage deep endothelial cells, as neutralisation by tears and tissue proteins can be quite rapid.
- 3.37. Alkalis, on the other hand, penetrate quickly through the corneal tissue, destroying one layer after another. Anhydrous ammonia, for example, can reach the <u>anterior chamber</u> in less than 1 minute.

Cement

3.38. Cement reacts with water to form an alkali and, as hydrolysis continues, the alkalinity increases. Tissue contamination with cement has therefore to be quickly cleaned if serious burn damage is to be avoided.

Petroleum

3.39. Petroleum and other hydrocarbons dissolve lipids and damage cell membranes, causing severe injury when in contact with body tissues. They are also rapidly absorbed once outer layers are penetrated, and can have widespread systemic toxic effects.

Sulphur mustard

3.40. Sulphur mustard, also known as mustard gas, yperite, or hd, was formerly used in munitions and produces severe blistering of exposed tissues. If exposure is prolonged, additional systemic effects include damage to blood cells and to the intestinal mucosa.

Phosphorus

- 3.41. Phosphorus, mainly the white form, was also once used in munitions. When in contact with tissues in the presence of air, particles of it ignite spontaneously at temperatures above 30 degrees C, causing burn injuries that are both chemical and thermal in type.
- 3.42. Local corrosion occurs and absorption can produce substantial <u>electrolyte</u> imbalances, and damage to liver and kidneys. The systemic effects are unpredictable and sudden death can occur in some victims after relatively small areas of skin damage.⁵ Skin lesions are slow to heal.

Radiation burns

Ultraviolet radiation

3.43. Ultraviolet radiation may cause burning of exposed tissues. It can originate from sunlight, sun lamps or sunbeds, and from certain types of welding. The subject is detailed in the Synopsis *Ultraviolet radiation and the skin*.

Microwave radiation

- 3.44. Accidental exposure to high-dosage microwave radiation can cause substantial damage to tissues.
- 3.45. Risk situations include microwave welding of plastics in industry and domestic use of faulty microwave ovens.^{6,7}
- 3.46. Even very brief exposure to domestic microwave oven radiation, (from reaching in when the oven has failed to switch off when the door is opened), can cause temporary dysfunction of skin circulation; longer exposure can cause more serious damage.

Ionising radiation

- 3.47. Burns due to exposure to <u>ionising radiation</u> vary according to the particle type.
- 3.48. Penetrating radiation of sufficient dosage many affect most body tissues, but nonpenetrating radiation, such as that produced by beta particle contamination, may cause injury only to the skin and underlying tissues, as internal organ damage is less likely.
- 3.49. Accidental exposure may arise from:
 - radiotherapy equipment

- some types of specialised equipment used in industry
- accidents involving nuclear reactors in power stations or warships
- spillage of radioactive materials in transit or during use in industry, research or medical therapy
- nuclear weapons testing
- use of weapons incorporating depleted uranium
- 3.50. Skin damage can arise from radiation exposure of as little as 2 <u>Gray</u> (Gy), equivalent to 200 <u>rad</u>s.
- 3.51. Low dose exposure of up to 15 Gy can produce <u>prodromal</u> symptoms of warmth, itching or mild erythema, followed by a latent phase of several weeks during which there is no evidence of tissue damage. Thereafter a manifest illness phase ensues, with redness of the skin and possible dry <u>desquamation</u>. Recovery is usually complete.
- 3.52. Higher dose exposure of 15 to 40 Gy causes more serious problems. The prodromal phase may be painful and may feature dry desquamation as well as itching and erythema. A shorter latent phase may last only one to three weeks, followed by damage similar to a second-degree thermal burn with pain, susceptibility to infection and slow healing.
- 3.53. Dose exposure of 40 to 550 Gy produces early erythema with severe pain and rapid development of blisters that lead to wet desquamation, ulceration and possible necrosis. The damage may be deep, resembling a third-degree thermal burn with associated complications. Complete healing may take years.
- 3.54. Massive dose exposure of over 550 Gy produces severe damage usually resulting in necrosis requiring extensive surgical management.
- 3.55. **Depleted uranium** (DU), used in modern weaponry, emits beta particles. A piece of DU in direct contact with the skin delivers in one hour a dose equivalent to the annual dose from natural radiation⁸, although DU as dust delivers a much smaller dose.⁸ Burns are unlikely to occur if decontamination is carried out promptly.

Friction burns

- 3.56. Abrasion of tissues can produce heat and burns may result, depending on the severity and duration of the friction injury.
- 3.57. Such burns are common in motor-cycle accidents, when the rider slides at speed along the road surface.
- 3.58. Severe burn injury can be inflicted if an accident victim is trapped in contact with moving machinery.

Cold injury

3.59. Skin tissue is more resistant to cold than to heat, and inflammatory reaction to cold injury is less. The topic is examined in more detail in the Synopsis *Cold Injury*.

Liquified gas

3.60. In industry, some gases, such as nitrogen, are maintained in their liquid form at very low temperatures. Spillage accidents can cause deep burns if exposure is excessive.

Frostbite

3.61. In cold climates, on mountains or in refrigeration units, prolonged exposure to excessive cold may cause injury to exposed extremities. The severity of damage depends on the environmental temperature and the duration of exposure.

4. Prognosis

- 4.1. The outlook for burn victims is very dependent on effective management from the earliest stages.
- 4.2. Reduction of the immediate damage being caused may require:
 - removal of the victim from the source
 - cooling burned areas
 - washing off chemicals
 - removing contaminated clothing
 - rewarming cold-exposed areas
- 4.3. Resuscitation measures may be needed and full thickness burns may require early surgery to prevent constrictive effects.
- 4.4. Thereafter, treatment varies according to the severity and type of damage caused and the body areas involved.
 - 4.4.1. Wounds need dressings and may require considerable surgical management beforehand. Of particular importance is the excision of damaged tissue and early wound closure using grafting techniques.
 - 4.4.2. Secondary infection has to be controlled, fluid balance and nutrition maintained, and other systemic effects carefully managed.
 - 4.4.3. Inhalation or ingestion burns pose management problems of special difficulty.
 - 4.4.4. Physiotherapy may be important from the early stages.
 - 4.4.5. Help with psychological problems may be required.
 - 4.4.6. Burns caused by exposure to ionising radiation require particularly careful management.
- 4.5. Later still, reconstructive procedures may be needed to deal with scarring that is unsightly or constrictive, and with contractures that are causing reduced range of movement.
- 4.6. The prognosis for burns patients varies according to the severity of the damage caused and the age and <u>physiological</u> fitness of the victim.
- 4.7. Minor burns usually heal completely with minimal alteration of function or appearance.
- 4.8. More serious burns carry a greater risk and death can occur in the most severe cases.
 - 4.8.1. The risk of death is related more to the area burned than to the depth, TBSA of over 40% carrying the most risk.^{9,10}

- 4.8.2. The risk of death is further increased if inhalation injuries have also been sustained,^{10,11} or if the patient is elderly.^{9,10,12} However, these risk assessments vary from study to study due to problems with comparability of data recording, especially with regard to the presence of other injuries and the treatment regimes employed.
- 4.9. Excision of damaged tissues and employment of grafting techniques can substantially improve rates of healing and reduction of scarring from severe burn injuries. However, the persistence of altered appearance may require multiple plastic surgery procedures over time to achieve some improvement.
- 4.10. Surgery may also be performed to deal with contractures and reduced mobility. This is particularly important in burns of the hands where substantial loss of function may occur due to impairment of skin flexibility, skin sensitivity, joint mobility, muscle strength and coordination.¹³
- 4.11. In burns involving the eye, damage to the cornea can often be successfully treated by grafting, but total loss of sight in one or both eyes can occur in severe cases.¹⁴
- 4.12. Severe chemical burns of the oesophagus can cause stricture formation. This may require intermittent dilatation or even reconstructive surgery.¹⁵
- 4.13. Malignant tumours can develop in unhealing burn scar areas many years after the burning incident.¹⁶ However, employment of the modern techniques of burned tissue removal and early wound closure with grafts is likely to reduce the incidence of such tumours in the future.
- 4.14. Months or years after burns due to ionising radiation exposure of at least 10 Gy, further effects may include:
 - dermal <u>atrophy</u>, recurrent ulceration or necrosis
 - <u>telangiectasia</u>
 - <u>lymphatic network</u> damage and <u>lymphostasis</u>
 - <u>fibrosis</u>
 - <u>keratosis</u>
 - <u>vasculitis</u>
 - skin pigmentation
 - persistent pain
 - skin cancer
- 4.15. Severe burn injury can cause a wide variety of long-term psychological problems resulting from loss of function, altered appearance and the stresses of the incident.^{17,18,19}
- 4.16. The patient's perceptions and emotional responses may have more effect on the psychological prognosis than the actual burn injuries and their physical outcomes. These

effects are not directly proportional to the size of the injury.^{19,20}

5. Summary

- 5.1. Burn injury can directly damage the skin and underlying tissues, the eyes, the respiratory tree and the gastrointestinal tract.
- 5.2. Damage to tissues varies with the type of burn, the quantity and characteristics of the burning material, and with the duration of contact.
- 5.3. After severe burn injury, marked systemic effects also occur.
- 5.4. Burns can be caused by hot liquids, steam or gases, by flames or by contact with hot solids or electricity of sufficient voltage. They can also be caused by corrosive chemicals, by various sources of radiation, by friction or by excessive cold.
- 5.5. The prognosis for burn victims is dependent on the severity of the damage and the systemic effects. Burns of more than 40% TBSA carry a substantial risk of death.
- 5.6. Various long-term effects may result. They include altered appearance, and reduction of limb function and sight.
- 5.7. Psychological after-effects can be substantial in some individuals.

6. Related Synopses

Ultraviolet Radiation And The Skin

Cold Injury

adducted	Moved towards the body.
alveolitis	Inflammation of the microscopic air sacs where gas exchange between air and blood takes place in the lungs.
anaesthetic	Lacking sensation.
anatomical	Pertaining to the structure of the body.
Anterior	At the front.
Anterior chamber	Fluid-filled area of the eye between the cornea and the pupil.
Atrophy	Wasting, diminution in size.
bronchoscopy	An examination of the interior of the tracheo-bronchial tree - the major air-tube system of the lungs.
Cells	The individual units from which tissues of the body are formed.
Ciliary	Pertaining to the cilia – fine hair-like structures protruding from cells lining the bronchial tubes of the lungs. They facilitate removal of debris.
coagulation	Formation of a solid or semisolid mass.
conduction	Transfer of heat through direct contact.
contractures	Shortening of muscles and associated tissues.
convection	Transfer of heat by circulatory movement within a quantity of liquid or gas due to variations in its density as it heats up.
Corneal	Pertaining to the cornea - the clear layer on the front of the eye.
cornified	Hardened.
dermal	Pertaining to the dermis.

dermis	Skin layer, underlying the epidermis, that provides structure, flexibility, sensation and blood supply.
desquamation	The shedding of the outer layers of the skin, in scales or small sheets.
distal	Further from the point of attachment to the body.
electrolyte	Substance dissolved in tissue fluids.
epidermis	Outer layer of skin that functions as a barrier.
epithelialisation	Formation of the surface cellular lining.
erythema	Redness of the skin that can arise from a variety of causes, including heat and irritation.
fascia	Flat layer of fibrous tissue that separates other body tissues.
fibrosis	The formation of fibrous scar tissue.
gastrointestinal	Pertaining to the stomach and intestine.
graft	Transplant of tissue or other material to reconstitute missing tissue. Hence <i>grafting</i> .
Gray	Unit of absorbed dose of radiation received by at least 10 cm^2 of the skin.
hyperaemia	Engorgement with blood.
intravascular	Within blood vessels.
ionising radiation	Radiation which dislodges electrons from an atom.
ischaemia	Suffering from a deficiency in the arterial blood supply.
keratosis	Benign but precancerous skin condition usually associated with ultraviolet irradiation.
lymphatic network	Network of microscopic vessels that convey <i>lymph</i> (a fluid that bathes tissues) to the blood circulatory system.

lymphostasis	Obstruction of the normal flow of lymph.
mucosa	Membrane that lines a body cavity.
myoglobin	Protein found in muscles.
necrosis	Tissue changes indicative of cell death. Hence <i>necrotic</i> .
oedema	Abnormal build up of fluid between tissue cells.
peripheral nerves	Nerves outwith the brain and spinal cord.
perineum	Area of the body between the anus and the urogenital structures.
peritoneum	Smooth membrane which lines the cavity of the abdomen. Hence <i>peritoneal</i> .
physiological	Referring to the functioning of the body.
plexus	Network.
posterior	At the back.
prodromal	Pertaining to early or premonitory symptoms of disease.
rad	Unit of absorbed dose of radiation received by at least 1 cm^2 of the skin.
saponify	Convert into soap.
shock	Failure of the circulatory system to maintain adequate blood flow to vital organs.
specific heat	Amount of energy needed to raise the temperature of one gram of a pure substance by one degree C.
subcutaneous	Underlying the skin.
subdermal	Underlying the dermis.
telangiectasia	Permanent dilatation of small blood vessels creating small red lesions in the skin.
thermal	Pertaining to heat.
tourniquet effect	Tightening around the whole circumference of part of the body that results in impaired

blood flow.

two-point touch discrimination

vascular

vasculitis

Ability to feel the presence of two separate points of contact with the skin.

Pertaining to blood vessels.

Inflammation of blood vessels.

8. References

⁹ Vico P, Papillon J. Factors involved in burn mortality: a multivariate statistical approach based on discriminant analysis. Burns 1992;18(3):212-5.

¹⁰ Ryan CM, Schoenfeld DA, Thorpe WP et al. Objective estimates of the probability of death from burn injuries. N Engl J Med 1998;338(6):362-6.

¹¹ Suzuki M, Aikawa N, Kobayashi K, Higuchi R. Prognostic implications of inhalation injury in burn patients in Tokyo. Burns 2005;31(3):331-6.

¹² Rashid A, Khanna A, Gowar JP, Bull JP. Revised estimates of mortality from burns in the last 20 years at the Birmingham Burns Centre. Burns 2001;27(7):723-30.

¹³ van Zuijlen PP, Kreis RW, Vloemans AF et al. The prognostic factors regarding long-term functional outcome of full-thickness hand burns. Burns 1999;25(8):709-14.

¹⁴ Schrage NF, Langefeld S, Zschocke J et al. Eye burns: an emergency and continuing problem. Burns 2000;26(8):689-99.

¹⁵ Davids PH, Bartelsman JF, Tilanus HW, van Lanschot JJ. Consequences of caustic damage of the esophagus. Ned Tijdschr Geneeskd 2001;145(44):2105-8.

¹⁶ Kowal-Vern A, Criswell BK. Burn scar neoplasms: a literature review and statistical analysis. Burns 2005;31(4):403-13.

¹⁷ Van Loey NE, Van Son MJ. Psychopathology and psychological problems in patients with burn scars: epidemiology and management. Am J Clin Dermatol 2003;4(4):245-72.
¹⁸ Pallua N, Kunsebeck HW, Noah EM. Psychosocial adjustments 5 years after burn injury. Burns

¹⁸ Pallua N, Kunsebeck HW, Noah EM. Psychosocial adjustments 5 years after burn injury. Burns 2003;29(2):143-52.

¹⁹ Kildal M, Willebrand M, Andersson G et al. Personality characteristics and perceived health problems after burn injury. J Burn Care Rehabil 2004;25(3):228-35.

²⁰ Bryant RA. Predictors of post-traumatic stress disorder following burns injury. Burns 1996;22(2):89-92.

¹ Smith JJ, Malyon AD, Scerri GV, Burge TS. A comparison of serial halving and the rule of nines as a prehospital assessment tool in burns. Br J Plast Surg 2005; [Epub ahead of print].

² Atiles L, Mileski W, Spann K et al. Early assessment of pediatric burn wounds by laser Doppler flowmetry. J Burn Care Rehabil 1995;16:596–601.

³ Kloppenberg FW, Beerthuizen GI, ten Duis HJ. Perfusion of burn wounds assessed by laser Doppler imaging is related to burn depth and healing time. Burns 2001;27:359–63.

⁴ Tekin A, Namias N, O'Keeffe T. A burn mass casualty event due to boiler room explosion on a cruise ship: preparedness and outcomes. Am Surg 2005;71(3):210-5.

⁵ Barillo DJ, Cancio LC, Goodwin CW. Treatment of white phosphorus and other chemical burn injuries at one burn center over a 51-year period. Burns 2004;30(5):448-52.

⁶ Ciano M, Burlin JR, Pardoe R. High-frequency electromagnetic radiation injury to the upper extremity: local and systemic effects. Ann Plast Surg 1981;7(2):128-35.

⁷ Tintinalli JE, Krause G, Gursel E. Microwave radiation injury. Ann Emerg Med 1983;12(10):645-7.

⁸ Burkart W, Danesi PR, Hendry JH. Properties, use and health effects of depleted uranium. International Congress Series 2005;1276:133-6.