People.Health.Care.

Welcome to the course

Wound and Disease Management!

Module:
Burns – General Information

About this module…

Burns can range from minor, common wounds to severe and life-threatening injuries. This first module describes the different causes of burn injury and the local and systemic changes that happen when the skin is burned.

Upon completion of this course

- you will recognize the **different causes** of burns
- you will understand the **pathophysiology** of a burn

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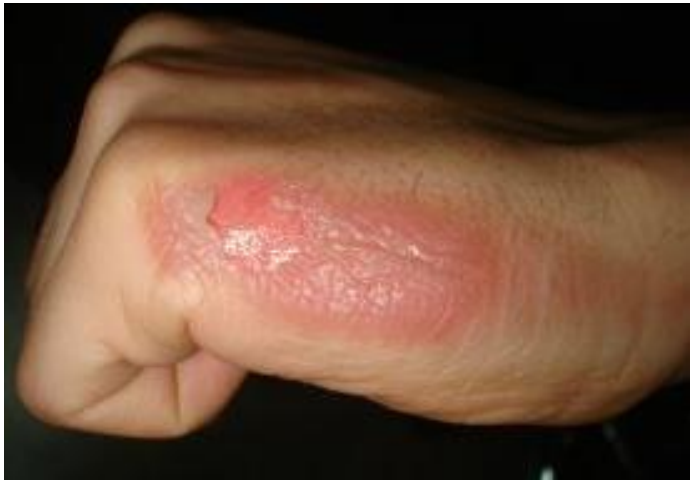
What is a burn?

A burn is defined as a **skin injury** caused by heat, electricity, chemicals, radiation or friction (NICE, 2015) that results in a **loss of skin integrity**.

This can range from superficial damage to the surface layer of the skin, the epidermis, through to severe damage involving all layers of the skin and the underlying structures, e.g. muscle and bone.



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Causes of burns

The cause of a burn can result in a **distinct pattern of injury**, which may require a specific approach to management. It is important to identify the cause of the injury so that the correct management can be implemented to optimize outcomes (Hettiaratch and Dziewulski, 2004; Stiles, 2015).

The causes of burns can be divided into the following **categories:**

- thermal
- electrical
- chemical
- radiation
- friction

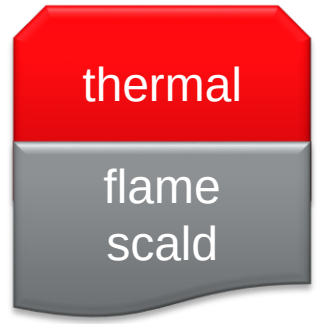
Causes of burns
Thermal

thermal

Thermal, or heat, injury can be caused by the following mechanisms:

- flame
- scald
- contact
- flash

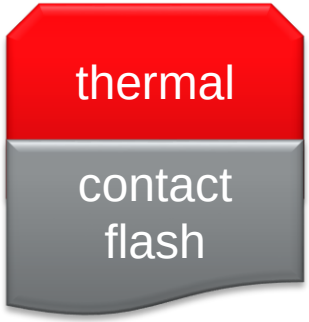
Causes of burns
Thermal



Flame burns are usually caused by **petrol, lighter fuel, or natural gas explosion** (Benson et al, 2006). They are often associated with inhalation injury and other concomitant trauma. Flame injuries are most commonly seen in adults of working age (Hettiaratchy and Dziewulski, 2004; International Best Practice Guidelines, 2014).

Scalds result from the spilling of, or immersion in**, hot liquids, or from steam**. Scalds are a common cause of burn injury in the elderly and in children, accounting for 70% of all burns seen in the latter group. In children who have pulled hot liquid onto themselves from a height, a ‘map of Africa’ shaped scald may be seen, affecting a large area of skin at the top of the body, with a smaller affected area beneath (International Best Practice Guidelines, 2014).

Causes of burns
Thermal



Contact burns are caused by the skin **touching an extremely hot object**, such as an iron or kettle, or by prolonged contact with a less hot surface, such as a radiator. The latter is the most common cause of contact injury, frequently seen in situations where the person is unable to break contact, for example, immobile people or those who have lost consciousness through drug/alcohol abuse, or epilepsy (Hettiaratchy and Dziewulski, 2004; Benson et al, 2006; International Best Practice Guidelines, 2014).

Flash burns are caused by an explosive ignition of a volatile substance, for example, accelerants used to light a fire, or resulting from gas explosions (Benson et al, 2006).

Causes of burns
Electrical

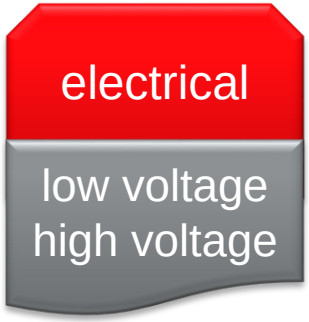
electrical

Electrical burns are caused by the heat energy generated as electricity travels through the body. There will be a burn at the entry and exit points, with the tissue in between potentially being damaged. Electrical burns vary in severity depending on the voltage involved. They can be classed as:

- Low voltage
- High voltage

Low, alternating or high voltage can interfere with cardiac rhythm, causing arrhythmia so cardiac monitoring may be required in this patient group (International Best Practice Guidelines, 2014).

Causes of burns
Electrical



Low voltage
Exposure to low voltage results in small, deep, contact burns at the points of entry and exit (Hettiaratchy and Dziewulski, 2004; International Best Practice Guidelines, 2014).

High voltage
Currents of more than 1000V can result in deep tissue injury, and possible limb loss. A large amount of soft and bony tissue necrosis will be seen (Hettiaratchy and Dziewulski, 2004; International Best Practice Guidelines, 2014).
Electrocution with more than 70,000V is usually fatal (Hettiaratchy and Dziewulski, 2004; International Best Practice Guidelines, 2014).
High-voltage flash burns may occur when high volt current passes by the body, as a result of the heat energy generated. Clothing may ignite, and result in more severe burns (Hettiaratchy and Dziewulski, 2004; International Best Practice Guidelines, 2014).

Causes of burns
Chemical

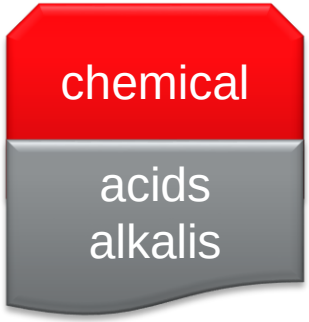
chemical

Chemical burns are commonly caused by industrial chemicals, but can also occur domestically. Chemical burns tend to be deep as the chemical agent will continue to corrode until it is completely removed from the skin (Benson et al, 2006). Some chemical agents may require specialist solutions to fully remove them (Benson et al, 2006). The most commonly encountered chemicals can be divided into:

- acids
- alkalis

Chemicals may be absorbed in toxic quantities, even when only in contact with a small area of skin. For example, hydrofluoric acid penetrates tissues deeply and can cause fatal systemic toxicity, even in small burns (Benson et al, 2006).

Causes of burns
Chemical



Acids such as sulphuric acid, nitric acid and phosphoric acid cause coagulative necrosis, denature proteins present in the skin and result in a painful burn (Hettiaratchy and Dziewulski, 2004).

Common alkali substances, such as bleaches, cleaning agents and cement, can cause deep burns, that can be more severe than those caused by acid (International Best Practice Guidelines, 2014). They result in liquefactive necrosis and can cause more damage than acids, as further injury occurs as exposure to alkali substances leads to cells dehydration and collagen and proteins in the skin are denatured. Often, the onset of pain can be delayed, leading to first aid being postponed and therefore allowing more tissue damage to occur through prolonged contact with the chemical (Benson et al, 2006).

Causes of burns
Radiation

radiation

Exposure to UV radiation from the sun, sunlamps, and tanning booths, can result in sun burn. This usually presents as superficial erythema that is very painful. Some blistering may also occur. With prolonged exposure, deeper changes within the structure of the skin may occur. High doses of ionizing radiation, usually for cancer treatment, can also result in radiation ‘burns’.

Causes of burns
Friction

friction

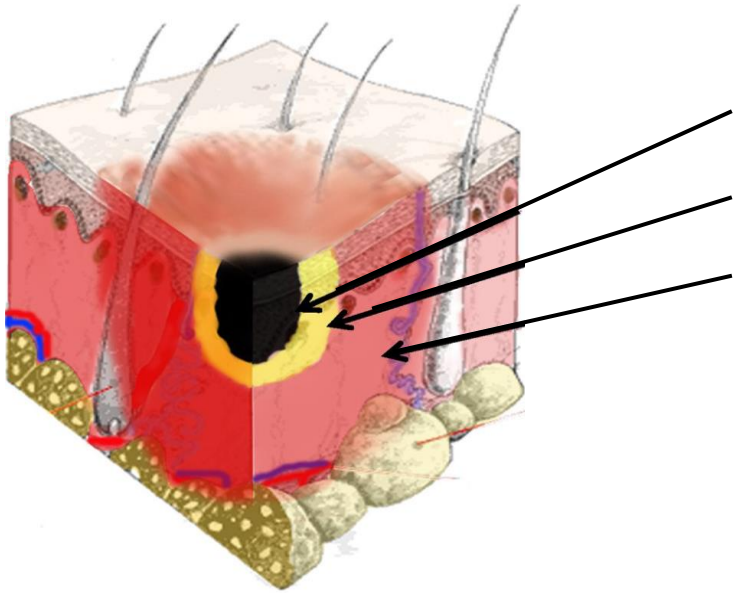
Friction burns occur when the skin is scraped off by contact with a hard object, e.g. a road surface and usually consist of both an abrasion and a thermal burn. They are commonly seen in road traffic accidents, sports injuries, or with rolling belts used in industrial and agricultural settings. The incidence is high but majority are minor burns, so they are not frequently seen in hospital settings (Agrawal et al, 2008).

Pathophysiology

When a burn occurs, it results in a **local response**, and when severe, a **systemic response**.
The local and systemic response is complex, and results in damage to both local tissue and tissue far removed from the site of injury. All damage is largely caused by the inflammatory mediators released upon injury.

Pathophysiology
Local response

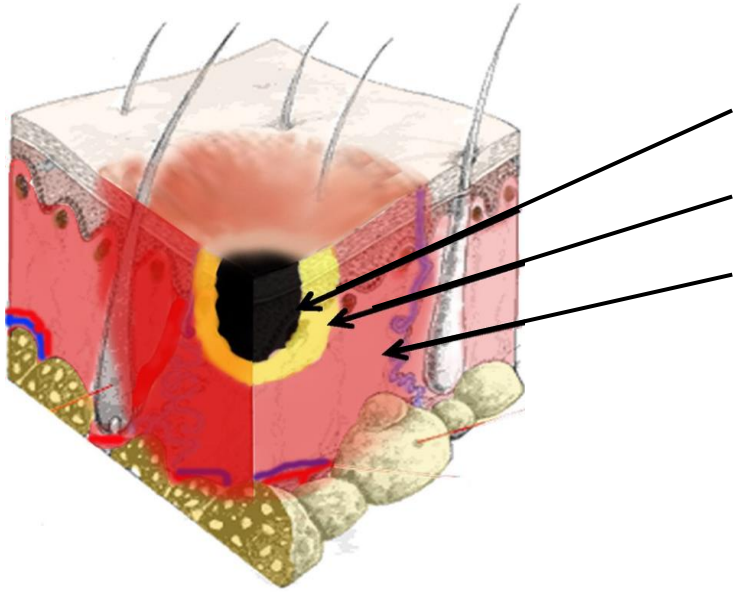
Within a thermal burn, the local response zone can be divided into **three specific areas** (Jackson, 1953):



- zone of coagulation
- zone of stasis
- zone of hyperemia

Source:
O'Neill (2020): Link

Pathophysiology
Local response



zone of coagulation

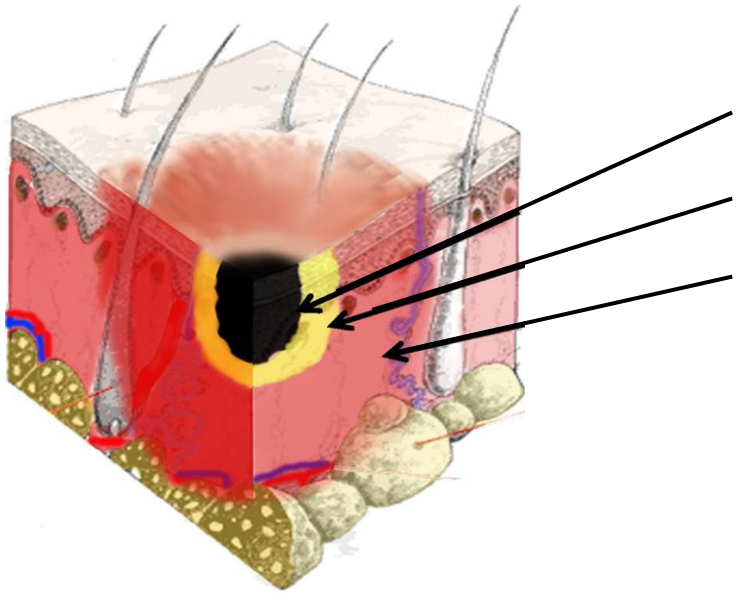
zone of stasis

zone of hyperaemia

Source:
O'Neill (2020): Link

The **zone of coagulation** is the most seriously damaged part of the burn. It is named as a result of the protein coagulation that occurs within the skin on burning, making the tissue necrotic or non-viable (Jackson, 1953). The tissue within this Zone must be removed, or debrided (Sterling et al, 2010), before healing can occur.

Pathophysiology
Local response



zone of coagulation

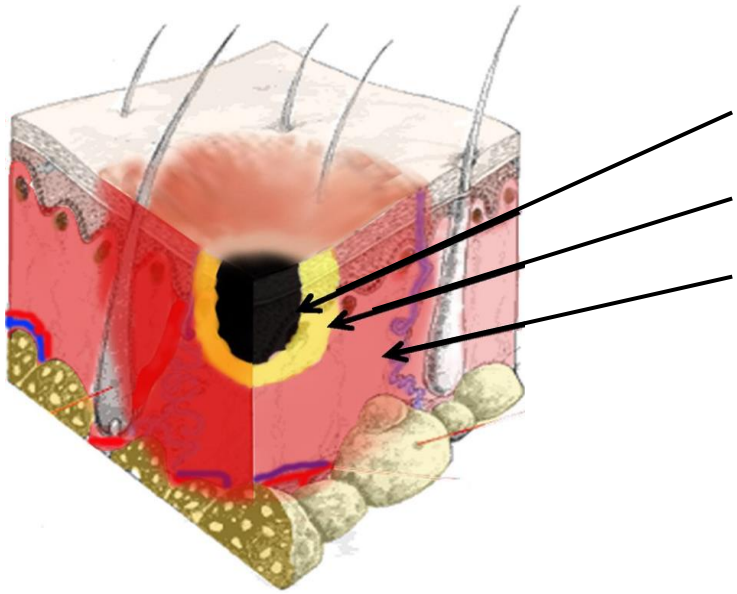
zone of stasis

zone of hyperaemia

Source:
O'Neill (2020): Link

The **zone of stasis** surrounds the central zone of coagulation, and consists of both viable and non-viable skin. The tissue in the zone of stasis undergoes both vasoconstriction and ischemia, which can lead to hypoperfusion, edema, and infection meaning that viable tissue in the zone of stasis may become necrotic. For this reason, wound reperfusion is a an important part of management to ensure healthy tissue can be saved. Systemic factors such as age, disease, edema, etc. may influence the survival of the zone of stasis. Therefore, this area is the one whose tissue survival is most influenced by management decisions.

Pathophysiology
Local response



zone of coagulation

zone of stasis

zone of hyperaemia

Source:
O'Neill (2020): Link

Hyperaemia occurs as a result of vasodilation caused by the release of inflammatory mediators on wounding. Hyperperfusion results, and the tissue is viable and will repair unless an adverse incident happens, such as infection or hypoperfusion (Hettiaratchy and Dziewulski, 2004).

Pathophysiology
Systemic response

In patients with large burns affecting more than 30% of the total body surface area (TBSA), a **systemic inflammatory response** may be triggered, which progresses with time, peaking 5-7 days after the burn occurs (Cakir and Yegen, 2004).

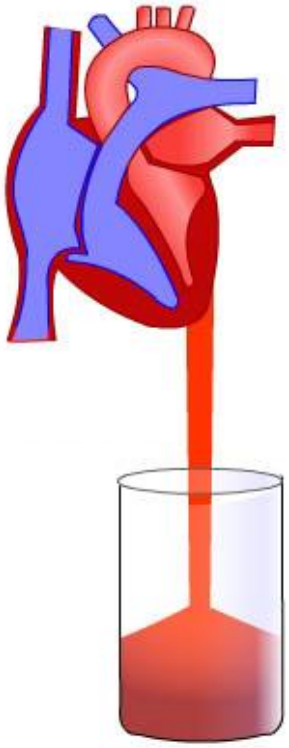
In this time, a number of changes may occur in the body that can lead to organ failure. These include:

- cardio-vascular
- respiratory
- renal
- metabolic
- immuno-logical

Pathophysiology
Systemic response

cardio-vascular

After burning, changes occur in the cardiovascular system that may result in hypovolemia. This decrease in blood plasma volume may be life threatening, and limiting blood volume loss is a management priority.
In the acute phase following initial injury, peripheral and splanchnic vasoconstriction results in decreased blood flow to the tissues and organs. The burn wound also triggers the release of inflammatory mediators that increase capillary permeability, enabling fluid to be released into the wound bed to promote wound cleansing and repair. This increased permeability, however, also promotes fluid and protein loss into the tissues, which reduces circulatory blood volume further (Hettiaratchy and Dziewulski, 2004; Cakir and Yegen, 2004).



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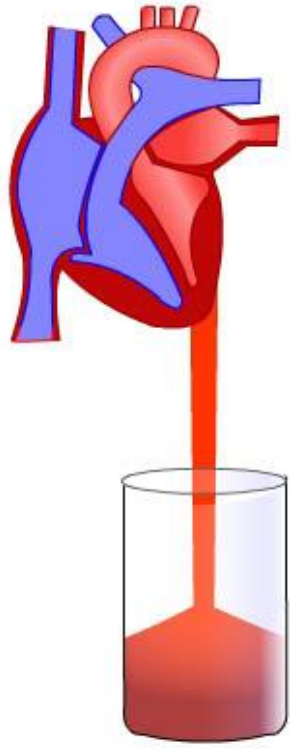
Pathophysiology
Systemic response

cardio-vascular

Within minutes of burn injury, cardiac output decreases in proportion to burn size. This lasts for approximately 48 hours and is followed by a hypermetabolic phase in which there is increased blood flow to the tissues and organs. In this phase, rapid edema formation occurs. Myocardial contractility decreases, and this, coupled with hypovolemia, results in cardiac instability (Cakir and Yegen, 2004).

Cardiac output is the volume of blood being pumped by the heart, by the left and right ventricle, per unit time. Cardiac output (CO) is the product of the heart rate (HR), i.e. the number of heartbeats per minute (bpm), and the stroke volume (SV), which is the volume of blood pumped from the ventricle per beat.
Values for cardiac output are usually denoted as L/min. For a healthy person weighing 70 kg, the cardiac output at rest averages about 5 L/min; assuming a heart rate of 70 beats/min, the stroke volume would be approximately 70 ml.



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Pathophysiology
Systemic response

respiratory

Respiratory failure is a major cause of death after burn injury. Inflammatory mediators can result in bronchoconstriction, and in severe burns, adult respiratory distress syndrome can occur (Hettiaratchy and Dziewulski, 2004).
Thermal injury results in significant lung changes, even without inhalation injury. Lung inflammation can persist for at least 5 days post injury. Inhalation injury is a primary cause of mortality for burns victims due to hypoxemia, and thermal and chemical effects which include pulmonary arterial hypertension, bronchial obstruction and increased airway resistance (Cakir and Yegen, 2004).

Pathophysiology
Systemic response

respiratory

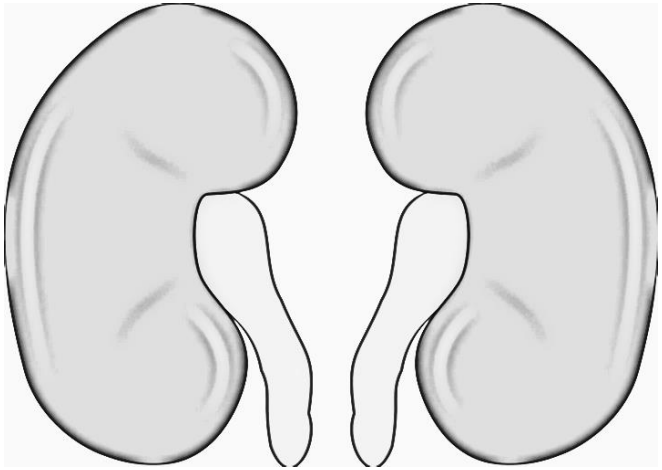
Hypoxia is a condition in which the body or a region of the body is deprived of adequate oxygen supply at the tissue level.
Hypoxia may be classified as either generalized, affecting the whole body, or local, affecting a region of the body.

Hypoxic effects on the body:

- on respiration: stimulate peripheral chemoreceptors and thus increase respiration
- increase in heart rate and blood pressure
- anorexia, nausea, vomiting
- the brain is affected in many ways: depressed mental activity, impaired judgment, drowsiness, disorientation, headache and coma
- reduced work capacity of the muscles
- decrease of kidney function

Pathophysiology
Systemic response

renal



The hypovolemia and reduced cardiac output that arises post burn causes a decrease in renal blood flow and glomerular filtration rate. If untreated, oliguria can progress to acute renal failure.
It is a complication in severely burned patients associated with high mortality rates (Cakir and Yegen, 2004).

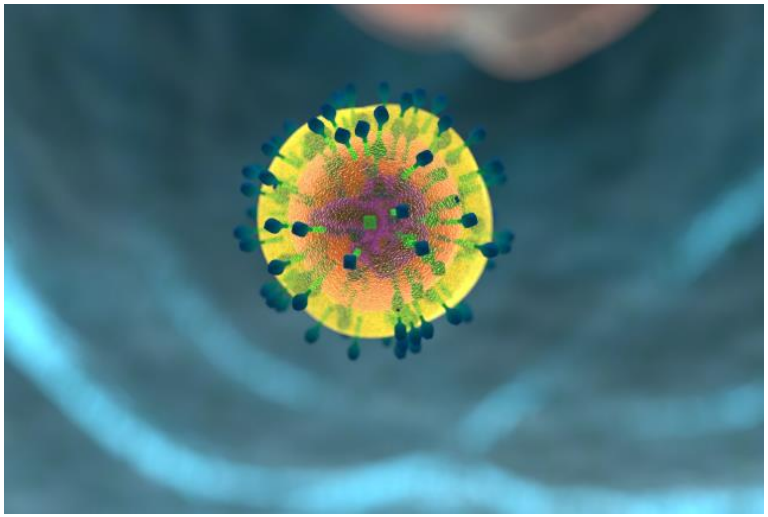
Pathophysiology
Systemic response

metabolic

The basal metabolic rate increases up to three times its original rate. This, with splanchnic hypoperfusion, necessitates early and aggressive enteral feeding to decrease catabolism and maintain gut integrity (Hettiaratchy and Dziewulski, 2004).

Pathophysiology
Systemic response

immuno-logical



Thermal injury causes immunosuppression in burns patients, predisposing them to sepsis and multiple organ failure, both of which are major causes of morbidity and mortality. Furthermore, damage to the epidermis makes the patient vulnerable to infection, while the presence of necrotic tissue and wound exudate at the injury site provides the ideal environment for micro-organism growth (Cakir and Yegen, 2004; Hettiaratchy and Dziewulski, 2004).

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Congratulations!

This slide concludes our presentation about general information on burns.

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