Southern African Journal of Anaesthesia and Analgesia. 2021;27(6 Suppl 1):S64-68
https://doi.org/10.36303/SAJAA.2021.27.6.S1.2701

South Afr J Anaesth Analg
ISSN 2220-1181 EISSN 2220-1173

Burns – head to toe of the ebb and flow

PM Brown

Department of Anaesthesiology, School of Clinical Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa
Corresponding author, email: *pduncan@tiscali.co.za*

Burns are the fourth most common type of trauma worldwide, with over 95 per cent of deaths attributed to burns occurring in low- to middle-income countries.[1] Anaesthetists are involved in the full spectrum of care for burn patients, from the initial resuscitation post injury through to reconstructive surgery and management of chronic pain that may result.

The World Health Organization (WHO) defines a burn as "an injury to the skin or other organic tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals".[2] Burns are classified according to severity through an assessment of depth, width and regions involved.[3] Significant burns result in distinct local pathophysiological changes, which then result in a systemic response that affects all major organ systems in the body.

The effect of burns on the cardiovascular system results in a distinct form of shock – burn shock.[3] The treatment for this is appropriately managed fluid resuscitation, especially within the initial 24 hours post injury. Fluid resuscitation that maintains tissue perfusion without being excessive has a survival benefit.[3] There is currently no robust data that dictates the type, amount or timing of the fluid used or the ideal endpoints of resuscitation.[4] Patients with major burns should be managed in specialised burn centres under the care of experienced clinicians for the best possible outcomes.

Keywords: burns, fluid resuscitation, burn shock

Burn injuries represent a significant global health burden.[2] Burns are the fourth most common type of trauma worldwide, with over 95 per cent of deaths attributable to burns occurring in low- to middle-income countries.[1] Anaesthetists are involved in the full spectrum of care for burn patients, from the initial resuscitation post injury to reconstructive surgery and management of chronic pain that may result. As the scope of this topic far exceeds what could possibly be covered here, the author aims to highlight the pathophysiology of severe burns, which ultimately informs the management thereof with specific reference to the initial fluid management.

Classification

Historically, burns have had issues regarding nomenclature. Various mechanisms of injury with distinct pathophysiology are all classified as burns, largely because of the similarity in appearance of the tissue damage that occurs.[5] The World Health Organization (WHO) defines a burn as "an injury to the skin or other organic tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals".[2] Burns are classified according to severity through the assessment of depth, width and regions involved. The classification and characteristics of burn wound depths are shown in Table I.[3]

The accurate assessment of the total body surface area (TBSA) of the wound remains challenging. The most commonly used methods include the Wallace Rule of Nines, the Palmar Method and the Lund-Browder chart.[6] All of these methods result in a subjective estimation of the affected area. The Lund-Browder chart is the most accurate, but it lacks clinical efficiency.[6] The Rule of Nines is clinically efficient but results in an overestimation.[6] The Palmar Method is useful for smaller wounds.

The various complex mechanisms that result from burn injuries have undergone extensive research. The driving forces

Table I: Classification and characteristics of burn wound depths

Burn depth	Layers involved	Characteristics	Healing
Superficial thickness (1st degree)	Epidermis	Painful, warm, erythematous, soft to touch, blanch	Desquamation ± 1 week later, no scaring
Superficial partial-thickness (2nd degree)	Epidermis and papillary dermis	Very painful, erythematous, soft, blistering, moist, blanch	2–4 weeks without scaring, pigment change may occur
Deep partial-thickness (2nd degree)	Epidermis, papillary and reticular dermis	Painful to pressure, blistering, wet (when blisters rupture) or waxy dry, firm, non-blanching, variable mottled appearance, mottled discolouration from white to red	> 4 weeks with scaring
Full-thickness (3rd degree)	Epidermis, dermis and hypodermis	Painless, white/brown, dry, firm and leathery, no blanching/blistering	Prolonged healing, scar formation, large areas need grafting

for the research have been to improve clinical therapies and thereby improving possible outcomes. Unfortunately, although the complexities have become clearer, this has not translated into improved treatment options with better outcomes.[7] An understanding of the pathophysiology associated with this unique trauma will, however, provide the basis for the knowledge required for the numerous challenges presented by its management.

Pathophysiology

The skin is the largest organ of the body.[6] It has numerous functions including acting as a barrier and regulating body temperature.

The initial burn injury results in three distinct zones which extend in all directions – as described by the Jackson's Burn Wound Model.[3,8] The central zone of coagulation is the area of maximal injury causing irreversible necrosis. The intermediate zone of stasis or ischaemia is characterised by decreased perfusion. This is a watershed area or penumbra where the tissue is potentially salvageable. The outer zone of hyperaemia is characterised by an increase in blood flow and inflammation. After the initial injury, these areas continue to evolve over the next 24–48 hours.[6]

The initial injury results in the local release and up-regulation of vasoactive and proinflammatory mediators (histamine, prostaglandins, bradykinin, serotonin, substance P, TNF-α, thromboxane and nitric oxide) from mast cells and macrophages.[7,9] This leads to local vasoconstriction and increased capillary permeability, which results in localised burn wound oedema. The initial injury causes microvascular dysfunction via thrombosis of damaged vessels and the release of proapoptotic factors (Bax, Bcl-xl and caspase-3).[7]

Numerous mechanisms result in the enhanced production and release of reactive oxygen species (ROS).[7,9] ROS cause local cellular membrane dysfunction and fuels the immune response.[3] With an injury involving a TBSA greater than 15–30% (major burn), the zone of hyperaemia extends to the rest of the body.[3,6,8] Systemic release of inflammatory mediators and cytokines (TNF-α and interleukins) lead to widespread increase in capillary permeability with associated extravasation of fluid and protein. The cellular membrane dysfunction also extends to uninjured tissue and leads to the disruption of the sodium-ATPase pump activity.[3] This results in an increase in intracellular sodium that worsens the hypovolaemia and cellular oedema.[9] The movement of fluid into the interstitium is exacerbated by an increased capillary hydrostatic pressure, decreased interstitial hydrostatic pressure, increased interstitial oncotic pressure and decreased capillary oncotic pressure.[9] The systemic effects induced by major burns affect all the major organ systems.[7]

Cardiovascular system

Cardiac dysfunction resulting from burns is complex and multifactorial. During the ebb (initial) phase, cardiac output is reduced. This results from the interplay of:

- hypovolaemia,
- increased systemic vascular resistance,
- decreased cardiac contractility (circulating humoral factors – TNF-α/ROS/endothelin 1/interleukins),
- decreased myocardial response to catecholamines (endogenous and exogenous due to decreased receptor affinity and a decrease in 2nd messenger production), and
- decreased coronary blood flow.[9,10]

Burn shock, which is the unique combination of distributive, hypovolaemic and cardiogenic shock, results from the effects of burns on the cardiovascular system.[3] The flow phase begins 24–96 hours post injury and is akin to the classic systemic inflammatory response. This hyperdynamic and hypermetabolic phase is characterised by an increased cardiac output, tachycardia, increased myocardial oxygen consumption and decreased systemic vascular resistance.[9,10] There is also direct cardiac damage, mediated by inflammatory mediators (macrophage migration inhibitor factor which is released by the injured skin and cardiac myocytes) and burn-induced oxidative stress which leads to mitochondrial damage.[7] Cardiac dysfunction results in impaired left ventricular systolic function, impaired isovolemic relaxation and a decrease in the diastolic compliance.[10] Left heart failure can ensue with increased filling pressure which exacerbates the extravasation of fluid into the lungs.[10] This leads to an increase in right ventricular workload with increased ventricular filling pressures and venous congestion, leading to right heart failure.[10]

Pulmonary system

Pulmonary complications from burns may result from the systemic complications of a major burn injury to the skin, direct inhalation injury or from burns to the chest wall that interfere with pulmonary function. The systemic effects of a distant burn lead to an increase in pulmonary vascular resistance and disruption of the pulmonary capillary alveolar membrane. There is increased extravascular lung water with impaired gaseous exchange.

Direct inhalation injury is classified in one of the following four ways:[9,11]

1. upper airway injury
2. lower airway injury
3. pulmonary parenchymal injury
4. systemic toxicity

Thermal injury and chemical irritants damage the upper airway. The injury results in the denaturing of proteins with activation of the complement system leading to histamine release.[11] Similar to the local effects of a burn wound to the skin, there is release of

multiple inflammatory mediators and ROS with resultant upper airway oedema.[7,11] This airway oedema evolves over the initial 24 hours post injury and can rapidly lead to airway obstruction.[7] Haemorrhage, ulceration and laryngospasm may result within the first 24 hours post injury.[7] The lower airways are protected from thermal injury (except from steam or blast injuries) by the upper airway which has efficient heat exchange mechanisms.[11] However, chemical irritants from the inhaled smoke result in injury to the lower airways. Surfactant functioning is immediately inactivated.[11] Chemical irritants are caustic and trigger a local inflammatory response.[11] There is a tenfold increase in the bronchial blood flow within minutes of the injury. This contributes to the increased capillary permeability and damage to the bronchial epithelium.[11] Exudate rich in protein, inflammatory cells and necrotic debris is produced by the damaged epithelium.[7] The inflammatory cells are chemotactic and cause migration of neutrophils through the glandular epithelium into the luminal airway.[7,11] The damaged epithelium inhibits the mucociliary apparatus of the trachea which allows the migration of upper airway material. This increases the risk for the development of airway obstruction and infection.[7] The extravasation of fluid leads to a decrease in the diffusion capacity resulting in a P/F (PaO2/FiO2) ratio of less than or equal to 200 after 24 hours post injury.[11] Initially, the secretions from the goblet cells are copious and foamy but in the hours and days post injury the secretions solidify, forming casts which leads to airway obstruction.[11] The chemical irritants can also result in an acute bronchospasm.[7] Lung parenchymal damage is delayed and will depend on the severity of the injury (duration of exposure) as well as the individual's unique response. Parenchymal damage is associated with exacerbation of the extravasation of fluid from the pulmonary vessels. There is an increase in the pulmonary microvascular pressure and a loss of hypoxic pulmonary vasoconstriction.[7] The combination of injury leads to ventilation–perfusion mismatch that worsens the hypoxaemia and leads to acute respiratory distress syndrome.

Systemic toxicity results from the inhalation of chemicals, mists, fumes, gases and cytotoxic liquids.[11] Common presentations include carbon monoxide and hydrogen cyanide poisoning.

Renal system

Acute kidney injury (AKI) occurs early during the ebb phase as a result of decreased renal perfusion during the resuscitation period.[3] Decreased renal perfusion is primarily attributable to burn shock. This can be further aggravated by an increase in intra-abdominal pressure, which can progress to intra-abdominal compartment syndrome.[7] The increased pressure is attributable to a decrease in abdominal wall compliance from circumferential burns in the region. This is associated with early multiple organ dysfunction and a higher mortality risk. If burn shock is managed appropriately through fluid resuscitation, early forms of AKI may be mitigated. Also, if there is significant injury to the soft tissue muscle, breakdown results in the release of myoglobin which is toxic to the kidney tubules and causes acute tubular necrosis. Late AKI is secondary to sepsis.[7] The mechanisms are multifactorial and poorly understood. The incidence of AKI in burn patients is as high as 30%.[3]

Neurological system

In the ebb phase, cellular hypoxia leads to an increase in intracranial pressure and cerebral oedema.[7] This hypoxia may present acutely with agitation, confusion, ataxic gait, abnormal posturing, decreased level of consciousness and seizures.[7]

Nerve regeneration begins in the recovery phase.[7] The sprouting and migration of nerve fibres is an imperfect process and this is the cause of the high rate (36%) of chronic pain post injury.[7]

Gastrointestinal system

After a major burn, blood flow to the bowel decreases by close to 60% of normal and remains low for up to 4 hours.[10] The gastrointestinal system is also susceptible to the effects of an increase in intra-abdominal pressure.

Management principles

Fluid management

Appropriate fluid resuscitation of major burn patients during the first 24 hours post injury has survival benefits.[3] The goal of fluid resuscitation is to maintain tissue perfusion in the setting of burn shock.[3] The most commonly used formula to estimate the amount of fluid required is the Parkland formula, which is as follows:

First 24 hours

Total amount of fluid (ml) = % TBSA burnt x patient weight x 4 with 50% administered in the first 8 hours post injury and the remainder in the next 16 hours.[3,10]

Next 24 hours

Colloids given as 20–60% of calculated plasma volume. No crystalloids. Glucose is added to maintain urinary output of 0.5–1.0 ml/kg/hr.[3]

The Modified Parkland formula is the same in the initial 24 hours, but in the subsequent 24 hours 5% albumin at 0.5 ml/kg/TBSA is administered.[3]

This formula provides only an estimation of the fluid requirement and is inaccurate for a number of reasons.[10] The formula contains two variables that are estimated – the TBSA affected (which should exclude areas with partial-thickness burns) and the patient's weight. The formula also does not take into account numerous other factors that determine fluid requirements, including the unique host response to the injury, mechanism of injury, presence of inhalation injury, comorbidities and the extremes of age.[10] The Parkland formula, however, remains the starting point for fluid resuscitation, with hourly assessments (dynamic period) in line with goal-directed resuscitation therapy. The amount of fluid needs to be sufficient to maintain

organ perfusion without being excessive. Excessive fluid, termed "fluid creep", is deleterious and results in injury to the glycocalyx, increased mortality, extension of the zone of necrosis, pulmonary oedema, AKI, impaired wound healing, acute respiratory distress syndrome (ARDS), and intra-abdominal and limb compartment syndrome.[4,8,10]

Type of fluid

The most commonly-used fluid in major burn patients is Lactated Ringers.[8] Although hypo-osmolar, it is effective at restoring extracellular sodium deficits. The lactate is metabolised to bicarbonate by the liver, assisting with the metabolic acidosis associated with severe burns.[4] In addition, Lactated Ringers is inexpensive, readily available, and has no specific storage requirements.[4] The use of a balance crystalloid as the primary fluid in resuscitation relates to the high volumes required. At these volumes, normal saline has a high incidence of hyperchloraemic acidosis and AKI.[8,10]

Hypertonic saline use in the resuscitative period gained popularity more than half a century ago.[4] The theory for its initial use was that it would act osmotically to draw fluid from the interstitium, thus limiting oedema formation and reducing the total fluid volume required for resuscitation. However, research has shown that the use of hypertonic saline is associated with hypernatremia, acute renal failure and increased mortality. [4]

The use of colloids, starches, albumin and plasma in burn resuscitation remains controversial.[4,10] During the initial 24 hours (resuscitative phase) after a burn injury, there is increased capillary permeability. Thus, the use of colloids, starches and plasma in this period may cause the passage of large molecules into the interstitial space exacerbating the oedema.[10] Within 5–8 hours after the initial injury, capillary permeability at distant sites returns to baseline.[4] Therefore, the use of albumin, plasma and synthetic colloids after this period could restore circulating volume with less fluid volume, which would translate to less tissue oedema.[4] The use of hydoxy-ethyl starch in critically-ill patients has been associated with a higher incidence of mortality and AKI, leading to judicious use.[10] Additionally, a Cochrane review showed that there is no mortality benefit when using colloids over crystalloids alone in critically-ill patients.[12] The use of albumin in the first 24 hours post injury has had conflicting results when the outcome of mortality was assessed.[10] The high cost, lack of optimal dosing, plasma concentration and patient selection, make it a less attractive option in this specific setting.[4] Further high-quality research is required to guide the use of albumin in burn injuries.

Expert opinion does advocate for the use of albumin under the following circumstances:[4]

- In a severe burn patient where the resuscitation requires high volumes of fluid to maintain tissue perfusion there is a dilution effect of the remaining albumin that can further exacerbate the extravasation of fluid. To prevent this, an albumin concentration above 12–15 g/l is required.
- When fluid resuscitation using the Parkland formula fails to maintain tissue perfusion, albumin administration may be considered.

Plasma is associated with transfusion-associated lung injury and allergic and anaphylactic reactions with no clear benefit.[4]

The mainstay of fluid resuscitation continues to be with Lactated Ringers.

Goal-directed fluid management

Regular assessments during the resuscitation period are required to optimise fluid management. The rate of fluid administration is titrated based on predetermined goals.[4] There is no consensus on the optimal goals used. However, common practice is to use the mean arterial blood pressure (> 65–70 mmHg) and urine output (0.5–1.0 ml/kg/min).[4,10] Adequate urinary output is thought to reflect sufficient renal perfusion and is, therefore, a surrogate of adequate cardiac output and tissue perfusion as a whole. These endpoints, although practical and easily measured, do not accurately reflect cardiac output or tissue perfusion.[10] There are numerous contributing factors to renal dysfunction in the severely burnt patient that may affect urine output.[10] Kidney injury may result from haemolysis, rhabdomyolysis, the inflammatory response or neurohormonal mediated intra-renal vasoconstriction.[10] Elevated lactate levels and the base deficit correlate with mortality in burn injuries.[4] Tracking these levels may offer insight into resuscitation efforts.

More invasive techniques to guide fluid resuscitation have been investigated; however, the literature is beset with small sample sizes and poor methodology.[8] Static measures of preload including intrathoracic blood volume and central venous pressure resulted in excessive fluid administration without benefit.[10] Lithium dilution cardiac output monitoring resulted in lower overall fluid administration without any benefit in organ dysfunction or mortality when compared to a control group which used dynamic preload variables using pulse contour analysis.[10] The pulmonary artery catheter has been used to guide resuscitation with improved survival; however, this technique is associated with numerous potential complications.[4] Transoesophageal echocardiography can add data to burn resuscitation through the assessment of cardiac parameters.[4] Additionally, real time responsiveness to resuscitation efforts can be assessed.[4] There is currently no data to validate transoesophageal echocardiography for this indication and it requires both a specialised skillset and the necessary equipment.[4]

Fluid management in the setting of severe burn patients remains challenging. A thorough understanding of the underlying pathophysiology guides management during the initial fluid resuscitation period and beyond.

Conflict of interest

The author declares no conflict of interest.

ORCID

PM Brown https://orcid.org/0000-0001-9502-9647

References

1. Peck M, Pressman MA. The correlation between burn mortality rates from fire and flame and economic status of countries. Burns J Int Soc Burn Inj. 2013;39(6):1054-9. https://doi.org/10.1016/j.burns.2013.04.010.
2. World Health Organization. Burns [Internet]. Geneva: World Health Organization. Available from: https://www.who.int/news-room/fact-sheets/detail/burns. Accessed 20 Aug 2021.
3. Haberal M, Abali AES, Karakayali H. Fluid management in major burn injuries. Indian J Plast Surg. 2010;43(Suppl):S29-36.
4. Gillenwater J, Garner W. Acute fluid management of large burns. Clin Plast Surg. 2017;44(3):495-503. https://doi.org/10.1016/j.cps.2017.02.008.
5. Lee RC, Astumian RD. The physicochemical basis for thermal and non-thermal 'burn' injuries. Burns. 1996;22(7):509-19. https://doi.org/10.1016/0305-4179(96)00051-4.
6. Kara YA. Burn etiology and pathogenesis. https://doi.org/10.5772/intechopen.71379.
7. Nielson CB, Duethman NC, Howard JM, Moncure M, Wood JG. Burns: pathophysiology of systemic complications and current management. J Burn Care Res. 2017;38(1):e469-81. https://doi.org/10.1097/BCR.0000000000000355.
8. Stapelberg F. Challenges in anaesthesia and pain management for burn injuries. Anaesth Intensive Care. 2020;48(2):101-13. https://doi.org/10.1177/0310057X20914908.
9. MacLennan N, Heimbach DM, Cullen BF. Anesthesia for major thermal injury. Anesthesiology. 1998;89(3):749-70. https://doi.org/10.1097/00000542-199809000-00027.
10. Soussi S, Dépret F, Benyamina M, Legrand M. Early hemodynamic management of critically ill burn patients. Anesthesiology. 2018;129(3):583-9. https://doi.org/10.1097/ALN.0000000000002314.
11. Jones SW, Williams FN, Cairns BA, Cartotto R. Inhalation injury. Clin Plast Surg. 2017;44(3):505-11. https://doi.org/10.1016/j.cps.2017.02.009.
12. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev. 2013;2:CD000567. https://doi.org/10.1002/14651858.CD000567.pub6.