Burns injury Part II: ICU Management of the burns. The injured patient

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Burns injury Part II: ICU Management of the burns of injured patient

2026 Update 2026

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Update Info

Intended Learning Outcomes

Burns injury Part II: ICU Management of the burns of injured patient

1. Describe the principles of fluid management following burns injury.
2. Describe the challenges and management of temperature control following burns injury
3. Outline the metabolic changes following major burns injury and the interventions used to attenuate these responses.
4. Describe the management of pain and sedation used for patients with burns injury in ICU.

eModule Information

Relevant Competencies from CoBaTrICE

Burns injury Part II: ICU Management of the burns of injured patient

- **1.1** Adopts a structured and timely approach to the recognition, assessment and stabilisation of the acutely ill patient with disordered physiology.
- **1.6** Assesses and provides initial management of the patient with burns.
- **2.1** Obtains a history and performs an accurate clinical examination.
- **2.2** Undertakes timely and appropriate investigations.
- **2.6** Obtains and interprets results from blood gas samples
- **2.7** Interprets chest x-rays
- **2.9** Monitors and responds to trends in physiological variables
- **3.1** Manages the care of the critically ill patient with specific acute medical conditions.
- **3.3** Recognises and manages the patient with circulatory failure.
- **4.2** Manages antimicrobial drug therapy
- **4.4** Uses fluids and vasoactive / inotropic drugs to support circulation.
- **4.9** Co-ordinates and provides nutritional assessment and support.
- **7.1** Identifies and attempts to minimise the physical and psychosocial consequences of critical illness for patients and families
- **7.2** Manages the assessment, prevention and treatment of pain and delirium

1. Executive Summary

As the largest organ in the body, the skin is vitally important to maintaining homeostasis. Skin loss due to burn injury therefore gives rise to a host of systemic sequelae. In this module, we discuss the acute physiological disturbances that follow burn injury and require judicious organ support within the intensive care unit. We also consider common complications, which occur across multiple systems and timeframes of hours to years.

2. Organ dysfunction and support

2. 1. Cardiovascular support

2. 1. 1. Fluid optimisation

Appropriate resuscitation with fluids is critical in the first 24 to 48 hours after a burn injury. Under-resuscitation may lead to impaired tissue perfusion, end organ damage and extension of burn depth (Guilabert et al. 2016). However, administering excessive fluids is also harmful; risks include electrolyte disturbances (such as hyponatraemia) and marked tissue oedema, which predisposes to gas exchange abnormalities, compartment syndromes and poor healing following surgical intervention.

The Parkland formula remains the most commonly used tool to calculate fluid requirements. Concerns have been raised that it may overestimate the volume needed, prompting bodies such as the American Burn Association to recommend less than 4 ml kg^{-1} $TBSA^{-1}$. However, it is unlikely that a single calculation will provide an accurate answer for the individual patient. Instead, fluid resuscitation should be commenced (we recommend using 4 ml kg^{-1} $TBSA^{-1}$ for initial calculations) and then amended following regular clinical reviews. The regular alteration in infusion rate based on individual patient physiology allows fluid volume to be escalated or de-escalated as appropriate, opposed to rigidly adhering to any one formula (McGovern, Puxty and Paton. 2022).

The most common and easy method of ensuring appropriate fluid resuscitation is by targeting an hourly urine output of 0.5-1 ml kg^{-1} ideal body weight. Failure to meet this target should prompt reassessment and adjustment of fluid delivery (Figure 1). However, an inadequate urine output is not always caused by volume depletion. Renal failure from acute tubular necrosis or rhabdomyolysis can result in oliguria, as can increased anti-diuretic hormone release in response to injury. Other causes such as vasoplegia, low cardiac output and abdominal compartment syndrome should also be considered. Conversely, urine volumes in excess of targeted values should prompt a reduction in the volumes of fluids given, mitigating against the phenomenon of ‘fluid creep’, whereby more than anticipated fluid is given.

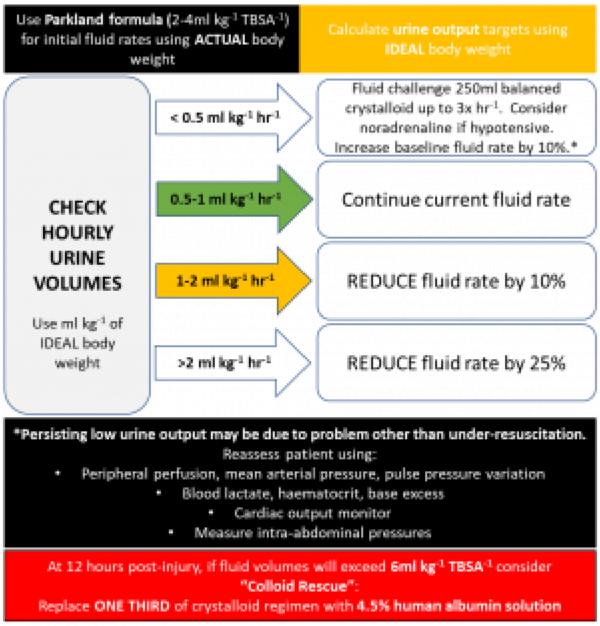

Figure 1: Adapted with permission from the COBIS (Care of Burns in Scotland) adult burn fluid resuscitation protocol.

Peripheral perfusion, serum lactate, acid base balance and haematocrit should also be used to help guide fluid therapy. The additional use of cardiac output monitoring to guide fluid delivery in patients with major burns has demonstrated improvements in cardiac output, oxygen delivery and organ dysfunction, but no mortality benefit has yet been identified.

Balanced crystalloids such as Hartmann's solution are the mainstay of fluid resuscitation in major burn injuries. Their use, when compared with 0.9% saline, has been demonstrated to reduce the incidence of significant electrolyte disturbances such as hyperchloraemic metabolic acidosis (Guilabert et al. 2016). The use of colloids such as human albumin solution in combination with crystalloids may reduce overall fluid volume requirements, mitigate against 'fluid creep' and lessen increases in intraabdominal pressures compared with crystalloids alone (Comish et al. 2021). We recommend the addition of 4.5% human albumin solution if fluid resuscitation volumes in the first 24 hours are projected at 12 hours to exceed 6 ml kg^{-1} $\%TBSA^{-1}$. This technique of 'colloid rescue' should be continued until 48 h after the burn injury.

2. 1. 2. Vasopressors

Hypotension following burn injury is often multifactorial in nature, potentially reflecting:

- Inadequate fluid resuscitation
- Peripheral vasodilation in the context of systemic inflammation
- Myocardial depression
- Diminution of sympathetic drive by drugs (e.g., propofol, benzodiazepines, opioids)

While fluid resuscitation is the keystone of initial cardiovascular support, many patients will require vasoactive agents during acute burn shock resuscitation. The optimal blood pressure target will vary between patients, particularly if other traumatic injuries have been sustained. However, mean arterial pressure should be sufficient to maintain renal perfusion. Vasopressors such as noradrenaline and vasopressin may be required to facilitate adequate blood pressure and limit the problems associated with over-resuscitation. Titration of fluid versus vasopressor support needs to be individualised for each patient with decisions based on physiological response.

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2. 2. Respiratory support

Respiratory failure is a common sequela of major burns injury. Inhalation injury can occur following inhalation of heat, smoke or chemical irritants (further details in Inhalation injury module). These patients are at risk of developing ARDS which is further compounded by the large volumes of fluid ad-ministered during resuscitation. Finally, the nature of the burn and dressings can impact upon ventilation in patients with burns injury.

2. 2. 1. Inhalation injury and the role of bronchoscopy

Fibreoptic bronchoscopy is considered the gold standard for diagnosing and assessing the severity of tracheobronchial injury in burns patients. This generally reflects chemical injury inflicted by noxious components of smoke in closed space fires. It is characterised by mucosal inflammation, hypersecretion and bronchial obstruction (Gill and Martin. 2015). The spectrum of inhalation injury will be dealt with in a sep-arate learning module.

The Abbreviated Injury Score (AIS) for gradation of inhalation injury can be used to assess severity of injury based on bronchoscopic appearances (Table 1 and Figure 2). The grading of inhalation injury has been demonstrated to predict outcomes, although this has not been consistent, possibly due to interobserver variability in grading. However, there is a strong association between the presence of any inhalation injury at bronchoscopy and increased duration of invasive mechanical ventilation, ICU length of stay and mortality. The revised Baux score equates the prognostic impact of inhalational injury to that of a burn affecting an addetional 17% TBSA.

Table 1: AIS gradation of inhalation injury

Grade	Incubation period
Grade 0: No injury	Absence of carbonaceous deposits, erythema, oedema, bronchorrhoea or obstruction
Grade 1: Mild injury	Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi (any or combination)
Grade 2: Moderate injury	Moderate degree of erythema, carbonaceous deposits, bronchorrhoea with or without compromise of the bronchi (any or combination)
Grade 3: Severe injury	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhoea, bronchial obstruction (any or combination)
Grade 4: Massive injury	Evidence of mucosal sloughing, necrosis, endoluminal obliteration (any or combination)

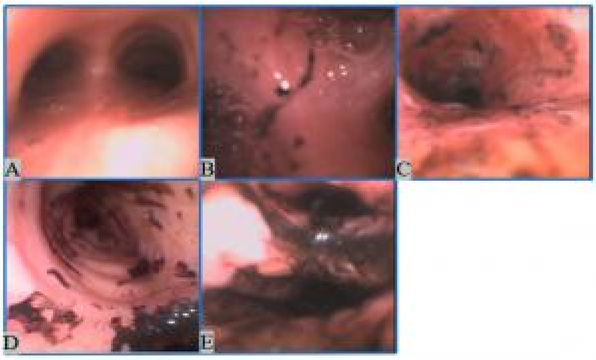

Figure 2: Examples of grading of inhalation injury using the abbreviated injury score (AIS). (A)—no injury; (B)—mild injury; (C)—moderate injury; (D)—severe injury; (E)—massive injury. Reproduced from Li et al under the terms of the Creative Commons Attribution NonCommercial License 4.0.

Preliminary studies suggest that bronchoscopy improves the diagnostic accuracy of inhalation injury compared to clinical history and examination alone. While the association between diagnosing inhalation injury on bronchoscopy and outcomes has been well documented, there is minimal evidence for bronchoscopic intervention such as therapeutic bronchoalveolar lavage or nebulised therapy.

While more research is needed to further establish therapeutic benefit of bronchoscopy in inhalation injury, it can be a valuable tool in diagnosing and managing respiratory complications in the burn injured patient e.g. with lobar collapse due to proximal bronchial obstruction. The decision to perform a bronchoscopy should therefore be made on a case-by-case basis by a suitably experienced medical professional.

2. 2. 2. ARDS

Lung parenchymal injury may occur in burns patients following inhalation injury or as part of a systemic inflammatory response. Acute Respiratory Distress Syndrome (ARDS) is characterised by non-cardiogenic pulmonary oedema and lung inflammation driven by the profound inflammatory response initiated by the burn injury (whether pulmonary or extra-pulmonary). Large volume fluid resuscitation may compound this situation with worsening extravascular lung water leading to further deterioration in gas exchange. The severity of ARDS is given by the ratio of the partial pressure of oxygen in arterial blood to the inspired oxygen concentration.

There are few specific treatment options other than supportive therapy for this heterogeneous condition that can occur in almost any critical illness. Supportive therapy involves limiting additional lung damage with lung protective ventilation. Prone positioning should be considered where possible although close liaison with the burns surgical team will be required as positioning the patient onto grafted skin may compromise graft take.

2. 2. 3. Ventilatory techniques and ECMO

The supportive measures employed in intensive care to manage ARDS include lung protective ventilation, conservative fluid management, neuromuscular blockade and prone positioning. The large volumes of fluid required in the resuscitation of burn injuries and difficulty in prone positioning due to the anatomical distribution of burns can make some of these interventions extremely challenging. Specific medical therapies such as corticosteroids may have benefit in the management of ARDS, but their use in the context of burn injury and potential risk of infectious complications mean that a careful risk/benefit assessment should be made before prescribing them.

Extracorporeal membrane oxygenation (ECMO) has been utilised by some centres in the context of burn injury with case reports of success in managing ARDS, both in the presence of inhalation injury and without (Fouché et al. 2023). The effectiveness of ECMO to improve mortality in ARDS of any cause has yet to be demonstrated in randomised controlled trials and the challenges of anticoagulation in the context of major skin loss add another layer of complexity in delivering this therapy to patients with major burns. However, ECMO may be regarded as a therapeutic option for the management of refractory hypoxaemia in major burn injuries or inhalation injury when other measures noted above have failed.

References

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- Fouché TW, Vrouwe SQ, Gottlieb LJ, Song TH, Mehta S, Tung A, Estimé SR. , Extracorporeal membrane oxygenation utilization in burn patients with severe acute respiratory distress syndrome., 2023, PMID:36357253

2. 3. Acute kidney injury

Acute kidney injury (AKI), as defined and categorised by a reduction in urine output and / or rise in serum creatinine level, can occur for a variety of reasons in burn injured patients. Egress of fluids and electrolytes due to the loss of skin integrity can result in hypovolaemia and therefore inadequate perfusion of the kidneys. Additionally, AKI can result from a combination of vasoplegia, low cardiac output and acute tubular necrosis. An increase in anti-diuretic hormone release as a normal physiological response to injury can also result in oliguria.

Other more specific causes of AKI include rhabdomyolysis when muscle tissue is damaged either directly by a burn (e.g. electrical burns) or as a consequence of a compartment syndrome. This results in the release of the intracellular contents of skeletal muscle, namely myoglobin, into the bloodstream. Myoglobin accumulates in the renal tubules, forming casts and obstructing flow through the renal tubules and collecting system.

Abdominal compartment syndrome can also occur, most commonly due to the significant tissue oedema following fluid resuscitation. Raised intra-abdominal pressures can impair first venous drainage from, and latterly arterial blood supply to the kidneys, causing reduced glomerular filtration and tubular perfusion.

In Chapter References

(Powell-Tuck et al. 2009; Li et al. 2022)

References

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- Li Y, Pang AW, Zeitouni J, Zeitouni F, Mateja K, Griswold JA, Chong JW., Inhalation Injury Grading Using Transfer Learning Based on Bronchoscopy Images and Mechanical Ventilation Period, 2022, PMID:36502127

3. Compartment syndromes

3. 1. Limb compartment syndromes

A compartment syndrome occurs when the pressure within a compartment increases to the point where blood flow is restricted causing distal tissue ischaemia and eventual necrosis. In burn injuries this most commonly occurs in limbs and extremities due to circumferential deep burns forming an inelastic eschar that prevents compartmental expansion as swelling occurs. This can also occur with profound oedema of deeper tissues such as muscle damaged by electrical burns.

Compartment syndrome is a medical emergency and requires the rapid release of the restricting tissues, either by escharotomy of burned skin (Figure 3) or fasciotomy of muscle compartments, to restore adequate perfusion to the affected tissues.

Figure 3: Escharotomy carried out on a leg with circumferential deep partial and full thickness burns. Reproduced with permission from Hettiaratchy 2004.

In Text References

(Hettiaratchy and Papini. 2004)

References

- Hettiaratchy S, Papini R., Initial management of a major burn: II--assessment and resuscitation., 2004, PMID:15242917

3. 2. Abdominal compartment syndrome

Circumferential burns to the abdomen can cause intra-abdominal pressure to rise above its normal value of 5-7 mmHg. As connective tissue proteins are denatured, deep burns have limited capacity for expansion with the reduction in abdominal wall compliance compounded by the fluid resuscitation, capillary leak and frequent ileus that follow. This rise in compartmental pressure leads to direct compression and potential thrombosis of abdominal viscera, manifesting as kidney injury and / or bowel ischaemia.

Intravesical pressure measurement is the most reliable means of identifying raised intra-abdominal pressure. In burns patients, this may be remedied by escharotomies, loosening of dressings and more general measures (e.g., sedation, neuromuscular blockade, gastrointestinal decompression, diuresis, and ultrafiltration).

3. 3. Others

Circumferential burns involving the thorax can restrict chest wall movement, thereby impairing respiratory mechanics and causing respiratory failure and difficulty in mechanical ventilation. As with other circumferential burn injury, escharotomy should be considered early following presentation to circumvent this issue.

4. Temperature management

4. 1. Early hypothermia

Hypothermia in burn patients is associated with mortality, with data registry analysis suggesting 5% increased odds of death for every 1 °C decrease in emergency department temperature (Stanton et al. 2023). Thus, proactive management of temperature should be instigated at an early stage. Patients and fluids should be actively warmed during resuscitation to avoid hypothermia. Initial assessment of burn severity will require patient exposure. However, to reduce heat loss, exposure should be for the shortest period of time by the fewest people and expose only the area to be examined. Careful monitoring of core body temperature should be initiated early. This is of particular importance in children and elderly burn patients, where heat loss can be pronounced and mechanisms to enhance thermoregulation are limited.

Interventions that limit early hypothermia include:

- Increasing ambient temperature to 28-33ºC (this can be achieved by caring for the burns injured patient in an isolation room or specialist theatre suite with increased temperature)
- Restricting patient exposure
- Removing wet clothes / bed linen
- Foil blankets and hats
- Forced air warmers - multiple devices may be required to cover the patient (Figures 4 & 5)
- Heat lamps
- Intravascular warming devices

Figure 4: Example of a forced air warming device.

Figure 5: Example of forced air warming devices being used in the theatre setting, both under patient and over the torso. Reproduced with patient consent.

References

- Stanton E, Gillenwater J, Pham TN, Sheckter CC., Temperature Derangement on Admission is Associated With Mortality in Burn Patients-A Nationwide Analysis and Opportunity for Improvement., 2023, PMID:36335477

4. 2. Late hyperthermia

Most patients with major burns will subsequently develop a raised core temperature. This reflects altering of the hypothalamic set point for thermoregulation by pyrogens such as interleukin-1 (IL-1) and tumour necrosis factor. More profound hyperthermia, with temperatures exceeding 40°C, can occasionally occur and may lead to multiorgan failure. This is most often seen in the context of superadded sepsis.

Steps to address potentially harmful hyperthermia should be taken in patients with a core temperature above 39.5°C. Techniques include debulking dressings, giving antipyretics such as paracetamol, applying ice to non-burned areas, infusing cooled intravenous fluids and irrigating the bladder and stomach with cold fluids. More invasive approaches such as intravascular heat exchange catheters (Figure 6) or extracorporeal circuits may also be considered.

Figure 6: Example of the equipment used to regulate core temperature with the use of an intravascular temperature control device.

In Chapter References

(McGovern, Puxty and Paton. 2022)

References

- McGovern C, Puxty K, Paton L. , Major burns: part 2. Anaesthesia, intensive care and pain management., 2022, PMID:35531075

5. Sepsis and antimicrobial therapy

5. 1. Sepsis following burn injury

Skin is the body's primary barrier to infection. Its loss due to burn injury, coupled with the degree of ensuing immunosuppression , results in increased risk of infectious complications. Infections account for 42-65% of deaths from burn injury and contribute significantly to the associated morbidity (Lachiewicz et al. 2017).

Burn wounds can become colonised with a variety of organisms, with low concentrations of bacteria frequently present on the surface of wounds. However, invasive wound infection, characterised by surrounding cellulitis, can result in extension of the existing burn depth, eschar separation or further necrosis of affected tissues. Patients may also develop systemic complications as a result of sepsis. Life-threatening organ dysfunction caused by a dysregulated host response to infection can occur and lead to multi-organ failure.

In the context of a major burn injury and ensuing systemic inflammation, the clinical diagnosis of sepsis can be difficult to make. The physiological characteristics of sepsis, such as fever and tachycardia, as well as a rise in biomarkers such as C-reactive protein (CRP), are often observed in burns patients. This makes it challenging to distinguish sepsis from the normal post-burns inflammatory response. Sepsis should be considered where the trends in these variables are deteriorating and burns-specific sepsis criteria may be informative.

References

- Lachiewicz AM, Hauck CG, Weber DJ, Cairns BA, van Duin D., Bacterial Infections After Burn Injuries: Impact of Multidrug Resistance., 2017, PMID:29194526

5. 2. Diagnosis of sepsis

Given the challenges, specific criteria for the diagnosis of sepsis in patients with burns have been proposed. The American Burn Association propose sepsis is likely where there is suspicion of infection with at least three of the following (Greenhalgh et al. 2007):

- Temperature >39°C or <36.5°C

- Heart rate >110 beats min^{-1}
- Ventilatory frequency >25 breaths per minute (or minute ventilation >12 L min^{-1} if invasively ventilated)
- Thrombocytopenia <100 x 109 L^{-1} (>3 days after initial resuscitation)
- Hyperglycaemia >11.1 mmol L^{-1} or insulin infusion dose requirement > 7 units h^{-1}
- Intolerance of enteral feed

5. 2. 0. 1. Biomarkers

The ideal biomarker would allow for early, accurate diagnosis of sepsis, risk-stratification, targeted intervention, disease monitoring and ultimately improved patient outcomes from an easily obtainable specimen subjected to readily available, low-cost analysis.

The inherent complexity of the dysregulated host response to infection renders such a biomarker elusive in critical care generally. The situation is further complicated in burn patients due to the:

- systemic inflammatory response syndrome (SIRS) almost universal in burns exceeding 30% TBSA;
- extensive colonisation of wounds;
- serial infective insults occurring over a protracted timeframe; and
- immunosuppressive element of hypermetabolism.

Conventional biomarkers therefore need to be interpreted with caution in burns patients. In a recent meta-analysis (Li et al. 2022), C-reactive protein and procalcitonin were both only moderately useful when measured at a single point to identify sepsis, and white cell count performed poorly. Biomarker trends, however, may be more useful than absolute concentrations breaching a certain threshold. Thus, in the Surviving Sepsis After Burn Campaign (Greenhalgh et al. 2023), rise in procalcitonin > 2 ng/ml from baseline is recommended as a trigger for prompting consideration of sepsis.

Novel means of identifying infection are the subject of much research in burns but many of these assays are not yet accessible to bedside clinicians, particularly in resource-limited settings.

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5. 3. Organisms

The most common pathogens, particularly early in the admission, are Gram-positive bacteria such as Staphylococcus aureus, Streptococcus and Enterococcus species.

Gram-negative bacteria such as Pseudomonas aeruginosa can translocate from the gastrointestinal tract or the environment and thrive in the moist environment of a burn wound. Infections from Pseudomonas will have a typically green/yellow colour and foul smell and can lead to invasive infection with necrosis. Other Gram-negative pathogens including Acinetobacter, Escherichia coli and Klebsiella can also cause invasive infections (Norbury et al. 2016).

Multi-drug resistant strains of pathogens including P. aeruginosa, Acinetobacter species and vancomycin-resistant Enterococcus (VRE) are an increasing concern. Recognised risk factors include the use of broad-spectrum antibiotics, colonisation at hospital admission, need for escharotomy, prolonged hospital or intensive care stay and multiple surgical procedures (Norbury et al. 2016). Fungal colonisation and invasive fungal infections are also significant problems.

Fungal wound infection is independently associated with increased mortality. Candida albicans is the most common pathogen, although Aspergillus and other invasive mould infections are becoming more common. Regular clinical review and sampling of wounds and other potential sources of colonisation and infection should guide antimicrobial therapy(McGovern, Puxty and Paton. 2022).

References

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- Norbury W, Herndon DN, Tanksley J, Jeschke MG, Finnerty CC., Infection in Burns. , 2016, PMID:26978531

5. 4. Topical Antimicrobial Therapy

Topical application of antimicrobial agents to burns has been commonplace since silver sulfadiazine dressings became the standard of care in 1968 (Klasen. 2000). However, a Cochrane systematic reviews has linked silver sulfadiazine to poorer healing than alternate dressings (Wasiak et al. 2013). This may reflect the requirement for regular dressing changes, poor eschar penetration or cytotoxicity of silver to keratinocytes and fibroblasts with consequent delay in wound healing. New methods of delivering existing agents continue to evolve, as do novel topical antimicrobials.

References

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- Wasiak J, Cleland H, Campbell F, Spinks A., Dressings for superficial and partial thickness burns., 2013, PMID:23543513

5. 5. Systemic Antimicrobial Therapy

When deemed necessary, systemic antimicrobial therapy is directed toward previous culture results. As major burn colonisation evolves over time, so too does the most appropriate antimicrobial agent. Thus, regular wound swab surveillance is important to inform best choice of antimicrobials in the event of superadded local infection or sepsis (Table 2).

Table 2: Common pathogens affecting burn injuries and corresponding antimicrobial therapy.

Type	Species	Antimicrobial Treatment
Gram positive	• Staphylococcus aureus • Streptococcus • Enterococcus	• Penicillins • Penicillins • Penicillins
Gram negative	• Pseudomonas aeruginosa • Acinetobacter baumanii • Enterobacteriaceae	• Piperacillin-tazobactam • Carbapenems • Cephalosporins

Fungi	• Candida • Aspergillus	• Azoles, amphotericin • Echinocandins
Muti-drug resistant	• Methicillin-resistant Staphylococcus aureus (MRSA) • Vancomycin-resistant enterococcus (VRE) • Extended-spectrum beta lactamases (ESBL) • Multi-drug resistant (MDR) Pseudomonas	• Vancomycin • Linezolid • Carbapenems • Colistin

Prolonged treatment with systemic antibiotics over time has propagated the evolution of resistant micro-organisms leading to treatment challenges. Increasing antibiotic resistance has driven the resurrection of old antibiotics, e.g., colistin, as well as the development of newer agents e.g., oxazolidinones. MRSA is a common resistant organism encountered in burns and is normally treated with vancomycin. The resistant gram-negative pathogens are becoming more problematic, with carbopenem resistant Pseudomonas and Acinetobacter particularly challenging (Branski et al. 2009).

Burns patients may gather particular benefit from the optimisation of antimicrobial drug delivery via continuous infusion (rather than intermittent dosing). The altered physiology and volume of distribution experienced by burns patients may profoundly lower antibiotic tissue concentrations. Monitoring of antimicrobial levels may therefore play a role in addressing antimicrobial resistance and mitigating further developments on this front (Ravat et al. 2011).

References

- Branski LK, Al-Mousawi A, Rivero H, Jeschke MG, Sanford AP, Herndon DN., Emerging infections in burns., 2009, PMID:19810827
- Ravat F, Le-Floch R, Vinsonneau C, Ainaud P, Bertin-Maghit M, Carsin H, Perro G; Société Française d'Etude et de Traitement des Brûlures (SFETB)., Antibiotics and the burn patient., 2011, PMID:20510518

6. Nutrition

6. 1. Energy expenditure following burn injury

Major burn injuries are associated with an increase in the individual's basal metabolic rate, more than doubling in patients with burns >40% TBSA (Williams, Herndon and Jeschke. 2009). This increased energy expenditure and need for increased substrate can result in loss of lean body mass, immune compromise and impaired wound healing. The impact on lean body mass correlates with an increased risk of adverse out-comes including infectious complications and death.

References

- Williams FN, Herndon DN, Jeschke MG., The hypermetabolic response to burn injury and interventions to modify this response., 2009, PMID:19793553

6. 2. Nutritional support

The aim of nutritional support following major burn injury is to deliver the correct amount of carbohydrate, protein and lipids to provide substrates for respiration, immune function and wound healing, and minimise loss of lean body mass while avoiding the risks of overfeeding (Figure 7). There is also recognition that vitamins and trace elements such as copper, selenium and zinc can be lost in large quantities in burn exudate. Prompt replacement of these micronutrients may improve antioxidant defences, prevent infectious complications and improve wound healing (McGovern, Puxty and Paton. 2022).

There has been widespread recognition that establishing early enteral nutrition in the hours immediately after injury can modulate the stress hormone response, maintain gut integrity, improve wound healing and help avoid infectious complications (Clark et al. 2017). The British Burn Association national standards (BBA 2018) state:

- Enteral nutrition should be started as soon as possible in major burns, ideally within 6–12 h after the injury.
- Total body weight loss should not exceed 10% of the patient's weight at admission.


Figure 7: Example of enteral feed.

As with most critically ill patients, the enteral route is preferred, routinely via continuous infusion through nasogastric tubes (Figure 7). Post pyloric feeding may be required if gastric stasis is present and impairing calorie delivery. Post pyloric feeding may also help reduce the risk of aspiration, especially in patients requiring multiple interventions under general anaesthesia. Parenteral nutrition is rarely required, but when used, caution should be exercised because of the associated risks of infec-tion and overfeeding.

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6. 3. Caloric requirements

Nutritional support in patients with major burns aims to meet the substantially increased caloric requirements of these patients while avoiding harmful overfeeding. The complications of overfeeding include hyperglycaemia, hypertriglyceridemia, hepatic steatosis, hypercapnia and prolonged duration of mechanical ventilation. Various methods have been used to assess caloric needs. The most accurate method is indirect calorimetry, but this remains mainly a research tool. In lieu of this, various formulae have been devised to guide clinicians (Table 3). Such formulae may under- or overestimate requirements at different times during a patient's admission. Given the complexities of providing optimum nutritional support, specialist input from a dietician within the burns team is essential.

Table 3: Examples of common formulas used to calculate caloric requirements in burn injured patients. Adapted from Clark et al, 2017.

Formula	Calculation (Kcal day^{-1})	Comments
Harris Benedict	• Male: 66.5 + 13.75 (weight in kg) + 5 (height in cm) - 6.76 (age in years) • Female: 66 + 9.56 (weight in kg) + 1.85 (height in cm) - 4.68 (age in years)	Multiplied by factor for burn size: • < 20% TBSA x1.5 • 20-40% TBSA x1.6 • > 40% TBSA x1.7
Toronto	-4343 + 10.5 (%TBSA) + 0.23 (caloric intake in last 24hrs) + 0.84 (Harris-Benedict unadjusted) + 114 (temp°C) - 4.5 (days after injury)	Adjusted by patient activity: • Confined to bed: x1.2 • Minimal ambulation: x1.3 • Moderate activity: x1.4
Hangang	867.542 - 5.546 (age in years) + 13.297 (weight in kg) + 4.879 (% TBSA) - 9.844 (PBD) + 500.612 (V)	Adjusted for each post-burn day (PBD) and if mechanically ventilated (V = 1 if ventilated, V = 0 if not)

6. 4. Macronutrients

The three main macronutrients (carbohydrates, proteins and lipids) provide substrates for adenosine triphosphate (ATP) biosynthesis, wound repair, immune function and maintenance of lean body mass. Carbohydrates are generally the preferred energy source, preventing a requirement for muscle proteolysis. However, relying solely on carbohydrates to meet caloric needs would result in hyperglycaemia caused by glucose delivery exceeding the rate at which it can be used. A relative insulin resistance, commonly observed in critically ill patients, may also exacerbate hyperglycaemia. Glucose should be limited to 55% of total energy requirements and hyperglycaemia managed with supplemental insulin as required (Rousseau et al. 2013).

Excessive lipid delivery can result in accumulation in the liver and impaired immune function. Therefore, lipids should account for no more than 30% of energy delivered, although some centres recommend a maximum of 15%. Given the potentially high sedation requirements of these patients, the lipid content of propofol should also be accounted for ((Clark et al. 2017; Rousseau et al. 2013).

Protein plays a crucial role in wound repair and maintenance of lean body mass. Increasing protein intake to supraphysiological values does not prevent catabolism of existing protein stores but does prevent a negative nitrogen balance and improve protein synthesis. Protein should be delivered at 1.5-2 g kg^{-1} day^{-1} in adults ((Rousseau et al. 2013). This often requires protein enriched feeds prescribed under the guidance of a dietetic team.

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6. 5. Micronutrients

Reserves of trace elements including copper, selenium, zinc and vitamins B, C, D and E may become depleted because of the intense inflammatory response, exudative losses and haemodilution accompanying fluid resuscitation. Micronutrients are essential for antioxidant defences, wound healing and immune function. Adequate supplementation frequently requires supraphysiological doses to be given, often parenterally because enteral absorption may be limited. Early replacement of these elements may reduce infectious complications, improve wound healing and reduce intensive care length of stay (Rousseau et al. 2013).

There has been much interest in the past regarding the role of glutamine on the metabolic and stress response following severe burn injury. A recent randomised-controlled trial (Heyland et al. 2022) evaluated enteral glutamine supplementation in 1200 patients with >10% TBSA burn (either full or partial thickness). This study failed to find any beneficial effect of glutamine on time to discharge alive from hospital or any of the secondary endpoints. Glutamine supplementation cannot therefore be recommended for use to attenuate the metabolic and stress response following burn injury.

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7. Hypermetabolism

7. 1. Pathophysiology

Hypermetabolism is a well-recognized physiological response to severe burns injury. It is characterized by an increase in the body's metabolic rate, leading to increased energy expenditure and changes in nutrient metabolism. This response is thought to be driven by a combination of factors, including systemic inflammation, tissue damage and stress hormone release.

One of the hallmark features of hypermetabolism following burns injury is an increase in resting energy expenditure (REE), which can be 2-3 times higher than normal. This increase in REE is thought to be driven by the release of stress hormones, such as cortisol and catecholamines, which activate metabolic pathways and stimulate protein breakdown.

Hypermetabolism following burns injury is also associated with changes in nutrient metabolism. Burn patients may experience increased proteolysis, decreased protein synthesis, and changes in the way the body processes glucose and fat. These changes can contribute to muscle wasting and weakness.

Hypermetabolism following burns injury is associated with several negative consequences, including increased muscle catabolism, loss of lean body mass and decreased immune function. These effects can prolong recovery time, increase the risk of infections, and contribute to long-term disability in burn patients.

Although changes in the metabolic and inflammatory response are most pronounced in the acute phase, multi-system pathophysiological changes can occur for years following burn injury, persisting long after the initial injury has healed (McGovern, Puxty and Paton. 2022). Research has demonstrated changes in urinary and plasma catecholamines, myocardial dysfunction and fibrosis, altered levels of various inflammatory mediators and changes in bone density for years after injury. These pathological changes may result in increased risk of various problems such as risk of infections from persisting immunoparesis, cardiovascular disease, cancer or early death (Table 4) (Barrett et al. 2019).

Table 4: Long-term effects of burn injury

Organ system	Effects

Immune	• Increased incidence of respiratory infections (influenza, pneumonia) • Increased hospital admissions with infectious diseases • Increased mortality from infections
Cardiovascular	• Increased risk of ischaemic heart disease, hypertension, heart failure and stroke • Reduced exercise tolerance • Myocardial fibrosis
Gastrointestinal	• Increased risk of disease of alimentary tract, gallbladder, biliary tract and pancreas • Increased hospital admissions with diabetes mellitus
Musculoskeletal	• Increased fracture risk • Joint pain and stiffness • Reduced mobility • Increased hospital admissions with musculoskeletal disorders
Central Nervous	• Increased hospital admissions with epilepsy, migraine and nerve problems
Miscellaneous	• Increased all-cause mortality • Increased cancer risk (perhaps worse in females)

Management of hypermetabolism following burns injury typically involves a combination of debridement (Figure 8) (to remove the stimulus), and grafting (Figure 9), nutritional support, pharmacotherapy and rehabilitative exercise. Nutritional support is critical to providing the body with the nutrients it needs to support healing and prevent muscle wasting. This may involve enteral or parenteral feeding, depending on the patient's condition and degree of gastrointestinal function.

Figure 8: Example of full thickness burn of an upper limb that has been debrided. Reproduced with patient consent.

Figure 9: Example of skin graft being applied to debrided area of skin. Reproduced with patient consent.

Physical therapy and exercise are also important in the management of hypermetabolism following burns injury. Regular exercise can help maintain muscle mass, improve functional outcomes, and reduce the risk of complications such as blood clots and pneumonia.

Pharmacological interventions may include the use of beta-blockers to reduce the metabolic response to burn injury and other medications, such as anabolic steroids and growth hormone, to prevent catabolism and support muscle recovery.

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7. 2. Beta-blockers

Beta-blockers are a class of medications that block the effects of adrenaline and noradrenaline at specific sites in the body. They are commonly used to treat conditions such as hypertension, heart failure and arrhythmias. In recent years, there has been growing interest in the use of beta-blockers in the treatment of burns, as they may have beneficial effects on inflammation, immune function, and other aspects of the body's response to injury.

The evidence for beta-blockers following burns injury is still emerging. However, several small studies and case series have suggested a benefit. The therapeutic use of beta-blockers to reduce the hypermetabolic response was first noted by a study in the 1970s (Szabó. 1979). This study evaluated the use of oxprenolol in burns patients with >20% TBSA and demonstrated less weight loss in the treatment group compared to control patients (5.4% vs. 9.2%). Further studies have confirmed a reduction in metabolism relative to the baseline resting metabolic rate in patients with severe burn injury following propranolol administration (Breitenstein et al. 1990). Comparing selective and non-selective beta-blockade, beta-1 adrenoreceptor blockers provide a similar reduction in heart rate, but do not prevent the lipolysis classically seen in burned patients (Herndon et al. 1994; Herndon et al. 2001). Non-selective beta-blockers such as propranolol are therefore the preferred drugs for attenuating the hypermetabolic response.

A recent randomised controlled trial evaluated the safety of propranolol administered in the first 72 hours following burn injury >20% TBSA (Lam, Khanh and An. 2022). Compared to the placebo group, propranolol led to significantly lower resting energy expenditure, less hepatomegaly and more favourable wound healing. During the first week of treatment, the average MAP was lower in the propranolol group, but still in normal physiological ranges. Significant hypotension occurred in 11.9% of the propranolol group and caution may therefore be required in patients with cardiovascular instability.

Propranolol can be used as a therapy for reducing the hypermetabolic response and comorbidities from burn injuries, however, data evidencing improvement in mortality or length of hospitalization are lacking. Further studies are needed to fully understand the potential benefits and risks of beta-blockers in critically ill patients with major burns – indeed this has been highlighted as research priority by the American Burn Association.

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7. 3. Androgens

Androgens are a class of hormones that include testosterone and are primarily known for their role in male sexual development and function. However, they also have important effects on muscle growth, bone density, and other aspects of the body's response to injury. Consequently, they may confer benefit in patients with major burns, with potentially advantageous effects on muscle wasting, immune function, and other aspects of recovery. Oxandrolone is an androgen receptor agonist, which stimulates protein synthesis and muscle growth with much less virilising activity than testosterone, making it more suitable for use in women and children.

Oxandrolone has been used from around Day 5 after burns, at a dose of 10 mg enterally twice daily in patients with ≥30% TBSA burns, to minimise weight loss, improve urinary nitrogen balance, increase muscle strength and reduce healing time. Some studies have shown benefits such as reduced ICU and hospital length of stay, maintained lean body mass and improved whole-body mass (Wolf et al. 2006). Adverse events include hepatic injury with increased liver transaminases, renal injury and skin complications such as cellulitis. Concerns regarding the risk: benefit ratio of oxandrolone prompted withdrawal of its FDA approval in 2023 and the drug is now seldom available, prompting a search within the burns community for other agents that may mitigate the hypermetabolic response.

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8. Management of pain and sedation

The pain experienced from a burn injury can be excruciating and is often difficult to manage. Some studies suggest higher pain scores during hospital admission are associated with poorer long-term outcomes such as mental health problems. Given the complexities and challenges of effective pain management, a specialist pain service should form an integral part of the burn team.

8. 1. Types of pain

Whilst the burn is usually the most significant source of pain, ancillary causes such as other injuries, tracheal tubes, invasive lines and catheters, skin autograft donor sites, pressure areas and interventions including position changes will all impact pain severity. Clinicians should adopt a structured approach to acute burn pain management, addressing the three main types of pain: background, breakthrough and procedural pain (table 5).

Table 5: Suggested approach to management of pain in burns patients

Type of pain	**Initial strategy**	**Step down (once enteral route established)**
Background pain	• Regular IV paracetamol • Opioid infusion • Ketamine infusion	• Regular PO paracetamol • Regular or modified release opioid • Gabapentinoid
Breakthrough pain	• Bolus IV opioid	• Immediate release opioid
Procedural pain	• Bolus short-acting IV opioid	• Fentanyl lozenges • Nitrous oxide (inhaled)

8. 1. 1. Background Pain

Patients will experience a degree of persistent pain after burn injury and multi-modal analgesia should be prescribed to maintain adequate control. Infusions should be titrated to a targeted effect, against pain scores. Multiple methods for pain assessment exist including the visual analogue scale (VAS), designed for cooperative and communicative patients. However, such linear scales have several limitations including what value should be targeted or considered acceptable to both patient and clinician. Such lack of clarity can result in unrealistic expectations with some evidence emerging that unidimensional pain scales can increase opioid use (Levy, Sturgess and Mills. 2018). As such, there has been a shift in focus, especially in perioperative practice, towards functional activity being the goal of analgesic practice, using tools such as the "Functional Activity Scale" (Tong et al. 2020; Levy et al. 2021):

- No limitation (do activity without limitation from pain)
- Mild limitation (do activity with moderate/severe pain)
- Significant limitation (unable to complete activity due to pain or adverse effects of analgesia)

However, there should be recognition that such shifts in pain assessment are primarily evident in the management of the post-operative patient, not those that suffer a major burn injury. Furthermore, both unidimensional and functional measures of pain are of limited value in critically ill, sedated or delirious patients where methods such as the Critical Care Pain Observation Tool (CPOT) can instead be used (Buttes et al. 2014).

Consideration should also be given to non-pharmacological measures such as appropriate dressings, comfortable positioning, cutaneous stimulation, acupuncture and techniques such as cognitive behavioural therapy and music therapy as a means to alleviate this form of pain.

8. 1. 2. Breakthrough Pain

Breakthrough pain can occur on top of well controlled background pain, either as an exacerbation of background pain or originating from another source. This type of pain can be managed with boluses of rapid acting agents, transient increases in the rate of opioid infusions or, if predictable, anticipatory doses of longer acting agents.

8. 1. 3. Procedural Pain

Interventions include dressing changes, position changes, line insertions and mobilisation can provoke procedure related pain. Appropriate timing of analgesic interventions should be considered to gain the maximum benefit from the agent being used. Opioid boluses, inhaled agents including nitrous oxide and methoxyflurane or occasionally sedative agents such as ketamine can all be considered for the management of procedural pain. Other non-pharmacological methods may be beneficial including hypnosis, virtual reality systems and other distraction techniques.

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8. 2. Pharmacological Management of Pain

8. 2. 1. Opioids

Opioids remain the mainstay of pain management in burn injuries. The choice of opioid and method of delivery will depend on local preferences and patient factors including renal function and conscious level. Patient Controlled Analgesia (PCA) may be used but requires a conscious and engaged patient able to use the delivery device. Continuous infusions of opioid are more usual in the initial phase of burn critical care admissions. However, patients with major burns often require large doses of opioids resulting in inevitable adverse effects such as ileus, respiratory depression, delirium, hypotension and potentially dependence. For this reason, a multi-modal analgesic approach is advised (Romanowski et al. 2020).
Given the severity of pain after burn injury often necessitating the use of opioid medication, combined with prolonged hospital admissions with multiple operative interventions, modified release opioids are sometimes used to facilitate step-down from parenteral opioids to enteral routes. It should be noted that multiple national and international guidelines have highlighted the increased risks associated with the use of modified release opioid preparations, prompting the UK Medicines and Healthcare products Regulatory Authority (MHRA) to remove the licenses for these medications in the management of post-operative pain. Given the frequent overlap in mental health conditions, drug and alcohol misuse in patients that suffer major burn injuries, the risk of developing opioid dependence may be high. The use of opioids, especially modified release preparations, should be used with caution, reduced and stopped at the earliest opportunity.

8. 2. 2. Non-Steroidal Anti-Inflammatory Drugs

NSAIDs such as ibuprofen and diclofenac are infrequently used in critically ill patients due to the risk of gastrointestinal haemorrhage and renal impairment. Whilst they may be of benefit in selected patients, they should be used with caution.

8. 2. 3. Ketamine

Ketamine is an N-Methyl D-Aspartate (NMDA) receptor antagonist with potent analgesic effects. Ketamine can be given as an infusion, often to reduce opioid requirements, or in sedative or anaesthetic doses for painful interventions. Ketamine may also have a role in preventing the development of neuropathic pain and has been shown to reduce secondary hyperalgesia and “wind-up” phenomenon in healthy volunteers (McGuinness et al. 2011).

8. 2. 4. Gabapentinoids

Gabapentinoids such as gabapentin and pregabalin have been used to manage acute and chronic burn pain and pruritus. Several observational studies have demonstrated an improvement in pain scores and reduced opioid consumption. However, given the increasing evidence of harm from dependence, abuse and overdose, their use should be considered cautiously, especially in patients with a history of alcohol or drug misuse (McGovern et al. 2021).

8. 2. 5. Dexmedetomidine

Dexmedetomidine is a highly selective α-2 receptor agonist with both analgesic and sedative effects. It can be used in the intensive care unit as part of an analgo-sedative regimen, in combination general anaesthesia, as a sedative in painful procedures and also as an adjunct in patient-controlled analgesia. Common side effects of dexmedetomidine are bradycardia and hypotension. As recent studies have highlighted the risk of pyrexia associated with dexmedetomidine use (Grayson et al. 2021), caution is advised in the context of hypermetabolism after burns.

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9. Psychological support

Given the distressing circumstances in which burn injuries can occur and the resulting potential for a prolonged hospital admission with significant pain and multiple interventions, the risk of psychological distress and harm in major burns patients is significant.

Post-traumatic stress disorder (PTSD) is characterised by symptom clusters including re-experiencing the traumatic event, avoidance of potential triggers, alterations in mood or cognition and a state of hyperarousal or perceived increased threat. Longitudinal studies have suggested that the incidence of PTSD after burn injury can be as high as 45%, with predictors of those being at increased risk hard to identify (Lodha et al. 2020). Surprisingly, the extent of burn injury, as measured by TBSA, does not seem to correlate with the risk of developing PTSD. However, an individual's perceptions, social support network and poor coping strategies are all thought to increase risk. The diagnosis of PTSD can be difficult to make as many patients exhibit no symptoms until after hospital discharge. Equally, reliable assessment of patients can be hampered by the distress caused by other factors such as pain and sleep disturbance.

Anxiety and depression are also common following burn injury, with some studies suggesting an incidence between 7 and 46%. Factors that can impact this include the traumatic nature of the injury, impaired physical functioning, altered body image and the financial implications of life-altering injury and protracted recovery.

A population based retrospective study conducted by Duke and colleagues found that patients with burn injury had a significantly increased risk of being admitted to hospital with a mental health disorder when compared to an uninjured cohort matched for age and gender (Duke et al. 2018). The authors reported an incident risk ratio of 4.89 (95% CI 3.52-6.79), with 4% of the burn injured cohort requiring a hospital admission for a mental health condition, predominantly self-harm or alcohol / drug dependence.

Additionally, the presence of pain will often impact an individual's mental health. Several studies have investigated whether there is a link between pain and psychiatric disease. One longitudinal study explored the link between acute pain and risk of suicidal ideation following a burn injury (Edwards et al. 2007). Of the 128 participants that completed the year-long follow-up period, 9.4% had active thoughts of suicide at discharge, 11.7% at 6 months and 14.8% at 12 months. Pain severity at hospital discharge was found to be the only predictor of suicidal ideation at follow-up.

Psychological support staff should form part of the burns multidisciplinary team. Routine psychological assessment should be undertaken, and robust care pathways should be in place to provide help and support to burn victims and their families. This might include access to support groups, charities, websites and opportunities for peer support. Early psychosocial screening may help identify patients at the highest risk of developing problems after burn injury, allowing prompt intervention to address the psychological, emotional and social challenges these patients may face.

In Text References

(McGhee et al. 2011)

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