Burns injury

Clinical problems

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Learning objectives

1. Know how to initiate early burn management in adults and children at the scene, during transport and in hospital (0-72 hrs).
2. Know how to identify patients that should be referred to a burn centre.
3. Know the origins and principles of early fluid management (0-72 hrs) in burn patients e.g. the rule of nines.
4. Know the dynamic nature of fluid resuscitation in burn patients including its practicalities and hazards.
5. Have knowledge of the epidemiology of burns, severity and mortality risk and know the principles of intensive care in burn patients.
6. Have knowledge of surgical interventions of burns, perioperative management and of the importance of multidisciplinary care including physiotherapy and rehabilitation.
7. Have knowledge of outcome and ethical considerations and of research priorities in burn injury.

FACULTY DISCLOSURES
The authors of this module did not report any disclosures.

DURATION
9 hours

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INTRODUCTION

Most emergency medical services (EMS) and hospitals will, from time to time, need to deal with the immediate management of patients with large burns. Care of these patients will be a major challenge for most doctors. Correct initial management can make the difference between a good and a poor outcome. Therefore, emergency medicine physicians, intensivists, plastic and general surgeons and anaesthesiologists require knowledge and competence in early burn assessment: depth of burn, % total body surface area (%TBSA), early fluid therapy, airway management, surgical principles, intensive care support and wound care. Knowledge of related pathophysiology and anatomy is important to the assessment.

The aim of this module is to give clinical guidance on how to manage patients with burn injury in the early and ongoing phases. Sections on basic knowledge (evaluation of depth and extent of burn), metabolic response, nutrition and pathophysiology provide a broad background for understanding unique aspects of burn management. Therapy suggestions are made from our clinical experience of burn patients and from literature where available.

Series of 12 articles in BMJ, 2004. PMIDs 15191982, 15205294, 15217876, 15242917, 15258073, 15271835, 15284153, 15297346, 15310609, 15321905, 15331482 15331482

See also the website of the American Burn Association and the European Practice guidelines for Burn Care http://www.euroburn.org/userfiles/users/36/pdf/guidelines/EBAGuidelinesBurnCareVersion1.pdf

Epidemiology

There is limited information available from European countries describing the epidemiology of burns. The American Burn Association (ABA), however, has provided extensive data collected from different centres across the USA. The majority of burn wounds are small and therefore treated in primary care and emergency care facilities. One in ten burn patients are referred to the hospital and of these 10-15% are transferred to a burn centre. In Norway, the rate of admission for burns was 15.5/100,000/year in 2007. In a review comprising data from more than 185,000 patients, the incidence of severe burns in Europe was reported as 0.2 to 2.9/10,000 inhabitants. Burn injury is more common in men than in women and flame and scald are the most common causes. Children <5 years of age are at risk, and the incidence of burns among boys younger than 5 years is about five times higher than in older age.

In patients with large burns, the mean stay in a burns unit is about 1 day per %TBSA.


**2011 National Burn Repository:** provides reports with demographic data, injury information and outcomes from US burn centres.
**Priorities including the primary survey**

The main responsibilities in the pre-hospital setting are safety for the patient(s) and the rescue crew, to stop the burning process and to perform ABCDEF (airway, breathing, circulation, disability, environment, fluid) assessment and treatment. Thereafter a gross estimation of the burned surface (%TBSA) should be carried out. Pre-hospital estimation of %TBSA is difficult and frequently an overestimation of smaller burns and an underestimation of larger burns takes place. For detail of the estimation of the %TBSA burned, see Task 2.


Collis N, Smith G, Fenton OM. Accuracy of burn size estimation and subsequent fluid resuscitation prior to arrival at the Yorkshire Regional Burns Unit. A three year retrospective study. Burns 1999; 25: 345-351. PMID 10431984

**Cooling**

Most textbooks advocate cooling of the burn injury until pain relief with 12–15 °C (never ice) clean water. It is claimed that cooling stops the thermal injury, reduces oedema and relieves pain. Studies have shown that the most significant reduction of temperature in burned tissue takes place within few minutes after the start of cooling. Cooling beyond two to three minutes is therefore of questionable value. Remember that cooling often causes hypothermia in patients with large burns.


Trupkovic T, Hoppe U, Kleinschmidt S, Sefrin P. Correspondence (letter to the editor): benefits of cooling are not known. Dtsch Arztebl Int 2010; 107(6): 101. PMID 20204125

**Hypothermia**

Hypothermia, defined as a core body temperature <35 °C, is rarely noted on initial presentation of burn patients but, particularly in large burns (>70% TBSA) the incidence may increase to 35%. Hypothermia is associated with increased fluid requirement, coagulopathy, depressed cardiac function, dysrhythmias, ventilatory depression, decreased oxygen delivery, acidosis, and a higher mortality.
**Airway management on scene**

If the patient has a facial burn or if there was a fire in a closed room, direct inspection of the oropharynx should be performed, preferably by a physician experienced in airway management. This procedure might be difficult to achieve in the pre-hospital setting. Carbonaceous sputum, hypoxia and hoarseness indicate inhalation injury (see the section on inhalation injury in Task 4, below) and most experts will advocate tracheal intubation in this setting. If there is doubt about the patency of the airway, intubation should be performed with a cuffed (endotracheal tube (ETT)) that has not been cut. Due to oedema formation, a smaller ETT may be required. A selection of ETT sizes and intubation aids should be available, including ‘difficult airway trolley’ if a difficult intubation is expected (usually not available on scene). Secure the tube properly after intubation. The indication to intubate should be broad; it is easier to extubate a patient when significant airway injury has been excluded than to intubate a patient with extensive airway burns which are identified later. Note that signs and symptoms of airway oedema may only become apparent when intravenous resuscitation is established - potentially during inter-hospital transport.

See the PACT module on Airway management.

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**NOTE** All (endotracheal tubes should be well secured, especially in patients with facial and neck burns. The tracheal tube may be very difficult or impossible to reinsert if accidentally dislodged.

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**Caution** Many use un-cut tracheal tubes to pre-emptively correct for anticipated oedema. If the tube is to be cut, do not cut the ETT to a standard ‘oral length’ as patients with facial and inhalational burns commonly develop marked oedema and a cut tube may ultimately become too short and be difficult to secure and/or become dislodged. [Images in interactive version]

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Eastman AL, Arnoldo BA, Hunt JL, Purdue GF. Pre-burn center management of the burned airway: do we know enough? J Burn Care Res 2010; 31(5): 701-705. PMID 20634705
Intravenous cannulae/needles and fluid resuscitation

On the scene one to two large peripheral cannulae should be inserted, preferably through un-burned skin (consider intraosseus access both in children and adults; video: http://www.youtube.com/watch?v=NiMREdptAww&feature=rellist&playnext=1&list=PL6DE21DEOA5B6EAB4). Secure the cannulae well. In adults with burns >20% TBSA, start intravenous crystalloid with 500 mL in one hour (limit hydration for smaller burns). In children with >10% TBSA burn injury, start with 50-100 mL of crystalloids in one hour dependent on the weight of the child. Mark the fluid bags with numbers but limit fluid administration until final assessment of burn size (overestimation of small burns on scene is frequent). After admittance to hospital, estimate fluid needs according to a validated formula e.g. Parkland - see below. [See Table 1 in http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3097558/ for formulae].

⚠️ Do not give too much fluid on site during the first 1–2 hrs after injury. A modest under-resuscitation is likely better than excessive fluids.


Wound care

Local treatment, except for irrigation (for chemical injury) and cooling of the burn wound, is not necessary at the scene. Wet and dirty clothes should be removed, and the wound should be covered with clean sheets. Do not use ointments, powders or complicated bandages as these have to be removed upon admission to allow adequate cleaning and assessment. Cling film (plastic wrap) may be used to cover burn wounds (video: http://www.youtube.com/watch?v=7kO2rlQT5X4). Put it on in layers, not circumferentially; it reduces pain and evaporative fluid losses.

Initial hospital management

When the patient is admitted to hospital, the primary (ABC) survey is re-checked and a secondary survey is performed. Emergency medicine physicians/intensivists, surgeons, anaesthesiologists and nurses work together to provide optimum initial care and to chart the injury and the condition of the patient. Clothes and bandages are removed, and the wounds are washed with tap water and soap or with sterile saline [Video of wound cleaning in interactive version]. A central venous catheter (preferred in adults with burns >20 %TBSA and in children with burns >15 %TBSA depending on hospital practice), arterial catheter and urinary catheter are positioned after the cleaning (preferably catheters placed before cleaning should be removed). Secure all lines carefully. Bloods for haemoglobin, electrolytes, creatinine, creatinine kinase, liver function tests and blood gas analyses are obtained and many units take swabs for microbiology, and blood cultures from the outset. It is important that the temperature in the admittance area be adjusted to above 30 °C to prevent hypothermia.

Burn centre referral criteria

Management of patients with large burns (Table in interactive version) is resource demanding. A team of intensivists, surgeons, anaesthesiologists, dedicated ICU nurses, physiotherapists and social workers are involved in the collaborative management of these patients to achieve the best cosmetic and functional result. It has long been demonstrated that patients benefit from a specialised burn centre if certain criteria are present. The patient should then be referred as soon as possible to such a centre.

The ABA has defined transfer criteria for burn patients:

- Second degree burns >10% TBSA
- Third degree burns
- Burns that involves face, hands, feet, genitalia, perineum and major joints
- Chemical burns
- Electrical burns including lightening injuries
- Any burn with concomitant trauma in which the burn injuries pose the greatest risk to the patient
- Inhalation injury
- Patients with pre-existing medical disorders that could complicate management, prolong recovery or affect mortality
- Hospitals without qualified personnel or equipment for care of critically burned children.

**Fluid resuscitation (0-24 hrs)**

Burns exceeding 20% TBSA (10% TBSA in children) are characterised by an early phase of massive capillary hyperpermeability proportional to the extent of the burn injury. [Images in interactive version]

**NOTE** The transient increase in permeability is caused by the massive liberation of numerous substances such as histamine, serotonin, cytokines, prostaglandins, leukotrienes, lipid peroxides, free radicals, myeloperoxidase, and complement. Studies in animals show that the leakage lasts 24-72 hrs, peaking between 12 and 24 hrs. The only similar clinical condition is anaphylaxis.

The resultant fluid leak causes an interstitial extravasation of intravascular fluid, potentially resulting in hypovolaemic shock which requires sodium containing isotonic crystalloids for treatment. During the first 24-48 hrs, molecules up to 100 kdalton in size escape into the interstitium. Colloids are discouraged in the early phase after injury because they will remain dispersed in the interstitium at the end of the hyperpermeability phase (their removal being dependent on an effective lymphatic transport).

According to recommendations, one should fluid resuscitate the burn patient with 2-4 mL/kg/%TBSA (the Parkland formula) using crystalloids. As a small burn size is frequently overestimated on scene, this formula may cause pre-hospital fluid overload. Other factors that contribute to the fluid creep (see Complications of fluid management) are over-sedation and volume guided fluid therapy. Dynamic protocols for fluid resuscitation managed by nurses may reduce fluid overload in the early phase.

The primary goal of fluid resuscitation is to maintain adequate tissue perfusion to the end-organs and the skin in an effort to conserve organ function/skin survival and to avoid ischaemic injury.

<table>
<thead>
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<th>Some supplementary critical care signs of fluid overload</th>
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<tr>
<td>Polyuria &gt; 1.0 mL/kg/hr</td>
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<tr>
<td>↑ cardiac preload (elevated CVP or ITBVI and PAOP if invasive monitoring instituted)</td>
</tr>
<tr>
<td>↓ PaO2/FiO2 ratio</td>
</tr>
<tr>
<td>Intra-abdominal pressure &gt;20 mmHg</td>
</tr>
<tr>
<td>Abdominal compartment syndrome manifested by</td>
</tr>
<tr>
<td>- Acute kidney injury</td>
</tr>
<tr>
<td>- Visceral ischaemia</td>
</tr>
</tbody>
</table>

CVP: Central venous pressure, ITBVI intrathoracic blood volume index, PAOP Pulmonary artery occlusion pressure

Remember, CVP (and PAOP) are more useful when used dynamically rather than as single stand alone measurements - see PACT module on Haemodynamic monitoring.
Over-resuscitation has become a major problem during the last 15 years, causing organ failure and death. Excessive fluid has been shown to worsen prognosis. This conclusion has led to the concept of ‘permissive hypovolaemia’.

Avoiding fluid overload

The simplest preventive measure is the prescription of a ‘half Parkland’ formula, i.e. 2 mL/kg/%TBSA instead of 4 mL/kg/%TBSA crystalloids (lactated or acetated Ringer’s) to initiate resuscitation, and to continue with a permissive hypovolaemia, aiming at delivering controlled amounts of fluids to compensate for a little more than the evaporative and exudative losses based on hard indicators of organ perfusion (see table below).

Lactated or acetated Ringer’s solution is preferred to normal saline as it carries less risk of hyperchloraemic acidosis.

Fluid administration can be guided by the calculation of the daily evaporative losses estimated according to the formula: \[3750 \text{ mL} \times \text{BSA (m}^2\text{)} \times \left(\frac{\% \text{burn}}{100}\right)\], combined with the clinical observation of signs of inadequate organ perfusion (low blood pressure and oliguria or anuria). Usually the aim for diuresis is 0.5-1 mL/kg/hr in adults - see table below. A simple measurement of haemoglobin concentration can be of guidance, and a concentration >17-18 g/100 mL indicates under-resuscitation.

<table>
<thead>
<tr>
<th>Objectives of fluid resuscitation in adults (combined fluid and pharmacological approach)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure &gt; 60 mmHg,</td>
</tr>
<tr>
<td>Heart rate &lt;120 bt/min</td>
</tr>
<tr>
<td>Central venous oxyhaemoglobin saturation &gt;60%</td>
</tr>
<tr>
<td>Adequate tissue perfusion</td>
</tr>
<tr>
<td>- Diuresis 0.5 mL/kg/hr, 1 mL/kg/hr in paediatric patients</td>
</tr>
<tr>
<td>- Near normal pH and base excess</td>
</tr>
<tr>
<td>- Near normal lactate levels</td>
</tr>
</tbody>
</table>


‘Permissive hypovolaemia’

The most recent iteration of this concept is the ‘Rule of 10’ developed at Fort Sam Houston (Texas, USA). The authors propose a three-step approach: the 1st step is to estimate burn size to the nearest 10% TBSA, 2nd step is to multiply this number by ten to derive the initial fluid rate in mL/hr (for every 10 kg body weight over 80 kg add 10 mL/h to this rate), and 3rd is to adapt the fluid rate to the signs of organ underperfusion.
**NOTE** Fluid resuscitation in patients with large burns is dynamic, and nurse driven protocols should be used to adjust fluid infusion rates. Preload (CVP, PAOP, or ITBVI) is never an objective! Do not use invasive or non-invasive haemodynamic monitoring to guide fluid resuscitation during the first 24 hrs after injury, but to monitor cardiac function.

In paediatric burns patients the same considerations apply to potential over-resuscitation, particularly as children additionally receive their normal hydration fluid.

**NOTE** Ongoing unexplained fluid requirements or persistent hypotension should raise the suspicion of unrecongnised associated injuries, missed inhalational burn injury, associated poisoning, or other complications such as myocardial infarction or sepsis.

In patients with very large burns, >40% TBSA, another tool to reduce fluid load is the possible use of high-dose vitamin C for the first 24 hrs (66 mg/kg/hr), while applying the above objectives: the study below suggests that fluid requirements, water tissue content and balance can be reduced by one third.

Tanaka H, Matsuda T, Miyagantani Y, Yukioka T, Matsuda H, Shimazaki S.  
Reduction of resuscitation fluid volumes in severely burned patients using ascorbic acid administration: a randomized, prospective study.  
Arch Surg 2000; 135(3): 326–331. PMID 10722036

After the first 12 hrs, colloids (albumin, gelatin, or starch solutions) may be used in case of persistent haemodynamic instability in burns patients with >40% TBSA or serum albumin <18 g/L. Fresh frozen plasma is used only for the treatment of coagulation deficiency/clotting disorders.

**Vasoactive infusion (vasopressor and inotropic) therapy**

The initial shock is multifactorial, combining hypovolaemic, cardiogenic and vasoplegic components particularly in burns >40% TBSA. Presuming euvolaemia has been achieved, haemodynamic support may require the early introduction of norepinephrine in doses up to 0.2-0.3 µg/kg/min to maintain an adequate perfusion pressure. At higher doses, caution is required as the microcirculation may be compromised. In the presence of persistent low cardiac output (elderly with cardiac comorbidity, electrical burns, very large burns), the addition of dobutamine may be required.

**Surgical assessment and procedures (0-24 hrs)**

In the initial phase, the main task for the surgeon is to evaluate the extent and the depth of the burn (Table in interactive version). Surgical procedures will be determined by the nature and depth of the burn (see Burn types below) and in addition, escharotomy is performed, if necessary - see below. [Further images in the interactive version].
Burn types

The skin


<table>
<thead>
<tr>
<th>Epidermis</th>
<th>Horn layer</th>
<th>Basal cells dividing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermis</td>
<td>Nerves</td>
<td>Blood vessels</td>
</tr>
<tr>
<td></td>
<td>Sweat- and sebaceous glands</td>
<td>Hair follicles</td>
</tr>
<tr>
<td>Subcutaneous tissue</td>
<td>Fat</td>
<td>Connective tissue</td>
</tr>
<tr>
<td></td>
<td>Muscle</td>
<td></td>
</tr>
</tbody>
</table>

Grade I

Grade IIa, b

Grade III

Scalding

Flame

Electrical injury

Chemical injuries
Electrical burn injury is addressed in Task 4.

**NOTE** Burn depth and size may not be initially clear and are often misinterpreted even by experienced surgeons. Regular reassessment is vital. The full extent and depth of the burn wound are often not clear for up to 48 hrs post injury.

**Escharotomy**

When extremities are burned, especially in circumferential injury, peripheral pulses should be evaluated. If pulses are absent (not caused by hypovolaemia), escharotomy is performed without delay. Escharotomy on the thorax and abdomen should be performed if there is circumferential deep injury, as the procedure will improve chest wall compliance and facilitate ventilation.

Escharotomy may be performed at the bedside, but is preferably be done under general anaesthesia in the Operating room/Burn unit using electrocautery to minimise bleeding.

In the upper limb, full-thickness incisions in the medial and lateral midaxial lines are performed. Ideally, the incision should extend just beyond the area of the full-thickness burn. Digital incisions are made in the midaxial line radially in the thumb and the little finger and on the ulnar side of the index, middle and ring fingers. Incisions can be made longitudinally in the spaces between the 2nd, 3rd, and 4th metacarpals and carpal tunnel release may be indicated.
Burn mass casualties

Mass casualty burn disasters are a highly challenging issue for several reasons: specialised burn beds are limited, the majority of healthcare personnel are not experienced in treating burn victims, and burn treatment is time, manpower and resource consuming. Analysis of several landmark fires in the US between 1900 and 2000 showed that most victims had fatal injuries and died on the scene or within 24 hours. Another large group was those patients with minimal burns that could be treated as outpatients suggesting that fire disasters produce relatively few patients requiring inpatient burn care. Since hospitals have limited surge capacities in the event of burn disasters, a special approach to both pre-hospital and hospital management of these victims is required to avoid overwhelming local resources; no objective criteria exist which define how to triage patients in such a situation. A table classifying patients according to their anticipated survival from burn injury was created some years ago to help and assist with this difficult task.

Specialised rescue and care can be adequately met at all levels of need by deploying mobile burn teams to the scene but such teams are not widely available. Burn specialists should nevertheless assist with both primary and secondary triage, contribute to initial patient management and offer advice to non-specialised designated hospitals that provide acute care for burn patients with TBSA <20-30%. It is also important to implement simplified treatment procedures which should be disseminated by specialised courses to hospitals that might be involved in a mass casualty network.

The resuscitation priorities remain the same: apply ABC principles.

**NOTE** An important issue in mass casualty is the availability of ventilators! The ‘normal’ liberal burn tracheal intubation policy may be applied to events with up to 20-30 casualties, but should be restricted with higher numbers of victims. Only patients with obvious inhalation injury and respiratory failure should be intubated on site. The early fluid management should be particularly restrictive to prevent oedema to the airway.

Analgesia favours the use of drugs that maintain spontaneous ventilation, such as ketamine. A second alternative for the same reason is morphine.

Patients should be triaged from the burn site to hospitals according to their burn size and requirements for specific airway management, transfer to specialised centres being effected only in those with burns >30% TBSA depending of course on numbers. A triage suggestion is provided below based on the Swiss burn plan.

<table>
<thead>
<tr>
<th>Type of hospital</th>
<th>Up to 50 victims</th>
<th>&gt;50 victims</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burn centre</td>
<td>Burns &gt;20% TBSA</td>
<td>Burns &gt;30% TBSA</td>
</tr>
<tr>
<td></td>
<td>Children: &gt;10% TBSA</td>
<td>Children: &gt;15% TBSA</td>
</tr>
<tr>
<td></td>
<td>Inhalation</td>
<td></td>
</tr>
<tr>
<td>University teaching or major</td>
<td>Burns 10-20% TBSA</td>
<td>Adults 10-30% TBSA</td>
</tr>
<tr>
<td>regional</td>
<td>Children: 5-10% TBSA</td>
<td>Children 5-15% TBSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inhalation</td>
</tr>
<tr>
<td>Smaller community hospital</td>
<td>None</td>
<td>Adults &lt;10-15% TBSA</td>
</tr>
</tbody>
</table>
Haberal M. Guidelines for dealing with disasters involving large numbers of extensive burns. Burns 2006; 32(8): 933-939. PMID 17081856


Saffle JR, Gibran N, Jordan M. Defining the ratio of outcomes to resources for triage of burn patients in mass casualties. J Burn Care Rehabili 2005; 26(6): 478-482. PMID 16278561

3/ ONGOING FLUID AND SURGICAL MANAGEMENT

Fluids (24-72 hrs)

After primary fluid resuscitation (0-24 hrs), the patients have oedema and are loaded with salt. At this stage, standard crystalloid solutions are replaced by hypotonic solutions (glucosaline, dextrose 5% or N/2 saline) and colloids are added \((0.5 \text{ mL x BW (kg) x } \%\text{TBSA Burned} = \text{ mL colloid/24 hrs})\) while aiming for 0.5 mL/kg/hr of urine output (1 mL/kg/hr in children). After 48 hrs, the goal is a negative salt and fluid balance and an intravascular volume that is adequate. The ambition is to bring the patient back to his/her usual weight within 7-10 days after the injury. Hypernatraemia is frequently observed during this fluid reversal phase and the use of thiazide diuretics may be considered. Due to evaporation it is important to give enough water e.g. as intravenous glucose 5% (glucose 50 mg/mL in water or as enteral water, usually via an NG tube). Other electrolyte disorders such as low potassium, phosphate and magnesium are common. The complication of most concern however is fluid overload due to ‘fluid creep’ - see below.


Greenhalgh DG. Burn resuscitation: the results of the ISBI/ABA survey. Burns 2010; 36(2): 176-182. PMID 20018451


Surgical procedures 0-24 hrs and beyond

Excision of the burn wound

Assessment of burn depth is often difficult and it may delay surgery as spontaneous healing is preferable in all cases where the healing is finalised before day 10-14 post burn. In cases of clear full-thickness burns, early excision is the state of the art recommendation.
Early tangential excision was first described in 1970. It has become the standard treatment for deep partial thickness and deep dermal burns. It requires careful attention to blood loss, patient body temperature, and viable tissue in order to be successful. In some patients with very deep burns, excision down to the fascial layer is indicated. Complete tangential excision should be accomplished within 72 hrs, or at latest by the end of the 1st week in very large burns. In large burns, excision might be done in two stages: one day in prone position followed by one in supine position.

In children and in burns to the face, debridement may be delayed as 2nd intermediate deep burns may recover with good resuscitation, which reduces grafting requirements. Bleeding and hypothermia are the limiting factors for the progress of surgery. The accuracy of the necrosectomy is crucial for successful wound closure.

Temporary coverage with cadaver skin or biological dressings may be necessary in larger burn wounds. This technique has been used in burn management since World War II and is still practised in major burn centres world wide. The use of allograft reduces water, electrolyte and protein loss, prevents desiccation of tissue, suppresses bacterial proliferation, reduces pain and promotes epithelialisation. Allogenic grafts are best applied unmeshed or minimally expanded in order to temporarily cover. The allografts are removed once the donor sites have healed sufficiently for reharvesting.
Removal of the burned tissue and wound closure as soon as possible is vital. Burned tissue is an ideal culture medium for bacteria and fungi, but even if not infected, the presence of burn tissue is a significant source of inflammatory mediators that support an ongoing systemic inflammatory response.

Wound grafting

The aim of the treatment is complete closure of the wound with autogenous split thickness skin grafts. If enough viable skin is available, skin grafting can be performed in the same operation after necrosectomy. In large burns, many surgeons prefer to do the necrosectomy and grafting in two procedures during two sequential days to make sure that necrosectomy is complete before grafting. Temporary coverage with cadaver skin or biological dressing as above may be necessary in larger burn wounds.

In burns exceeding 40-50% TBSA, autologous cell cultures (keratinocytes, fibroblasts) may provide good coverage of the burned surface. The definitive role of this therapy is still to be confirmed.

American Burn Association White Paper. Surgical Management of the Burn Wound and Use of Skin Substitutes


Complications of fluid management (‘fluid creep’)

The Parkland formulae (2-4 mL/kg/%TBSA) for burn injury resuscitation was launched in the late sixties, and has been the cornerstone for fluid management in burns ever since. In later years there has been a tendency to give larger volumes of resuscitation fluid for a number of reasons:

First, and possibly most important, is the use of central circulation surveillance techniques (pulse contour measurement and echocardiography) to assess fluid status of the patients in parallel to using the endpoints suggested by the Parkland formulae (urine output and mean arterial pressure). It is then evident that in the normal patient there is central circulation hypovolaemia if the Parkland formulae is adhered to, especially at 12 hrs post burn. Thus, conventional burn fluid resuscitation (Parkland formula) is a permissive ‘hypovolaemia’ strategy. Secondly, it is claimed that patients are currently more likely to be intubated and sedated with large doses of sedatives and analgesics. This practice leads indirectly to larger fluid volume needs to maintain blood pressure and urine output. Thirdly, as mortality for patients with the largest injuries is declining, resuscitative measures are more often
performed for these individuals and their fluid needs are very large. We emphasise that larger volumes than recommended by the Parkland formula may lead to significant complications – most importantly, compartment syndromes involving both extremities and the abdomen. The latter complication carries a significant mortality when it occurs and underlines the need for abdominal compartment pressure monitoring in major burns. In an often cited paper by Oda, the risk for abdominal compartment syndrome is lower if less than 300 mL/kg/24 hrs are provided (other authors set the risk at 250 mL/kg/24h = Ivy index).

**NOTE** Keep in mind that large fluid volumes and water deposition in the tissues lead to hypoperfusion and ischaemia. Hypoperfusion may be especially unfavourable for the burn-injured skin and therefore should be avoided.


**Preventing unwanted ‘fluid creep’**

First, the utilisation of urine output is a robust endpoint (0.5-1 mL/kg/hr) can be relied upon in most cases. Exceptions to this strategy are patients with altered kidney function, cardiovascular morbidity or systemic sepsis if later in the clinical course. In such patients, the use of colloids (so called ‘colloid rescue’) before 24 hrs may be considered.

The ‘permissive hypovolaemic’ strategy requires close medical supervision, and implies tolerating moderate hyperlactatemia and acidosis. Vasopressors (norepinephrine or vasopressin) and inotropes (dobutamine) are recommended if the blood pressure and/or cardiac output is low. It has been suggested however that avoiding colloids for the first 8 hrs post burn is preferable in the expectation of minimising colloid leak into burn injury-induced, capillary leak tissue – thus risking aggravation of tissue oedema. It is important to stress that vasopressors can have negative effects on the skin as the skin has a high density of alpha-1 receptors. There is a potential risk for tissue ischaemia when such drugs are used extensively and in high doses.


Inhalation injury

Incidence and diagnosis

The incidence of inhalation injury ranges from 5% to 30% of ICU admissions after burn injury and is suggested by the history and the examination of the patient. Hot gases can damage the lung as far as the terminal bronchioles, while smoke injury can extend more distally. The reflex vocal cord closure is not always sufficient to prevent penetration. The chemical products harm the respiratory mucosa. Diagnosis of the extent of the inhalation injury should be carried out on admission to the specialised burn facility with a separate Ear, Nose and Throat (ENT) specialty evaluation of the oro-pharynx and tracheobronchial tree - overlooking an ENT injury carries the risk of inappropriate early extubation.

Standardising the description of inhalation injury - a descriptive score (See Ikonomidis C et al. 2012 below)

<table>
<thead>
<tr>
<th>Signs and symptoms</th>
<th>Mucosal airway appearance at the oro-pharyngeal and/or tracheobronchial levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial burns</td>
<td>Grade I: Erythema + mucosal oedema</td>
</tr>
<tr>
<td>Hoarseness</td>
<td>Grade II: Bullous mucosal detachment, erosions, exudates</td>
</tr>
<tr>
<td>Singed nasal vibrissae and oropharynx</td>
<td>Grade III: Stenosis, profound ulcers, partial necrosis</td>
</tr>
<tr>
<td>Carbonaceous deposits and sputum</td>
<td><strong>NB!</strong> Presence or absence of soot with any grade</td>
</tr>
<tr>
<td>Cough, stridor</td>
<td></td>
</tr>
<tr>
<td>Wheeze</td>
<td></td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td></td>
</tr>
<tr>
<td>Depressed mental status</td>
<td></td>
</tr>
</tbody>
</table>

Airway management and ventilator treatment

**Immediate action:** Any evidence of inhalation injury should prompt tracheal intubation - in controlled circumstances. Delay in securing the airway can potentially compromise oxygenation and access to the airway very rapidly, rendering intubation impossible due to the development of facial and upper airway oedema during fluid resuscitation. Suxamethonium (together with sedation/anaesthesia) can be used safely to facilitate intubation until 48 hrs after a major burn injury.

In patients with deep injuries and/or oedema, intubation may be impossible. Case reports indicate that both percutaneous and surgical tracheostomy are safe in the acute setting when performed by skilled doctors. Importantly, in oedematous patients, the conventional tracheostomy tube might be too short, so that a tube with a adjustable flange will be required. There is no clear consensus on indications or the correct timing of tracheostomy in patients with large burns.

Inhalation injury prolongs time on mechanical ventilation and distal injury to the tracheobronchial tree has a major effect on the duration of intubation. No mechanical ventilation technique has proven superior: local team competencies being the most important determinant in the choice of the mode.
See the PACT module on Airway management

**NOTE** When inhalation injury is suspected, the indication to intubate should be broad and intubation carried out promptly. Late intubation is sometimes impossible leading to death by asphyxia, while extubating a patient without injury is simple and can be done rapidly after confirmation of absence of inhalation injury.

### Specific measures after inhalation injury

Lack of diagnostic criteria and large randomised trials make specific therapeutic recommendations in patients with inhalation injury difficult. Since 100% oxygen reduces the half-life of carbon monoxide (CO) it should be initiated as soon as possible (see CO intoxication). Once carboxyhaemoglobin levels have normalised, FiO₂ should be reduced. In a review article from 2011 on modern burn treatment, Kasten suggests that aggressive pulmonary toilet, nitric oxide, nebulised heparin, N-acetylcysteine, and/or bronchodilators may be considered in patients with inhalation injury. However with the exception of for aggressive broncho-pulmonary toilet, these measures are not standard care.


### Carbon monoxide poisoning

A high index of suspicion should always be maintained for carbon monoxide (CO) poisoning, particularly in high-risk injuries such as burns suffered in enclosed spaces and in patients with associated injuries which may have altered the level of consciousness. CO poisoning is responsible for many early deaths in burn victims due to anoxic encephalopathy.

**Diagnosis**

Carbon monoxide poisoning requires blood gas analysis by CO-oximeter, which will give accurate measurements of oxyhaemoglobin, carboxyhaemoglobin (COHb) and methaemoglobin. Arterial or venous blood can be used. Arterial blood gas analysis using the conventional blood gas machines may only demonstrate a metabolic acidosis.
Clinical signs of CO poisoning related to the blood level of COHb:

<table>
<thead>
<tr>
<th>Blood Level of COHb</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.3-0.7%</td>
<td>None</td>
</tr>
<tr>
<td>2.5-5.0</td>
<td>↓ blood flow to vital organs, angina on exertion</td>
</tr>
<tr>
<td>10-20</td>
<td>Headache, slight dyspnœa, lethal to fetus</td>
</tr>
<tr>
<td>20-30</td>
<td>Nausea, severe headache, throbbing temples, gushing, ↓ manual dexterity</td>
</tr>
<tr>
<td>30-40</td>
<td>Severe headache, vertigo, nausea, vomiting, weakness, irritability, impaired judgment, syncope</td>
</tr>
<tr>
<td>40-50</td>
<td>As above but more severe with syncope and collapse</td>
</tr>
<tr>
<td>50-60</td>
<td>Coma, convulsions, Cheyne-Stokes respiration</td>
</tr>
<tr>
<td>60-70</td>
<td>Depressed respiration and cardiac function, death possible</td>
</tr>
<tr>
<td>70-80</td>
<td>Generalised depression, death</td>
</tr>
</tbody>
</table>

Note: non-smokers may have up to 3% COHb, while smokers may have up to 10-12% COHb

Oxygen saturation by pulse oximetry will be normal, even when there is tissue hypoxia due to severe CO poisoning, because of similar wave length absorption. All burn patients should be given supplemental oxygen pending availability of carboxyhaemoglobin (COHb) levels.

Pathophysiology

CO acts by binding tightly to haemoglobin displacing oxygen, and ultimately resulting in cellular hypoxia and acidosis. Intracellularly, CO also inhibits mitochondrial cytochrome c oxidase function, and such inhibition could participate in acute symptoms and clinical outcome of CO poisoning, classically attributed to COHb-mediated hypoxia.

Treatment

As CO has an affinity for haemoglobin which is 250 times greater than for O₂, the management of any suspected CO poisoning entails the administration of supplemental O₂ at as high a concentration as possible. The administration of 100% O₂ reduces the half-life of carboxyhaemoglobin (COHb) from 4-6 hours to 60-90 minutes.

As CO binds even more avidly to fetal than adult haemoglobin, infants and pregnant patients present further management difficulties.

Hyperbaric Oxygen therapy: Some would regard severe metabolic acidosis and myocardial ischaemia as possible indications but most clinicians only consider hyperbaric oxygen therapy in the following circumstances:
Altered mental state
- Loss of consciousness
- Focal neurological deficits
- Seizures
- Pregnancy with COHb levels >15%

**NOTE** While hyperbaric oxygen treatment was considered a standard after an early (prematurely interrupted) trial, more recent evidence has caused enthusiasm to fade. In 2011, the debate continued (see references below) as repeated hyperbaric treatment had the potential to worsen outcome in comatose patients. Oxygen (100%) administration remains the primary treatment.

**Outcome**

**NOTE** Survivors of severe CO poisoning are at risk of delayed neurological sequelae. About 40% patients develop mild to severe symptoms 3 to 240 days after apparent recovery.


**Cyanide poisoning**

Cyanide (CN) poisoning is still an often-overlooked diagnosis in fire victims, particularly in closed space burns. Clinical features are dose-dependent, starting with anxiety, progressing to dyspnoea, seizures, coma and cardiorespiratory arrest.

**Diagnosis**

CN poisoning presents a diagnostic dilemma for first-response emergency personnel. While clinicians are often able to diagnose CO poisoning by either arterial- or venous blood sampling to measure COHb or by oximetry (with reasonable accuracy), CN poisoning is less clear cut. Baud et al. found that the concentration of lactate increases proportionally with the amount of CN poisoning because of the related metabolic acidosis. An early arterial lactate level >5 mmol/L should raise suspicion.
Although diagnosing CN poisoning is a challenge in the emergency setting, immediate treatment is of utmost importance. Given the fact that methods to detect and measure CN in blood are usually not readily available and that patients may often be exposed to both CO and CN, clinicians have to rely on the presenting symptoms and the general status of the patient. In patients hospitalised with a history of a fire accident, combined with severe neurological compromise (reduced Glasgow Coma Scale Score), soot particles in the mouth or from tracheal expectoration raise the suspicion of concomitant CN poisoning.

Any unconscious fire victim should be considered as CO or CN poisoned in absence of any evidence of traumatic brain injury and treated as such.

Pathophysiology

Hydrogen cyanide (HCN) is easily absorbed from all routes of exposure. Since HCN is a small lipid soluble (mainly undissociated) molecule, distribution and penetration of HCN into cells is rapid. In most severe cases the symptoms are unconsciousness, convulsions, cardiovascular collapse followed by shock, pulmonary oedema and death. Virtually all patients with severe, acute CN poisoning die immediately. Autopsy findings include petechial, subarachnoid or subdural haemorrhages.

The similarity between CO and CN is the ability to bind iron ions. However, where CO impairs the ability of erythrocytes to transfer oxygen, CN binds to erythrocytes but does not affect the oxygen transfer. The primary effect of CN is a blocking of the mitochondrial respiration chain and thereby blocking the formation of intracellular adenosine triphosphate (ATP). The result is cytotoxic hypoxia caused by the inhibition of Cytochrome C Oxidase (CCO) by the high affinity of CN to heme a3 of the enzyme. The effect is a structural change, reduced activity of the enzyme and an increase in lactate production resulting in metabolic acidosis.

Treatment

In the emergency setting, treatment is given based on the presumptive clinical diagnosis. Oxygen in combination with a recommended antidote should be administered immediately, the first to reduce cellular hypoxia and the second to eliminate cyanide. A specific antidote is hydroxocobalamin, which can be given intravenously (70 mg/kg via a peripheral IV line - the usually described initial dose being 5g) with nearly no side effects. Hydroxocobalamin turns the urine and sometimes skin purple. Other antagonists (nitrites and thiosulfate) can be used but are more difficult to administer.
Emergency treatment includes 100% O₂, tracheal intubation in case of unconsciousness, symptomatic treatment of seizures and hypotension, and administration of an antidote as soon as available. A commonly used antidote is hydroxocobalamin (Cyanokit).

Cyanide may contaminate the rescuers!
In the case of chemical disasters, caregivers should wear chemical-protective clothes with positive-pressure breathing apparatus when they are at risk of being contaminated. Any contact with salts or solutions containing cyanide or inhaling hydrogen cyanide gas from vomitus must be avoided. Contaminated clothes should be removed and bagged.


Electrical injuries

Electrical injury can occur in isolation, or in association with thermal injury. Burn casualties with associated electrical injuries should be referred to a specialised burn centre. See http://www.ameriburn.org

Severe electrical injuries, excluding lightning strikes, tend to occur in the workplace. Injuries are caused by current passing through the body and by the heat generated, and secondarily as a result of prolonged muscle contractions stimulated by the current. An electrically injured patient should be managed as a multiple trauma patient. For more information see the PACT module on Multiple trauma.

Rescuer safety, always a priority, is especially important at the scene when treating these patients. Always ensure the electricity source has been made safe.

Prolonged muscle contractions in these patients can result in secondary fractures and dislocation injuries, including injuries to the vertebral column. Full spinal immobilisation precautions are indicated.

NOTE: Immediate death from electrical injury occurs as a result of ventricular fibrillation, asystole, or because of respiratory arrest secondary to central nervous system damage or sustained respiratory muscle contraction.

The severity of the injury sustained depends on the following:

- Voltage encountered
  - >1000 V: high voltage injury
  - 220-1000 V: intermediate voltage injury
  - <220 V: low voltage injury
- Resistance of the patient (moisture will reduce the resistance)
- Route taken through the body
- Duration of contact

Low and intermediate voltage injuries may be complicated by local tissue destruction but are not commonly associated with multisystem damage. High voltage injuries can be complicated by severe burns, myocardial necrosis, cardiac dysrhythmias, central and peripheral nervous system damage, visceral injuries, limb ischaemia, compartment syndromes, rhabdomyolysis, and ultimately multiple organ failure. The extent of injury may not be initially evident, as the injury at the contact or entry and exit points may not appear very severe.

NOTE: The terminology ‘contact points’ is used for most i.e. alternating current injuries. Entry and exit point terminology is used for lightening or other direct current injury.

There should be a low threshold for exploring muscular compartments.[Further images in interactive version]. Serial estimations of creatine kinase, myoglobin and frequent measurement of potassium levels may help in detecting rhabdomyolysis and the urine may have a characteristic colour - see image.
Symptomatic patients (including those with loss of consciousness), those with abnormal ECG or a history of cardiac disease, and all patients exposed to intermediate and high voltages require continuous cardiac monitoring for a minimum of 24 hours post injury.

There is no specific therapy for electrical injuries. Management is symptomatic and supportive.

High voltage electrical injury patients need higher volumes of resuscitation fluid than other burn injuries. The goal of resuscitation is a urine output of >100 mL/h to prevent myoglobin toxicity causing acute renal failure.


5/ CRITICAL CARE CONSIDERATIONS SPECIFIC TO THE BURNED PATIENT

Infection

Surveillance and blood cultures

For baseline purposes, obtain wound surveillance cultures on admission in patients with large burns. If the patient has been hospitalised in another facility, most units will take specimens to identify multiresistant microbes e.g. MRSA (methicillin resistant staphylococcus aureus), VRE (vancomycin resistant enterococcus) and other pathogens. Blood cultures may also be taken.

Thereafter, specimens should be taken at the time of septic work-up when indicated by clinical infection. If respiratory infection is suspected and the patient is intubated, it may be wise to include a protected (brush or other) specimen via the endotracheal tube.

Ongoing surveillance cultures may be taken once a week. Wound surveillance swabs are preferably accomplished after wound cleaning, but despite correct sampling interpretation of surface cultures may be confounded by extensive colonisation. Skin biopsies are much more specific of infection, but seldom used.

More than 50% of the patients have transient bacteraemia after dressing changes and surgery. Such cultures are not regarded as indicative of systemic infection or of a need for antimicrobial therapy. Therefore as a guide to starting antibiotic therapy, blood cultures should not be taken immediately after dressing changes and surgery.

THINK Do not take blood cultures from established arterial or venous lines, since these are often contaminated due to hub colonisation with bacteria and fungi. Cultures from new, aseptically inserted venous and arterial catheters are of course the equivalent of ‘clean-stab’ blood cultures.


**Invasive infections**

Infection is a major cause of morbidity and mortality in burn patients. Due to the broken skin barrier, central i.v. catheters and invasive ventilator therapy, patients with burn injury are susceptible to infections as early as 24-72 hrs after the incident. Wound and pulmonary infections are the most common. Early extubation is important to avoid ventilator associated pneumonia (VAP). Patients with inhalation injury are particularly susceptible to lung infections, and some of them develop the Acute Respiratory Distress Syndrome (X-rays in interactive version). Initially, Gram-positive bacteria (skin organisms) predominate, and the most common microbes are Staphylococcus epidermidis and aureus. With increasing length of hospital stay, Gram-negative organisms such as Pseudomonas sp. [image in interactive version] are common pathogens. Another major problem is the more resistant Gram-positive organisms such as MRSA and VRE. Fungal infections also appear later, especially if broad spectrum antibacterials have been used for a long time.

The common signs of infection such as pyrexia, leukocytosis, and other elevated markers of inflammation are frequently seen in burn injury patients in the absence of invasive infections. This makes diagnosis tricky. It is important for the clinician to monitor the patient over time to tailor antibiotic treatment. In addition to the classical signs of invasive infection, evaluate the general clinical condition of the patient [higher fever (> 39 °C), tachycardia, hypovolaemia, increasing CRP, PCT, blood sugar and increased gastric residual volume] as indirect signs that might point to an invasive infection. Always do a septic work-up incorporating new swabs, protected (brush) sampling (if indicated and the patient is intubated), urine and blood cultures before antibiotics are introduced or changed.

**NOTE** Sepsis is the major cause of death in patients who survive the initial burn injury. Remember that central catheters, arterial lines, gall bladder, sinuses, endocarditis and decubitus ulcers might be the source of an intercurrent infection.

**Antibiotics**

Prophylactic antibiotics should not be given. Aim for short courses of narrow-spectrum antibiotics targeted to clinical, invasive infection and modify/refine therapy based on microbiological results.

Perioperative antibiotic use during wound debridement and grafting remains controversial, but is commonly administered for 24 hrs perioperatively in patients with large burns. The antibiotics used should be dependent on the microbes identified in that particular patient or those usually present in the unit. A common combination is an aminoglycoside and penicillinase resistant penicillin.

The pharmacokinetics are different in burn patients requiring larger doses and more frequent dosing of antibiotics. A longer therapeutic course may be indicated if the burn wounds are purulent; the agent of choice being guided by recent (septic work-up or surveillance) culture results. As the volume of distribution is unpredictable and generally significantly increased, antibiotic concentrations should be monitored; subtherapeutic concentrations may contribute to the selection of resistant microorganisms.

For more information see the PACT modules on Severe Infection and Sepsis and MODS.
**Metabolic alterations and nutrition**

The metabolic response to major burns (>20% TBSA) is characterised by an initial ‘ebb phase’ followed within 24 to 48 hrs by the ‘flow phase’, i.e. to a hypermetabolic response.

The metabolic disturbances are qualitatively similar to those of other critically ill patients (hypermetabolism, increased gluconeogenesis, insulin resistance, increase in endogenous lipolysis and net loss of the lean body mass) but are much more intensive and persistent (weeks to months).

The cytokine and mediator release is characterised by the intensity and the prolonged persistence of the oxidative stress. This response is due to the time required for burn wounds to heal with the response abating after closure of the wounds. The increases in stress hormones (catecholamines) are particularly marked.

The body’s response includes an elevation in body temperature, cardiac output and substrate turnover rate, causing an important increase in energy demand. The elevation in energy expenditure (EE), is directly related to several factors including the extent of the burn injury, time elapsed since injury and the presence of septic complications. There is an important variation over time in the EE which is difficult to predict, rendering indirect calorimetric measurements particularly valuable. Current burn injury treatments appear to have attenuated the hypermetabolic response compared to data from the 1980s. The peak of hypermetabolism lasts from 5 to 21 days after injury, and declines progressively thereafter.
Nutritional support

See the PACT module on Nutrition.

Energy requirements

Several predictive energy requirements equations have been developed for burn injury with the intention of preventing the acute loss of lean body mass. A typical example was the Curreri equation which has led to massive and deleterious overfeeding and these equations should be definitively abandoned.

Setting an energy target in patients with major burns is challenging in the absence of indirect calorimetry. However, the Toronto equation (further detail within interactive version) based on the study of a large patient population represents a good alternative to indirect calorimetry, and includes several important factors such as the burn size (%TBSA), previous day’s feed (last 24hrs’ caloric intake = CI), Age, Sex, and Weight (derived from the predicted resting EE via the Harris & Benedict equation), fever (T°C), and time after injury.

\[
\text{Total EE} = -4.343 + (10.5 \times \%\text{TBSA}) + (0.23 \times \text{CI}) + 0.84 \times \text{REE H-B}) + (114 \times T^{\circ\text{C}}) - (4.5 \times \text{days})
\]

Nutrition route

In burns, early enteral nutrition (EN) is particularly important as it contributes to improved splanchnic perfusion during the early phase of resuscitation, prevents ileus, and attenuates the metabolic response to injury. Gastric EN should be introduced within the first 12 hrs after injury. Postpyloric feeding may be required in patients with the largest burns.

Parenteral nutrition should be limited to the cases of intestinal failure with contraindication to EN. Failure is defined as the incapacity to deliver more than 60% of requirements calculated by the Toronto equation for more than four consecutive days by the postpyloric route, which is extremely rare in burn patients, who tolerate feeding rates up to 150 mL/hr for ‘catch up’ feeding after interruptions such as after repeated daily surgical procedures.
Lam NN, Tien NG, Khoa CM. Early enteral feeding for burned patients--an effective method which should be encouraged in developing countries. Burns 2008; 34(2): 192-196. PMID 17804169

**Macronutrients**

The nutritional requirements are particularly increased for glucose and proteins. There is a maximal oxidation rate for glucose of 5 mg/kg/min for adults and children that should not be exceeded to avoid the development of fatty liver. Protein requirements are increased to 1.5-2 g/kg/day.

Lipid delivery should probably be tightly controlled to <1.5 g/kg/day. A recent Cochrane survey shows that there may be a possible clinical benefit to high-carbohydrate enteral feeds compared with high-fat enteral feeds in the treatment of burn injuries with a reduced risk of pneumonia.


**Micronutrients**

Trace elements are lost in large amounts with the exudative losses in adults and children. The losses cause trace element deficiencies in patients with major burns, if not compensated (particularly selenium, zinc and copper). Early substitution, with doses that represent 6-10 times the recommended parenteral doses, is associated with improved wound healing and reduction of infective complications.

Vitamin requirements are increased in parallel with anabolic requirements. Deficiencies, particularly of vitamin C and vitamin D, have been repeatedly demonstrated and can be prevented by systemic early administration in addition to amounts present in enteral nutrients.

High-dose ascorbic acid (66 mg/kg/hr for the first 24 hours) has been used to stabilise the endothelium and reduce the capillary leak.


Non-nutritional modulation of metabolism

Decreasing the loss of lean body mass requires a multimodal approach that includes nursing in a warm environment (28-32°C) particularly during surgery and in case of open wound therapy, early surgical debridement and grafting, appropriate analgesia and sedation, pharmacological attenuation of the hypermetabolic response, and stimulation of anabolism.

Propranolol, by attenuating the sympathetic response and its non-selective metabolic beta-2 blocking actions, strongly reduces catabolism and reduces cardiac work load in children and adults. Anabolic agents, such as insulin and oxandrolone, accelerate wound healing and shorten hospital stay. Oxandrolone started within 7 days of injury has been shown to improve long term outcome in both adults and children in prospective randomised trials. Recombinant growth hormone (GH) probably has its place in the treatment of children (but not in adult burns patients) as children with burn injuries have a proven GH deficit.


**NOTE** Patients with large burns are strongly catabolic, and weight loss is considerable; this response can be reduced by beta-blockade and liberal protein supply.

Williams FN, Herndon DN, Kulp GA, Jeschke MG. Propranolol decreases cardiac work in a dose-dependent manner in severely burned children. Surgery 2011; 149(2): 231-239. PMID 20598332


Multiple organ failure

It was appreciated early in the burn community that infective complications lead to multiorgan failure (MOF), poor outcome and even death. Sheridan and colleagues, were the first to report that burn patients dying in MOF did not have a high rate of positive blood cultures in the later phase of their care, but succumbed during so called ‘sterile’ conditions. This finding was important as it underlined that infection is not always the immediate cause of death for this patient group. Some patients also develop sepsis, having had MOF of a different aetiology. This led to the idea that burn injury and the inflammatory reaction secondary to it were important and significant contributors to the development of MOF in burn injury.

The Sheridan paper also described the order in which each organ failure occurred. Early effects were seen on lung function (ARDS) and later effects on kidney function (AKI). However, the strength of any conclusion of this finding is affected by the specific circumstances of burns where inhalation injury may concomitantly have been present and therefore blurred the diagnostic specificity. In these studies, and especially important from an outcome perspective, organ effects on the lung-kidney axis were detected. Today, evidence has been put forward that suggests common underlying mechanisms for the effects on these two organs. From the kidney perspective, it is also important to stress that there does not seem to be an on-off phenomenon for kidney injury as a part of MOF but more of a continuum ranging from no injury, to a full-blown acute renal failure (RIFLE criteria; see the PACT module on Oliguria and anuria (AKI part I). Despite improved survival in burn-related organ failure patients, high mortality rates still apply to overt kidney failure patients post burn. Another important finding in burn critical care has been the close correlation between age and the development of MOF. This finding has surpassed in importance the previously identified close relationship between %TBSA and MOF. This finding is interesting as it suggests that genetic factors are also important in the development of MOF.

In the last 20 years, burn care has improved significantly as measured by a decrease in length of stay and most importantly by a decrease in mortality. One important consequence of the present picture of MOF is that it frequently is curable and the mortality rates of patients with increasing number of organs failing is less than previously documented. This has led to the aggressive surgical approaches used in larger burn injuries and to a reduction in do not resuscitate (DNR) orders in some TBSA% ranges. See ethics below.
Analgesia, sedation and delirium

Also read the PACT module on Sedation and Analgesia.

Pain - General aspects

Pain is a major concern in patients with burn injury, and both adults and children experience tachyphylaxis to opioids. This effect is most evident in patients on the ventilator. The need for analgesics varies considerably between patients. In the early phase, most patients need general anaesthesia during dressing changes (see Perioperative management section below on general anaesthesia) due to otherwise unbearable pain and anxiety. However, burn wounds heal and over time the need for analgesics decreases as the general condition of the patient improves. To avoid complications associated with opiate and benzodiazepine withdrawal, most burn patients need weaning from medication rather than abrupt cessation. A significant number of burn patients have pre-existing addiction problems. Long-standing addiction issues may impact on their analgesic requirements and increase the likelihood of withdrawal problems.

Treatment - Clinical aspects

1. In non-ventilated patients: Combine peripheral and centrally acting analgesics. Start by combining paracetamol (10-15 mg/kg) given four times daily (QID) with morphine - starting with low dosage and titrate to effect. Different formulations of paracetamol are available, the preferred being by the oral/enteral route. Very large doses of morphine (>100 mg/24hrs) even in small children might be needed. When the opioid dose is stable, preferably use long acting drugs. Alternatives to morphine are oxycodone, ketobemidon and alfentanil. Usually intermittent injections and not infusions are used in spontaneously breathing patients due to the risk of respiratory depression. Both in children and adults one can use morphine infusion, 10-30 µg/kg/hr in spontaneously breathing patients - always monitor to avoid respiratory depression.

2. In ventilator dependent patients: For short-term sedation, propofol may be used (maximum dose 4 mg/kg/hr) combined with an opioid (morphine, fentanyl, alfentanil, remifentanil). For long-term sedation, midazolam in combination with an opioid (morphine, fentanyl, alfentanil, remifentanil, 

Fitzwater J, Purdue GF, Hunt JL, O'Keefe GE. The risk factors and time course of sepsis and organ dysfunction after burn trauma. J Trauma 2003; 54(5): 959-966. PMID 12777910

Steinvall I, Bak Z, Sjoberg F. Acute respiratory distress syndrome is as important as inhalation injury for the development of respiratory dysfunction in major burns. Burns 2008; 34(4): 441-451. PMID 18243566


Hydromorphone is preferred. In the presence of ‘creeping requirements’ for opioids, ‘opioid rotation’ should be considered enabling the halving of the previous dose for equivalent pain reduction. Later the introduction of methadone (via the nasogastric tube) with its NMDA effects may also help control analgesia.

3. To reduce tachyphylaxis, ketamine 1 mg/kg/24 hrs as a continuous infusion in addition to opioids is an option.

4. Some units use patient controlled analgesia (PCA).

5. Some units use subcutaneous infusions containing a mixture of opioid, alpha-2 agonists, ketamine, and even the non-analgesic haloperidol to attenuate agitation.

6. Intravenous infusion with dexmedetomidine (0.2-0.7 µg/kg/hr) during weaning seems promising.

7. Gabapentin may be used to relieve phantom pain.

As soon as the patient is extubated, the use of nonpharmacological methods such as hypnosis should be considered: hypnosis enables the reduction of anaesthesia requirements for painful procedures.


**Delirium**

**Definition and prevalence**

Delirium is defined as an acute change in or fluctuating mental status that includes inattention, disorganised thinking, and an altered level of consciousness, with or without agitation (Diagnostic and Statistical Manual of Mental Disorders, 4th ed.; DSM-IV). In intensive care patients, delirium is associated with a poorer clinical outcome and death. However, data on delirium in burn patients are scarce, and in a recent study Argarwal et al. found that delirium occurred at least once in 77% of the study population. In this study, exposure to benzodiazepines was an independent risk factor, while i.v. opioids and methadone were associated with a lower risk of delirium.

**Prevention**

Optimal pain control is probably a main measure to prevent delirium in general intensive care and burn patients. Noise reduction and making a distinction between day and night are other important factors. Ensure that hearing aids and glasses are updated and in good condition.

**Treatment**

Experts consider that antipsychotic drugs are the most effective in all types of delirium (haloperidol 0.5 mg -10 mg x 2), the dose to be reduced when symptoms improve; alternatively or additionally olanzapine 5-10 mg). However, in delirium caused by alcohol or sedative hypnotic withdrawal, benzodiazepines are the treatment of choice, complemented in time by clonidine (600-1200 µg/day). Levomepromazine is not advocated to treat delirium in ICU patients.

www.icudelirium.org


See also the PACT module on Sedation and Analgesia.
6/ PERIOPERATIVE MANAGEMENT OF INTERCURRENT SURGICAL EVENTS

Most of this Task does not deal with critical care considerations per se but more specifically refers to the anaesthetic management during dressing change and wound surgery which is likely to be conducted by specialist anaesthesiology staff. The details are included here however for the sake of critical care staff in situations where they are also the anaesthesiologists (as in Scandinavia) and to provide some general background information on perioperative considerations for non-anaesthetic, critical care staff.

Anaesthesia

During dressings

In the initial phase of burn treatment, dressing changes are usually performed under general anaesthesia and the expertise/assistance of an anaesthesiologist will be required. If the patient is on the ventilator, one can add propofol in anaesthetic doses and additional opioids (alfentanil, fentanyl). The needs for opioids might be large. Later, when the patient is extubated, dressing changes are performed during spontaneous breathing. In this setting propofol and alfentanil (titrate the doses, slow induction with propofol to avoid apnoea) are a good combination in adults. The most important issue is to give adequate doses of analgesics. Due to tachyphylaxis, the needs might become very high (>0.7 mg of fentanyl in a 70 kg patient for a 60 min procedure). In children, ketamine 2 mg/kg with additional doses of 0.5-1 mg/kg after 10-15 minutes works well. To calm the child and the parent, it is smart to start anaesthesia with a small dose of i.v. midazolam, 0.5-2.5 mg dependent on the weight of the child. If venous access is difficult to achieve, i.m. ketamine or rectal ketamine (5-10 mg/kg) and midazolam (0.5-2.5 mg) mixed in the same syringe are options in the youngest children.

During burn surgery

Together with wound revisions/dressing change procedures, surgical debridement constitutes the mainstay of wound care. These events pose significant challenges for both the patient and the burn team and may involve significant risks. It is therefore mandatory that these procedures are undertaken by a prepared team with well planned protocols. There are a number of issues that are specific for the perioperative care and anaesthetic handling of these patients that needs to be appreciated for a good outcome.

The main problems specific for burn surgery are related to the risk of circulatory compromise due to:

- The early large fluid needs due to the burn injury in both burn-injured and in non-injured tissue due to the negative imbibition pressure (injured tissue) and the vascular permeability increase (non-injured tissue) that occurs early after the injury.
- Extensive and difficult to quantify blood loss during burn surgery is common, especially involving surgery of the face, neck, hands and torso.
- Septic complications. These are often less frequent very early in the course
but this risk increases over time. Also, the patients may have ongoing septic problems, where the treatment is surgery and wound revision.

Added to these points are other specific needs of these patients.

- Difficulties in maintaining body temperature.
- Specific needs for ventilator support as, in the larger injuries, there is a risk for early ARDS and the possible effects of inhalation injuries.

With these listed problems, good outcome crucially depends on good patient monitoring. Therefore most units use:
- Pulse oximetry/capnography;
- Invasive blood pressure measurements,
- Central venous pressure monitoring,
- Cardiac output/systemic vascular resistance assessment.
- Repeated measurements of haemoglobin, (or haematocrit) and blood gas assessment including Na/K and glucose are common during surgery in the most severely injured patients.

In cases with concomitant respiratory compromise, conventional lung mechanics parameters (airway pressures; dynamic and static compliance) provided by the ventilator are also used for surveying the status of the patient.

**Perioperative considerations**

*Early fluid treatment during surgery*

In cases where surgery is performed within 24 hrs post burn, the regular strategy for fluid treatment is followed (Parkland formulae). This strategy is not altered as surgery is performed. Many units provide blood transfusion according to losses and with no further addition. At times evaporative losses may be larger and these should then be included also in the calculation of fluid losses. It is important to note that after the start of surgery, the diuresis often decreases due to the added stress response of surgery and attention should be directed to the fluid balance. When the necrotic skin is removed, diuresis may increase.

**NOTE** Significant intraoperative fluid loss is common in this group of patients. Haemorrhage may be complicated by coagulopathy, requiring transfusion of blood and blood products. Care is taken to limit the potential for hypothermia.

*Perioperative blood loss and severe sepsis*

The dominant risk and difficulty with burn surgery has been the blood loss encountered in these events, when at the same time it is difficult to assess quantitatively how large this loss is. Furthermore intraoperative bacteremia and sepsis may contribute to the hypotension.

A common rule is to pay extra attention to the blood pressure recordings in addition to the assessment of the wounds. Decreasing blood pressure is a sign of a negative transfusion balance. Other measures that can help in this assessment are pulse pressure variations (with respiration) and effects on the plethysmographic part of the oximetry signal. Blood loss may vary substantially between patients and burn excision in the face, neck, torso and hands constitutes the major problem. It underlines the need to follow the circulatory surveillance parameters.
Know your hospital guidelines for the management of massive haemorrhage.

Choice of anaesthetics/techniques

For intubation, proper muscle relaxation is needed and suxamethonium can be used for the first 48 hrs post burn. Thereafter it should be avoided as it causes hyperkalaemia and may lead to malignant dysrhythmias.

Some units base their anaesthesia on propofol and an opiate whereas others use inhalation agents. There is no evidence for any advantage of either. Postoperatively, pain is a major problem and proper analgesia needs to be administered prior to the end of surgery. Also it is important to dose the anaesthetics according to effect rather than on a mg/kg or minimal alveolar anaesthetic concentration (MAC) % basis as the pharmacokinetics may be altered by the burn injury.

Regional blocks may be recommended for cases where such techniques are practical i.e. regionalised injuries in areas feasible to anaesthetise. In general such blocks are made by single injections rather than leaving indwelling catheters because these may become infected over time as burn victims often have repeated bacteraemia.

Haemostasis and co-operation with the surgical team

One important task for the anaesthetic team is to optimise circumstances for a good surgical outcome. There are a number of issues that need to be addressed in this aspect. For burn surgery the most important features include:

- Maintaining tissue perfusion so that the burn depth assessment is facilitated. If the patient is hypotensive, necrosectomy may be too shallow and also haemostasis may be inappropriate when normotension is achieved later.
- Aiding in provision of coagulation support. Coagulation support can be managed by transfusing platelets, coagulation factors (plasma) or desmopressin in cases of large blood volume losses. International practice in recent years has favoured repeated smaller excisions, i.e. 20% TBSA daily for a number of days rather than one very large excision at the cost of a significant blood loss. Biological dressings have also been increasingly used for similar reasons, as they seem to have coagulostatic properties themselves.

SURGEONS: it is important to undertake actions to reduce blood loss. Techniques include using tourniquets in extremity surgery; using adrenaline/noradrenaline soaked gauze intra-operatively along with tempered sodium chloride solutions and subcutaneously infiltrating noradrenaline. In developing countries, intravenous vasopressin has been used for these purposes successfully. However, vasoconstrictive drugs may negatively affect skin blood flow and reduce skin graft take rate. Unfortunately few studies are available to guide treatment strategies.

There is a shortage of good evidence-based guidelines on how to address perioperative blood loss and coagulation in burns.
Another important complication during burn surgery is the septic effects on the circulation and haemostasis. Such effects may be significant but are most common in large burns and later during the course of treatment. In such cases infection surveillance and diagnosis and the choice of appropriate antibiotics are essential.

**Active temperature control during surgery**

One very important topic in burn surgery is temperature control. The patient is losing heat for several reasons: evaporative losses as dressings are removed; a nonfunctional skin barrier for temperature control; and cooling as surgical washing of the skin is made. These reasons make it mandatory to have a close temperature surveillance (e.g. via bladder catheter) and using active heating devices, the most successful being convective heat devices. Other means are keeping the operating room warm, warming the ventilator breathing system (circuit), warming all fluids given to the patient and limiting the amount of time in the operating room.

![Warning](image)

**Hypothermia prevention: give this high priority!**

Hypothermia favours coagulation disorders with bleeding and worsens outcome! Increased mortality has been shown in patients with delayed recovery of normothermia after surgery!

See PACT module on Bleeding and thrombosis.

Yowler CJ. Recent advances in burn care. Curr Opin Anaesthesiol 2001; 14(2): 251-255. PMID 17016410


Epidemiology and prevention

Burns are the most common cause of accidental death in children 0-14 years of age in the United States after car accidents and drowning. In the western world, scald is the most common reason [images in interactive version].

In 1946 Emlyn Lewis, Gloucester City General Hospital, wrote in the British Medical Journal urging legislative interventions to prevent burn injuries in small children from electric fires and gas stoves, a matter still in focus today. Measures to prevent burn injury in children are to teach children about fire prevention and to keep dangerous materials out of reach in particular in the kitchen. Supervision during fireworks is critical.

Child abuse

Distinguishing intentional injuries from accidents is challenging. Maguire and colleagues performed a systematic review to characterise whether the mechanism of injury in burns was suspected abuse. Dipping injuries caused by hot tap water which were identified in children with intentional scalds, were symmetrical and with distinct upper margins, located on extremities and/or buttocks. Unintentional scalds were most often localised in the upper right part of the body, i.e. the face, neck and right upper extremity. Typically these wounds had irregular margins and depths (see figs 3,4,5 in second Maguire reference, below)


http://ep.bmj.com/content/95/6/170.long

How does burn care in children differ from burn care in adults?

Airway and intravenous access

The smallest children often need general anaesthesia (with or without intubation) if central venous access is needed. Most experts advocate a CVC in burns >15%-20% of TBSA or if peripheral venous access is difficult. Further, the airway is small and if the burn is located in the face, neck and upper part of the trunk, the child is often intubated. If one suspects a compromised airway, intubation should be performed without delay. However, it is important to be aware that children that stay intubated are easily overhydrated as they have an increased production of antidiuretic hormone (ADH). SIADH (syndrome of inappropriate anti diuretic hormone secretion) may occur. Fluid overload often leads to respiratory failure and ventilator-associated pneumonia.
(VAP) prolonging ventilator therapy. When the injury involves the lower part of the body, try to avoid intubation except during necrosectomy and grafting. Dressings can be performed under ketamine anaesthesia under spontaneous ventilation. If intubation is needed for procedural purposes; the anaesthesiologist is usually willing to extubate after finishing the procedure.


Circulation

Delayed venous access and a late start with volume resuscitation increases mortality. If venous access is difficult, an intraosseous access is a good alternative in the acute phase. An indwelling urinary catheter is mandatory in the resuscitation phase in children with injury >12-15 %TBSA. Aim for a urinary output of 1 mL/kg/hr in children and 2 mL/kg/hr in infants. Be aware that children have a major ability for physiological compensation, and hypovolaemia is often not recognised before >25% of circulating volume is lost. To diagnose hypovolaemia in a child, one should assess mental status, pulse pressure, arterial blood gases (low base excess), increasing lactate and colour/temperature of the extremities.

A child with clammy, cool extremities but with normal blood pressure and heart rate is a child in danger.

Surface area and fluid resuscitation

Small size makes children challenging for physicians and nurses, who are often more used to working with adults. It is important to recognise that fluid losses are proportionally larger in children than in adults due to a large surface area compared to fluid weight. The rule of nine does not apply to children less than 15 years of age, and the Lund and Browder chart should be used to calculate burned surface area. Venous access may be particularly difficult in pre-hospital settings; intraosseous access is particularly well suited.

To calculate body surface, one can use a simple nomogram or a formula [interactive version].

The Parkland formula is unsuitable for children less than 30 kg BW but a modified version is shown below. The most common formula used for fluid resuscitation in children is based on surface area:

\[ 5000 \text{ mL/m}^2 \%\text{TBSA burned (due to the injury)} + 2000 \text{ mL/m}^2 \text{ TBSA (maintenance fluid)}. \]

This formula favours over-resuscitation which is a risk in children as much as in adults. The maintenance fluid should probably be reduced (as outlined in the adult section) but there is not specific, strong evidence on this point.
One can also use a modified Parkland formula; 3 mL/kg/%TBSA and add basal needs.

**Monitoring:** Urinary output and capillary refill are good measures for volume status in the burned child. Measurement of intra-abdominal pressure is frequently not possible for technical reasons, which complicates assessment of over-resuscitation.

**NOTE:** Fluid losses are proportionally greater in children than in adults. Evaporative water loss in a 20%TBSA burn in a 10 kg child is 475 mL or 60% of circulating blood volume. The corresponding fluid loss in a 70 kg adult is 1100 mL or 25% of blood volume.

Children seem more sensitive to circulating protein (albumin) dilution than adults. An alternative in the early phase is the more liberal use of fresh frozen plasma whenever coagulation laboratory abnormalities are detected. Nevertheless, as in adults, the abnormal coagulation tests reflect dilution and primarily prompt limitation of fluid administration. The argument used to support a broader use of albumin is that children seem to be particularly sensitive to oncotic pressure shifts, but this requires further research as data are conflicting.


Zdolsek HJ, Lisander B, Jones AW, Sjöberg F. Albumin supplementation during the first week after a burn does not mobilise tissue oedema in humans. Intensive Care Med 2001; 27(5): 844-852. PMID 11430540

**NOTE:** Always keep the child warm. A drop in body temperature is deleterious.


**Delirium and pain**

Children often express pain differently from adults and may become agitated or depressed. Further, regression is a common problem. After extubation the child is often agitated and experiences withdrawal; this might compromise the airway and ventilation so that re-intubation may become necessary. To avoid re-intubation due to pain and delirium, small doses of continuous morphine (20 µg/kg/hr) combined with dexmedetomidine (0.2 µg/kg/hr) may be considered in such children.
Insufficient analgesia and withdrawal after extubation may lead to re-intubation.

In a specialised burn unit, mortality is 5-7%, including patients where life-sustaining treatment is futile and is withheld. Age and %TBSA are strongly related to survival. A patient 18 years of age with a 80 %TBSA burn injury may have a probability of survival of >50%. Survival rate declines with increasing %TBSA and increasing age. Inhalation injury also significantly contributes to a reduced chance for survival. Mean length of stay (LOS) in the burn unit is about 1 day per percentage TBSA affected by burn injury.

Quality of life is reduced in former burn patients and there is an over-representation of later accidental death in patients surviving severe burn injury. Also many burn patients have pre-existing psychiatric or addiction problems which may complicate recovery. Even if they did not have these problems beforehand, they frequently have them after a large or life-threatening burn injury. To handle this problem, a multidisciplinary team approach is advocated.

To facilitate physical rehabilitation it is important that burn patients, both children and adults, have the benefit of physiotherapy from the day of admission, including when on a ventilator. The main goal is to prevent contractures and restore function, as well as minimising scar formation. Physiotherapists are important and necessary members of the multidisciplinary team treating burn patients.


Ethics

Patients with large burns that survive carry the sequelae of the injury for life. The injuries are often overwhelming both for the patients and next of kin. Due to a good prognosis, even in patients with very large burns (>80% TBSA), primary treatment with escharotomy and necrosectomy are instituted. Active treatment may evidently become futile leading to their withdrawal in some patients. Palliation is a team/consensus decision performed in full transparency with relatives and sometimes the patient. Palliation and withdrawal of interventive treatment in a severely burned patient is demanding, especially if the decision is made in a local hospital with limited experience in such desicions. Usually the family expects that all treatment options are performed. Doctors and nurses may also have different opinions on when to stop active treatment in patients with poor prognoses and very limited rehabilitation potential. When the decision to withdraw active treatment is made, it will often take some time before the patient passes away. This situation puts a lot of stress on doctors, nurses and family. Burn patients without a realistic hope of survival according to clinical evaluation and to survival criteria should be offered palliation and good general care at the local hospital near family and friends. The decision for palliation should preferably be made in collaboration between the local hospital and the burns unit. A transfer to the burn centre will sometimes be
performed in this setting. If prognosis appears poor and palliation seems to be the best choice after an overall evaluation, the decision to terminate active treatment should be made as early as possible in the treatment course.

For further reading see the PACT modules on Ethics and Communication.


Burn care research

Although the research activities have increased in the burn care community throughout the years, a significant portion of usual burn care practices still lack a strong scientific basis. Despite this, mortality has decreased significantly, length of stay has dramatically been shortened and long-term outcome assessed as health related quality of life has improved. However, mainstream burn care does still not fulfill the highest evidence-based medicine (EBM) criteria. At the same time, it is important to realise that a significant amount of knowledge used in burn care and other care specialties, especially trauma and critical care, are based on early experiences in the care of the burn injured. This applies especially to fluid therapy, knowledge on the trauma response and the complications in the post trauma care period and the nutritional support. Lately, important research within burn care has been conducted in the field of modification to the trauma response - such as pharmacological modulation of this specific trauma response.

Within the burns specialty, the focus is now directed at improving research quality and there is a strong demand for more randomised controlled trials supporting present care and identifying better care strategies.

http://www.elsevier.com/wps/find/journaldescription.cws_home/30394/description #description) and the journal of Burn Care Research http://journals.lww.com/burncareresearch/pages/default.aspx


In patients with burns covering more than 10-15 % of TBSA, fluid resuscitation should be initiated early.

Administer the fluid orally or intravenously. Fluid resuscitation in the initial phase is dynamic. Nurse driven protocols may reduce the susceptibility to over-resuscitation. Aim for a urinary output of ½-1 mL/kg/hr in adults. Never use thermodilution techniques to measure fluid needs in the initial phase (0-24 hrs) as it causes fluid over-resuscitation.

Fluid induces oedema, and facial burns may necessitate early orotracheal intubation to secure the airway.

Impaired ventilation and/or compromised peripheral circulation due to deep or circumferential burns should be managed by early escharotomy at the primary hospital.

Respiratory distress is most often due to vigorous fluid resuscitation, secretions, pneumonia and/or other cause of systemic sepsis. Fibre optic bronchoscopy may reveal inhalation injury and enables removal of sequestered material or retained secretions from the airways.

In the acute, initial phase, hypotension is usually caused by hypovolaemia. Subsequently a massive inflammatory response (SIRS) causes vasodilatation and hypotension despite an increased cardiac output.

Wound and airway infections are common. SIRS may cause CRP levels above 100 mg/L and a body temperature of 38- 39 °C which makes it difficult to define the precise onset of infection and to identify the right time to start antibiotic treatment. Prophylactic use of antibiotics is not encouraged.

Definitive surgery, excision and grafting, should be performed early, preferably within the first week.

Patients with large burns should be treated according to general principles for intensive medical care, preferably in units with special experience in treatment of burns.
PATIENT CHALLENGES

A 22-year-old female was admitted to the Emergency Department following an indoor bioethanol fire. On site, her wounds which involved her face and neck were cooled. At admission, she was awake with a GCS of 15 and her body temperature was 34 °C. Her BP was 95/71 mmHg, heart rate 75/min, respiratory rate 22/min, and SpO2 on mask oxygen (35%) was normal. Body weight was estimated at 60 kg, height 160 cm.

Q. What is your initial response?
A. Resuscitate according to ABCDEF principles.

Q. Outline your specific approach under each of these six headings:

1. Airway:
A. Ensure a patent airway with cervical spine protection. Look for signs of inhalational injury or airway burns. Give oxygen by face mask at as high a concentration as possible. This is especially important where there is a possibility of carbon monoxide or cyanide poisoning, when SaO2 can be misleading.

2. Breathing:
A. Ensure adequate air entry and symmetrical chest movement.

3. Circulation:
A. Establish peripheral intravenous access and put in a urinary catheter. Prepare for central venous access dependent on the extent of the burn injury.

4. Disability:
A. Assess neurological deficit.

5. Exposure:
A. Assess %Total Body Surface Area (%TBSA) burned, is the injury circumferential, what is its depth? Consider treating/preventing further hypothermia.

6. Fluid:
A. Calculate fluid volume already delivered: if burns appear to be >20% BSA, administer calculated fluid needs according to 50% of the Parkland formula. Aim for a urinary output of 0.5-1 mL/kg/hr. At the moment, the size of the burn has not been formally evaluated. Resuscitate adequately but be cautious concerning administered fluid - do not over-resuscitate.

Learning Issues

Think ABC first
Initial resuscitation specific to burns (The Parkland formula) and the dynamic approach to fluid resuscitation

Hypothermia in burn patients

Blood was drawn for full blood count, urea and electrolytes, coagulation screen, cross-match and arterial blood gases. You consider the likelihood of carbon monoxide and cyanide poisoning when requesting laboratory testing. A blood culture was also taken.

**Learning Issues**

Carbon monoxide poisoning

Cyanide poisoning

Primary and secondary survey revealed extensive burns to upper and lower extremities, chest, back, neck and face – estimated at 65% of total body surface area. There was no definitive sign of inhalation injury.

**Q. How will you assess this patient and her burns?**

A. Burn wounds should be evaluated for extent, depth, and circumferential components – as urgent escharotomy may be required. The ‘Rule of Nines’ is commonly used for rapid initial estimation of burn extent. Remember the possibility of non-burn injuries.

**Q. How would you modify this assessment in a child?**

A. The ‘Rule of Nines’ is less accurate in children as the head represents a much larger proportion of total body surface area. The use of an age-specific grid such as the Lund-Browder diagram will give the best estimation of burn extent in children.

**Learning Issues**

Assessment of the burn injury – evaluate %TBSA

Area of burn – ‘Rule of Nines’. The palm of the hand with fingers is 1% of TBSA

Depth of burn

The need for escharotomy

Non-burn injuries

Paediatric differences (Lund-Browder)
Q. What are the warning signs for airway burns injury?
A. Those with facial burns, those caught in enclosed areas or who were intoxicated or asleep when burned or who have clinical signs indicative of airway burn injury viz. hoarseness, singeing of nasal vibrissae, soot-staining, oropharyngeal burns.

Q. In patients with obvious evidence of airway burns, what is the approach to airway management?
A. These patients should have their airway secured with a tracheal tube.

Q. Why is early/pre-emptive tracheal intubation indicated?
A. A delay in airway management may result in a difficult or impossible intubation later when tissues become grossly oedematous secondary to the burn injury.

Q. Does the oxygen saturation of 98% with a respiratory rate of 22 per minute and a normal chest X-ray (CXR) rule out a respiratory injury in this patient? Does this patient require respiratory intervention?
A. The possibility of heat, and/or chemical injury to the respiratory tract and upper airway must be considered in every burn patient. Due to the history and nature of the injury including the involvement of the face and neck, most clinicians would intubate this patient.

Q. Will a conventional approach to the intubation be appropriate? Are there any specific concerns? Which drugs will you use?
A. You can use a conventional technique, but be aware that she possibly is hypovolaemic due to the injury. Her blood pressure will probably drop lower than it currently is after the administration of I.V. sedative/anaesthetic medication. You probably do not need neuromuscular blocking agents (NMBAs) for intubation. Suxamethonium can be used without danger of hyperkalaemia up to 48 hrs after the injury.

**Learning Issues**

Airway inhalational injury

Early pre-emptive tracheal intubation

Choice of N MBA if necessary
With due preparation and skilled assistance, the patient was uneventfully sedated, intubated and ventilated. Bronchoscopy was normal. Initial burn injury estimation of size showed:

40% full-thickness burns
33% partial thickness burns

Regarding ongoing fluid therapy, you are aware that the Parkland formula is the most widely used of the many regimens advocated (all based on the area of burn injury) and you continue fluid therapy according the ‘50% Parkland formula’ above. You know that some use crystalloid as the sole resuscitation fluid, whereas others use a combination of crystalloid and colloid but that no one formula has been shown to be superior to another.

Q. Are there clinical issues to be factored into the fluid therapy plan or is straightforward adherence to the formula appropriate?
A. While the formulae are useful in the initial resuscitation, they must be used in conjunction with ongoing haemodynamic and clinical monitoring to avoid over-resuscitation.

Q. Given that you are using the ‘50% Parkland formula’, give the precise calculation of this patient’s fluid requirements for the next 24 hours and how will you monitor?
A. 2 mL (instead of 4 mL)/kg × 65% × 60 kg = 7,800 (instead of 15,600 mL) and reassess according to haemodynamic response.

Q. What will be the primary, single monitor of the effectiveness of your fluid regimen?
A. A targeted urinary output =0.5 mL/kg/hr (and not preload) will be the primary monitored measurement.

Q. Presuming you will use crystalloid resuscitation, which fluid will you give in the next 24 hours? How will you space out the fluid administration?
A. Ringer’s Lactate (or Acetate). Of the calculated volume, 50% will be prescribed for the first 8 hrs, the rest in the next 16 hrs.

Q. How will you monitor ongoing fluid requirements and adequacy of resuscitation?
A. Urine output should be 0.5-1 mL/kg/hr. Serial base excess (BE), lactate and haemoglobin measurements can be useful. Fluid resuscitation in the initial phase is a dynamic process.
The importance of regular reassessment of adequacy of resuscitation cannot be overstated. Guidance of therapy based primarily on measurement of preload (with non-invasive haemodynamic monitoring or PA catheter) may lead to too much fluid administration.

**Learning Issues**

Initial fluid resuscitation in a severely injured burn patient

Monitoring fluid requirements

How to deal with the ‘fluid creep’

Blood pressure is low and urinary output is nearly zero, and you have administered fluid well in excess of the volume calculated by the Parkland formula.

**Q. How can this situation be improved?**

A. Presuming euvolaemia has been achieved, consider vasoactive infusion therapy via a CVC - the agent(s) chosen being guided by the clinical pointers to the primary cause for the hypotension (vasoplegic vasodilation or cardiac dysfunction).

**Learning Issues**

Use of pressors/inotropes for hypotension and oliguria

**NOTE** Diuretics are not indicated in this phase.

The burns on the thigh and right forearm are circumferential, compromising the vascular supply to that hand, therefore requiring urgent escharotomy. She is therefore transferred to the operating room for an escharotomy during which procedure, she remained stable.

**Learning Issues**

Urgent escharotomy for limb salvage
After wound debridement and escharotomy, the patient is stable and normothermic and you now consider that this patient should be transferred to a burns centre. You call the centre and begin to organise the patient transport.

Q. How do you describe your patient and the clinical scenario to the burns centre?

A. Inform them that you have a patient that you want to transfer. Describe the patient’s clinical situation on arrival to your hospital (awake, stable, mechanism of injury, depth and extent of injury), intubated after admission, her resuscitation and monitoring procedures to date, how much fluid and the surgical procedures she has had.

Learning Issues

Planning medical management including consultation and referral

Transfer to the Burns Unit

PACT module on Patient Transportation

The patient is transferred to the Burns unit less than 24 hrs after the injury. Reassessment of the wounds is performed, and she is scheduled for surgery the next day.

Intra-operatively she remained haemodynamically stable although blood loss reached 2000 mL requiring transfusion of two units of RBC and four litres of crystalloid in addition to her ongoing burn resuscitation fluid. On return to the Burns unit, she becomes hypotensive (BP 75/40 mmHg) with tachycardia of 130 bpm, including frequent ventricular ectopics.

Q. Given that the most likely aetiology in this situation is hypovolaemia, describe your initial management?

A. Give an i.v. fluid bolus: 500 mL crystalloid or colloid and assess for ongoing bleeding and check the haemoglobin (Hb).

Q. The haemoglobin was <70 g/L, and the patient was given red blood cells. Is this a reasonable approach?

A. Yes, although most would probably administer colloid/plasma in this setting. However, it may be argued that the haemoglobin trigger was too low (especially where bleeding continued) and that further blood transfusion was indicated. Some burns centres aim for a Hb of 10 g/dL (100g/L).
Q. If bleeding continues despite an absence of a surgical bleeding site, what is the approach?
A. Check platelet and coagulation indices (in the aftermath of a substantial fluid/blood transfusion) and prescribe therapy (usually fresh frozen plasma and platelets) as indicated.

**NOTE** Old hydroxyethyl starch HES solutions are associated with long lasting pruritus.

After surgery she is hypothermic and hypotensive, but after some hours of fluid management she stabilises. On day four, temperature approaches 40 °C, CRP is 300 mg/L and PCT is 10 µg/L. Blood pressure is normal. PaO₂/FiO₂ ratio is declining (20 kPa).

Q. Sepsis is the apparent cause of this deterioration. What is the likely aetiology?
A. In view of her deteriorating respiratory state, a pulmonary source is likely but, in patients with thermal injuries, a wound infection is always a possibility. Differentiation will require clinical evaluation.

**Learning Issues**
- Sepsis
- Burn wound infection
- Respiratory infection

Q. In your search for the source of infection, what will be the target(s) of your clinical evaluation?
A. The search for a source of sepsis will include: respiratory and urinary systems; operative site, (in this case extensive burn wounds); indwelling catheters including CVC access and arterial access and other bloodstream infections (including cardiac valves). Other possibilities are acalculous cholecystitis, *Clostridium difficile* infection (particularly if the patient has been on antibiotics and/or has diarrhoea), sinusitis, otitis media, and decubitus ulcers.

**NOTE** If, on clinical examination there is no other evident source for the sepsis, and CRI (catheter-related infection) is suspected, the CVC, arterial catheter or other intravascular device is removed or changed (and catheter tip and blood cultured).
Sepsis diagnosis

Catheter-related infection (CRI)

See PACT modules on Sepsis and MODS, Severe infection and Pyrexia (for diagnosis of CRI).

Specimen from a protected brush, (large volume of purulent sputum noted), urine, blood and swabs from burn sites have been sent to lab for microscopy, culture and sensitivity (MCS) analysis. Apart from an increasing leukocytosis, all other bloods including liver function tests are unremarkable. The first of three faecal samples for *C. difficile* toxin has been collected. All catheters have been changed. CXR shows a new left-sided consolidation; she now requires FiO₂ >80% to maintain saturation and she has become hypotensive despite increasing doses of vasopressor.

Q. She has an evident respiratory infection. How will you treat it?

A. Once the diagnosis of sepsis is made, immediate empirical antibiotic therapy is indicated pending laboratory results from the diagnostic specimens sent at the time of the septic work-up. Augmented supportive respiratory therapy will likely also be required.

Q. Given that this is a nosocomial pneumonia, what will be your approach to antimicrobial therapy?

A. Antibiotic therapy may be guided by urgent sputum microscopy or by recent microbial surveillance data, but should be broad and incorporate good Gram-negative cover. If resistant organisms such as MRSA are common in the unit, then antibiotics active against these organisms should be considered. Fungal infection is common in burn patients, usually several days post injury.

Q. Although, a pulmonary infection is the apparent cause of this deterioration, what other infections may co-exist in this burn injury patient?

A. In burn injury patients, always consider a wound infection and see that all necrotic tissue is excised. If there is clinical evidence or suspicion of *C. difficile* infection, oral metronidazole is indicated, pending toxin results.

PACT module Sepsis and MODS

PACT module Severe infection

She was commenced on piperacillin/tazobactam together with fluconazole and oral metronidazole and rapidly improved in the following 48 hours. Cultures from the protected brush specimen showed a heavy growth of sensitive Pseudomonas sp. as did her burn site.
In the absence of significant fungal growth, with all samples negative for *C. difficile* toxin, the fluconazole and metronidazole were discontinued.

**On reflection:**

Burns injury may be severe and life-threatening and require standard, prompt resuscitative management from the outset. Peculiar to Burns injury is the specific need for pre-emptive airway control when there is evidence of inhalation injury and for fluid management to take account of the percentage of total body surface area (TBSA) involved. Remember that non-burn injuries and related (carbon monoxide and cyanide) poisoning may co-exist.

In circumferential burns on extremities, thorax and/or abdomen, it is highly important to perform escharotomy at the admitting hospital. Escharotomy facilitates ventilation and saves peripheral tissue. Although standard formulaic fluid therapy is appropriate, fluid over-resuscitation should be avoided. Fluid therapy in the early phase (0–24 hrs) is dynamic and aims for a urinary output of 0.5–1 mL/kg/hr in adults.

If indicated, surgery with excision and grafting should start within 24–72 hrs. During and after surgery, hypovolaemia is likely due to considerable blood-loss and requires active management.

There is a potential for hypothermia and because of the broken skin barrier and the need of artificial ventilation, the burn patient is particularly susceptible to sepsis due to wound infection and ventilator-associated pneumonia. Sepsis is promptly and actively treated with supportive and specific measures but pre-emptive (prophylactic) antibiotics are not advocated.

Multiorgan failure is however common and is related to patient mortality but historical evidence suggests that outcome is improving. Length of stay in hospital is 1–1.5 days per percentage of burn injury.