EC EMERGENCY MEDICINE AND CRITICAL CARE
Review Article

Recent Advances in Burns Critical Care

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Abstract

The critical care management of burned patients has evolved over last 2 decades and like other critical diseases, burned patients have a fortunate outcome. The time has come when modern burn care should be institutionalized in all tertiary care centres dealing with burns. We reviewed over 76 articles on burns intensive care and proposed some conclusive guidelines that may help a lot of burn care doctors. The important aspects of burn intensive care are fluid management, early recognition of sepsis, maintenance of organ perfusion and early intubation in those with inhalation injuries.

Keywords: *Burns; Critical Care*

Introduction

The critical care of burns has revolutionized over the last two decades. Correct initial management can make the difference between a good and a poor outcome. Therefore, emergency medicine physicians, Intensivists, plastic and general surgeons and anesthesiologists 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 [1].

Modern burn care may be divided into the following 4 general phases [2]:

- Initial evaluation and resuscitation; occurs on days 1 - 3 and requires an accurate fluid resuscitation and thorough evaluation for other injuries and comorbid conditions.
- Initial excision and biologic closure; includes the maneuver that changes the natural history of the disease; this is accomplished typically by a series of staged operations that are completed during the first few days after injury.
- Definitive wound closure; involves replacement of temporary wound covers with a definitive cover; there is also closure and acute reconstruction of areas with small surface area but high complexity, such as the face and hands [2].
- Rehabilitation, reconstruction, and reintegration; although this begins during the resuscitation period, it becomes time consuming and involved toward the end of the acute hospital stay [2,3].

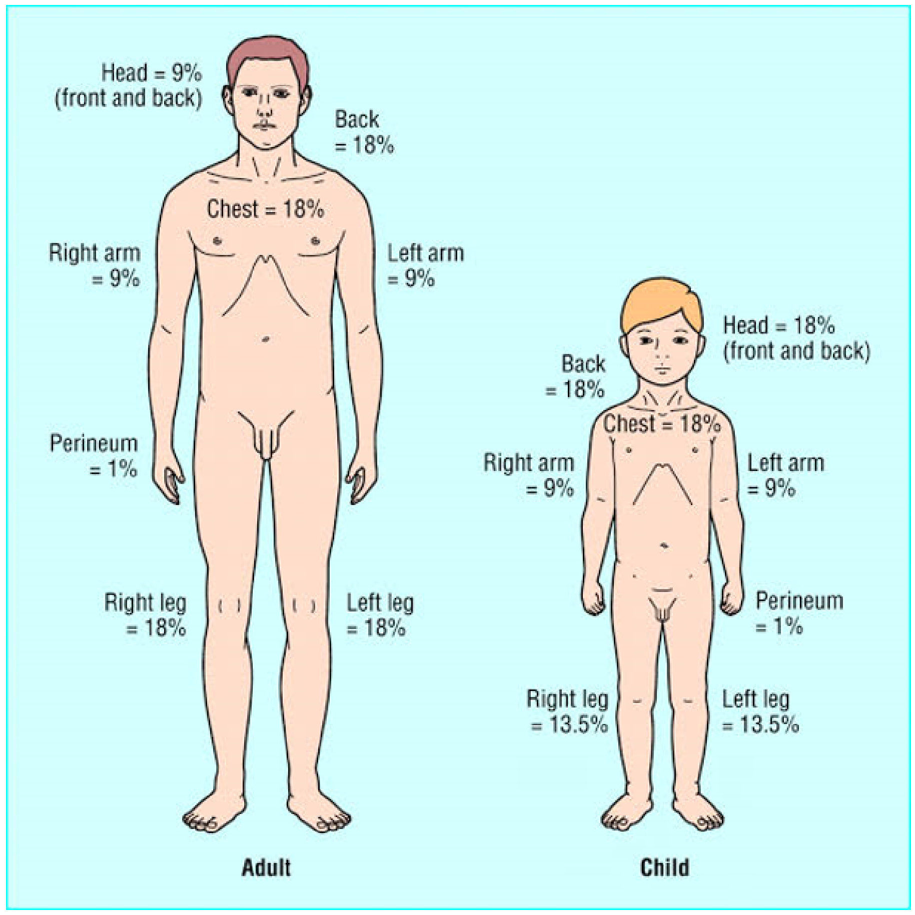
Pathophysiology of burn shock

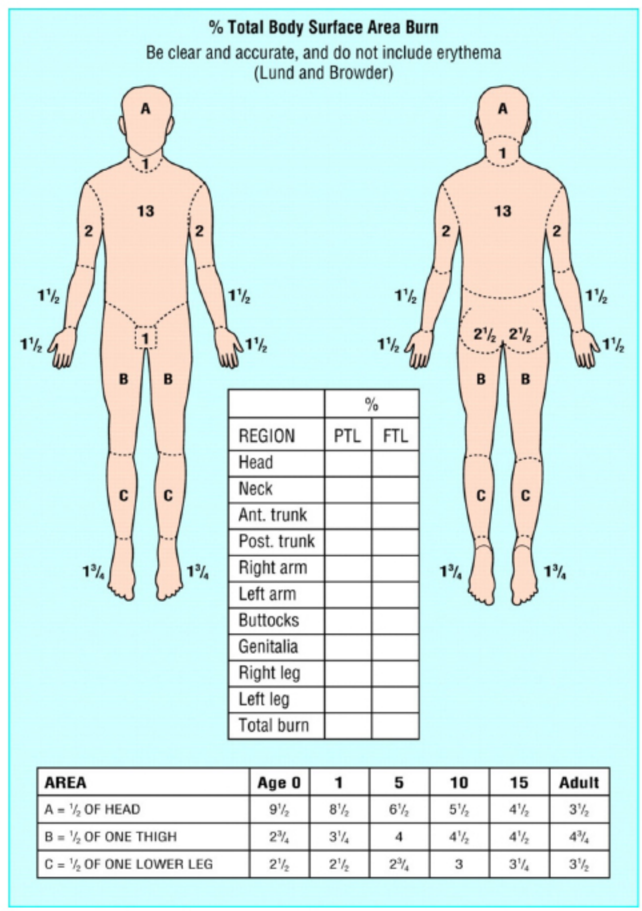
Burn injury results in cardiogenic, hypovolemic and distributive shock. The intravascular volume becomes depleted primarily due to increased capillary permeability and fluid shifts. Above 30% total body surface area (TBSA), only partial compensation can be achieved by fluid resuscitation due to a generalised reduction in sodium ATPase activity and disruption of the cellular transmembrane ionic gradient that persists for several days. Microvascular injury secondary to inflammatory mediators such as histamine, bradykinin, prostaglandins, leukotrienes, vasoactive amines, platelet activation products and complement allows protein loss into the interstitium. The intravascular colloid osmotic pressure falls and fluid escapes the vascular system. The result is a loss of intravascular fluid, electrolytes and proteins with rapid equilibration with the interstitial compartments. Clinically, this is manifested by hypovolaemia, haemoconcentration, oedema, reduced urine output and cardiovascular dysfunction. Adequate resuscitation from burn shock is a critical therapeutic intervention in burn management [4].

Burn sepsis

Preliminary diagnosis of post-burn sepsis can be made if six out of the first 11 criteria below are met. This preliminary diagnosis can be confirmed if any one aspect described in the last criterion is met.

The diagnostic criteria are: (1) mental excitement, hallucinations, disorientation or depression; (2) abdominal distension, diminished bowel sound; (3) rapidly deteriorated burn wounds, exhibited as wet, dark and/or deepened wounds with necrotic spots, and so forth; (4) core temperature > 39.0°C or < 36.5°C; (5) increased heart rate - adults > 130 times/minute, children of all ages > 2 standard deviations of normal value; (6) increased respiratory rate - adults >28 times/minute (without mechanical ventilation), children of all ages > 2 standard deviations of normal value; (7) thrombocytopenia - adults < 50 × 10^9/l, children of all ages < 2 standard deviations of normal value; (8) peripheral white blood cell count - adults >15 × 10^9/l or <5 × 10^9/l, in which neutrophil percentage > 80% or immature granulocytes > 10%, children of all ages >2 or <2 standard deviations of normal value; (9) blood procalcitonin > 0.5 μg/l; (10) blood sodium levels > 155 mmol/l; (11) blood glucose > 14 mmol/l (no history of diabetes); and (12) positive blood culture or positive response to antibiotic therapy [5,6].





% Total Body Surface Area Burn

Be clear and accurate, and do not include erythema

(Lund and Browder)

	%	
REGION	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	Age 0	1	5	10	15	Adult
A = ½ OF HEAD	9½	8½	6½	5½	4½	3½
B = ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C = ½ OF ONE LOWER LEG	2½	2½	2¾	3	3¼	3½

Fluid management (0-24 hours) and permissive hypovolemia

Fluid management has always been a cornerstone in critical care of burned patients. 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. Estimating the total burned body surface area is a key component for resuscitation. Fluid resuscitation by formula is recommended if the TBSA is more than 15%. All current formulas and methods for resuscitation in burned patients are based on body weight and the percentage of TBSA burned. The initial history and physical exam should also include the body weight and an estimate of the second and third degree burn areas used to calculate resuscitation. The "rule of nines" allows a simple, rapid estimate of TBSA: (in an adult [units = % TBSA]): entire head and neck = 9; each arm = 9; upper 1/2 torso front = 9; lower 1/2 torso front = 9; upper 1/2 torso back = 9; lower 1/2 torso back = 9; each leg = 18- anterior leg = 9; and posterior leg = 9 [9-11].

Over-resuscitation has become a major problem, 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 consensus or '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 mL x BSA (m^2) x (% burn/100)], 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. Objectives of fluid resuscitation pharmacological approach) in adults (combined fluid and Mean arterial pressure > 60 mmHg) [9].

Mean arterial pressure	> 60 mmHg
Heart rate	< 120/min
Central venous oxyhaemoglobin saturation	> 60%
Diuresis/output	> 0.5 ml/kg/hr > 1 ml/kg/hr in paediatrics
pH	7.30 - 7.45
Lactate	< 2 mmol/l

Table1: *Therapeutic end-points of fluid administration.*

There continues to be no consensus regarding the timing of colloid initiation. Fluid extravasation has been shown to stop by 8 to 12 hours, and there is no evidence to indicate the need to delay colloid administration beyond this. Early colloid actually appears to have a pulmonary volume-sparing benefit]. O'Mara and colleagues randomised patients to either a plasma or crystalloid-only resuscitation group, using the Parkland formula to calculate initial fluid requirements and measured intra-abdominal pressures [13]. Although they did not find an improvement in overall outcomes using plasma resuscitation, they did show a reduction in overall fluid loading and significantly lower intra-abdominal pressures (26.5 vs. 10.6 mmHg, P <0.0001) in the plasma treated group [13].

After 24 hours begin colloid infusion of 5% albumin 0.3–1 ml/kg/%TBSA.

Dextrose water or dextrose saline is used for additional fluid requirements to maintain output > 0.5 ml/kg/h [15].

Role of vitamin-c in fluid management

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 [10].

However, more RCTs are required to implement its routine use in large burns intensive care.

Normoglycemia and intensive insulin therapy

The hypermetabolic state provides glucose to glucose-dependent tissues, it ultimately contributes to immune dysfunction, sepsis and organ failure. Holm and colleagues identified increased adverse outcome in adults following hyperglycaemia in the first 48 hours post injury [13-15].

The paediatric burn literature showed that hyperglycaemia is associated with increased catabolism, bacteraemia/fungaemia, skin graft loss and mortality whilst intensive insulin therapy was associated with survival [17-21]. The Normoglycaemia in Intensive Care Evaluation and Survival Using Glucose Algorithm Regulation Investigators assessed glucose targets in a mixed ICU population, using intensive insulin control versus conventional insulin control (4.5 to 6 vs. 8 to 10 mmol/l) in more than 6,000 patients and reported lower mortality in the conventional group (24.9 vs. 27.5%, P = 0.02) [21]. Following subgroup analysis, however, the trauma group (not stipulated whether this included patients with burns) benefited from tight glycaemic control. Control of hyperglycaemia can be difficult in burn patients and intensive insulin treatment can lead to a higher risk of hypoglycaemia. Studies examining the effect of tight glycaemic control in burns are limited, but mortality benefits as evidenced by Van den Berghe and colleagues [21,22] and others has led to the control of hyper glycaemia becoming a standard of care. Insulin has additional immunomodulatory effects beyond increasing cellular uptake of glucose. Insulin has a trophic effect on mucosal and skin barriers, reducing bacterial invasion and translocation, improves wound matrix formation and inhibits the production of proinflammatory mediators. The glucose levels should be kept as normal as possible and if required, insulin should be started [20].

Vasopressors and inotropes

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 improve hemodynamics [23].

Surgical management

Surgical management of burns is beyond the scope of this article. 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 [26]. 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 mid-axial lines are performed. Ideally, the incision should extend just beyond the area of the full thickness burn. Digital incisions are made in the mid-axial 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 [23-27].

Nutrition

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 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 24 hrs' caloric intake = CI), Age, Sex, and Weight (derived from the predicted resting EE via the Harris and Benedict equation), fever (To), and time after injury [29].

$$\text{Total EE} = -4.343 + (10.5 \times \%\text{TBSA}) + (0.23 \times \text{CI}) + 0.84 \text{ days}) \text{ REE H-B}) + (114 \times T°\text{C}) - (4.5 \times \text{days}).$$

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 [23,24].

Hypermetabolism

Burn-induced catabolism results in accelerated protein breakdown and reduced protein synthesis, culminating in a negative net nitrogen balance. Demling and de Santi reported that oxandrolone (an oral anabolic agent) improved weight gain and was an unquantified measure of muscle function [24] and in a later study showed a reduction in weight loss, urinary nitrogen loss and a shortened time to wound healing [25]. A prospective multicentre randomised double-blind trial of 81 patients with burns of 20 to 60% TBSA found that starting enteral oxandrolone 5 days post injury significantly reduced the hospital stay (45.3 vs. 32 days, $P = 0.035$) without significant adverse events [26]. The hypermetabolic response is in part mediated by endogenous catecholamines: shortly after severe burn or trauma, plasma catecholamine levels can increase up to 10-fold. β-blockade following severe burns can reduce supraphysiologic thermogenesis, tachycardia, cardiac work and resting energy expenditure. Herndon and colleagues conducted a prospective randomised control trial in paediatric patients with severe burns to evaluate the effect of propanolol on muscle catabolism [28]. The net muscle–protein balance increased by 82% from baseline values in the propanolol group ($P = 0.002$) and was significantly higher with respect to the control group ($P = 0.001$). The mechanism of action of propanolol remains unclear but appears to be a result of increased protein synthesis during a period of persistent protein breakdown and reduced peripheral lipolysis [29].

Burns related acute kidney injury

Burn-related kidney injury is typically classified as early (0 - 3 days after injury) or late (4 - 14 days after injury). Early burn AKI is typically due to hypovolemia, poor renal perfusion, direct cardiac suppression from TNF-alpha, and precipitation of denatured proteins, while late AKI is often due to sepsis, multi-organ failure, and nephrotoxic drugs. Diagnosis can be difficult as UOP and biochemical markers can be relatively normal even with significant renal injury [27].

A sensitive and specific biomarker for the early diagnosis of AKI is sorely needed, and multiple potential biomarkers are being investigated. For treatment, the reversal of the underlying cause is the first intervention. The advent of renal replacement therapy has significantly improved the mortality of burn patients with AKI and should be initiated early if injury progresses despite initial maneuvers. Unfortunately, no beneficial pharmacologic agents have been identified, despite multiple investigations. Of burn patients who survive AKI, the vast majority do not receive long-term hemodialysis and they are generally thought to have a good renal prognosis [29].

Sedation, pain and delirium management in burn ICU

1. In non-ventilated patients, combine peripheral and centrally acting analgesics. Start by combining acetaminophen (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 [30].
2. In ventilated patients; for short-term sedation, propofol may be used (maximum dose 4 mg/kg/hr) combined with an opioid (morphine, fentanyl, alfentanyl, remifentanil). For long-term sedation, midazolam in combination with an opioid (morphine, fentanyl, alfentanyl, remifentanil, 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 [31].
3. To reduce tachyphylaxis, ketamine 1 mg/kg /24 hrs as a continuous infusion in addition to opioids is an option [32].
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 [33,34].

6. Intravenous infusion with dexmedetomidine (0.2 - 0.7 μg/kg/hr) during weaning seems promising. (31)
7. Gabapentin may be used to relieve phantom pain [34].
8. Delirium affects 60 - 80% of mechanically ventilated (MV) intensive care unit (ICU) patients and is independently associated with prolonged MV, ICU length of stay, on-going neuropsychological dysfunction and a three-fold increased risk in death at six months. Unfortunately, there is limited information regarding the prevalence and risk factors associated with delirium in critically ill burn patients. A study conducted in burn intensive care patients showed delirium occurred in 77% of ventilated burn patients. Majority of these patients had hypoactive delirium. Exposure to benzodiazepines was an independent risk factor for delirium while opiates and methadone reduced the risk of developing delirium, possibly through reduction of pain in these patients [36].

Blood transfusion in burns

Transfusion Requirements in Critical Care study by the Canadian Clinical Trials Group prospectively randomised critically ill patients to a restrictive strategy of red cell transfusion (maintenance of haemoglobin at 7 to 9 g/dl) or a liberal strategy (maintenance of haemoglobin at 10 to 12 g/dl). Unfortunately, this study excluded burns. The study found the restrictive strategy to be at least as effective as a liberal strategy with the possible exception of patients with acute myocardial infarction and unstable angina [36]. Following a multiple-centre cohort analysis highlighting an increased mortality associated with blood transfusion, there is increasing implementation of a restrictive transfusion strategy [38]. Implementation of a restrictive transfusion policy appears to have not adversely affected outcome in paediatric or adult burn patients [39]. Palmieri and colleagues undertook a study evaluating the effects of transfusion on outcome and revealed that the mean haemoglobin level in patients receiving their first unit of blood was significantly lower outside the operating theatre (8.9 vs. 10.2 g/dl, $P < 0.05$), which is higher than that perceived in medical practice (8.1 g/dl) [38]. Non survivors received significantly more blood transfusions than survivors (17.9 vs. 13, $P < 0.05$). This association was found after adjusting for multiple variables including burn size using multi-logistical regression. Patients with larger transfusions and poorer outcomes also had larger burns so increased transfusion requirements may simply be a surrogate marker for the severity of burn injury. In addition, the total number of units transfused correlated with the number of infectious episodes (Spearman rank correlation = 0.647; $P < 0.01$) [35]. A prospective, randomised trial of restrictive (7 to 8 g/dl) versus liberal (9 to 10 g/dl) blood transfusion policy in burns >20% TBSA is currently being undertaken by the American Burn Association. The burn patient will intermittently need surgery that may need large or massive transfusion and sets the burn patient apart from the more general critical care patient. Red cells facilitate haemostasis through a rheological effect by pushing platelets to the periphery of the vessel lumen to better interface at the endothelium as well as through direct effects on platelet biochemistry. Haematocrit <30% can therefore lead to significant extra blood loss [39]. Transfusion requirements also need to reflect the surgical and rehabilitation plan. If a large burn excision requiring major transfusion is planned, the patient needs to be optimised for theatre and a low starting haemoglobin concentration seems unwise. If patients are entering the rehabilitation phase, anaemia compromising relevant activities should be avoided: the consequences of delayed or impaired rehabilitation are profound in this patient group [40].

Inhalation injury

Significant inhalation injury, characterised by signs of hypoxia despite adequate arterial oxygen tension. Various agents such as nitrates and hydroxycobalamin have been used, but some are themselves toxic, and there is little evidence from randomised trials to support their routine use. Carbon monoxide levels higher than 15 to 20% should be treated with 100% endotracheal oxygen. There is little consensus regarding parameters or indications for hyperbaric oxygen, and availability is limited [41].

Almost all patients with significant inhalational injury would require intubation. So earlier elective approach is superior. As the times passes the swelling of face and neck increase and laryngedema also cause difficult intubation.

Ventilator management in burns ICU

In mechanically ventilated patients, there has been an increase in the use of volume-controlled modes over time. Despite this, ventilation parameters when patients are mechanically ventilated have not changed. Importantly, there was no difference in tidal volumes delivered during mechanical ventilation. Furthermore, both groups were ventilated with mean tidal volumes at the upper limit of the recommended ‘‘lung-protective’’ value of 6 - 8 ml/kg. This might indicate awareness of the importance of low-volume ventilation strategies (hence an increase in volume-controlled ventilation) among clinicians, but continued concern regarding hypercapnia despite evidence it causes no harm [41].

Moreover, burns patients are different from other intensive care patients. The patients suffering from inhalational injury required increased mechanical ventilation with higher peak airway pressures than those without. This was matched by a trend toward higher ASB and inspiratory pressure in the inhalational injury group. This likely represents the ventilation settings required to achieve adequate gas exchange in patients with inhalation injury. There was also a trend towards higher PEEP and lower tidal volumes in the inhalational injury group, though this did not reach statistical significance. This could represent a tendency towards high PEEP, low volume ventilation [41].

Tracheostomy

Indications and management in burns Earlier the better specially in those with neck and face burns and those with significant inhalation injury. Evidence suggests percutaneous tracheostomy is safe in burn patients, but caution should be employed in those with severe head and neck burns or upper airway edema because airway loss could result in serious complications [42-54].

Burn surgeons are experienced at these challenging tracheostomies and coordination of tracheostomy (closed or open) with grafting of the neck reperfusion mismatch and diminished transpulmonary oxygen transfer [55].

Discussion, Conclusions and Guidelines for Burns Critical Care

1. Use the consensus or ‘half Parkland’ formula, i.e. 2 mL/kg/%TBSA instead of 4 mL/kg/%TBSA crystalloids (lactated Ringer) to initiate resuscitation, and to continue with a permissive hypovolaemia, the primary end-points should be achieved soon.
2. After 24 hours begin colloid infusion of 5% albumin 0.3 - 1 ml/kg/%TBSA Dextrose water or dextrose saline is used for additional fluid requirements to maintain output > 0.5 ml/kg/h.
3. Norepinephrine is vasopressor of choice in fluid refractory shock in burns. If second inotrope is required to maintain hemodynamics, dobutamine is added to norepinephrine.
4. Blood glucose should be kept as normal as possible with insulin if required.
5. Surgical interventions like escharotomy and debridgement should be done after 48 hours of burns injury and the hematocrit>30% is recommended before starting surgery.
6. Burn sepsis should be diagnosed as early as possible by detecting 6 out of 11 criteria given below.
7. Therapeutic end points of resuscitation should be targeted. They are MAP > 60 mmHg, Pulse < 120, output > 0.5 ml/kg/h, pH 7.35 - 7.45, $SCVO_2$ >65% and lactate < 2 mmol/l.
8. Vitamin C (ascorbic acid) at 66 mg/kg/h for 24 hrs can reduce fluid requirements to 1/3rd in > 40% TBSA.
9. Early enteral nutrition is the cornerstone of burns and should be started within 12 hours of burns. Postpyloric or nasojejunal feeding is preferred over nasogastric tube feedings.
10. Opioids are the best option for pain management in large TBSA burns. In many instances they’re also used for sedation mostly in pediatric burns intensive care and many times in adults as well. In non-ventilated patients, intermittent doses are preferred and in ventilated patients infusions can be employed as well. Benzodiazepines are usually avoided due to their propensity to cause delirium in burned patients. Dexmedetomidine is recently used in a number of burn units and is found to be very beneficial so

far in reducing and sometimes replacing opioids. The authors use dexmedetomidine exclusively in burned patients and experience excellent results.

11. Acute kidney injury mostly occurs due to hypovolemia in burns. Patients who fail to improve after adequate intravascular volume replacement should be offered hemodialysis. Dopamine has no role in treatment of renal failure in burned patients.
12. Almost all patients with significant inhalational injury would require intubation. So earlier elective approach is superior. As the times passes the swelling of face and neck increase and laryngedema also cause difficult intubation.
13. Airway protection is cardinal in patients with face or neck burns or very large burns. Intubation is mostly difficult and its complications increase by delay. Early intubation and tracheostomy are shown to decrease number of days on ventilator.
14. Ventilator management is with volume control ACMV, which is universal and low tidal volumes 4-6 ml/kg with high PEEP. Plateau pressures are maintained below 30 cm of water. Permissive hypercapnea of up to 75 mmHg except patients with severe inhalation injury in which hypercapnea is avoided and pCO_2 levels are maintained in normal range of < 40 mm Hg.
15. Surgical management of burns involves regular assessment of burn wounds and prompt diagnosis of compartment syndrome can significantly reduce both morbidity and mortality. Escharotomy should be done early and incisions should be given in circumferential burns. Regular monitoring of peripheral pulses is important to diagnose acute limb ischemia.
16. Oxandrolone and propanolol are used for hypermetabolic phase of burns in many burn units however, their routine use is not recommended in every critical patient.
17. Transfusion threshold of 7.0 g/dl is followed in all burn ICU patients. A recent large multicentre RCT organized by American burn association on burn critical care patients found no difference in outcome of restrictive (7 - 8 g/dl) versus liberal (9 - 10 g/dl) transfusion groups.
18. Preliminary diagnosis of post-burn sepsis can be made if six out of the first 11 criteria below are met. This preliminary diagnosis can be confirmed if any one aspect described in the last criterion is met.

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