24 February 2017
No. 3

AN OVERVIEW OF ACUTE MANAGEMENT IN BURNS

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AN OVERVIEW OF ACUTE MANAGEMENT IN BURNS

INTRODUCTION

There are estimates that over a million patients are burnt annually on the African continent, and they contribute to about eighteen percent of hospital admissions. The mortality ranges between 6 and 10%. [1] It is vital to know and understand the pathophysiological changes that occur in the acute phase (24-48 hours) and during the hypermetabolic or hyperdynamic phase (>48 hours) of burns. [2] Anaesthetists form part of the multidisciplinary team involved in the management of the patient who has sustained burns. These involvements being early as admission, as these patients often require surgical treatment. To provide optimal care one must pay attention to risk factors (for example; burn shock, fluid resuscitation, changes in the anatomy of the airway) that make burns patients susceptible to increased morbidity and mortality. A comprehensive preoperative assessment is required. [2]

TERMINOLOGY

Ebb phase being the early phase of burn injury, within 12 to 48 hours
Flow phase or the polyuric phase, at 2-7days and lasting up to 12 months
Burn Shock is the inability of the circulation to meet the needs of tissue oxygenation, supply of nutrients and the removal of the metabolites.
Fluid creep is the difference between the estimated fluid requirements and the infused amount of fluid.

MECHANISMS OF BURN

Burns can be sustained from many causes and by different mechanisms. These can be:

- Thermal injury
  It can be due to Hot (water, fire) or Cold (ice) injury. This mechanism also includes iatrogenic heating and non-accidental injury.
- Chemical
  This type occurs with Acids and Alkalis
- Electrical
  Mains, High Tension, Railways and Lightning
- Radiation
  This type can occur from welding or from exposure to ultraviolet light
- Inhalational
  When a burn occurred in a closed space

CLASSIFICATION OF BURNS

Burns can be classified according to severity (using Total Body Surface Area involved), the presence or absence of inhalational injury and depth of the burn. The most commonly used classification is adapted from the American Burn Society.

The severity grading system includes minor, moderate and major burn. There is a different criteria for each grade.[2, 3]

1. Minor Burn:
   Is a burn area that is less than 10% Total Body Surface Area (TBSA) in adults,

Less than 5% TBSA burn in young or old patient,
Less than 2% full-thickness burn

- These patients can be managed as outpatients.

2. Moderate Burn:
   When there is 10% to 20% TBSA burn in adults,
   5% to 10% TBSA burn in young or old,
   2% to 5% full-thickness burn,
   A high voltage burn
   When inhalation injury is suspected
   A burn that is circumferential
   Co-morbidities predisposing to infection (for example; diabetes mellitus, sickle cell disease)

- Patients under this grade have to be admitted in hospital.

3. Major Burn:
   Burn more than 20% TBSA burn in adults,
   More than 10% TBSA burn in young or old
   More than 5% full-thickness burn
   A high voltage injury
   When an inhalation injury is known
   Burn to face, eyes, ears, genitalia, hands, feet or joints that is significant, and associated injuries (for example; the presence of a fracture or having sustained other major trauma)

The classification using depth of skin burn is described as first, second, or third degree, based on whether there is superficial, partial thickness, or full-thickness destruction of the skin.
Fourth degree is when deeper structures, such as muscle, fascia, and bone have been injured. Superficial burns heal without surgical treatment. Deep second- and third-degree burns require surgical debridement and grafting. [2, 3]

Table1. **Classification of burn depth** [2]

Depth	Level of Injury	Clinical Features	Result/Treatment
Superficial (first degree)	Epidermis	Dry, red; blanches; painful	Healing time 3–6 days, no scarring
Superficial partial thickness (superficial second degree)	Papillary dermis	Blisters; moist, red, weeping; blanches; severe pain to touch	Cleaning; topical agent; sterile dressing; healing time 7–21 days; hypertrophic scar rare; return of full function
Deep partial thickness (deep second degree)	Reticular dermis; most skin appendages destroyed	Blisters; wet or waxy dry; reduced blanching: decreased pain sensation to touch, pain present to deep pressure	Cleaning; topical agent; sterile dressing; possible surgical excision and grafting; scarring common if not surgically excised and grafted; earlier return of function with surgery
Full thickness (third degree)	Epidermis and dermis; all skin appendages destroyed	Waxy white to leathery dry and inelastic; does not blanch; absent pain sensation; pain present to deep pressure: pain present in surrounding areas of second-degree burn	Treatment as for deep partial-thickness burns plus surgical excision and grafting at earliest possible time; scarring and functional limitation more common if not grafted
Fourth degree	Involves fascia and muscle and/or bone	Pain to deep pressure, in the area of burn; increased pain in surrounding areas of second-degree burn	Healing requires surgical intervention

ESTIMATION OF TOTAL BODY SURFACE AREA

The TBSA burned in adults can be estimated using the “rule of nines”, where major body surface areas are divided into multiples of nine. However this method tends to overestimate burns less than 40% and underestimate the larger burn in the pre-hospital setting.[39]There is also a modified version for children and infants. The Lund–Browder chart is a diagram specific for age and accounts for the changing body surface area relationships. [2]

Rule of Nines Diagram [5]

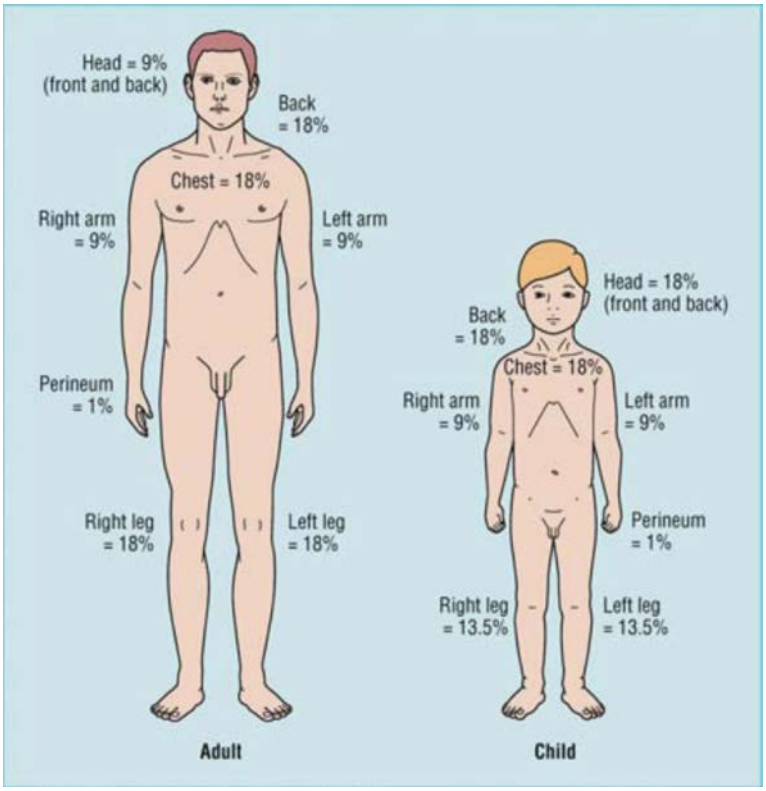


Figure1. Wallace Rule of Nines. Reproduced from: Hettiaratchy S, Papini R. Initial management of a major burn: II - assessment and resuscitation. Br Med Jnl 2004; 329: 101–3.

Lund-Browder Diagram and Table

Burn Estimate and Diagram Age and Area

Initital evaluation*

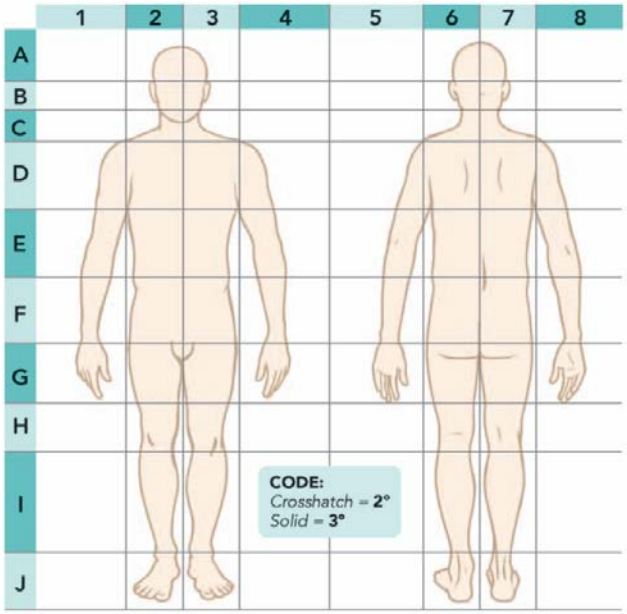
Signature: _____

Date of burn: _____

Date completed: _____

*To be completed by the admitting physician or Licensed Independent Practitioner on admission

This is a working burn estimate diagram only, and is not as accurate as photography.



Area	Birth-1 yr.	1-4 yrs.	5-9 yrs.	10-14 yrs.	15 yrs.	Adult	2°	3°	TOTAL
Head	9	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	13	13	13	13	13			
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	2.5	2.5	2.5	2.5	2.5	2.5			
Left hand	2.5	2.5	2.5	2.5	2.5	2.5			
Right thigh	5.5	6.5	8	8.5	9	9.5			
Left thigh	5.5	6.5	8	8.5	9	9.5			
Right lower leg	5	5	5.5	6	6.5	7			
Left lower leg	5	5	5.5	6	6.5	7			
Right foot	3.5	3.5	3.5	3.5	3.5	3.5			
Left foot	3.5	3.5	3.5	3.5	3.5	3.5			
***Only 2° and 3° burns are included in the total TBSA burn percent*						*TOTAL*			

Figure 2. Lund–Browder chart suggested to be used in pediatric patients because the body surface area relationships vary with age. The letters and numbers on the y and x axes can be used to demarcate site-specific changes.[2]

BURN CENTRE REFERRALS

Referral is done for patients with complex burn injuries including any of the following: [5]

a. Extremes of age (less than 5 years of age or older than 60 years)
b. Partial thickness burns greater than 10% TBSA
c. All full-thickness burns
d. All burns of the face, hands, feet, face or perineum
e. Or any flexure particularly the neck or axillar; or any circumferential dermal or full-thickness burn of the limbs, torso, or neck
f. All electrical, inhalation & chemical burn injuries
g. Suspicion of non-accidental burn injury
h. Pre-existing co-morbidities (significant cardio-respiratory disease, diabetes, pregnancy, immunosuppression, hepatic impairment, and cirrhosis
i. Associated fractures, and crush, head plus penetrating injuries

PATHOPHYSIOLOGY OF BURN INJURY

There is massive tissue destruction caused by major burns; resulting in activation of a cytokine-mediated inflammatory response that leads to pathophysiologic effects at different sites, and which are local plus distant from the burn. [2]

Early phase 24-48hrs [2, 4]

Cardiac and circulation:

- There is tachycardia, and normal or low blood pressure.
- There is decreased cardiac output from the depressed myocardial function, and also due to hypovolemia secondary to tremendous fluid extravasation from increased capillary permeability plus hypoproteinemia as well as evaporative losses.
- Systemic vascular resistance (SVR) is increased because of vasoactive substance release.
- Pulmonary vascular resistance is also increased.
- There is hemoconcentration that results from fluid loss induced by burn, occurring in the absence of marked red cell volume loss.
- Metabolic acidosis plus venous desaturation ensures.
- Echocardiogram or Ultrasound shows small heart chambers and decreased contractility.

Patients, whom have sustained electrical burn injuries, including both low-voltage and high-voltage, are at risk for dysrhythmias and direct damage to myocardium. The nonspecific ST-T changes and atrial fibrillation are seen commonly on ECG with these types of burns. [4]

Understanding the above changes is important to us as anaesthetists because most medications used for induction of anaesthesia decrease SVR and cardiac preload. Thus it is vital to maintain baseline hemodynamic parameters and loading conditions. Positive pressure ventilation used will also exacerbate decreased preload, especially in those who are not volume resuscitated adequately. [4]

Pulmonary and inhalational injury:

- The pulmonary system is affected in major burns by the inflammatory response.
- Inhalation injury most likely occurs if the burn was sustained in a closed space and there may be carbon monoxide poisoning.
- The injury to the lung and irritation result from smoke, flame, or damaging gases. This can result to laryngospasm, bronchospasm, bronchitis, shunt, and decreased pulmonary compliance.
- There is impaired gas exchange as a result of reduced surfactant, oedema, bronchial casts, sloughing of tissues, decreased clearance of debris and bacteria by mucociliary transport.
- Circumferential burns result in a restrictive thoracic and abdominal physiology.
- Acute Lung Injury or ARDS result from the inflammatory response and capillary leak that occurs along with fluid resuscitation.

Chest radiographs are usually normal at initial presentation. Changes are seen later due to secondary complications of inflammation, infection, or development of atelectasis.

Central nervous system:

- There is hypoxia induced by pulmonary injury and/or carbon monoxide toxicity.

- Altered mental status.
- Encephalopathy may be seen in children.
- Seizures that are isolated and self-limiting occur after carbon monoxide poisoning.
- Direct injury to the spinal cord and brain may be seen with electric burns.
- There is cerebral oedema.
- Increased intracranial pressure
- There is increased anti-diuretic hormone release (ADH)
- Pain response is also increased.

Renal system:
- Renal tissue gets damaged because of hypotension and a low cardiac output with inadequate fluid resuscitation.
- Renal artery vasoconstriction and decreased blood flow to the kidneys occur as a result of catecholamine release and vasoactive substances after burn injury.
- There is decreased glomerular filtration rate (GFR) and urine production.
- There is release of myoglobin and myoglobinuria caused by massive muscle tissue breakdown with electrical and crush injuries, resulting in renal injury.

Haematology:
- Hemoconcentration results from hypovolemia and capillary leak; and in turn cause increased haematocrit and blood viscosity.
- Anaemia results as a consequence of dilution during fluid resuscitation, in the absence of other trauma.

Other:
- There is mottled clammy non burn skin
- Non blanching burned skin
- Generalized oedema is seen with more than 25% burns. Oedema formation begins after 5 min to 1 hour in burned skin, gets maximal after twelve to twenty four hours, and is related to increased permeability plus leakage from the capillaries. [12]
- There is increased cortisol
- Aldosterone is increased
- There is decreased gastric perfusion
- Compartment syndrome develops in the presence of a circumferential burn without escharotomy being performed. This syndrome can also occur in the abdomen, extremities, or orbits without local or circumferential burns.

The late or hypermetabolic/hyperdynamic phase more than 48hrs

The following physiological changes are seen: [2, 3, 4, 5]

Cardiac
- There is still tachycardia
- Cardiac index and cardiac output increased
- The presence of subclinical myocardial dysfunction is seen
- There is altered contractility, seen on ECHO
- Increased venous desaturation
- Decreased SVR
- Systemic hypertension

Pulmonary
- ARDS

- Pneumonia
- Chest wall restriction

Neurological
- Altered mental status
- Hallucinations
- Delirium
- Coma

Renal
- There is increased GFR
- Decreased tubular function

Haematology
- Bone marrow suppression
- Anaemia
- Decreased haematopoiesis
- Thrombocytopenia

Endocrine and metabolic
- There is increased metabolic rate (with increased oxygen consumption and production of carbon dioxide)
- Core body temperature increases
- There is increased muscle catabolism
- Insulin resistance is increased, resulting in hyperglycaemia
- There is increased lipolysis, and glucolysis
- Thyroid and parathyroid hormone levels are reduced
- Electrolyte abnormalities have to be monitored

Other
- These patients get stress ulcers, and there is increased gut permeability to bacteria
- The lack of epidermis and dermis leading to development of wound infections
- There is immunosuppression
- There is increased blood flow to the liver, gluconeogenesis increased, reduced coagulation clotting factors, and low albumin
- Osteoporosis
- Deep vein thrombosis [5]
- Pharmacological responses are altered
- Infective complications in association with the management of a major burn can occur, such as: excessive burn oedema of the eyes (secondary to infection of corneal burns), urinary tract infection (from urinary catheters and burns to the perineum), sinuses and middle ear infection (as a complication of nasogastric feeding), and infective endocarditis
- At a later stage these patients experience;
  - Chronic pain
  - Post-traumatic stress disorder
  - Psychological effects (of severe illness, disfigurement and loss of independence)
  - Suicidal tendencies
  - Depression
  - Sleep disorder

Pharmacological changes

The pharmacokinetics and pharmacodynamics are different compared to non burn patients. The pathophysiological changes as mentioned above such as: fluid compartment alterations, changes in cardiac output, changes in organ perfusion, decreased renal and hepatic function, changes in serum protein levels and hypermetabolism; contribute to the differences. The response to drugs is irregular, and it is important that medication given is titrated to effect. [4] The volume of distribution is increased acutely and this should be taken into account when giving medications such as opioids, because serum concentrations will be reduced after a single bolus. [3]

The number of extrajunctional acetycholine receptors is increased significantly, in about twenty four hours after a burn, and severe hyperkalemia may occur with the use of a depolarizing muscle relaxant as intracellular potassium is released. [3, 4]
Figure 3 shows the difference between mature innervated and immature denervated acetylcholine receptors (AChRs).

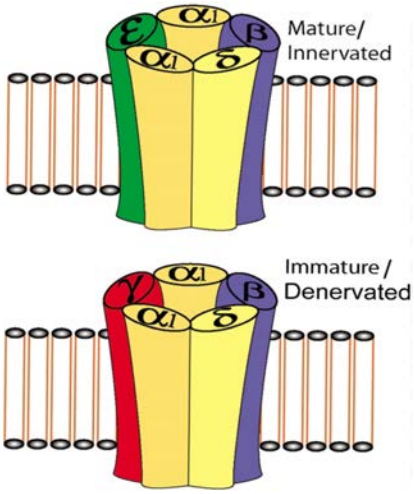


Figure 3. Acetylcholine receptor channels. [6]

The increase of AChRs is thought to be related to inflammation and local denervation of muscle. The up-regulation of AChRs at distant sites from burn is most likely related to the concomitant immobilization. The involvement of extensive body surface area from a major third-degree burn may up-regulate AChRs throughout the body, without the presence of infection, and this occurs because of its extent and direct inflammation/injury to muscle. There are reports published that even after a burn injury involving a single limb with the TBSA of 8%; hyperkalemia has been observed, and this indicates the size of the burn alone cannot be the only contributing factor. [6] It is showed in the figure below, how potassium is released after succinlylcholine administration in the innervated and denervated muscle.

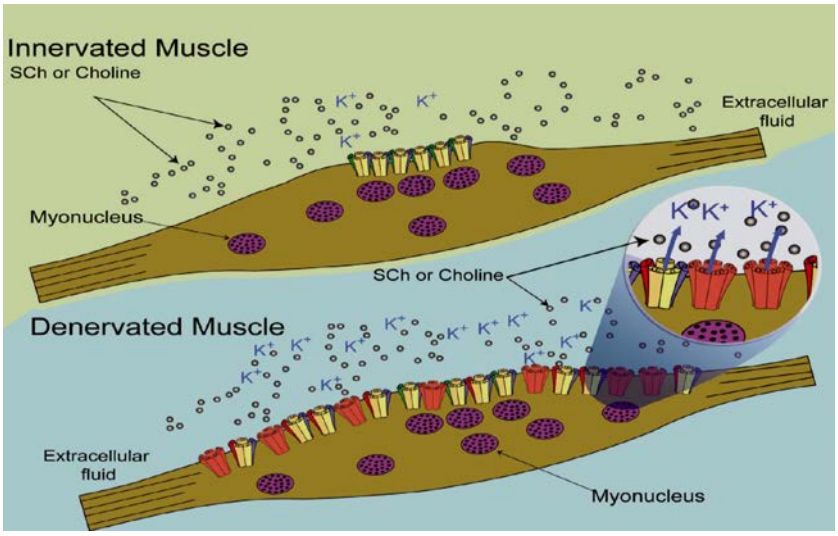


Figure 4 Succinylcholine (SCh) - induced potassium release in an innervated (top) and denervated muscle (bottom). [6]

In the innervated muscle, the administration of succinylcholine reaches all muscle membranes and only the junctional receptors (α1, β1,δ,ε) are depolarized as acetylcholine receptors (AChRs) solely located in this area. [6] With denervation muscle, both extra junctional (α1, β1,δ,γ) AChRs and α7AChRs are expressed by the nuclei throughout the muscle membrane. The administration of succinlylcholine in the system can lead to massive efflux of intracellular potassium as all of the up-regulated AChRs gets depolarized. The metabolite of succinylcholine (choline plus succinylmonocholine) can maintain this depolarization via α7AChRs and further enhancing the potassium release, thus hyperkalemia maintained. [6] It is reported that this response may persist up to 18 months. Therefore this drug should be avoided after 24 hours in these patients. [3, 4, 11] The usage is also discouraged in recent electrical burn injury because there may be hyperkalemia from tissue destruction and that of the muscle. [11]
This up-regulation of AChRs can persist as long as the condition that induced it continues to be present. It is suggested that AChRs return to normal levels when wounds have healed, catabolism of proteins subsided, and the patient mobile. The persisting immobilization as a result of severe contractures or other reasons, will lead to the up-regulation not being abated.[6]

There is resistance to non-depolarizing muscle relaxants (NDMR). This change occurs in about a week after the burn, and commonly seen if greater than 20% TBSA burn. These differences will persist for months to years after a burn injury. [3, 4, 7]This is seen with a slower onset of paralysis when normal doses administered, and muscle relaxation that is inadequate or faster recovery time. This change is thought to be caused by proliferation of peri-junctional and expression of the immature, fetal type, or both alpha 7 neuronal acetylcholine receptors (AChRs) on the muscle membrane. [7]

INITIAL EVALUATION AND MANAGEMENT

The management of the patient with burn injury to be successful has to begin at the scene of injury and continue in casualty or the emergency department. It starts with an assessment, which is thorough and based on the Advanced Trauma Life Support guidelines. The aim is to maintain vital organ function and perfusion. The airway should be assessed and protected, resuscitation initiated, and the evaluation for coexisting injuries required.[2, 5] The possibility of intoxication must also be ruled out.[39] The burn must be cooled (avoiding

hypothermia), clothing and jewelry removed and history obtained (nature of Burn Injury, allergies, Tetanus immunization and significant Past Medical History).[2]
A circumferential eschar on the chest or abdomen will decrease pulmonary compliance plus interfere with respiration, and therefore a chest wall escharotomy may be necessary. [2] The prophylactic use of antibiotics has no role and not recommended in acute burn injury. Antibiotics should only be given when the infection is proven or highly suspected, with the plan of reviewing management in 48hours.[39]

Inhalational injury
Inhalation injury should be recognized early at evaluation. The diagnosis is based on history of exposure to fire and smoke (especially if combined report of entrapment in an enclosed space or loss of consciousness) along with the physical examination (such as burns over face, singed nasal or facial hair, carbonaceous sputum, hypoxia, hoarse voice, and bronchorrhea, and/or bronchospasm). Management involves administration of 100% O2, obtaining and monitoring HbCO levels & ABG's, monitoring for hypoxia and/or airway obstruction, and anticipating endotracheal intubation. Fibre-optic bronchoscopy may be done to support the diagnosis and may show carbonaceous debris, erythema, or ulceration. After performing a thorough airway examination and the airway found to be compromised, intubation should be done as early as possible. [2, 8]

Clinical signs mentioned above indicate possible involvement of the airway, but are unreliable and poor predictors of injury severity. The patients with clinically significant airway involvement may go undetected if clinicians rely on these signs. [9, 1] A prospective observational study by Ikonomidis C et al; included hundred patients with clinically suspected inhalation injury and the results showed that twenty one percent of the fibre-optic evaluation done did not have evidence of upper airway pathology and in thirty nine percent there was no evident tracheobronchial pathology, while the evaluated 38% with noted upper airway injury did not present with singed nasal hairs. [9] In a retrospective study by Madnani et al looking at 41 patients; it was found that while body burns including the face were predictive for laryngeal oedema, there was no significant correlation with carbonaceous sputum and soot in the oral or nasal cavity, stridor, hoarseness, drooling or dysphagia. [10]

Intubation
Intubation of burn patients, if being transferred to another hospital must not be seen as a benign intervention because securing an endotracheal tube (ETT) may be difficult once edema develops, this occurring as a result of facial burns or glottic edema.[2] In patients where difficult airway is anticipated or unexpected; supraglottic airway, video laryngoscopes, and flexible intubation endoscopes are extra valuable devices that should be capitalized. [11] The nasogastric feeding tube should also be placed after intubation.[2]

The use of induction agents in the emergency department for airway management should be guided by the hemodynamic status of the patient. Propofol doses should be reduced as low as possible due to its vasodilatory effect, and also to remember that patients may present in hospital with pre existing fluid deficits. Therefore giving large dosages of propofol may lead to blood pressure decreasing significantly and cardiovascular collapse.[11]
Outside the operating theatre, etomidate is the induction agent that still remains the most frequently used for rapid sequence induction and intubation. The reason for its use is because of hemodynamic and cardiac stability. It is known that adrenal cortical suppression via the inhibition of 11-beta hydroxylase result from using etomidate; however there is lack of evidence showing neither increased morbidity nor mortality after administering a single dose in burn patients. Its use should not be repeated and must be avoided in these patients as they turn to require multiple operative procedures and anaesthetic. [11]

Ketamine has gained popularity as an induction agent in burn patients because of its several properties such as analgesic effects and likely increasing or maintaining blood pressure. The catecholamine levels are depleted in burn patients; therefore using large doses of ketamine may lead to myocardial depression and also result in hypotension.[11]
The muscle relaxants commonly used for emergency intubation are succinylcholine (within 24 hours of injury and discouraged in electrical injury due to hyperkalemia from muscle and tissue destruction), or rocuronium (1.2 mg/kg).[11]

FLUIDS

Intravenous fluid resuscitation is required if the burn involves more than 15% BSA or 10% with smoke inhalation in adults. [5] Delays or inadequate fluid resuscitation will result in hypovolemia, hypoperfusion of tissues, burn shock, multiple organ failure, and the effects of smoke inhalation can also be exacerbated. [2] There are different formulas used to calculate fluids, and regardless which one chosen, it should serve as a guide, with the aim of titrating to physiological endpoints. [2]

The formulae used are:
a. Parkland = 4 ml/kg/%TBSA Burn of Ringer Lactate
b. Brooke = 1.5 ml/kg/%TBSA Burn of Ringer Lactate
= 0.5 ml/kg/%TBSA Burn of Colloid
c. Modified Brooke = 2ml/kg/% TBSA Burn of Ringer Lactate

The Parkland formula is the most commonly used resuscitation guideline and is 4ml/kg/%burn; where fluid requirements for the first 24 hours after injury is predicted. This guideline means starting calculating from the time of burn injury and not the presentation time at the hospital, and half of the fluid given in the first 8 hours whilst the remaining half is given over the next 16 hours. The fluid of choice is Lactated Ringer solution. The already given fluid prior calculation should be subtracted from the calculated requirement.[5] The guide for resuscitation is measuring urine output (aim 1 mL kg^{-1} $hour^{-1}$), and fluids increased by 25% after each assessment if deemed insufficient.[12] Colloids are allowed during the second 24 hours of resuscitation. Volume is gradually decreased if this form of resuscitation deemed adequate. It is important to note that urine output is not the ideal resuscitation endpoint because at a microvascular level; the end-organ or tissue hypoperfusion may not be reflected.[12]

Fluid creep

There are multiple reports that there is excess fluid administration during resuscitation of burns patients. [13] This reported fluid excess leads to morbidity and multiple complications associated with fluid overload such as pulmonary edema, wound healing that is delayed, ileus, compartment syndrome of the limbs, orbital compartment syndrome, intra-abdominal hypertension (IAH), and abdominal compartment syndrome (ACS) leading to multiple organ failure. [12, 13]
A retrospective study conducted for a period of three years by Chung et al. compared fluid resuscitation in military burn casualties using the Parkland formula and the modified Brooke formula. The fluid requirements were significantly more in the Parkland group patients than those whom the modified Brooke formula was used (5.9 vs 3.8 mL kg^{-1} $\%TBSA^{-1}$, $P < 0.0001$). This study demonstrates that the total volume given is affected by the starting fluid rate, and this can result in resuscitation morbidity. With modified Brooke formula there was less 24 hour fluid volume given, and this did not result in higher morbidity and mortality. [14]
There is considerable evidence, stated by Saffle, showing excessive administration of crystalloid whilst colloids not used, and that this could contribute greatly to fluid creep. [15]

In a study looking at the factors for predicting increased fluid requirements in burn patients by Cancio and colleagues; physicians were considerable noted to less likely titrate the rate of infusion down in response to adequate urine output than titrating infusions up when output inadequate. [15, 16] An inaccurate assessment of TBSA in burn injuries, may also contribute to inappropriate fluid volumes administered during resuscitation.[12]

Resuscitation targets and evaluation

- Urine output that is 0.5–1.0 ml /kg^{-1} / h^{-1} (cannot be used as the only parameter, as this can result in under or over estimation of fluid resuscitation).[2]
- Blood pressure within normal range for age.[2]
- Heart rate that is variable.[2]
- Cardiac output monitoring.[2]
- Central venous pressure between 3–8 mmHg (the use is controversial).[2]
- Invasive arterial monitoring and pulse contour analysis (using pulse pressure variation [PPV] and stroke volume variation [SVV]) have been used with success to assess fluid responsiveness in mechanically ventilated patients. PPV or SVV greater than 12% has been reported in most publications as highly predictive of fluid responsiveness. However we must not interpret increased PPV and SVV that is caused by intra-abdominal hypertension [IAH] or abdominal compartment syndrome [ACS] as hypovolemia with fluid responsiveness, as this will lead to over-resuscitating of patients even further. The reason is that the SVV and PPV value doubles if IAP increases by 18mmHg.[38]
  Intra-abdominal pressures should be monitored on patients with major burns.
  Passive leg raise test may be used as an indicator of fluid responsiveness even on spontaneously breathing patients. However this test may be inaccurate when IAP elevated.[38]
- Fractional excretion of Na+ (FeNa) less than 1%; indicating hypovolemia.[2]
- BUN/Cr ratio greater or equal to 20; indicating hypovolemia.[2]
- Echocardiogram/ultrasound showing normal stroke volume and ejection fraction.[2]
- Arterial blood gas to assess hypoperfusion, checking the base deficit and lactate.[2, 38]

Types of resuscitation fluids

Crystalloids

Normal Saline

The adverse effect of 0.9% saline solutions after large volume infusions contribute to the development of hyperchloremic metabolic acidosis. [18] Fluid resuscitation in burns requires large volumes to be infused and therefore saline cannot be used as the first choice. [12, 19]

Lactated Ringers or Hartman's solution

The lactated Ringer's or Hartmann's solutions reported to be balanced or physiological solutions, replace the anion bicarbonate into lactate, acetate or gluconate forms, and providing a strong ion difference better from an acid base perspective. Based on evidence it has been concluded that balanced crystalloid solutions in majority of ill and burns patients, are a pragmatic initial resuscitation fluid. [12]

There is still not enough evidence in literature regarding the appropriate crystalloid to use in burn patients. [17]

Colloids

During the first 24 hours of burn resuscitation, the use of synthetic colloids has been controversial ever since the theory stating that edema formation will increase because of the existing capillary leak, as large molecules of colloid fluid exert an osmotic pull through

leaking in the extravascular space. [20] Colloid fluids contain large molecules in a carrier solution and these are less likely to leak into the extravascular compartment and while in the intravascular compartment will increase plasma oncotic pressure. [12, 17]

Colloids are categorized as natural and semi-synthetic. The natural category is derived from blood; albumin and fresh frozen plasma being examples. Cost is the limiting factor to using natural colloids. The semi-synthetic category has major subclasses such as hydroxylethyl starches (HES), gelatins and dextrans.[12, 17] The most commonly used in this class is Hydroxylethyl starches (HES) and the molecules are slowly metabolized resulting in intravascular volume expansion that is prolonged but can potentially accumulate in the reticulo-endothetial tissues such as kidneys, skin and liver.[21, 22] The concerning factors are altered blood coagulation, incidence of renal failure, renal replacement and increased mortality rate associated with using HES.[12, 17]

A prospective randomized trial by Michael S. O'Mara et al in 2005 showed the possibility to significantly lower IAP using human colloids such as plasma in comparison to crystalloids, but the study had a small sample size (31 burn patients).[24] However, a 2014 systematic review by Strang et al observed no benefits in preventing IAH despite the effect of colloids on decreasing the needs of resuscitation volume.[13] The use of colloids in burn patients cannot be recommended as initial resuscitation fluid; given the recent published data.

Albumin

A retrospective observational study reported in 2010 by Lawrence et al. found that there was rapid reduction of fluid requirements hourly during resuscitation, the normal resuscitation ratios restored, and fluid creep ameliorated with the addition of albumin to the Parkland formula.[17, 25] A study by Cochran and colleagues was conducted in 2007 on burn patients with TBSA equal to or greater than 20%, comparing those who received albumin because of increased fluid requirements with a cohort comparable for age and burn injury who did not require albumin administration. Albumin administration was found to be a protective factor for mortality on multivariate analysis.[27]

In patients who sustained greater than 20% TBSA burn, and receiving albumin within 24 hours after injury were reported by Park et al. to have reduced mortality and decreased duration of mechanical ventilation.[30] The albumin 20% (based on evidence available) may be recommended for use in severe burns, more during the de-resuscitation phase. Its use is guided by indices such as capillary leak, weight of the patient, a positive fluid balance, extravascular lung water, fluid overload, and IAP.[26]

Hypertonic saline

There is an existing risk of severe hypernatremia, renal failure and acute cerebral fluid shifts when large quantities of hypertonic saline are infused.[28] In 1995 a large retrospective cohort study was published by Huang and colleagues, where burn patients who received hypertonic saline versus controls who had a crystalloid resuscitation were compared.[29] The rate of acute renal failure (40% vs 10.1%, $P < 0.001$) and mortality were significantly higher (53.3% vs 26.6%, $P < 0.001$) on patients who received hypertonic saline.[29] There is lack of evidence regarding usage of hypertonic saline in burns. The reported use has been associated with lower volume requirements during initial resuscitation, lower intra-abdominal pressure, and the incidence of compartment syndrome reduced.[17] Close monitoring of sodium levels is encouraged whenever using this solution. [12]

Adjunctive therapy

Vitamin C

Ascorbic acid (an antioxidant) is reduced due to pathophysiological changes that occur in the liver after burn injury.[12] The effects of ascorbic acid are (osmotic) diuretic and that may lead to hypovolemia. In a prospective randomized study; it was found that using a high dose of ascorbic acid during the first 24 hours reduced fluid volume requirements during resuscitation (3.0 vs 5.5 mL kg^{-1} %$TBSA^{-1}$, $P < 0.01$), reduced weight gain, worsening respiratory dysfunction and wound edema. Reduced inflammatory response may be causes by the decreased insensible fluid losses, and this reduction also lead to earlier mobilization of fluid. [31] During the initial 24 hours of resuscitation, it is indicated that ascorbic acid be infused at 66 mL kg^{-1} $hour^{-1}$ (25 grams of ascorbic acid added in 1000 mL of Plasma-Lyte® solution and a black bag used to cover the mixture in order to prevent auto-oxidation induced by light). [12]

Plasmapheresis

In burn resuscitation plasma exchange or plasmapheresis has been described as a rescue therapy previously. A humorally mediated systemic inflammation is initiated in burn shock and these inflammatory mediators can be removed mechanically by plasmapheresis. There is removal of the patient's plasma during this strategy, which is replaced with albumin or fresh frozen plasma.[12] During the early resuscitation period, the benefits of plasmapheresis used as a salvage therapy has been confirmed by the more recent retrospective studies. There was reduced fluid administration associated with this strategy, and increased urine output seen. [12] Klein and Neff found a decrease in fluid administration per hour after therapeutic plasmapheresis by 28.3% and 25% in the groups of patients they studied.[32, 33] It is an invasive plus expensive procedure, and use should be considered once other measures have been tried. However this benefit of plasmapheresis still needs to be verified by large prospective randomized trials to see outcomes in burn patients.[12]

NUTRITION

There are associated metabolic alterations with severe burn injury and can persist for up to 2 years post burn.[34] The persistent and prolonged hypercatabolism provoke an unpleasant chain of events; such as loss of weight, growth retardation, immunosuppression, catabolism of muscle and bone.[34, 35] Sustained protein losses lead to failure to fully rehabilitate.[34] There is gut mucosal damage that is significant in the acute phase, and increased bacterial translocation that inclusively lead to reduction in absorption of nutrients. The optimal nutrition support is best accomplished after injury by initiation of enteral nutrition (EN) within 24 hours (that is early initiation) for the severely burn patient. This early initiation of enteral feeding can major modulate the hypermetabolic response to severe burn, and this demonstrated by multiple studies.[34, 35]

The integrity of the gut mucosa, its motility, and intestinal blood flow is also preserved by early EN and these relevant to preventing intestinal hypoperfusion or ileus when resuscitation delayed or due to reperfusion. The effects of post burn ileus affects the stomach and colon primarily, it is then advised to feed 6 hours after burn via enteral tubes to the small bowel (duodenum or jejunum), and this independent of total gastro-duodenal function in patients with severe burn injury.[34]

SEDATION AND ANALGESIA GUIDELINES FOR ACUTE BURNS

In patients with burn injury pain relief is of paramount importance and tachyphylaxis to opiods is experienced by both adult and pediatric patients. Multiple centres have formulated

an analgesic and sedation strategy for acute burn patients. It is always wise to choose what is best for different patients under your care. Examples of strategies used listed below:

Bittner and colleagues strategy

Stage of Injury	Background Anxiety	Background Pain	Procedural Anxiety	Procedural Pain
Acute burn ventilated	#1 Midazolam infusion	Morphine infusion	Midazolam boluses	Morphine boluses
	#2 Dexmedetomidine infusion	Morphine infusion	Dexmedetomidine higher infusion rate	Morphine boluses
	#3 Antipsychotics	Morphine infusion	Haloperidol (very slow) boluses	Morphine boluses
	#4 Propofol infusion (<48 h)	Morphine infusion	Propofol boluses	Morphine boluses
Acute burn not ventilated	Dexmedetomidine IV or scheduled lorazepam IV or PO	Morphine IV or PO	Lorazepam IV/PO	Morphine IV/PO or Ketamine IV
Chronic acute burn	Scheduled lorazepam or antipsychotics (PO)	Scheduled morphine or methadone	Lorazepam or antipsychotics (PO)	Morphine PO or oxycodone

Fentanyl infusions could be substituted for morphine infusions. In view of the increased incidence of delirium with benzodiazepines, minimal use of them is advocated.

Figure 5. Sedation and analgesia in acute burns [2]

Guttormsen and colleagues have published options: [37]

In non-ventilated patients the aim is to combine peripheral and centrally acting analgesics.

1. Paracetamol (10–15 mg/kg) is given four times daily (QID) with morphine started at a low dose and titrated to effect. Intermittent injections are recommended due to the risk of respiratory depression. It is important to always monitor patients when infusions used to avoid respiratory depression.

In ventilated patients:

2. Propofol may be used for short term sedation (maximum dose 4 mg/kg/hr) combined with an opioid (morphine, fentanyl, alfentanyl, remifentanil).
3. Midazolam combined with an opioid (morphine, fentanyl, alfentanyl, remifentanil) for long term sedation.
4. Ketamine infusion 1 mg/kg /24 hrs, as an option in addition to opioids, to reduce tachyphylaxis.

Other options:

5. Patient controlled analgesia (PCA) can be used.
6. Subcutaneous infusions with mixtures of an opioid, alpha-2 agonists, ketamine, and even the non-analgesic haloperidol to attenuate agitation.
7. Intravenous infusion with dexmedetomidine (0.2–0.7 μg/kg/hr) during weaning of infusions
8. Gabapentin as an option to relieve phantom pain.

Hypnosis should be considered once the patient extubated; as it enables the reduction of anaesthesia requirements for painful procedures.

Nikki Allorto is the president of South African Burns Society and the founder of the Burn Care Trust. She has developed protocols for sedation and analgesia for the Pietermaritzburg Metropolitan Burn service. The strategy is as follows:

	Drug	Paediatric	Adult
	these are oral doses unless otherwise stated		
Mandatory	Paracetamol (syrup = 120mg/5ml)	15 mg/kg 6hrly	1g 6 hrly
Mandatory	Tilidine (1 drop = 2.5 mg)	1 mg/kg 6 hrly	-
Add if pain not controlled and for donor site pain	Ibuprofen (100mg/5mls)	10mg/kg 8 hrly	400mg 8hrly
Mandatory if > 15%TBSA and no Tilidine	Morphine syrup 1mg/ml	0.1 mg/kg 6 hrly	-
Mandatory	Tramadol	-	50 - 100mg 6hrly
Add if pain not controlled OR neuropathic pain	*Clonidine (25mcg tablets that cannot be broken) (O/S currently)*	*25mcg 8 hrly increase to maximum 50mcg 8 hrly*	*75mcg 8hrly increase in increments of 25mcg per dose*
Add if pain not controlled OR neuropathic pain	Tegretol 200mg tabs	-	200mg 12 hrly increase to max 1200mg/day (400mg 8 hrly)
Add if pain at night/ difficulty sleeping	Amitryptiline	-	25mg nocte
For neuropathic pain and or severe itch	Lyrica 75 or 150mg tabs mixed into suspension for paeds	start at 25mg 12hrly, increase in 25mg increments to max 75	start at 75mg 12 hrly, increase to max 150mg 12hrly
	Gabapentin	10mg/kg 8hrly, increments of100mg/dose up to 600mg 8 hrly	300mg 8 hrly, increase up to 600mg 8 hrly
For ICU patients/large TBSA burns **(MORPHINE mixed as a 1mg/ml solution ie. 10 mg in 10 mls or 50mg in 50 mls)**	Morphine **IVI** Remember this needs to be weaned and not stopped suddenly! (wean the infusion rate then move to bolus dosing and increase the dose interval over time)	0.1mg/kg loading dose then 0.1mg/kg/hour infusion increase to effect, reload and increase rate by 0.05mg/kg	0.1mg/kg loading dose then 0.1mg/kg/hour infusion increase to effect, reload and increase rate by 0.05mg/kg
Last resort if pain not controlled	Morphine **IMI**	0.1mg/kg 4-6 hrly	10-15mg 4-6hrly
For PTSD OR anxiety OR opioid withdrawal	Valium	2.5 mg nocte, titrate to effect can be increased to 8 hrly	5 mg nocte, titrate to effect can be changed to 2.5 mg 8 or 12 hrly
For Delirium	Haloperidol	-	2.5 - 5mg 8hrly

Figure 6. Sedation and analgesia strategy by Nikki. Permission obtained

ETHICAL ISSUES

There are often ethical dilemmas present when managing burn patients. The International Society of Burn injury (ISBI) practice guidelines have been published in 2016, with multiple recommendations. The recommendation for ethical issues is as follows:

"Patient autonomy must be respected, with the patient him/herself making decisions regarding treatment. If the patient is unable to speak for him/herself, then a responsible surrogate must be appointed to provide decisions regarding care. The treatment team role resides in providing the best information to the patient and/or his/her surrogate regarding the likely course of care, alternatives, and prognosis".[36]

QUESTIONS TO ASK DURING MANAGEMENT OF MAJOR BURN

1. What is the mechanism of burn?
2. How long ago was the burn?
3. Have you estimated the total body surface area correctly?
4. What is the classification of the burn?
5. Which formula to use when calculating fluids?
6. What are the resuscitation targets?
7. Does the patient need to be intubated? Has Fiberoptic bronchoscopy been done?
8. Have you ruled out other injuries and intoxication?
9. Is the NGT inserted?
10. Have you made the plan to start enteral feeds?
11. Has analgesia been given?
12. Does the patient need referral?

CONCLUSION

The TBSA burn should be assessed properly at initial presentation to prevent morbidity and mortality. The aim should be to preserve organ perfusion and function. The avoidance of over and under resuscitation must be part of the goals. The airway should be assessed, maintained and an early decision for intubation must be made. Other injuries and intoxication must be ruled out at initial presentation. The nasogastric tube should be inserted for every patient with major burn and enteral feeding commenced within 24hours. It is vital to commence analgesia as soon as possible, and early referral of patients fitting the criteria is advised.

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