

appropriate in the polytrauma patient. It may also be difficult to maintain during transfer and repatriation to the UK. The main dilemma for the military intensivist is whether we should be measuring ICP in this patient group, and if so, how. External Ventricular Drains are the gold standard and are easy to monitor with current ITU monitors and in-flight but require neurosurgical intervention to insert. An ICP bolt can be inserted, after training, but the technology is temperamental, prone to drift and not appropriate for use in flight.

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CRITICAL CARE OF MILITARY BURN CASUALTIES AT ROLE 3 FACILITIES

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Abstract

Burn casualties will inevitably occur in the military environment during both conflict and peacetime. The number and type of casualties will vary on the nature of warfare and the type of troops deployed. New preventative measures have decreased the number and severity of burns found on the battlefield however with new weapon systems casualties suffering from thermal injuries are still to be expected in modern warfare. Over the last 4 decades great advances have been made in the treatment of thermal injuries. These advances are reviewed here with emphasis on those that can be accomplished in the Role 3 facility by non-specialist clinicians. It is beyond the scope of this review to produce didactic treatment protocols but it is hoped that in the near future Clinical Guidelines for Operations will soon reflect these. Where advances have occurred that can not be mirrored in the field hospital early evacuation to specialist facilities back at Role 4 facilities should be a priority.

Introduction

During armed conflict military personnel are at high risk of thermal injury from both battle and accidental injury. The increase in risk is dependant on both the weapon systems deployed and the type of combat engaged in. In conflicts over the last six decades the incident of thermal injury has ranged from 2.3% to as high as 85% (Table 1).

The detonation of a nuclear weapon at Hiroshima in 1945 produced an estimated 57,700 burn victims. This equated to 85% of the total casualties [1]. In contrast during the Panama police

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Conflict	Number of burn casualties (% of total injured)	
World War II Hiroshima 1945	45,500 – 59,500	(65-85)
Vietnam Conflict 1965-1973	13,047	(4.6)
Israeli Six Day War 1967		(4.6)
Yom Kippur War 1973		(10.5)
Falklands War 1982		
- UK Casualties	140	(18.0)
- Argentine Casualties	34/194	(17.5)
Lebanon War 1982		(8.6)
Panama Police action 1989	6/259	(2.3)
Operation Desert Shield/Storm	36/458	(7.9)

Table 1. The Incidence of Burn Injury in Armed Conflict.

action in 1989 only 2.3% of total casualties involved thermal injury. This has been attributed to the fact that only infantry and airborne forces were deployed using small arms.

Battle casualties are not the only cause of burn injuries during armed conflict. In the Vietnam conflict due to the total air supremacy and a lack of armoured warfare, patients with thermal injury constituted only 4.6% of total casualties for the period of 1963- 1975 [2]. The majority of these (58%) were non-battle injury [3].

Traditionally conflict involving armoured vehicles and naval forces has been associated with a higher association of burns victims, highlighted by the casualty figures from the British Army of the Rhine at the end of the second world war and the Israeli conflicts of 1973 and 1982 [4,5]. In the Falklands campaign 34% of all casualties from the Royal Navy ships were burns victims, equating to 18% of all casualties [6].

Reductions in the number of thermal injuries and a reduction in the seriousness of the individual injury have been made by technological advances such as flame retardant clothing and fire suppression systems in vehicles. In the Israeli conflict of 1973 29% of casualties with burns had injuries above 40% total body surface area. In the following conflict of 1982 this figure had reduced to 18%.

Modern weapons systems have also reduced the differential incidence of burn injury between armoured vehicle personnel and personal from other units. This is highlighted by the similar casualty rates from Argentine and British forces during the Falklands campaign where little armoured warfare took place [6].

What is clear from these figures is that there is a high incidence of thermal injury occurring in military personal in both conflict and peacetime. This will produce a group of casualties suffering from extensive burns who require tertiary centre burn care to ensure optimum outcome and survival.

The challenge to the military medical services on operations is to bring the elements of the tertiary burns centre to the deployed field hospital that will have the greatest impact on the outcome of burns victims and those that are not achievable because of environmental constraints must be mitigated for (Figure 1). Following this line of thought it would be useful to review the major advances in burn care that have occurred over the last few decades that have had the maximum impact on burns survival.



Figure 1. 20% total body surface area partial thickness cutaneous burn due to scald in coalition soldier during Op Telic being treated in Role 3 facility.

Advances in burn care

Most advances in burn care have occurred in the last 50 years. Between 1942 and 1952 shock, sepsis and multi organ failure have caused a 50% mortality rate in children with burns of 50% of total body surface area [7]; presently burn care survival in children has improved such that a 95% total body surface area injury can be survived by 50% of victims.

The main areas that have impacted on patient survival have been the improvement in resuscitation, infection control, support of the hypermetabolic response, nutritional support, treatment of inhalational injuries, prevention of stress ulceration, early debridement with wound closure and the multidisciplinary approach to burn care. The majority of these interventions can be initiated at the field hospital.

Resuscitation

The history of modern resuscitation in major burns dates back to the 1921 Rialto theatre fire in the USA. Underhill noted that blister fluid had a composition similar to that of plasma. This was then replicated by a salt solution containing proteins - from this work Underhill suggested that burns victims died from loss of fluid [8]. In 1944 Lund and Browder estimated burn surface area and developed charts to derive the percent of total body surface area burned [9]. This led to the development of fluid replacement strategies based on the amount of burned surface area.

Cope and Moore postulated that the interstitial space was a major recipient of the plasma loss in burned and unburned tissue [10]. Baxter discovered [11] that after a cutaneous burn fluid is not only distributed into the interstitial space but there is an accumulation of intracellular oedema due to a failure of the sodium potassium pump in the cell wall leading to an inability to remove water from the cells. It was postulated that any protein given in the first 24 hours would leak out into the interstitial space and cause further oedema. This was confirmed in patients with inhalational injury at a later date [12].

It is now accepted that if untreated, cutaneous thermal injuries of the order of 30% total body surface area invariably result in a severe and specific derangement of cardiovascular function called burn shock. Shock may be defined as an abnormal physiological state in which blood flow is insufficient to maintain adequate tissue perfusion. Burn shock is both hypovolaemic and cellular in origin. Clinically signs of hypodynamic instability including decreased cardiac output, extracellular fluid sequestration, reduced plasma volume and oliguria. The primary aim of resuscitation is to restore tissue perfusion. In burn shock this is complicated by burn oedema and voluminous transvascular shifts. These are unique to thermal injury. The primary goal of therapy is to replace the fluid sequestered outside the vascular space. The most important concept to understand in burn shock is that massive fluid shifts occur even though the total body water remains unchanged. It is the volumes of specific compartments that change.

Intracellular and interstitial volumes increase at the expense of plasma and blood volume. This oedema process is accentuated by resuscitation fluid, the degree of which is dependant on the amount and type of fluid given [13]. The replacement fluid should replace the extracellular salt loss that occurs into the burned and non-burned tissue at cell level [14-16]. The amount given should be enough to maintain adequate organ perfusion and be continually titrated to avoid both under and over resuscitation [17, 18].

Crystalloid resuscitation

Ringers lactate is clinically the most popular resuscitation fluid administered. The most common reason given for not using colloids is that even large proteins leak from capillaries following thermal injury [19]. The quantity of fluid given depends on the parameters used to identify adequate resuscitation clearly a lot more fluid will be needed to generate a target urine output of 1ml/kg/hr rather than 0.5ml/Kg/hr. The Parklands formula recommends 4mls/Kg/% burn as a target for fluid resuscitation, at this level however hypoproteinaemia will occur and lead to further oedema.

Hypertonic saline

These salt solutions have been known for many years to be effective in treating burn shock [20,21]. Rapid infusions produce two positive outcomes - serum hyperosmolarity and hypernatraemia [22]. The hypertonic serum reduces shift into the extravascular space of vascular water which should decrease tissue oedema with fewer complications such as compartment syndrome. Monafó [20] and Demlimy [23] and colleagues demonstrated that fluid intake was less when titrated to urine output in burns victims when resuscitated with hypertonic solutions compared to Ringers Lactate. However soft tissue interstitial oedema in burned and non-burned tissue was increased with hypertonic saline to a level seen with Ringers Lactate. This can be explained by a shift of intracellular water into the extracellular space due to the hyperosmolar solution used. Therefore extracellular oedema can occur at the same time as intracellular fluid deficit giving the clinical impression of less oedema. This fact makes the use of hypertonic saline in burns resuscitation controversial to date.

Colloids

Plasma proteins are extremely important in the circulation as they generate the oncotic pressure correcting the capillary hydrostatic pressure. Considerable confusion exists for their role in resuscitating burn victims. Slater *et al* [24] recently utilised FFP during burn shock, titrating to physiological effect.

Monitoring of fluid resuscitation

Monitoring traditionally relies on clinical assessment of cardiovascular, renal and biochemical parameters as indicators of organ perfusion. Heart rate, blood pressure and electrocardiography are the primary modalities. Fluid balance during burn shock resuscitation is typically monitored by measuring urine output of 0.5-1.0 mls/Kg/hr. Baseline laboratory findings are essential to monitor the progress of resuscitation. Full blood count, serum electrolytes, glucose, albumin and acid base balance are required. These should be regularly repeated. Monitoring should include central venous pressure and invasive blood pressure monitoring as a minimum and further invasive monitoring may be required but a balance of risk should be made.

All the various resuscitation formulas are only guidelines. There is a temporal relationship between the time of the burn and the fluid resuscitation required. Resuscitation is only required up to the point at which no more accumulation of oedema fluid occurs, this is usually 18-30 hours post injury.

Fluid requirements following burn shock resuscitation.

Maintenance fluid requirements will vary in burns patients for several reasons but the amount can be estimated. This estimation reflects the normal basal requirements plus a volume for the Burn Maintenance, reflecting the evaporation loss from the burn wound. Warden suggested that at the 24-48 hour period post-burn, urine output was an unreliable sign of adequate hydration [25]. Respiratory losses, osmotic diuresis and deranged ADH all contribute to the increased fluid loss despite adequate urine output. Patients more than 24 hours after thermal injury require higher than normal urine outputs. Plasma sodium is a more useful indicator of dehydration. [26]. Due to these complicating factors meticulous fluid balance is essential in these patients.

Control of infection

One of the major advances in burn care is the decreased mortality due to the control of infection. Between 1966 and 1975 60 – 80 % of patients with burns over 50% TBSA died of bacterial sepsis. The introduction of topical antimicrobials decreased wound sepsis significantly. Early excision and coverage of the burn wound further decreased mortality and morbidity. The use of silver

containing antimicrobials with their low incidence of side effects has become the mainstay of topical antimicrobial therapy [14].

Pattern of wound infection

Burn wounds should be recognised as sites of microbiological colonisation, which if left unchecked will proceed to wound invasion and systemic dissemination; this colonisation initially arises from patients own resident bacteria [27-30]. Bacteria on the surface of skin will be killed by the original thermal injury, but bacteria in hair follicles and sebaceous glands may survive. These bacteria multiply and when they reach a level of 10^5 bacteria per gram of tissue they will begin to migrate through tissue invading dermal and subcutaneous levels. Perivascular bacterial growth causes localised thrombosis and necrosis of remaining dermal elements transforming partial thickness burns to full thickness injury. Therefore bacterial levels above 10^5 per gram of tissue produce wound infection. As levels of bacteria increase so does the incidence of septicaemia.

Maintaining wounds at low contamination levels reduces the frequency and duration of septic episodes. Preventing infection in burns patients requires two defensive strategies. The first is the establishment of techniques which obstruct burn wound contamination [27,31,32]:

- Total area disinfection including wound and the surrounding area.
- Topical antimicrobial therapy to restrain local wound contamination and potential sepsis [27].
- Excision of potential source of infection by debridement and wound cover; this requires tertiary level of care.

The second requires

- Targeted topical and systemic antimicrobials based on microbiological surveillance
- Improve burn patient's local and general ability to resist microbiological wound invasion. This resistance is greatly enhanced by appropriate resuscitation and nutritional therapies.

Administration of systemic antimicrobials

A plethora of experimental and clinical data highlights that in vivo use of antibiotics when delayed more than 3 - 4 hrs after surgery is ineffective. The earlier antibiotics are given the more likely they are to reduce wound colonisation and infection. Although initially the burn wound will be sterile it will quickly become colonised and may become contaminated in the pre-hospital environment; prophylactic antibiotics should be seriously considered in all major burn victims.

Hyper metabolic response to Burn Trauma

Reduction in mortality has also occurred due to a better understanding of how to support the hypermetabolic response to severe trauma. The response itself comprises of an increase in the metabolic rate and peripheral catabolism. Sneve [16] described this as exhaustion and emaciation. Cuthbertson described the negative nitrogen and phosphorus balance. Moore [33] advocated the maintenance of body cell mass by continuous feeding to prevent catabolism after trauma. It is now known that the hypermetabolic response to burn injury causes increased metabolism, negative nitrogen balance, glucose intolerance and insulin resistance. In 1974 catecholamines were identified as the primary mediator of this response [34]. Hypermetabolism leads to a five or six times elevation in catecholamines which increases peripheral lipolysis and peripheral protein breakdown. Hart *et al* [35] further showed metabolic response increased with the burn size, reaching a plateau at 40 % total body surface area.

In 1984 Bessey stated that the hypermetabolic response also was due to an increase in cortisol and glucagons. Wilmore [17]

found that burn patients required an ambient temperature of 33°C and strived for a core temp of 38.5°C. Warming the environment decreased the hypermetabolic response. The stress response to burn injury is similar to all critical illness or trauma, it differs only in its magnitude and duration. Burn patients experience an ebb phase immediately following thermal injury which lasts for two to three days [36-38]. During this phase the metabolic rate and cardiac output may even decrease. After this ebb phase patients pass the hypermetabolic phase. This is characterised by a hyperdynamic circulatory response [39] with increased body temperature, oxygen and glucose consumption [40]. This response starts around day five post-burn and lasts up to nine months [41] causing erosion of lean body mass, muscle weakness and immune suppression and poor wound healing [42,43].

The increase in metabolic requirements in patients with major burns can cause major tissue breakdown leading to nitrogen loss and a lethal depletion of essential proteins. This response is temperature sensitive and may be attenuated by environmental warming.

The stress response is influenced by both the nervous and endocrine systems. The limbic system is activated by fear and nociceptive stimuli via the thalamic pathways. Cardiovascular and respiratory reflexes are altered by hypoxemia, hypercapnia and hypotension. Inflammatory markers also stimulate the hypothalamus directly. All these reset the thermoregulation set point.

Catecholamines involved in the hypermetabolic response are released from sympathetic nerves and adrenal glands. There is close correlation between plasma catecholamine levels and metabolic rate [34,44]; these levels remain elevated until wound healing is complete. Cortisol levels are also increased and facilitate the action of catecholamines.

Understanding the principles of the increased metabolic demands caused by the thermal injury allows changes to the supportive therapies that are normally instigated to critical care patients with other pathologies and types of trauma. The recovery from burn injury requires a large amount of tissue regeneration. This tissue regeneration is much more prolonged and less effective in a catabolic state. Once again limiting the effects of the hypermetabolic response is vital to a good outcome.

Hormonal manipulation of burn hypermetabolism.

The metabolic response to burn injury is produced by increases in several catabolic hormones. The strategy of hormone manipulation including blocking catecholamines, administering agents with counter-regulatory effects or the use of anabolic hormones is controversial at present. Some of these methods show promise it must be remembered that the hormonal milieu which accompanies burn injury is extremely complex and incompletely understood.

Nutritional support

The importance of increased nutritional support with high calorific feeding was identified as early as 1909 by Shaffer and Coleman. Curreri *et al* [45] retrospectively studied a number of burns patients to quantify the amount of calories required to maintain body weight. Sutherland [46] suggested that children required even higher numbers of calories per total body surface area burnt. Herndon [47] subsequently showed that supplemental parenteral feeding increased both immune deficiency and mortality and thus recommended continuous enteral feeding as a standard of burn treatment.

It is now clear that that early enteral feeding has enormous value in the treatment of significant burn patients; it can be started safely within hours of burn injury and that doing so will improve the

nitrogen balance and overall nutrition [48]. With gradual increases in infusion rates, tube feeding are usually well tolerated even during the first day post burn. The use of nasal-jejunal tubes will allow feeding during surgery or transfer of patients without increased risk of aspiration or other complications. Such policies reduce the interruptions in feeding, assure adequate nutrition in patients undergoing multiple procedures and may reduce the incidence of infectious complications [49].

Specific nutrient requirements

Carbohydrates

The major source of calories for thermally injured patients should be carbohydrates. Glucose is the preferred fuel for healing wounds and accessory metabolic pathways to provide glucose including the Alanine and Cori cycles. The major complication with high carbohydrate diets is glucose intolerance. Patients may require significant amounts of supplemental insulin to improve glucose absorption.

Protein

The hormonal environment of burn injury greatly increases proteolysis and provision of carbohydrate and fat calories only partially reduces protein catabolism. Increased quantities of protein must be supplied to satisfy both ongoing demands and to provide amino acids for wound healing, synthesis of enzymes, immune competence and other functions. Proteins should not be administered primarily as an energy source. Calculated energy requirements should be met by non-protein calories.

Lipids

A small quantity of fat is an essential component of nutritional support however the hormonal environment of burn patients of the burn patient suppresses lipolysis and limits the extent to which lipids can be utilised for energy. Most authorities recommend that fat comprise no more than 30 % of non protein calories.

Trace elements

In addition to the major nutrients metabolism and excretion of so called micronutrients, vitamins and trace elements, which are important in wound healing and immunity, are greatly affected by the burn injury. Limited clinical data has been collected on the amounts required however attention to these components of a balanced diet should be made. These nutrients include Vitamins A and C, Iron, Zinc and Selenium.

Inhalational injury

The latest area to advance in the treatment of burn injury is that of concomitant pulmonary injury – a vast topic that would merit a review in its own right but Shirani, Pruitt and Mason in 1987 reported that smoke inhalation and pneumonia in addition to age and burn size greatly increased burn mortality. Of 1256 burns patients treated at the US Army Institute of surgical research from 1985-1990 330 received a significant inhalational injury with 97 of those 330 (29.4%) succumbed to their injuries [50].

Inhalational injury is an acute respiratory tract insult caused by steam or toxic irritants such as fumes, gases and mists. Inhalational injury may occur with out cutaneous burn injury though the two often occur together. The physicochemical properties of the causative agent, the amount of smoke inhaled, any pre-existing disease which may lower patients immunological resistance will all go to determine to site and degree of injury.

Inhalational injury continues to be one of the most serious associated injuries complicating the care of the thermally injured patient. Prevention or early diagnosis and treatment of associated complications are necessary to decrease its associated morbidity and mortality.

Treatment algorithms for pathophysiologic events resulting from inhalational injury		
Problem	Diagnosis / Treatment	Seen in
Hypoxia	Supplemental oxygen	All injuries
Reactive Bronchorrhoea, Copious secretions	Incentive Spirometry, Chest physiotherapy, Nasotracheal suctioning	All Injuries
Inspissated Secretions	Humidification nasotracheal suctioning	Moderate and severe injuries
Wheezing	Diagnostic bronchoscopy to distinguish endobronchial obstruction (plugging) from bronchospasm	Moderate to severe injuries
Plugging (inspissated mucus or mucosal slough)	Humidification, therapeutic bronchoscopy as needed, aerosolised heparin	Moderate to severe injuries
Bronchospasm	Nebulized B 2 agonist, if ineffective then intravenous aminophylline	Moderate to severe injuries
Respiratory failure	Intubation, mechanical ventilation, permissive hypercapnia, tracheotomy if failure prolonged	Severe injury

Table 2. Treatment Algorithms for Inhalational Injury.

The realisation that patients with inhalational injury should not be under resuscitated has been emphasised already. A patient with a major inhalational injury is likely to require 2 mls / kg / % total burn surface area more fluid in the first 24 hours compared with a similar burn without the inhalational injury [51]. It should be considered standard care that these patients should receive protective ventilatory strategies. High frequency oscillating ventilation is becoming increasingly accepted as a useful treatment modality [52]. Employing treatment regimes to support the respiratory system is now common practice however there are various regimes being employed. The majority include nebulized heparin, N-acetylcysteine and bronchodilators either in conjunction or in isolation (Table 2).

Stress ulceration

For many years it has been known that there is a significant risk of gastric ulceration in burns patients. Czaja, McAlhany and Pruitt studied [53] the stomachs of burns patients with burns over 40% total body surface area and found that 86% had signs of significant gastric erosions. However the introduction of antacids and later gastric acid prophylaxis has virtually eliminated gastric ulcers as a significant problem in patients with major burns. Continuous enteral feeding has also helped to maintain the integrity of the gut mucosa, decrease bacterial translocation and minimises the need for antacids.

Early wound excision

One of the most effective therapies in decreasing mortality from major thermal injuries has been the early excision of the burn wound and its coverage by various techniques. Excision and grafting was pioneered by Jackson and colleagues in 1954 starting with small burns and progressing to burns of up to 30% [54]. Janzekovic working alone in Yugoslavia in the 1960s developed the technique of tangential excision of burn wounds [55]. Dr Burke during the 1970s and 1980s [56] reported an unprecedented survival after massive excision to the level of fascia in children with burns over 80%. He practiced early burn excision using a combination of tangential excision for the smaller burns and excision to the level of fascia for the larger burns. He demonstrated a reduced length of hospital stay and mortality in massive burns. Herndon *et al* [57] demonstrated a decrease in mortality in massively burned adults with full thickness burns when they were treated with early excision of the total burn wound as opposed to conservative management. Herndon *et al* also demonstrated that massively burned children with 95% total body surface area burns had a 50% survival rate after early wound excision. The process of early wound excision is extremely resource intensive and requires experienced clinical input on a multidisciplinary basis only found at tertiary centres (Figure 2).



Figure 2. Multi disciplinary teams includes multiple surgical and anaesthetic teams treating a single casualty for early burn wound excision. The increase in resources required to undertake early burn wound debridement in a tertiary referral unit is substantial.

At present these resources are not available at the military field hospital. As a result emphasis is placed on early repatriation of burns victims to Role 4 facilities.

Conclusion

Thermal injury will always be a cause of military casualties. The number of casualties receiving thermal injury on the battle field will vary for several reasons including the type of warfare, the weapon systems deployed and the preventative measures undertaken.

Major advances in burn care have produced a dramatic drop in mortality and morbidity over the last 5 decades. A lot of these advances can be initiated at the military field hospital by clinicians not particularly experienced in treating major burns. Those techniques that are not able to be deployed due to the environmental constraints can be mitigated by prompt evacuation of the casualty to appropriate tertiary care centres in the UK. Where evacuation to tertiary facilities is not possible, such as with local nationals, will pose a dilemma. The decision to undertake definitive treatment, including early wound excision, with the use of scant resources in a suboptimal environment with reduced likelihood of a successful outcome will have to be balanced against the ethical decision about withholding treatment on patients with potentially survivable injuries if they were treated in the UK.

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