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# On scene first aid and emergency care for burn victims

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## Abstract

Burn injury is damage to the skin or other body parts caused by extreme heat, flame, or contact with heated objects or chemicals. In United Kingdom (UK), it accounted for 175,000 emergency department attendances and 15,000 admissions. A further 250,000 burn patients were managed in the community by general practitioners or other healthcare professionals. A survey in 1998 showed that up to 58% of UK ambulance services had no specific treatment policy for burn patients. In Ireland and Australia, only 23% and 39% respectively, had employed the correct first aid burn management in studies conducted on their primary careers. Early burn management, both on scene and upon arrival to the hospital, are important to reduce the potential morbidity and mortality of burn victims. The initial management of burn, from removal of patients from zone of incident to the topical administration of cool water, has been shown to significantly reduce the extent of injury of burn patients. Further critical and timely assessment and management of these patients, pre-hospital and on arrival to emergency department, improve their chances of survival through adequate airway management and resuscitation. The need for emergency surgical procedure from emergency department to operating theatre should also be instituted when warranted without delay. Here, we review the pathophysiological rationale and evidence of practice behind each of these steps, from the first aid burn treatment to their assessment and resuscitation, and finally emergency procedure, together with their ancillary treatment, in practice.

***Keywords:*** First aid, emergency burn care, inhalation injury, burn referral criteria, burn resuscitation.

## Introduction

Burn injury is damage to the skin or other body parts caused by extreme heat, flame, or contact with heated objects or chemicals. The World Health Organization estimated that 322000 people die each year from fire related burns with >95% of these occurred in developing countries [1]. The mortality rates were much greater at both ends of the age spectrum. Thirty-

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eight percent of all burn deaths were due to multiple organ failure and only 4.1% were due to burn wound sepsis [2]. The biggest factor in burn mortality was inhalation injury with increased mortality to 26.3% in the 6.5% of burn patients admitted with inhalation injury [2].

In United Kingdom (UK), burns accounted for 175,000 emergency department attendances and 13000 admissions, and a further 250,000 patient were managed in the community by general practitioners and allied professionals [3]. Although recent data from Burn Repository in USA showed significant improvement of overall burns mortality, there are still many areas that can be improved especially in the early stage of burns treatment to optimize chances of survival and outcome [2]. A survey in 1998 showed that up to 58% of UK ambulance services had no specific treatment policy for burn patients [4]. In Ireland and Australia, only 23% and 39% respectively, had employed the correct first aid burn management in studies conducted on their primary carers [5,6]. Equally, a review of the management of minor burns within the emergency departments of hospitals in Ontario, Canada, showed that 70% of responding physicians would not measure the extent of the burn area when making an assessment, whereas 45% failed to discuss analgesic requirements [7]. Based on these data, there is still much room to improve in the basic care of burn victims.

## Pathophysiology of burns

Burn from thermal source inflicts damage through various mechanisms, both locally and systemically, through direct injury and reactive physiology. The natural functions of skin are to act as a barrier to bacteria and prevention of water loss through its epidermal layer. The deeper, dermal layer provides mechanical strength and integrity through its abundant connective tissue composition, and nourishment to epidermal and dermal layers through myriad of vascular plexuses. The dermal layer is also the home to many dermal appendages such as sweat glands and hair follicles, which houses regenerative epidermal cells that are capable of replenishing injured skin. When such functions of skin are compromised, attending physician should institute treatments that will minimize the injury and compensate its deficits through adequate first aid and resuscitation. Early burns management is one of the most important determinant factor in reducing the potential morbidity and mortality of burn victims. In USA, approximately 75% of severe burn deaths occur on scene or during initial transport [8].

The pathophysiology of thermal burn injury is related to the initial distribution of heat within the skin with its severity dependent upon the temperature of the source of insult and the duration of contact [9]. On a cellular level, the initial response to thermal injury involves direct heat-induced protein denaturation and cell death. This is followed by inflammation and ischemia-induced injury, which resulted in burn of varying skin depth. Because skin is a good insulator, most burns generally involve only the epidermis or part of the dermis. Only with prolonged exposure do burn encompasses the entire dermis or extend beneath the dermis into subcutaneous tissue such as fat, muscle, and bone. However, similar extent of burn can also occur with short exposure to an object of high temperature. For instance, a thermal blistering injury can occur after 5 minutes of exposure to water at 48.9 °C (120 °F) or after just 1 second of exposure to water at 68 °C (155°F).

The depth of burn is a dynamic process, as popularly described by Jackson [10]. The histopathology of burn consists of three concentric zones with the zone of coagulation being the area closest to the heat source. The tissue in this zone is either entirely necrotic or undergoes severe denaturation of proteins and forms an eschar. The injury in this zone is irreversible. Just below this is the zone of stasis, where there is only modest denaturation of macromolecules but characterized by hypoperfusion of tissue due to significant edema and stasis. This is attributed to capillary leak and cell membrane disruption in this zone. Beneath the zone of stasis is an area of hyperemia, where blood flow gradually increases, becoming particularly prominent by about 7 days after the injury [11]. Although tissue in the zones of stasis and hyperemia are at risk for necrosis, they are potentially salvageable given optimal intervention that preserves perfusion of these zones [10]. Conversely, a burn that appears superficial may become deeper over a period of 48 to 72 h, with

the zone of stasis becoming necrotic in the face of suboptimal treatment [12]. This is especially likely to happen if the burn wound becomes infected or poorly perfused. Adequate fluid resuscitation is essential to maintain tissue perfusion in this dynamic zone [13,14].

In addition to primary tissue loss due to local protein denaturation and tissue necrosis in burn, this is rapidly followed by activation of toxic inflammatory mediators, especially in the perfused subsurface [12]. The release of vasoactive mediators translates local edema into both local and systemic fluid shifts that exacerbate hypoperfusion in vulnerable tissue, specifically in the zones of stasis and hyperemia [12-15]. Oxidants and proteases further damage skin and capillary endothelial cells, potentiating ischemic tissue necrosis [12-15]. When the burn exceeds 20% of the patient's body surface, the local tissue response becomes systemic with potential hazardous effect on distant tissues and organs [16-18]. Sequelae such as edema and altered perfusion promote progression of injury beyond the degree of initial necrotic area through worsening fluid regulation and systemic inflammatory responses [12-15]. Activation of complement and coagulation systems causes thrombosis and release of histamine and bradykinin, leading to an increase in capillary leak and interstitial edema in distant organs and soft tissue [16]. Secondary interstitial edema and organ dysfunction from bacterial overgrowth within the eschar can then result in systemic infection. Activation of the pro-inflammatory cascade and the counter-regulatory anti-inflammatory reaction then lead to immune dysfunction [16]. This increases patient's susceptibility to sepsis and multiple organ failure. The systemic response to burn also leads to a hypermetabolic state, doubling normal physiologic cardiac output over the first 48 h post-burn [19]. This is mediated by hugely increased level of catecholamines, prostaglandins, glucagons, and glucocorticoids, resulting in skeletal muscle catabolism, immune deficiency, lipolysis, reduced bone mineralization, and reduced linear growth. These systemic metabolic changes in the burn patients may continue for up to a year after injury. Therefore, mitigating factors in early treatment to decrease these effects, which include adequate first aid and resuscitation, early excision of burn and grafting, control of sepsis, supplemented nutrition through high-carbohydrate or high-protein diet, and the use of anabolic agents, need to be instituted as early as appropriate.

## The first aid and pre-hospital care

All emergencies started on scene, at the site of incident. Emergency care should therefore, includes first aid, pre-hospital and en route care before actual arrival to emergency department. The first aid is emergency care or treatment given before regular medical aid can be obtained, and it serves to provide analgesia and halt the progression of burn. Walker et al [20] summarized the consensus of the first aid management and pre-hospital care for burn victims to serve as a reminder for the carer as to the priorities in caring for burn victims (Table 1).

Historically, first aid in burns treatment ranges from the use of natural to traditional or folk remedies over the centuries, to more recent recommendation from regulatory bodies based on clinical studies. The concept of 'first aid' was believed to be initially described by the Prussian surgeon (Surgeon General) Friedrich Von Esmarch (AD 1823–1908), with his work on 'Erste hilfe' first translated from German to English in 1882 [21]. Modern first aid concept, as a set of trained drills and skills, dated only from the late nineteenth century [22].

The use of cold water as first aid for burn has the greatest volume of supporting literature in comparison to other therapies, and it has been a popular treatment throughout history. Early literature as far back as Galen (AD 129–199), Rhazes (AD 852–923), and Ibn Sina (980–1037) can be found to have used cooled or cold water for burn [23]. In 1969, St John Ambulance first aid manual advocated irrigation of burn wound with cold water even though no duration was specified [24]. More recent studies by Rose among others indicated that treatment with cold water decreased pain and mortality by reducing damage to tissues [25-32]. Other reports subversive to the use of cooled water concerned the lack of clear guidelines on the temperature of the coolant, duration of application and the effect of delay between burn injury and onset of cooling [33].

**Table 1. Consensus on first aid management of burn**

| | |
|---|---|
| SAFE approach | • Shout or call for help<br>• Assess the scene for dangers to rescuer or victim<br>• Free or remove from danger<br>• Evaluate the casualty |
| Stop burning process | • Stop burning process by allowing patient to roll on the ground, use of blanket, water or fire extinguisher<br>• Remove clothing and accessories unless adherent to the patient. Remove any jewelry, which may become constrictive<br>• Bring all clothing articles to the hospital for examination |
| Cool burn | • Irrigate the burn area for up to 20 minutes with cool running tap water [178]<br>• Ice and very cold water should be avoided<br>• Place a cold wet towel over area of small burn (<5%) on top of polyvinylchloride film (e.g. Clingfilm) dressing<br>• Caution on hypothermia especially in children |
| Dress burn and victim | • Polyvinylchloride film dressing to keep burn area clean and help in pain relief<br>• Use small sheet of dressings rather than large circumferential wrapping to avoid constricting effect<br>• Alternatively use water gel dressings to cool and dress [20]<br>• Wrap the patient in blankets or a duvet to keep warm |

Studies conducted by Cuttle et al showed that the immediate use of 2-15 $^{o}$C running cold water for 20 min duration can increase healing by limiting the depth of burns, and promoted re-epithelialization over the first 2 weeks post-burn with decreased scarring at 6 weeks [34,35]. This benefit was noticeable in treatment for as little as 10 min duration with maximum benefit observed at 3 h even in cases where the onset of treatment was delayed by up to 30 min to 1 h [33,35].

Cold water reduces the extent of burn injury by cooling the tissue below the damaging temperature and subsequently assist burn healing by preventing cells from undergoing progressive necrosis 24–48 h after burn in the zone of stasis [10,25,36]. It causes a decrease in cell metabolism, which allow the compromised cells to survive a hypoxic wound environment, stabilize the vasculature by decreasing capillary leakage, increasing dermal perfusion and re-establishing blood flow, and dampens the inflammatory response to facilitate cell survival in the burn wound. Over the years, ice application has also been described to confer benefit in burn treatment [37,38]. Modern first aid recommendations however, advise that ice deepens or worsens burn injury [39]. It also increases the risk of hypothermia especially in children or patients with large body surface area burn as demonstrated by Ofeigsson's experiments [25]. Because of the perceived potential for hypothermia after cold treatment, some researchers advocated treating burn wound with lukewarm or body temperature water [40].

Warm water as first aid burn treatment was popular in the late 1800s to early 1900s. It was recommended that burned limbs should be soaked in body temperature water of 36.9 $^{o}$C (98.4 $^{o}$F) until suitable dressings could be found [41-43]. This was to decrease pain, and also prevent shock and infection. It was believed that the application of heat in smaller doses restores the tissue to normal. However, it was also recognized as early as 1899 that further heat application to burn simply causes more harm [44].

Other substances used for first aid burn treatment by native cultures are aloe vera and tea tree oil. Aloe vera has been shown to improve treatment of first and second degree burns in a clinical review by Maenthaisong et al [45]. It significantly shortens the wound healing time by modulating collagen response and inhibits the inflammatory process in the healing wound [46-49]. There are several hydrogel products on the first aid market based on tea tree oil such as Water-Jel®, Burnshield®, Burnaid® and Burnfree®.

These products are recommended as first aid for burns, and have been adopted by Australian ambulance and paramedic services. They contain ≥90% water and melaleuca oil in a proprietary gel. To date, there is limited evidence that these products are beneficial for burn treatment, although they are reported to soothe the burn [50,51]. Their effects were attributed to their anti-inflammatory, antibacterial and antifungal properties [52-54].

Several other oils have also been recommended for first aid treatment of burn, namely lavender and thyme oil [55,56]. There are some evidence that oils, especially those derived from plants, may act as antioxidants and either directly or indirectly scavenge oxygen-derived free radicals [57,58]. One study has demonstrated that lavender oil possesses local anaesthetic, antibacterial, and antifungal properties, which makes it effective in healing wounds including burns [55,59]. Thyme oil, from the herb thyme is used in Turkey as an excellent remedy for the treatment of burns and has been shown to also have antibacterial, antifungal and antioxidant properties [56, 60]. Other oils and creams used over the years to protect against excessive burn wound exposure to air were painted grease or butter, almond or cod-liver oil, olive oil, salad oil, castor oil or carron oil, linseed, lamp oil, or carbolic acid or thymol oil soaked cotton wool dressing, and Vaseline® or lanoline cream [21,41,43,61]. Most other common household liquids have also been used as first aid to treat burn. There are reports in the literature of treatment with toothpaste, soy sauce, eggs, honey, ink, and traditional African wound treatments such as leaves, mud, burned snail shell, a mixture of urine and mud, and cow dung [62-67]. Many of these folk remedies are based on the idea that air should be excluded from a burn as quickly as possible.

## Assessment upon arrival ± referral

Most minor burns can be managed by general practitioner in primary care, but complex and major burns warrant a specialist and skilled multidisciplinary approach to optimize clinical outcome. Upon arrival to emergency department, the first decision is whether a burn can be managed at the local facility or should be transferred to a designated burns center. Table 2 outlines the criteria for referral to specialized burns center [68].

**Table 2. Criteria for referral to specialized burns center**

| |
|---|
| • If 10-40 years old: ≥15% total body surface area (TBSA) partial thickness burn or ≥5% TBSA full thickness burn |
| • If <10 or >40 years old: ≥10% TBSA burns |
| • Special area burns involving face, hands, feet and/or perineum, genitals, joints |
| • Circumferential burns |
| • In extremes of age |
| • Polytrauma |
| • Significant co-morbid disorder |
| • If surgical management indicated (deep partial thickness, full thickness burns) |
| • Electrical burns |

Other factors to consider include the presence of inhalational injury, adequate pain relief, home circumstances, nutritional requirement and continuity of care of the patient at home or in primary setting. If in doubt of adequate access to the necessary meticulous burn care, the burn patient should be referred to burns center. Because burn wound evolves over several days, it should be re-evaluated within 3 to 5 days [12,69]. Evaluation should confirm that the burn wound is healing, and no risk of joint contracture or infection. Even small area burn that take more than 14 days to heal need to be referred to specialized center [68]. Although it is possible to successfully treat the majority of partial thickness burns in the general hospital or community setting, full thickness burns should always be referred [70]. It is also advisable to refer to specialized burns center if the primary physician anticipates a potential need for physical or occupational therapy for their burn patient. Any failure to foresee such need is detrimental to the patient's recovery.

## Early burn management in hospital

Annually in the United Kingdom, around 175000 people attend emergency departments with burns from various causes [3]. Of patients referred to the hospital, some 16000 are admitted, and about 1000

patients need active fluid resuscitation [68,71]. The number of burn related deaths average 300 a year [68]. All major burns should be managed initially according to the guidelines of the American College of Surgeons Committee on Trauma and the Advanced Trauma Life Support (ATLS) manual [72-74]. The survival data analysis from 1665 burns patients from the Massachusetts General Hospital identified three risk factors for death: age over 60 years, more than 40% total body surface area (TBSA) burn, and inhalation injury [75]. Table 3 outlines the principles of emergency burn care as outlined in Emergency Management of Severe Burns (EMSB) course [69,72].

**Table 3. Emergency management of severe burns (EMSB)**

| | |
|---|---|
| Airway | • Humidified oxygen ($O_2$) at 8L/min (40%) ± Bronchodilators<br>• 100% $O_2$ supplement if suspecting CO poisoning (COHb ≥15% or altered level of consciousness suspicious of CO poisoning). If a patient has an isolated burn injury that is small and when no inhalation injury is suspected $O_2$ may not be necessary<br>• Arterial blood gas (ABG) and other bloods (as indicated)<br>• Intubation as indicated |
| Burn assessment | • TBSA<br>• Depth |
| Circulation and resuscitation | • Parkland formula 4ml/kg/%TBSA burn (first ½ in the first 8 h from the time of incident and second ½ in the next 16 h) if large burn (≥15% TBSA adult, ≥10% TBSA child)<br>• Holliday-Segar formula for maintenance fluid (4ml/kg/h for the first 10kg, 2ml/kg/h for the second 10kg, 1ml/kg/h for subsequent kg of body weight) in children<br>• Two 16G IV cannulas but the cannulation itself should not unnecessarily delay the transfer time. This should be limited to two attempts only<br>• Ideally warm resuscitation fluid used<br>• Urinary catheterisation if ≥20% TBSA adult and ≥15% TBSA children. Urine output maintain ≥0.5ml/kg/h for adult, ≥1.0ml/kg/h if child >1 year old or <30kg, and ≥2.0ml/kg/h if <1 year old<br>• Reassessment every 15-30 min<br>• Fluid boluses of 5-10ml/kg in 15-20 min and/or increase the next hour fluids to 150% of calculated volume if necessary |
| Dire or emergency surgical procedure | • Escharotomy<br>• Fasciotomy |

## *A) Assessment and parallel management of immediately life-threatening injuries*

Approximately 10% of all burns present with concomitant trauma [76]. These should be suspected, diagnosed, and managed following the guidelines of ATLS [73]. An important point to note is that in the emergency situation, attending surgeon should institute the steps in ATLS in parallel, often with the assistance of other medical personnel.

### *I) Airway*

#### I. Inhalation injury

Inhalation injury is the most frequent cause of death in burns patients [77,78]. It is a greater contributor to overall morbidity or mortality than either percentage of body surface area affected or age. It has been reported recently that the presence of inhalation injury increased burn mortality by 20% [77]. Children and the elderly are especially vulnerable due to their limited physiologic reserve. Aggressive diagnosis and early prophylactic intubation can be life saving. All

patients with facial burn or burn in an enclosed space should be assessed by an anesthetist, and the need for early intubation ascertained before transfer to a specialized burn center [79].

The clinical diagnosis of inhalation injury has traditionally rested upon high index of clinical suspicions, and a group of clinical observations [80]. The clinical warning signs observed include facial burns, singed nasal vibrissae, and a history of burn in enclosed space. Other tell-tale signs which should not be overlooked are changes in voice quality, hoarse brassy cough, croup-like breathing, productive cough with or without carbonaceous sputum (sputum containing soot), inspiratory stridor, and respiratory difficulty with flaring of alar nasae, tracheal tug and rib retraction [80-82]. Hypoxia, rales, rhonchi and wheezes are seldom present on admission, occurring only in those with the most severe injury and implying an extremely poor prognosis [83].

Chest radiograph taken on admission for the diagnosis of inhalation injury is generally of little value in immediate burn assessment [84]. It does however, provide a good baseline investigation for evaluation of progress as almost two-thirds of patients with inhalation injury develop focal or diffuse infiltrates or pulmonary edema within 5–10 days of injury [84]. Fiberoptic bronchoscopy is the current standard in diagnosing inhalation injury [81, 82]. It identifies upper airway injury through observation of soot, charring, mucosal necrosis and airway edema. A positive or negative finding on upper airway injury however, does not reflect on the possibility of lung parenchymal injury [82,85]. To evaluate lung parenchyma damage, Xenon scanning has been utilized to demonstrate areas of the decreased alveolar gas washout, which identifies sites of small airway obstruction caused by edema or fibrin cast formation [86].

Acute upper airway or supraglottic injury occurs in approximately ⅕ to ⅓ of hospitalized burn patients with inhalation injury and is a major hazard because of the possibility of rapid progression from mild pharyngeal edema to complete upper airway obstruction [87]. It is usually the clinical consequence of direct thermal insult to the upper airway as well as chemical irritation, especially during the first 12 h of injury [69]. The worsening of upper airway obstruction is marked by supraglottic structures edema with obliteration of the aryepiglotic folds, arytenoid eminences, and interarytenoid areas, which prolapse to occlude the airway [88]. Whenever a supraglottic injury is suspected, the most experienced anesthetist in airway management should perform endotracheal intubation. Securing the endotracheal tube can become increasingly difficult if not carried out immediately due to the rapid swelling that occurs within the next 72 h [89]. Acute upper airway obstruction is also exacerbated by systemic capillary leak, bronchospasm from aerosolized irritants, and decreased lung and chest wall compliances due to swelling or burn to the chest wall [90-94]. Although upper airway edema usually resolves in 2 to 3 days, it can continue to worsen and patient intubated for supraglottic injury should be monitored closely. Extreme care should continue be taken if these patients are extubated over the next 48 to 72 hours [79].

The pathophysiologic changes in the parenchyma of the lungs that are associated with inhalation injury are not the result of direct thermal injury. The damage to lung parenchyma or infraglottic injury is caused by the incomplete products of combustion which causes lower airways chemical tracheobronchitis and bronchospasms [95]. The small airway becomes occluded with sloughed from endobronchial debris and loss of ciliary clearance mechanism. Occluded segments of the lung causes increased intrapulmonary dead space and difficulty in gaseous exchange from intrapulmonary shunting, and interstitial and alveolar flooding. This predisposes the patient to serious infection of the already denuded tracheobronchial tree and poorly compliant pulmonary parenchyma over the next few days of admission [90-94].

Many toxic gases in house or industrial fire are hazardous to the lung parenchyma, in particular, the aldehydes and oxides of sulphur and nitrogen [96, 97]. The sulfates, phosphates, and chlorides derivatives are acidic and induce rapid pulmonary edema, as well as systemic acidosis. Burning polyvinylchloride (PVC's) yields at least 75 potentially toxic compounds, including hydrochloric acid and carbon monoxide [98]. Patients with pre-existing reactive airway diseases are particularly vulnerable to irritative gaseous exposure.

The treatment for inhalation injury demands vigorous pulmonary toilet and ventilatory support to

limit rapid lung deterioration. Airway clearance techniques are an essential component of respiratory management of patients with inhalation injury and it demands the involvement of respiratory therapists, nurses and physicians who play a central role in its clinical management [99]. Bronchial hygiene therapy employs several modalities such as therapeutic coughing, chest physiotherapy, early ambulation, airway suctioning, therapeutic bronchoscopy and pharmacologic agents to achieve adequate respiratory clearance [100-107].

Among the pharmaceuticals employed are bronchodilators and mucolytics. Bronchodilators have been employed with good outcome in many cases [108,109]. Most of them act on the biochemical mechanism, which controls bronchial muscle tone. Aerosolized sympathomimetics are effective in relaxing bronchial muscle tone and stimulating mucociliary clearance. Racemic epinephrine is used as an aerosolized topical vasoconstrictor, bronchodilator, and secretion bond breaker [100]. Beta antagonists such as salbutamol may assist with exacerbation of reactive airway disease, which is very common due to the inhalation of toxins and particulate debris [109]. Water, employed as a diluent for racemic epinephrine, lowers both the adhesive and cohesive forces of the retained endobronchial secretions, thus serving as a bond-breaking vehicle [100].

## II. Carbon monoxide poisoning

Combustion of carbon in an oxygen-deficient environment results in the production of carbon monoxide [98,110]. Carbon monoxide is an invisible and odourless gas with nearly 200 times greater affinity for haemoglobin than oxygen. It competes with oxygen binding sites on the haemoglobin to form carboxyhemoglobin (COHb) and thus, reduces haemoglobin oxygen carrying capacity. The deprivation of oxygen at the tissue level is made worse by a concomitant leftward shift of the oxyhemoglobin dissociation curve. Because of its high affinity for haemoglobin, only a minimum level of carbon monoxide present in fires in enclosed space can cause significant carbon monoxide poisoning. Clinical findings of headache, nausea, and behavioral disturbances occur only at COHb level above 30% [95]. The pathognomonic cherry red skin discoloration is unreliable as a sign of carbon monoxide poisoning as it only occurs at COHb level above 40% [95]. Carbon monoxide poisoning can be evaluated by measurement of serum COHb [95]. The level however, is a poor indicator of poisoning since most burn patients are placed on 100% oxygen ($O_2$) on scene and upon arrival to emergency department [111]. The time from injury to measurement is very important because it takes 4-5 h for levels to fall by ½ while patients breathe room air, and less than 1 h on 100% $O_2$ [95]. Clark et. al. has developed a nomogram to estimate the original level of COHb at the time of extrication from the fire, based both on time from extrication and $O_2$ concentration delivered between time of extrication and time to first blood gas [111].

Normal COHb levels are generally <3% but can rise to 10-15% in heavy smokers. Levels <10% are generally not considered harmful to a healthy person but can be deleterious in those with cardiovascular disease. As levels increase to >15%, symptoms such as headache and lethargy become common. At ≥30%, these symptoms are supervened by dizziness, nausea, and impaired vision, and unconsciousness develops at levels between 40% and 50%. Carbon monoxide poisoning at ≥60% is frequently associated with death.

Carbon monoxide can dissociate from hemoglobin, with the speed of dissociation determined by arterial oxygen content. Carboxyhemoglobin has a half-life of approximately 5 h in room air, reduced to 1 h in 100% $O_2$, and further reduced at hyperbaric levels of $O_2$ at 3 atmospheres to approximately ½ h [95,112]. Therefore, all suspected or significant exposure to carbon monoxide should be treated with administration of 100% $O_2$ continued for several half-lives, either by facemask or by following endotracheal intubation.

The use of hyperbaric $O_2$ in the treatment of carbon monoxide poisoning is controversial [112,113]. Physiologically, the rationale is clear as the half-life of COHb is reduced by almost 90% at 3 atmospheres compared with room air but a recent meta-analysis failed to support the use of hyperbaric $O_2$ therapy as a beneficial standard practice [114]. When considered against the backdrop of transferring a critically ill patient to a poorly accessible hyperbaric

oxygen chamber and without a clear benefit of treatment, it is hard to justify its usage in common practice.

***II) Burn assessment***

Burn should be gently cleaned and debrided on initial assessment to determine its depth. The European working party of burn specialists recommended cleaning burn with soap and water or disinfectant to remove loose skin, including open blisters [115]. The raised epidermis of bullae should be removed or deroofed, followed by excision of sloughed tissue. All blisters should be deroofed apart from the isolated lax blisters of <1 $cm^2$ [116]. Although the clinical evidence for 'deroofing' of blisters is poor, without 'deroofing' burn depth cannot be adequately examined.

**III. Burn size**

Burn size assessment is an important exercise during early burn management as it determines the amount of fluid, which the burn patient requires. There are a few commonly accepted methods to estimate the percentage of TBSA burn, with some more practical than others in their use in emergency department or specialized burns center. The initial assessment of burn size should be performed with a standardized Lund and Browder chart (Figure 1) [117]. It takes into account changes in body surface area with age and growth, therefore making it very useful across all age groups. It also has good interobserver agreement. Whilst Lund and Browder chart gives more accurate TBSA burn estimation, its use in pre-hospital setting is uncommon and they are not, therefore, as widely used in non-specialized center. The simpler Wallace's rule of nines is helpful for rapid assessment but less accurate (Figure 2) [117]. It tends to overestimate the percentage of TBSA burn by about 3%. It is taken to be the current standard technique for assessing burn area in pre-hospital setting, and hence the tool that most management decisions are based on.

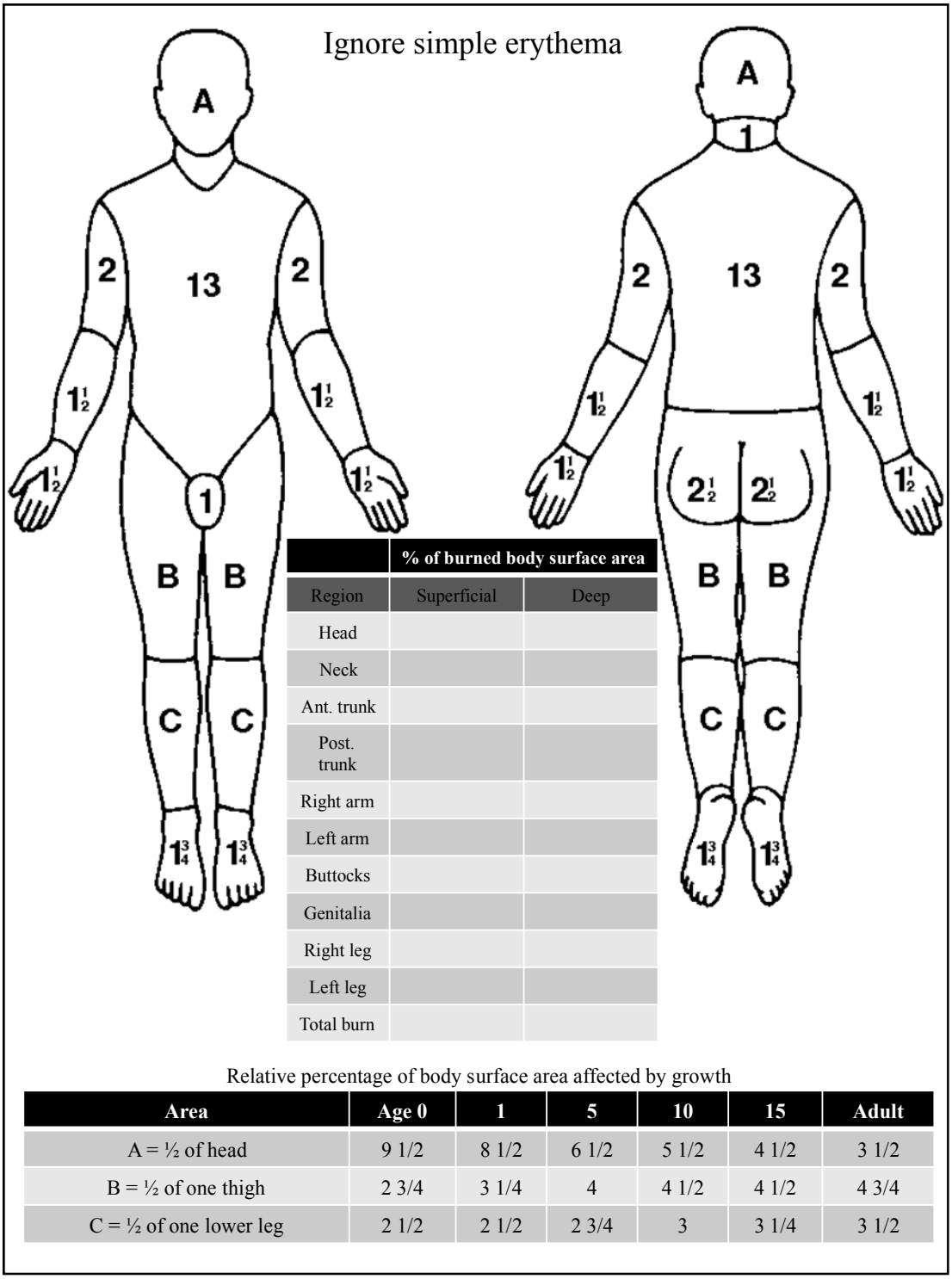
Another method of burn assessment is based on the palm being taken as a gauge of 1% TBSA. But studies of body surface area have shown that the adult palm with fingers corresponds to 0.8% of TBSA in adults and 1% in children [118]. This method is useful for estimating small burns (<15%) or large burns (>85%). In very large burns, the burnt area can also be quickly calculated by estimating the area of uninjured skin and subtracting it from 100 [119].

More recently, a serial halving method has been described [120]. This latter technique is effective in burn size estimation in pre-hospital assessment due to its rapidity and ease of use. The approach is based on serial halves of >50%, <50%, 25–50%, or <25% [120]. When compared with rule of nines, it allows management decisions to be made that are equivalent to those that would have been made using the rule of nines. This is particularly valuable in pre-hospital assessment, where a key end point is appropriate disposal to further care. However, it is advocated that the serial halving method only be used as an initial assessment tool or with conjunction with other existing methods.

**IV. Burn depth**

Quantifying the depth of burn is an important initial step in the treatment of burn as the attending surgeon weighs on the decision for resuscitation, transfer, and surgical debridement [121]. A common mistake in burn depth assessment in the inexperienced is inclusion of erythema. Only de-epithelialized area should be included in burn assessment calculation. Although it is ideal to assess burn depth accurately in emergency situation, the distinction between superficial and deep partial dermal burns is not always precise and burn wound may not be homogeneous with respect to depth. Clinical estimation of burn depth is often a subjective process with independent blinded comparison among experienced surgeons showed only 60-80% concurrence [122]. Fortunately, a detailed formal depth assessment is not necessary for partial thickness burn, as the distinction between superficial and deep partial thickness dermal burns is based largely on their healing times. Under normal circumstances, superficial partial thickness dermal burns do not require surgery and heal within 10–14 days by epithelialisation without scarring [115, 123]. Deep partial and full thickness dermal burns take longer to heal and are likely to scar. Deep partial thickness dermal burn requires excision and skin grafting. A retrospective cohort study by Cubison et. al. examined 337 children with up to a five year follow-up found hypertrophic scarring occurred in less than 20% of superficial scalds that healed within 21 days but in up

to 90% of burns that took 30 days or more to heal [123]. In order to achieve good aesthetic outcomes, all partial thickness dermal burns that have not healed by 10-14 days should be referred to a specialized burns center [115].




| | % of burned body surface area | |
|---|---|---|
| Region | Superficial | Deep |
| Head | | |
| Neck | | |
| Ant. trunk | | |
| Post. trunk | | |
| Right arm | | |
| Left arm | | |
| Buttocks | | |
| Genitalia | | |
| Right leg | | |
| Left leg | | |
| Total burn | | |

Relative percentage of body surface area affected by growth

| Area | Age 0 | 1 | 5 | 10 | 15 | Adult |
|---|---|---|---|---|---|---|
| A = ½ of head | 9 1/2 | 8 1/2 | 6 1/2 | 5 1/2 | 4 1/2 | 3 1/2 |
| B = ½ of one thigh | 2 3/4 | 3 1/4 | 4 | 4 1/2 | 4 1/2 | 4 3/4 |
| C = ½ of one lower leg | 2 1/2 | 2 1/2 | 2 3/4 | 3 | 3 1/4 | 3 1/2 |

Figure 1. Lund and Browder chart for percentage of total body surface area burn estimation.

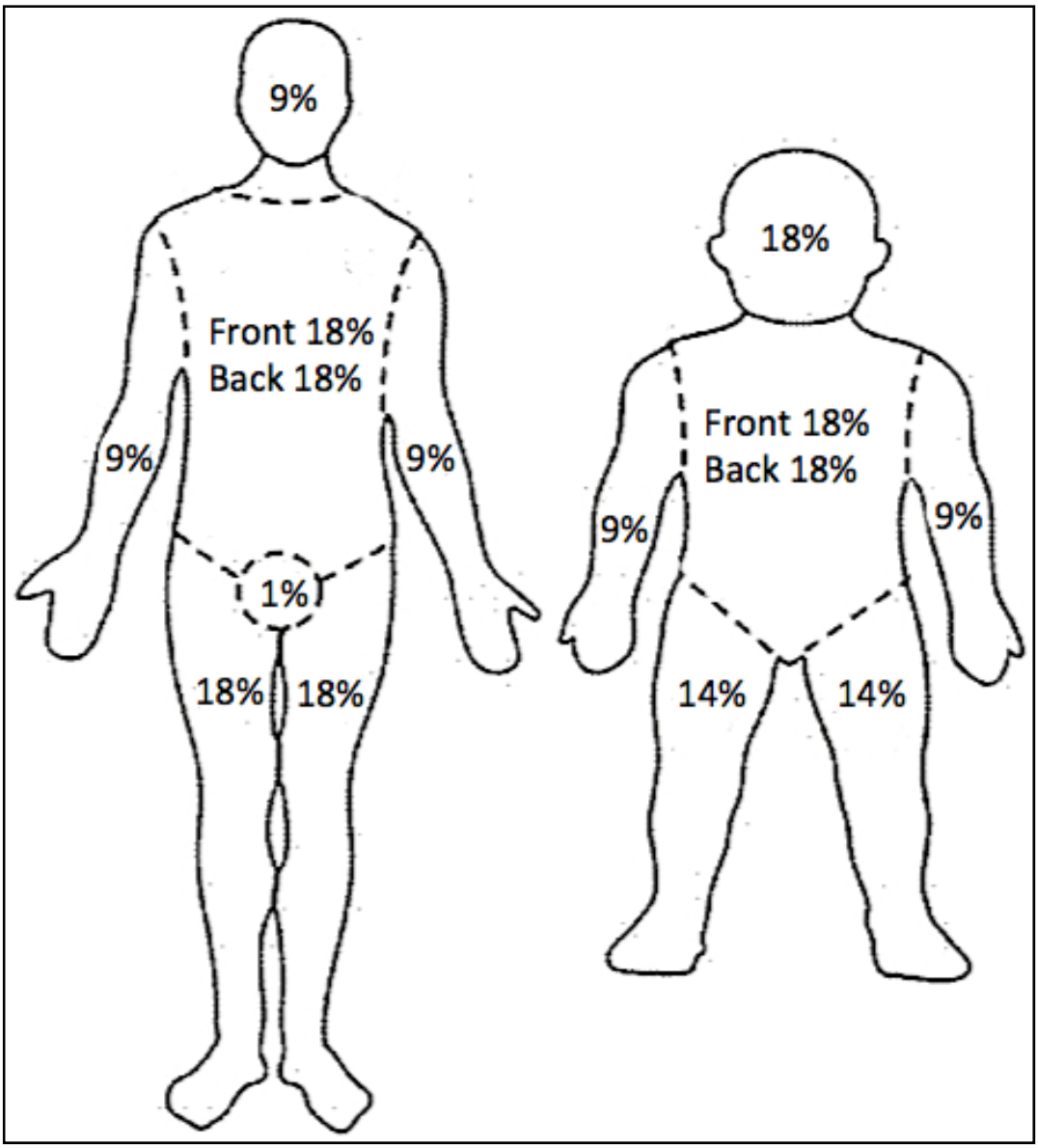



Figure 2. Wallace's rule of nines for percentage of total body surface area burn estimation. In children, the head and neck account for 18% and lower limbs 14% each. Note that for every year above one year old, the head decreases by about 1% and each lower limb gain about 0.5%. The adult proportion will be attained at 10 years old.

In superficial partial thickness dermal burns, the layer of necrosis occupies only the upper (papillary) dermis, with normal underlying reticular dermis. Clinically, such burns are pink or red, may have blistering, are painful, and have a good blood supply. These are usually managed conservatively without excision and grafting. In contrast, in deep partial thickness dermal burns, the layer of necrosis extends into the reticular dermis, with the zone of stasis extending deep into the dermis. Clinically, these burns tend to be less red with poor blood flow. Table 4 summarizes the key characteristic of various burn depth appearances as outlines in EMSB course [69]. It is important to be aware that burn wounds are dynamic and need reassessment in the first 24-72 hours because the depth can increase especially if inadequately treated or infected [69]. There have been various methods described to improve the accuracy of burn depth assessment but the high outlay costs for these equipments preclude their use outside specialized burns centers [122, 124, 125].

**Table 4. The key clinical characteristics that distinguish different level of burn depth**

| Depth of burn | Color | Blisters | Capillary refill | Sensation | Healing |
|---|---|---|---|---|---|
| Superficial | Red | No | Present | Present | Yes |
| Superficial partial thickness dermal | Pale pink | Small | Present | Painful | Yes |
| Deep partial thickness dermal | Dark pink or blotchy red | + / − | Absent | Absent | No |
| Full thickness dermal | White | No | Absent | Absent | No |

### *III) Circulation and resuscitation*

Thermal burn injuries pose a significant local and systemic insult. The presence of burn is associated with significant fluid loss due to the damage and loss of protective keratin layer of the skin and their systemic reactive changes. Effective fluid resuscitation remains the cornerstone of management in major burns. It is widely accepted that one of the principal factors responsible for the reduction in mortality from acute burn is the introduction of fluid resuscitation. In the UK, expert consensus recommends that fluid resuscitation be initiated in all children with ≥10% TBSA burns and in adults with ≥15% TBSA burns (Table 3) [14 78]. Fluid replacement should also be started on scene for burn >25% TBSA and/or if time to hospital is more than one hour from the time of injury. Children who had fluid resuscitation within 2 h had a lower incidence of sepsis, renal failure, and overall mortality [14, 69].

Over the years, several formulas based on body weight and area burnt, estimate volume requirements for the first 24 h. Although none is ideal, the Parkland formula and its variations are the most commonly used (Table 3) [126]. The aims are to maintain vital organ perfusion and tissue perfusion to the zone of stasis to prevent extension of tissue necrosis. Although multiple studies have reported the inadequacy of standard fluid resuscitation formula, with needs routinely exceeding the calculated requirements, this is likely due to variations in body mass index, accuracy of the calculated size of burn, and differences in mechanical ventilation [127-129]. It may also be that none of the present protocols are ideal because simply that different protocol suit different patient in different situation.

The preferred resuscitation fluid varies greatly. This is reflected by the evolving protocols for resuscitation from the plasma infusions of the 1940s to crystalloid resuscitation using the Parkland formula-guided Hartmann's infusion today [130-132]. There is no robust scientific evidence to support the adoption of one particular protocol over the others. Many believe that resuscitation fluids should be isotonic for the first 24 h, and then colloid added after 24 h, when capillary integrity returns [126, 133, 134]. Although in theory, the addition of colloids in burn resuscitation may decrease total volume requirement, randomized controlled trials are still needed to evaluate its full benefits [135]. A recent Cochrane meta-analysis of 65 randomized controlled trials of trauma, burns, and post-surgery patients found no evidence that colloid resuscitation reduces mortality more effectively than crystalloids [136]. Currently, the most popular type of fluid is crystalloid Hartmann's solution, which effectively treats hypovolemia and extracellular sodium deficits [126, 133]. Many burn centers continue to add colloid after the first 12 h for large burns [137]. Sodium chloride solution (0.9%) should be avoided because it causes hyperchloremic metabolic acidosis [138]. A recent survey of burns centers in USA and Canada revealed that 78% of centers used the Parkland formula to estimate resuscitation fluid volumes and that Ringer's lactate (similar to Hartmann's solution) was the most popular type of fluid used [133]. In UK and Ireland, the estimated resuscitation volumes were also calculated using the Parkland formula in 76% of units, and Hartmann's solution remained the most widely used [126]. Minor burns (<15-20% TBSA) need 150% of normal maintenance intravenous fluids [139-141]. Approximately half of the centers did not routinely change the type of intravenous fluid administered after the initial period of resuscitation [126]. Resuscitations were discontinued after 24 h in 35% of centers and after 36 h in 30% of centers [126].

Resuscitation starts from the time of injury, and thus any delay in presentation or transfer to the

hospital or specialized burns center should be taken into account, and fluid infusion rate calculated accordingly. The goal of resuscitation is to achieve enough volume to ensure end organ perfusion while avoiding intra-compartmental edema and joint stiffness. Care should also be taken not to over-infuse small, frail, elderly patients with a history of cardiac failure. Resuscitation formulas are only guidelines, and the volume must be titrated against monitored physiological parameters such as urine output, lactate, base excess, peripheral temperature, blood pressure, and heart rate [142, 143]. In UK, it is recommended that adults with burn >20% TBSA and >15% TBSA in children requires burn resuscitation monitored with urinary catheter for adequate urine output (Table 3) [69]. Patients with pre-existing conditions that may affect the correlation between volume and urine output require invasive monitoring for circulatory end points such as mean arterial blood pressure, central venous pressure, and if a pulmonary artery catheter is placed, pulmonary artery wedge pressure [128, 142, 143]. Central venous pressure or pulmonary capillary wedge pressure should be considered in patients with known myocardial dysfunction, age greater than 65 years, severe inhalation injury, or fluid requirements greater than 150% of that predicted by the Parkland formula [140].

### *IV) Dire or emergency surgical procedure*

Any deep partial thickness or full thickness dermal burns that encompass or almost encompass a region of the body can form a tight tourniquet as a result of eschar formation which limits tissue elasticity and excursion [69]. On initial examination, there may be no signs of compromise, but with the development of edema from the burn injury and fluid resuscitation, problems will eventually be compounded through the combination of external splintage and internal structural compression. As edema in the region continues to worsen, this tourniquet effect is enhanced. In the extremities, it will deprive the limbs of blood supply by the circumferential banding of eschar. Any burns involving extremities must be checked for circumferential burn and compartment syndrome [69]. The release of a restricting eschar and sometimes, fasciotomy will improve perfusion of distal extremity.

As the resuscitation continues, chest and abdominal walls may also become edematous. This causes constriction to the trunk by the existing eschars, which makes ventilation more difficult especially in patients with an already compromised, direct pulmonary injury. Ventilation insufficiency is due to the presence of burn eschar on the chest, forming a tight cuirass that restricts movement of the chest wall. These truncal constrictions require rapid and complete escharotomies to allow the underlying viable tissue to springs through the eschar and facilitate respiratory excursion. Often, circumferential burn of the neck, especially in children, can worsen respiratory embarrassment due to unyielding eschar that externally compresses and obstructs the airway. Escharotomy to the neck will be helpful in reducing the tight collar eschar and therefore, decrease the pressure exerted on the trachea. The elevated intra-abdominal pressure reduces the excursion of the diaphragm causing diminished functional reserve capacity of the lung. Raised abdominal compartment syndrome also has a negative impact on splanchnic, renal, and limb perfusion.

Theoretically, escharotomy should be a painless procedure, requiring no anaesthesia. However, it is rare for a burn to be full thickness in its entirety without areas of partial thickness loss that are exquisitely painful. Escharotomy bleed profusely and therefore, need to be performed in a monitored environment equipped with electrocautery and conscious sedation [76]. For this reason, it is good practice to infiltrate the proposed line of incision with a solution of 0.5% lidocaine with 1:200,000 epinephrine (maximum dose of 7 mg/kg), leaving it for at least 10 min before incising. The addition of hyalase (1500 iU per 50 ml) to the solution will improve penetration of the eschar. The incision should extend into normal tissue in both extent and depth, with the healthy tissue bulging through the release, forcing the eschar apart. Fasciotomy may also need to be undertaken in patients with deep burn, or those caused by high voltage current. Fasciotomy for release of edematous muscle should be performed in controlled environment such as in operating room to allow for appropriate visualization of the anatomy [76]. Although escharotomy may be needed to avert respiratory distress or vascular compromise of the limbs from constriction in full thickness

circumferential burns involving the neck, chest, abdomen, or limbs, a proportion of patients will have no signs of any compromise to circulation or ventilation. In these cases, escharotomies should be performed prophylactically if they are to be transported any distance without ability for regular monitoring or to act on new onset clinical findings. In the limbs, prophylactic escharotomy was proven to be beneficial [100]. Whilst the value in the fingers is controversial, a controlled study demonstrated a statistically significant number of phalanges were salvaged in circumferentially burned fingers with early escharotomy [144].

Care should be taken in the placement of incisions for escharotomy to insure adequate release and to avoid damaging vital structures such as nerves and vessels. The concept and techniques for escharotomy and fasciotomy have been well described by Burd et. al. based on the fundamentals of decompression [145]. Figure 3 illustrates in brief, the position of escharotomy incisions as taught in EMSB course in UK [69].

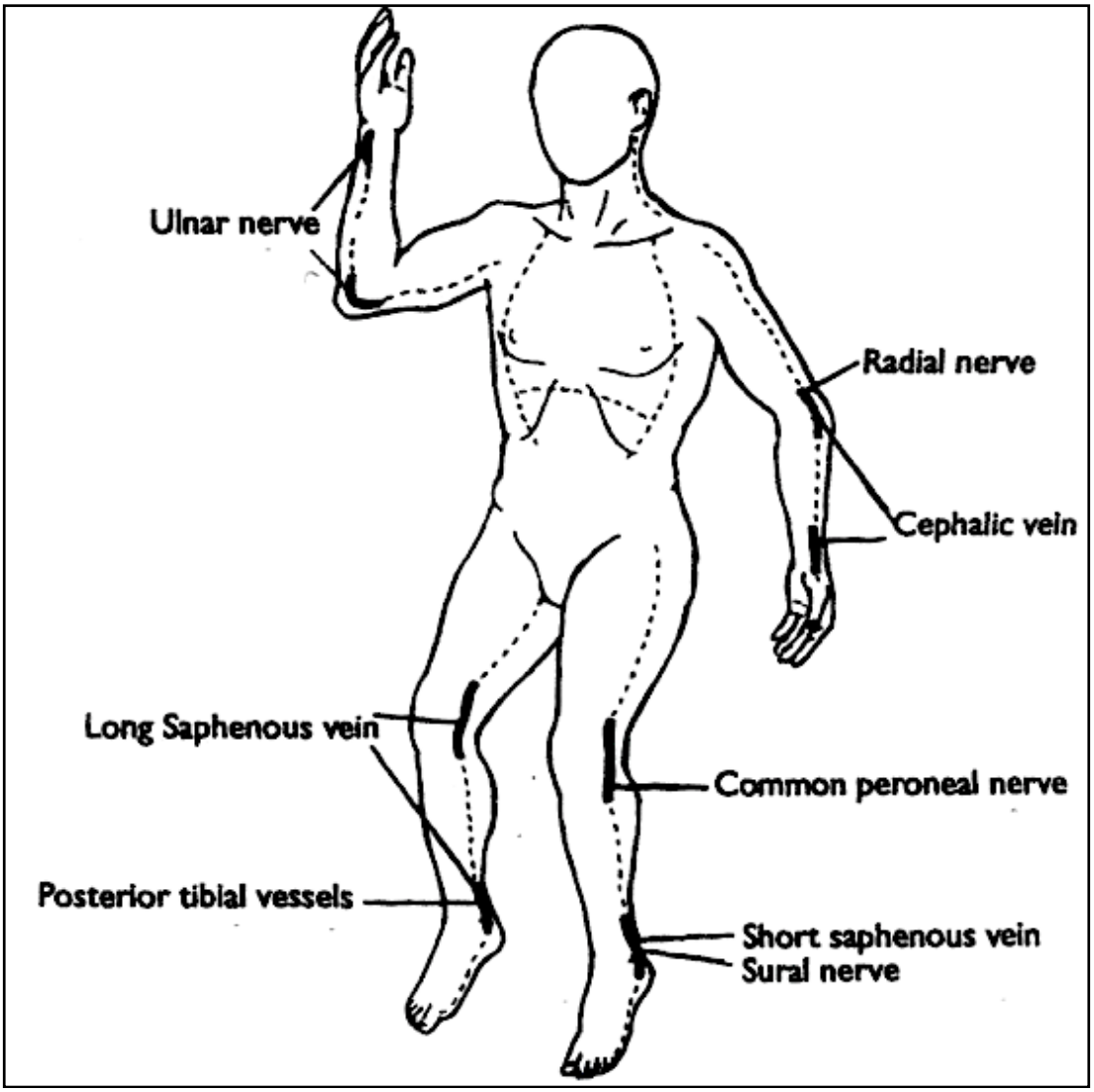



Figure 3. Sites of escharotomy incision (dotted lines) with particular attention over areas where vital nerves and vessels (thick lines) are adjacent to the site of incisions.

### *B) Analgesia*

The pain threshold temperature for skin is 42 $^{o}$C [20]. Control of burn pain must begin upon initiation of medical care. As previously indicated, analgesia is best accomplished by cooling and covering the burned area [4]. Once intravenous access is gained and resuscitation started, intravenous opioids should be administered. Intravenous morphine is the first line of acute pain management in burns (Table 3). Its effect is immediate with peak analgesia at 10 min. The recommended intravenous dose for adult is 1–2 mg repeated every 3–5 min, and 20 mcg/kg repeated every 3–5 min in children above one year old [146]. The opioid dose occasionally exceeds the standard weight-based recommendations and is necessary to achieve adequate pain control. Intravenous opioids can be titrated to make the adult patient more comfortable and should be accompanied by an antiemetic. There is no evidence that opioid addiction occurs more often in burn patients than in other populations requiring opioids for acute pain (approximately 1 in 3000) [147]. Oral opioids can also be administered in adults if no intravenous access available. Entonox should only be used when these options are unavailable as it is difficult to administer, has varying efficacy, and decreases the oxygen delivery. It is typically self-administered by an awake, cooperative patient via a mouthpiece or face mask in a concentration of 50% nitrous oxide and 50% oxygen [148]. In children, intranasal diamorphine is another alternative option that may be considered [149, 150].

### *C) Antibiotic use*

The use of systemic prophylaxis antibiotic in burn is controversial. In the acute setting, Ravat et. al., through their review recommended that systemic antibiotic prophylaxis could be used in patients needing invasive surgery but not in those for dressing changes. They recommended oxacillin or cloxacillin (30 mg/kg) or first generation cephalosporin (30 mg/kg) to target methicillin-sensitive Staphyllococcus. In case of allergy, clindamycin should be used (10 mg/kg) [151]. Another recent systematic review and meta-analysis by Avni et. al. reported that systemic antibiotic prophylaxis, given for 4 to 14 days after admission, cut all-cause in-hospital mortality by 46% [152]. But the authors cautioned that none of those findings are definitive as the overall methodological quality of the studies were poor. Other available data from adult, paediatric and mixed population studies had demonstrated that systemic antibiotic prophylaxis in burn patients has no role in the prevention of bacterial infections [153-160]. There has also been a paucity of evidence on the relevance of post-operative antibiotic use in the management of paediatric burn and with the lack of support of the role of prophylactic antibiotic in the surgical management of paediatric burn, there is diminishing role for peri-operative antibiotic use [153, 155, 156, 161-163]. Peri-operative antibiotic prophylaxis had not been shown to decrease the incidence of graft or donor site infection [164]. There has been significant evidence that antibiotic therapy prescribed to prevent infection of burned skin did not actually prevent it and even facilitated the emergence of multi-resistant bacteria [159]. It is likely that diffusion of antibiotic in the burned skin is questionable and cannot achieve bactericidal concentrations so that bacteria can grow and develop resistances. The compromise to skin barrier and the overgrowth of bacteria in the burn eschar leading to sepsis have led to a high rate of antibiotic resistance in common organisms.

On the other hand, early application of burn with topical antimicrobial has been shown to decrease the bacterial overgrowth and incidence of burn wound sepsis. Topical antimicrobials also keep the wound moist and control pain. Local burn wounds management with topical agents has a well documented efficiency in both preventing and treating burn infections [165]. Current treatment regimens include silver sulfadiazine, Bactroban, or Sulfamylon in conjunction with daily cleaning and debridement. Its use however, is no substitute for a timely surgical intervention. Without eventual surgery, the benefit of topical antimicrobial preparations on mortality is only minor on burns less than 40% TBSA [166].

Patients who show signs of sepsis should receive a complete work-up, with a special focus on the most commonly encountered bacteria in burn centers, including Staphylococcus aureus, enterococci and Pseudomonas aeruginosa [167, 168]. Diagnosis of infection in burn patients is not easy because clinical

and biological infection criteria are poorly relevant, especially in larger burn patients. Clinical parameters such as hyperthermia, hyperleukocytosis and increased C-reactive proteins can be part of physiological reactions to large thermal insult. A major burn triggers a systemic inflammatory response syndrome (SIRS), which mimics usual clinical and biological signs of infection [169]. Children with significant burn can often have moderate fever in the absence of infection and in this circumstances, administration of broad-spectrum antibiotic is not appropriate and may ultimately worsen outcomes in previously uninfected children [162, 163]. On the other hand, there is also insufficient data to show judicious use of antibiotics in febrile paediatric victims being unacceptable. As long as the infection is not documented, antibiotic therapy is empiric. Therefore, broad-spectrum antibiotics should be chosen for maximum efficacy. In established burn wounds sepsis, targeted antibiotic usage is helpful to eradicate the bacteraemia or septicaemia, and reduces mortality rates [163].

## Transfer

There are a number of published guidelines and requirements concerning the transport of the critically ill and injured [170, 171]. These should be used in devising local policies with the inclusion of specific requirements of burn patients as laid down in EMSB course [172]. Of primary importance is the need to avoid any delay in the transfer of someone with burn injury to a place of definitive care. All treatment should be carried out with the aim of reducing on scene times and delivering the patient to the appropriate treatment center. This should be the nearest appropriate emergency department, unless local protocols allow direct transfer to a specialized burns center. The use of retrieval teams and/or aeromedical transfer needs to be balanced in each case. Communication with the recipient hospital should give essential information only such as age, sex, incident time and mechanism, ABC problems, relevant treatment received and expected time of arrival [3, 173-177]. Important issues to also consider when transporting children with serious burn include maintenance of body temperature, fluid administration if transport time >1 h, accurate documentation, notification of family, and identification of the child's legal custodian. In some cases, the possibility of non-accidental injury should also be borne in one's mind. It is recommended by the National Burn Centre Response Committee (NBCRC) in UK that all complex burns referred for admission reach the specialized burns center within 6 h of injury across the British Isles and within 4 h of injury if referred from an urban site [3]. A failure to achieve these targets should be regarded as a critical incident and the reasons investigated.

## Conclusion

Care to the burn victims by healthcare professionals begins on scene with first aid before arrival to the emergency department. Emergency burn management during transport and on arrival to hospital should follow guidelines as set up by Advanced Trauma Life Support (ATLS) and Emergency Management of Severe Burns (EMSB) to optimize patients' outcome. Criteria for referral and transfer for severely burn patients to specialized burns center should be adhered to allow satisfactory emergency care with its ancillary procedures. Many healthcare professionals continue to require continuous education to facilitate adequacy of their performance in caring for these patients. Awareness and emphasis of prompt first aid care to burns and their subsequent referral to specialized burns center should continue to be part of healthcare professionals continuous medical education in the general hospital and primary care setting. The ever-evolving field of emergency care for burns requires primary care physicians' active participation in providing first line service to the general public. The importance of adequate emergency burn care and contributions made by primary healthcare professionals in the management of burns cannot be overemphasized as they are vital gatekeepers to the outnumbered, specialized burns centers in the world.

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