Chapter for
Emergency Surgery: Principles and Practice

Burns
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The frequency of burn injury and its omni-system effects make the treatment of burn patients a commonly encountered management challenge for the emergency/trauma surgeon. The biphasic pathophysiologic organ system changes evoked by burn injury represent the stereotypic response to injury and make the burn patient the universal trauma model (Table 1). The emergency surgery components of initial burn care include fluid resuscitation, and ventilatory support, as well as preservation and restoration of function of the other organ systems. Following resuscitation, burn patient management is focused on wound care and provision of the necessary metabolic support. The involvement of the emergency/trauma surgeon in burn wound management is dependant upon the extent of the wound, depth of the wound, and site of care, i.e. a general hospital in which an emergency/trauma surgeon might provide definitive wound care for a patient with burns of limited extent as opposed to a burn center where the wound care of a patient with extensive burns would be provided by a burn surgeon. The emergency/trauma surgeon must be able to identify those patients who are best cared for at a burn center and ensure the safe expeditious transport of those patients requiring transfer.

Epidemiology

The precise number of burns that occur in the United States each year is unknown because only 21 states mandate the reporting of burn injury and in 9 of those states only specific burns defined by etiology or extent must be reported. An estimated total number of burns has been obtained by extrapolation of those data collected in less than half of the states to the entire population. At present, 1.25 million is regarded as a realistic estimate of the annual incidence of burns in the United States.\(^1\) The vast majority (more than 80%) of those burns involve less than 20% of the total body surface, and only 190 -263 patients per million population are estimated to require admission to a hospital for burn care each year.\(^2\) Statewide hospital discharge data for 1994 in Pennsylvania identified three distinct age-related peak hospital discharge rates for burns, i.e. less
than five years, 25 to 39 years, and 65 years and older. Scalds and hot substances caused the burn injury in 58% of patients and fire and flame sources caused the burn in 34%. Personal assault and self inflicted injuries each accounted for 2% of the burns.

Within the population of burn patients requiring hospital care, there is a smaller subset of approximately 20,000 burn patients who, as defined by the American Burn Association (Table II), are best cared for in a burn center each year. That subset consists of 42 patients per million population with major burns and 40 patients per million population having lesser burns but a complicating co-factor. Those patients are typically cared for in the 132 self-designated burn care facilities in the United States and the 14 similar facilities in Canada, the geographic distribution of which correlates closely with population density. That distribution may necessitate the use of aeromedical transfer by either fixed wing or rotary wing aircraft to transport burn patients to those facilities from remote areas.

There are identifiable populations at high risk for specific types of injuries that will require treatment by the emergency/trauma surgeon. Scald burns, which cause over 100,000 patients to seek treatment in hospital emergency departments annually, are the most frequent form of burn injury overall. Sixty-five percent of children, age four and under, who require in-hospital burn care, have scald burns, the majority of which are due to contact with hot foods and liquids. In children of all ages treated in emergency rooms for burns, thermal burns outnumber scald burns by more than a factor of 2. The occurrence of tap water scalds can be minimized by adjusting the temperature settings on hot water heaters or by installing special faucet valves that prevent delivery of water at unsafe temperatures.

In adults, flames and the ignition of flammable liquids are the most common causes of burns. In the octogenarian population, scalds and flames each cause approximately 30% of burn injuries. In that elderly group of patients, preexisting disease contributes to the injury event in approximately two-thirds of patients and their morbidity and mortality rates are higher than in younger patients.
One-fifth to one-quarter of all serious burns are employment related. Kitchen workers are at relatively high risk for scald injury, and roofers and paving workers are at greatest risk for burns due to hot tar. In 1988, there were 236,200 patients treated in emergency rooms for chemical injuries. Employees involved in plating processes and the manufacture of fertilizer are at greatest risk for injury due to strong acids, and those involved with soap manufacturing and the use of oven cleaners are at greatest risk of injury due to strong alkalis. Employment also defines those at greatest risk to injuries by phenol, hydrofluoric acid, anhydrous ammonia, cement and petroleum distillates.

Injuries due to white phosphorus and mustard gas are most frequent in military personnel. Civilian recreational explosive devices, fireworks, are a seasonal cause of burn injury. The highest incidence of firework injuries occurs during the 4th of July holiday in the United States, and during religious celebrations in countries such as India.

Electric current causes approximately 1000 deaths per year. One-quarter of electric injuries occur on farms or industrial sites, and one-third occur in the home. Young children have the highest incidence of electric injury caused by household current as a consequence of inserting objects into an electrical receptacle or biting or sucking on electric cords and sockets. Adults at greatest risk of high voltage electric injury are the employees of utility companies, electricians, construction workers (particularly those manning cranes), farm workers moving irrigation pipes, oil field workers, truck drivers, and individuals installing antennae. National death certificate data document an average of 107 lightning deaths annually. The vast majority (92%) of lightning-associated deaths occur during the summer months when thunderstorms are most common. Slightly more than one-half of patients killed by lightning were engaged in outdoor activities such as golfing or fishing, and a quarter of patients who died from lightning injury were engaged in employment-related activities.

Child abuse is a special form of burn injury, typically inflicted by parents but also perpetrated by siblings and child care personnel. The most common form of thermal injury abuse in children is caused by intentional application of a lighted cigarette. Burning the dorsum of a hand by
application of a hot clothing iron is another common form of child abuse. The burns in abused children who require in-hospital care are most often caused by immersion in scalding water with the injury typically involving the feet, posterior legs, buttocks, and sometimes the hands (Fig. 1). It is important that the emergency/trauma surgeon identify and report child abuse because if abuse is undetected and the child returned to the abusive environment, repeated abuse is associated with a high risk of fatality. In recent years, elder abuse has become more common and it too should be reported and the victim protected.

**PATHOPHYSIOLOGY**

**Local Effects:** The cutaneous injury caused by a burn is related to the temperature of the energy source, the duration of the exposure and the tissue surface involved. At temperatures less than 45°C tissue damage is unlikely to occur in either adults or children even with an extended period of exposure. In the adult exposure for 30 seconds when the temperature is 54°C will cause a burn injury. In the child with relatively thinner skin exposure to this same temperature for 10 seconds produces a significant degree of tissue destruction. When the temperature is elevated to 60°C, a not uncommon setting for home water heaters, tissue destruction can occur in less than 5 seconds in children. At 71°C a full thickness burn can occur in a near instantaneous manner. It is no surprise that when patients come in contact with boiling liquids, live flames or are injured in industrial accidents where temperatures can exceed 100°C significant depths of injury occur. The systemic consequences of the injuries are related to the depth and the extent of the body surface area involvement as well as the patient’s underlying physiological status and whether any other associated traumas occur. The combination of these variables of tissue involved, intensity of the heat source and duration of exposure will determine whether the patient has a full thickness injury or partial thickness injury.

The burn injury may cause three zones of damage. Centrally located is the zone of necrosis. Surrounding this is an area of lesser cell injury, the zone of stasis, and surrounding that an area of minimally damaged tissue, the zone of hyperemia, which abuts undamaged tissue. In a full thickness burn, the zone of coagulation involves all layers of the skin extending down thru the dermis and into the subcutaneous tissue. In partial thickness injuries, this zone extends down
only into the dermis and there are surviving epithelial elements capable of ultimately re-surfacing the wound. In the zone of stasis blood flow is altered but is restored with time as resuscitation proceeds. If thrombosis were to occur in a patient who is not adequately resuscitated, the zone of stasis can be converted to a zone of coagulation. The zone of hyperemia is best seen in patients with superficial partial thickness injuries as occur with severe sun exposure.

Along with the changes in wound blood supply there is significant formation of edema in the burn-injured tissues. Release of local mediators from the burned tissue as well as from leukocytes causes alterations in local tissue homeostasis. Factors elaborated in the damaged tissues include histamine, serotonin, bradykinin, prostaglandins, leukotrienes and interleukin-1. Complement is also activated which can further modify transcapillary fluid flux. The changes in tissue water content have been ascribed to increased capillary filtration as well as changes in interstitial hydrostatic pressure.13, 14, 15, 16 The net effect of these various changes is significant movement of fluid into the extravascular fluid compartment. The ongoing development of edema fluid in the burn-injured tissue conceptually represents increased vascular permeability. Subsequent changes in lymph flow from burned tissue have been ascribed to changes in lymphatic vessel patency with obstruction occurring due to serum proteins that have transmigrated from the damaged capillaries.17 Maximum accumulation of both water and protein in the burn wound occurs at 24 hours post injury.18 This accumulation in tissues can remain beyond the first week post-burn. In addition to the changes in transcapillary fluid movement within the burn injured tissues, patients who have greater than a 20-25% body surface burn have similar fluid movement in undamaged tissue beds. This may in part be related to the changes in transcapillary fluid flux and also be in response to the volume of resuscitation fluids administered.19, 20

The injuries that will be apparent on examination are the consequences of the level of tissue destruction (Fig.2). When the wounds are superficial they are associated with hyperemia, fine blistering, increased sensation and exquisite pain upon palpation. The wounds are hyperemic, warm and readily blanch. These types of injuries represent first-degree burns or alternatively are
termed superficial partial thickness injuries. With a second degree or deeper partial thickness burn the wound presents with intact or ruptured blisters or is covered by a thin coagulum termed pseudoeschar. The key physical finding is preservation of sensation in the burned tissue although it is reduced (Table III). With proper care superficial and even deeper partial thickness injuries are capable of healing. Burn blister fluid represents a near pure acellular filtrate of plasma. Interestingly when studied this fluid does not necessarily promote wound healing. Infection risk in deep partial thickness wounds is significant and if an infection develops it can lead to a greater depth of skin loss. When the injury penetrates all layers of the skin or extends into the subcutaneous or deeper tissues the wound will appear pale or waxy, be anesthetic, dry and inelastic and contain thrombosed vessels (Fig. 1). Occasionally in children or young women, the initial appearance of a wound may be more that of a brick red coloration. Such wounds will have significant edema, are inelastic and insensate. Over the subsequent days the extravasated hemoglobin, which is still fully oxygenated immediately post injury and responsible for the wound color, undergoes reduction and the wound appearance is more characteristic of that of a full thickness wound. Full thickness wounds are infection prone wounds, as they no longer provide any viable barrier to invading organisms and if left untreated become rapidly colonized and a portal for invasive burn wound sepsis.

**Systemic Response:** The organ system response to a major burn injury results in some of the most profound physiologic changes that a person is capable of enduring. The magnitude of the response is proportional to the burn size reaching a maximum at about a 50% body surface area burn. The duration of the changes is related to the persistence of the burn wound and resolves with wound closure. The organ specific response follows the pattern that occurs with other forms of trauma with an initial level of hypo-function followed by the hyperdynamic flow phase. The changes in the cardiovascular response are some of the more critical ones and directly impact the initial care and management of the burn patient. Following burn injury there is a transient period of decreased cardiac performance in association with elevated peripheral vascular resistance. This can be further compounded by failure to replace adequately the patient’s intravascular volume loss leading to further impairments of cardiac filling, decreased cardiac
output and worsening organ hypoperfusion. Systemic hypoperfusion can result in further increases in systemic vascular resistances and reprioritization of regional blood flow. The notion that the burn is responsible for causing a myocardial depressant to appear in circulation or whether the impaired cardiac performance is simply a consequence of inadequate volume restoration remains an open question. What seems to be clear from experimental studies is that when there is a failure to resuscitate a burn patient adequately there is substantially impaired myocardial performance. Conversely the provision of adequate resuscitation volumes can preserve cardiac performance. Patients receiving appropriate volume restoration during the course of their resuscitation, develop normal cardiac performance values within twenty-four hours of injury and by the second twenty-four hours those values further increase to supranormal levels. It is not uncommon to see adult patients with cardiac outputs in excess of 10 liters per minute. In association with changes in cardiac output, there is a reduction in the systemic vascular resistance to 30 – 40% of normal values. The patient at this juncture is in a hyperdynamic flow phase as part of the hypermetabolic response to the injury, which will revert back to more normal levels with wound closure. However there may be some element of increase present in major burn victims for months after recovery until the wound is fully mature.

Pulmonary changes following burn injury are the consequences of direct parenchymal damage as occurs with inhalation injury and those changes, which occur solely, related to the burn injury. With isolated burn injury neutrophil sequestration occurs in the lungs and may mediate lung injury. One potential mediator of this response may be platelet-activating factor, which serves to prime neutrophils. The changes in the pulmonary vascular response parallel those of the peripheral circulation though the increase may be to a greater degree and with a longer duration of change. Capillary permeability in the lung appears to be mostly preserved following burn injury with the primary change being an increase in the lung lymph flow but no change in the lymph to plasma protein ratio. Lung ventilation increases in proportion to the burn size with the patient having both an increase in respiratory rate and tidal volume. The increases are primarily related to the overall hypermetabolic response to the burn injury. Further perturbations in the patient’s ventilatory status not related to the presence of an inhalation injury would indicate
a supervening process. Such common events include fever, sepsis, pneumonia, occult pneumothorax, pulmonary embolism, congestive heart failure and an acute intra-abdominal process. In patients without these events, pulmonary gas exchange is relatively preserved and there is relatively little change in pulmonary mechanics.

The renal response to burn injuries is largely orchestrated by the cardiovascular response. While initially there may be a reduction in renal blood flow, this is restored with resuscitation. If the resuscitation is delayed or the fluid need underestimated, renal hypoperfusion will occur with early onset renal dysfunction secondary to renal ischemia. If the patient also experiences myoglobinuria or hemoglobinuria, which are capable of causing direct tubular damage, sequential injury can occur leading to further impairment of renal function. The changes in renal blood flow following burn injury require that the doses of certain medications such as aminoglycoside antibiotics be adjusted to attain therapeutic levels. In patients who are receiving nutritional support, large doses of carbohydrates can cause glycosuria resulting in an inappropriate osmotic diuresis necessitating therapeutic intervention, i.e. reduction of glucose load and/or administration of insulin. Daily urinary outputs in burn patients who are receiving protein loads greater than normal need to be relatively greater than in nonburn patients in order to excrete the increased solute load.

Burn induced changes in gastrointestinal tract motility and a reduced capacity to tolerate early feedings previously had been thought to preclude the use of the GI tract as the primary route for nutritional support. With near immediate institution of enteral feedings via nasogastric or nasoduodenal tubes, gastrointestinal motility can be preserved, mucosal integrity protected and effective nutrient delivery achieved. It seems that delay in the initiation of enteral feeding is associated with the onset of ileus, which can also occur when the burn resuscitation has been complicated. Patients who are under resuscitated will have alterations in GI tract motility and mucosal integrity as a consequence of intestinal hypoperfusion. Patients who have received massive resuscitation volumes will have significant edema of the retroperitoneum, bowel mesentery, and bowel wall leading to a paralytic ileus. In patients who are intoxicated at the time
of their burn injury there may be further alterations in the gastrointestinal tract with changes in the mucosal barrier function and alterations in local immunity. In the past the major gastrointestinal complications following burn injuries were related to upper gastrointestinal ulceration and bleeding. However, there has been a relative shift in the site of post-burn gastrointestinal complications with the small bowel and colon now being more often affected.

Burn injury results in an elevated hormonal and neurotransmitter response similar in magnitude to that of the "fight or flight" response. The duration of the neurohumoral response is prolonged and it can be further increased with surgical stress. This can adversely impact the burn induced metabolic changes and immune response. The increases in glucocorticoids and catecholamines correctly support the stress response of the injured patient except where this response becomes dysfunctional. In pathologic studies in humans as well as in animals when there is an insufficient stress hormone response an otherwise survivable insult becomes fatal.

Many of the multi-system changes occurring post-burn can be related in part to the alterations in catecholamine secretion particularly the changes in resting metabolic expenditures, substrate utilization and cardiac performance. As wound closure is accomplished, the altered neurohumoral response abates as the catabolic hormones recede and the anabolic hormones become predominant.

Burn injury results in the loss of balance in both leukocyte and erythrocyte production and function. Burns of greater than 20% total body surface area are associated with alterations in red cell production resulting in anemia. Patients with major thermal injuries may lose up to 20% of their red cell mass in the first twenty-four hours due to thermal destructions of red cells in the cutaneous circulation. Such loss can be further compounded by frequent blood draws, blood loss related to surgical procedures, hemodilution with resuscitation and transient alterations in erythrocyte membrane integrity. Longer-term changes appear to be related to hypo-responsiveness of the erythroid progenitor cells in the bone marrow to erythropoietin. Burn patients manifest increased circulating levels of erythropoietin following injury and attempts to augment those levels to improve red cell production have met with little success. During the early stages of resuscitation, reductions in platelet number, depressed fibrinogen levels, and
alterations in coagulation factors return to normal or near normal values with appropriate resuscitation. Subsequent changes if they occur may be related to a septic process or in the case of platelets, heparin induced platelet antibodies if heparin flushes are used as part of the maintenance protocol for intravascular devices. Changes in white cell number occur early with an increase in neutrophils due to demargination and accelerated bone marrow release. With uncomplicated burn injury, bone marrow myelopoiesis is relatively preserved. With a septic complication there appears, based on experimental evidence, to be a reduction in granulocyte formation and a relative shift to monocytopoiesis. This defect appears not to be related to a lack of granulocyte colony stimulating factor but a growth arrest within the bone marrow of granulocyte precursor cells.

In addition to the changes occurring in the bone marrow and the non-specific aspects of the host defense mechanisms there are significant further depressions in the immune response. Burn injury causes a global impairment in host defense mechanisms. Alterations of the humoral immune response include reductions in IgG and IgM secretion, decreased fibronectin levels, and increases in complement activation. Cellular changes include alterations in T-cell responsiveness, changes in the T-cell sub-populations favoring the cytotoxic/suppressor T-cell, alterations in antigen processing and presentation, reductions in IL-2 release, and impairment of delayed type hypersensitivity reactions. In addition to the changes noted in granulocytes and monocytes and their release from the bone marrow, there are corresponding functional changes. Granulocytes have been noted to have impaired chemotaxis, decreased phagocytic activity, decreased antibody dependent cell cytotoxicity, and a relative impairment in their capacity to respond to a second challenge. The relative shift to monopoiesis is associated with an increase in secretion of PGE₂. More recently dendritic cells, a critical component in the immune response, have been found to be significantly altered following burn injury with infection. The clinical importance of these observations is that the burn patient is at significant risk for post burn infectious complications. This mandates the strictest adherence to aseptic technique in the management of the wounds and the insertion of intravascular devices, the judicious use of antimicrobial agents, aggressive nutritional support, and the achievement of rapid wound closure.
Resuscitation Priorities

In the immediate postburn period the changes induced in the cardiovascular system by burn injury receive therapeutic priority. If the early postburn plasma volume loss is unreplaced, burn shock may occur accompanied by kidney and other organ failure and even death. In all patients with burns of more than 20% of the total body surface area and those with lesser burns in whom physiologic indices indicate a need for fluid infusion, a large caliber intravenous cannula should be placed in an appropriately sized peripheral vein underlying unburned skin. If no such sites are available, a vein underlying the burn wound may be cannulated. If there are no peripheral veins available, the cannula can be placed in a femoral, subclavian, or jugular vein. Lactated Ringers solution should be infused at an initial rate of 1 liter per hour in the adult and 20 ml per kg per hour for children who weigh 50 kg or less. That infusion rate is adjusted following estimation of the fluid needed for the first 24-hours following the burn.

Fluid Administration

Resuscitation fluid needs are proportional to the extent of the burn (combined extent of partial and full thickness burns expressed as a percentage of total body surface area) and are related to body size (most readily expressed as body weight), and age (the surface area per unit body mass is greater in children than in adults.) The patient should be weighed on admission and the extent of partial and full thickness burns estimated according to standard nomograms (Fig.3) or, in the adult, by the use of the rule of nines which recognizes the fact that the surface area of various body parts represents 9% or a multiple thereof of the total body surface area, i.e. each upper limb and the head and neck 9%, each lower limb, posterior trunk and buttocks, and anterior trunk 18%, and the perineum and genitalia 1%. Those surface area relationships differ in children in whom the head and neck represent 21% of the total body surface area and each lower limb, 14% at age one. The fraction of the total body surface area represented by the head decreases progressively, and that represented by the lower limbs increases progressively to reach adult proportions at age 16. The fact that the palmar surface of the patient’s hand (palm and digits) represents 1% of his or her total body surface can be used to estimate the extent of irregularly
distributed burns, i.e. the number of the patient’s “hands” needed to cover the patient’s burn wounds.39

The fluid needs for the first 24-hours can be estimated on the basis of the Advanced Burn Life Support and Advanced Trauma Life Support consensus formula:40

**Fluid required for the first 24 hours postburn**

- Adults = 2-4 ml LR/%TBSAB/Kg, BW
- Children = 3-4 ml LR/%TBSAB/Kg, BW

TBSAB = Total body surface area burned
LR = Lactated Ringers
BW = Body weight

Because of the greater surface area per unit body mass in children, the volume of fluid required for the first 24-hours is relatively greater than that for an adult. One of the authors (BAP) prefers to make the estimate using 2 ml/%TBSAB/Kg BW for an adult and 3 ml/%TBSAB/Kg BW for children to minimize volume and salt loading. The infused volume is increased only as needed to achieve adequate resuscitation. The other author (RLG) makes the estimate using 4 ml/%TBSAB/Kg BW for all burn patients out of concern about delayed initiation of infusion. One-half of the estimated volume should be administered in the first eight hours after the burn. If the initiation of fluid therapy is delayed, the initial half of the volume estimated for the first 24-hours should be administered in the hours remaining before the eighth postburn hour. The remaining half of the fluid is administered over the subsequent 16-hours.

The limited glycogen stores in a child may be rapidly exhausted by the marked stress hormone response to burn injury. Serum glucose levels in the burned child should be monitored and 5% dextrose in ½ normal saline administered if serum glucose decreases to hypoglycemic levels. In the case of small children with small burns, the resuscitation fluid volume as estimated on the
basis of burn size may not meet normal daily metabolic requirements. In such patients maintenance fluids should be added to the resuscitation regimen.

The infusion rate is adjusted according to the individual patient's response to the injury and the resuscitation regimen. The high circulating levels of catecholamines evoked by the burn and the progressive edema formation in burned and even unburned limbs commonly make measurements of pulse rate, pulse quality, and even blood pressure difficult and unreliable as indices of resuscitation adequacy. Since the hourly urinary output is a generally reliable and readily available index of resuscitation adequacy, an in-dwelling urethral catheter should be placed and the urinary output measured and recorded each hour. The fluid infusion rate is adjusted to obtain 30-50 ml of urine per hour in the adult and 1 mg per kg body weight per hour in children weighing less than 30 kg. To avoid excessive fluctuation of the infusion rate, the administration of fluid is increased or decreased only if the hourly urinary output is one-third or more below or 25% or more above the target level for two or three successive hours. If in either adults or children the resuscitation volume infused in the first 12 hours to achieve the desired urinary output or other indices of resuscitation adequacy exceeds estimated needs by more than twofold or will result in administration of six or more ml per percent body surface area burned per kg body weight in the first 24-hours, human albumin diluted to a physiologic concentration in normal saline should be infused and the volume of crystalloid solution reduced by a comparable amount. Inasmuch as functional capillary integrity is gradually restored during the first 24-hours postburn, such use of colloid-containing fluid is best reserved for the latter half of the first postburn day.

Restoration of functional capillary integrity and the establishment of a new transvascular equilibrium across the burn wound are manifested by the fact that both protein and water content of the burn injured tissue reach maxima at or near 24-hours after injury. Consequently, the volume of fluid needed for the second 24 hours postburn is less and colloid-containing fluids can be infused to reduce further volume and salt loading. Human albumin diluted to physiologic concentration in normal saline is the colloid-containing solution infused in a dosage of 0.3 ml per
percent burn per kg body weight for patients with 30-50% burns, 0.4 ml per percent burn per kg body weight for patients with 50-70% burns, and 0.5 ml per percent burn per kg body weight for patients whose burns exceed 70% of the total body surface area. Electrolyte-free, 5% glucose in water, is also given in the amount necessary to maintain an adequate urinary output. The colloid-containing fluids for the second 24-hours for burned children are estimated according to the same formula, but half normal saline is infused to maintain urinary output and avoid inducing physiologically significant hyponatremia by infusion of large volumes of electrolyte-free fluid into the relatively small intravascular and interstitial volume of the child.

During the second 24 hours after injury, fluid infusion “weaning” should be initiated to minimize further volume loading. In a patient who is assessed to be adequately resuscitated, the volume of fluid infused per hour should be arbitrarily decreased by 25% to 50%. If urinary output falls below target level, the prior infusion rate should be resumed. If urinary output remains adequate, the reduced infusion rate should be maintained over the next three hours at which time another similar fractional reduction of fluid infusion rate should be made. This decremental process will establish the minimum infusion rate that maintains resuscitation adequacy in the second postburn day.

As resuscitation proceeds and edema forms beneath the inelastic eschar of encircling full thickness burns of a limb, blood flow to underlying and distal unburned tissue may be compromised. Circulatory compromise can occur in limbs with mixed depth partial thickness burns and on occasion in limbs with less than completely circumferential burns. Edema and coolness of distal unburned skin on a burned limb are normal accompaniments of the injury and are not indicative of circulatory compromise requiring surgical release. Cyanosis of distal unburned skin and progressive parasthesias, particularly unrelenting deep tissue pain, which are the most reliable clinical signs of impaired circulation may become evident only after relatively long periods of relative or absolute ischemia. Those limitations can be overcome by scheduled (q 1-2 hours) monitoring of the pulse signal in the palmar arch vessels and the posterior tibial artery using an ultrasonic flowmeter. In an adequately resuscitated patient, absence of pulsatile flow or progressive diminution of the pulse signal on repetitive examinations are indications for escharotomy. Since the full thickness eschar is insensate, the escharotomy can be performed as a
bedside procedure without anesthesia using a scalpel or an electrocautery device. On an extremity, the escharotomy incision, which is carried only through the eschar and the immediately subjacent superficial fascia, is placed in the mid-lateral line and must extend from the upper to the lower limit of the burn wound (Fig.4). The circulatory status of the limb should then be reassessed. If that escharotomy has not restored distal flow, another escharotomy should be placed in the mid-medial line of the involved limb. A fasciotomy may be needed when there has been a delay in restoring the patient's limb circulation and in particular if the patient is receiving a massive fluid load. Mistakes in the performance of escharotomies include injuries to extensor tendons and digital neurovascular bundles, insufficient depth and length of the incision, and delay in performing the escharotomy. Additionally, delayed bleeding from previously thrombosed vessels transected during the escharotomy should be promptly controlled. Continuous elevation and active exercise of a burned extremity for 5 minutes every hour limits edema formation and may eliminate the need for escharotomy.41

Edema formation beneath encircling full thickness truncal burns can restrict the respiratory excursion of the chest wall. If the limitation of chest wall motion is associated with hypoxia and progressive increase in the work of breathing, and peak inspiratory pressure, escharotomy is indicated to restore chest wall motion and improve ventilation. These escharotomy incisions are placed in the anterior axillary line bilaterally, and if the eschar extends onto the abdominal wall, the anterior axillary line incisions are joined by a costal margin escharotomy incision (Fig.4). Rarely, encircling full thickness burns of the neck or penis will require release by placement of an escharotomy incision in the mid-lateral line(s) of the neck or the dorsum of the penis.

Fluid management after the first 48 hours postburn should permit excretion of the retained fraction of the water and salt loads infused to achieve resuscitation, prevent dehydration and electrolyte abnormalities, and allow the patient to return to preburn weight by the 8th to 10th postburn day.39 Infusion of the large volumes of lactated Ringers required for resuscitation commonly produces a weight gain of 20% or more and a reduction of serum sodium concentration to approximate that of lactated Ringers, i.e. 130 mEq/l. Such patients do not need additional sodium and, in fact, have an elevated total body sodium mass in association with increased total body water. Correction of that relative hyponatremia is facilitated by the
prodigious evaporative water loss from the surface of the burn wound which is the major component of the markedly increased insensible water loss that is present following resuscitation. Insensible water loss, which must be taken into account in post-resuscitation fluid management, can be estimated according to the formula:

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\text{Insensible water loss (ml per hour)} = (25 + \% \text{TBSAB}) \times \text{TBSA in square meters}^{42}
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\text{TBSA} = \text{Total body surface area burned}
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\[
\text{TBSA} = \text{Total body surface area}
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That water loss should be replaced only to the extent that will permit a daily loss of 2-2.5% of the resuscitation associated weight gain (as measured 48 hours after the burn) until preburn weight is attained. Inadequate replacement of insensible water loss makes hypernatremia the most commonly encountered electrolyte disturbance in the extensively burned patient following resuscitation. Such hypernatremia should be managed by provision of sufficient electrolyte-free water to allow excretion of the increased total body sodium mass and replace insensible water loss to the extent needed to prevent hypovolemia.

During resuscitation hyperkalemia is the most frequently encountered electrolyte disturbance and is typically a laboratory sign of hemolysis and muscle destruction by high voltage electric injury or a particularly deep thermal burn. Hyperkalemia may also occur in association with acidosis in patients who are grossly under-resuscitated. Hyperkalemia in the burn patient is treated as in any other surgical patient. In the case of patients with high voltage electric injury, emergency debridement of nonviable tissue and even amputation may be necessary to remove the source of the potassium. Hypokalemia may occur after the resuscitation period in association with alkalosis as a consequence of hyperventilation and may also accompany post-resuscitation muscle wasting. Potassium losses are increased by the kaluretic effect of the mafenide acetate in Sulfamylon® burn cream and, as noted below, by transeschar leaching in patients treated with 0.5% silver nitrate soaks.

Significant depression of ionized calcium levels is uncommon, but total calcium levels may be depressed if calcium binding proteins such as albumin decrease as may happen in patients
receiving a high volume of resuscitation fluid. In extensively burned children, hypocalcemia has been associated with hypoparathyroidism and renal resistance to parathyroid hormone. Symptomatic acute hypocalcemia should be treated with intravenous calcium (90-180 mg of calcium infused over 5-10 minutes) to control cardiac dysfunction and neuromuscular hyperactivity. Prolonged administration of parenteral nutrition and/or failure to supply sufficient phosphate to meet the needs of tissue anabolism following wound closure may cause hypophosphatemia. Administration of large volumes of antacids for stress ulcer prophylaxis is an infrequent cause of hypophosphatemia now that acid secretion inhibitors are used for that purpose. Hypophosphatemia can be prevented and treated by appropriate dietary phosphate supplementation.

The timely administration of adequate fluid as detailed above has essentially eliminated acute renal failure as a complication of inadequate resuscitation of burn patients. In a recent 10-year period, at the U. S. Army Burn Center, only two out of 2132 burn patients treated at the U. S. Army Burn Center developed early renal failure, and those patients had established anuria when they were received in transfer from other institutions. Far more common today are the complications of excessive resuscitation, i.e. compartment syndromes and pulmonary compromise. Compartment syndromes can be produced in the calvarium, muscle compartments beneath the investing fascia, and the abdominal cavity. Cerebral edema which may efface the epidural space and compress the ventricular system is most apt to occur in burned children and is manifested by obtundation and changes evident on computerized tomographic scans. Such changes should be addressed by maintaining cerebral perfusion pressure and minimizing further edema formation by reducing fluid infusion rate and inducing diuresis. Anterior ischemic optic neuropathy manifested by visual field defects and even blindness, is another manifestation of excessive fluid infusion. This complication has typically occurred in association with other compartment syndromes and anasarca in critically ill patients, particularly in those who are nursed in a prone position. Consequently, prone positioning should be avoided in severely injured, critically ill burn patients requiring large volumes of parenteral fluids. The occurrence of visual field defects should prompt alteration of fluid therapy and induction of a diuresis.
Excessive fluid administration may also cause formation of enough ascitic fluid and edema of the abdominal contents to produce intraabdominal hypertension. The abdominal compartment syndrome represents progression of intraabdominal hypertension to the point of organ dysfunction, i.e. typically oliguria, and/or altered pulmonary mechanics. Infusion of resuscitation fluid volumes in excess of 25% of body weight has been associated with a high incidence of abdominal compartment syndrome. In all patients who have received that volume of resuscitation fluid, hourly monitoring of intracystic pressure should be instituted. Elevation of intracystic pressure above 25 mm of mercury, as measured through a urethral catheter, should prompt therapeutic intervention beginning with adequate sedation, reduction of fluid infusion rate, diuresis and paracentesis. If organ failure becomes evident a mid-line abdominal incision should be made to reduce the elevated intraabdominal pressure. That incision can be temporarily closed with a polyethylene “bag,” or a vacuum-assisted closure device. The abdomen can be definitively closed as soon as visceral edema resolves.

Compartment syndromes may also occur in the muscle compartments underlying the investing fascia of the limbs of burn patients, even in limbs that are unburned. To assess compartment pressure the turgor of the muscle compartments should be assessed on a scheduled basis by simple palpation. A stony hard compartment is an ominous finding which should prompt further evaluation. The adequacy of arterial flow in all limbs of a burn patient should be monitored on a scheduled basis using the ultrasonic flowmeter. Ultrasonic detection of pulsatile flow is reassuring but the ultrasonic flow signals can be misleading since flow in large vessels may be maintained even though microcirculatory flow is severely compromised within a muscle compartment. Direct measurement of intracompartmental pressure using either a wick or needle catheter is much more reliable. A muscle compartment pressure of 25 mm of mercury or more necessitates performing a fasciotomy of the involved compartment in the operating room using general anesthesia.

Edema causing airway obstruction, in the absence of inhalation injury, has also been attributed to excessive resuscitation fluid. This complication, which necessitates endotracheal intubation, has been of particular concern in small children with extensive scald burns who have received in excess of 6 ml of lactated Ringers per percent body surface area burned per kilogram body
weight. Pulmonary edema is rare during the initial 48-hour resuscitation period, but may occur in patients with extensive burns following resuscitation when the edema fluid is being resorbed and the protective early postburn pulmonary vascular changes have dissipated. The treatment of pulmonary edema in the burn patient is the same as for other patients.

VENTILATORY SUPPORT AND TREATMENT OF INHALATION INJURY

Pathophysiology: Patients suffering both inhalation injuries and thermal burns have a significantly increased incidence of complications and probability of death. While an inhalation injury alone carries a mortality of 5%-8%, a combination of a thermal injury plus inhalation injury can easily result in a mortality 20% above that predicted on the basis of age and burn size. Patients who have an otherwise survivable injury may succumb to their burn as a consequence of their inhalation injury and the complications that occur, particularly a gram negative pneumonia.47 Injuries to the airway are due to the direct damage of inhaled products of combustion or pyrolysis that cause inflammation and edema. Damage to the airway, while in part related to the heat content of the inhaled material in the oropharynx is, in the more distal airways, principally related to the particulate material contained within the smoke and the chemical composition of inhaled materials. Moist heat, which occurs with steam, has four thousand times the heat carrying capacity of dry smoke and is capable of causing more extensive thermal damage of the tracheobronchial tree.48

Presenting patient signs and symptoms are stridor, hypoxia and respiratory distress.49 The probability that a patient has suffered an inhalation injury is highly correlated with being burned in an enclosed space, having burns of the head and neck, and having elevated carbon monoxide levels. The extent and severity of the inhalation injury are directly related to the duration of exposure and the various toxins contained within the smoke. Injury due to heat is typically confined to the upper airway and supraglottic structures.50 Particulate material within smoke is the vehicle by which the toxic materials are carried to the distal airway. Particles of less than five microns in size can reach the terminal bronchi and the alveoli. Upper airway injuries involve the mucous membranes, nasopharynx, hypopharynx, epiglottis, glottis and larynx. The lining mucous membranes as well as the cartilage of the glottis are easily damaged and can cause acute airway
compromise. Direct thermal injury to the lower airway is uncommon as rapid dissipation of heat occurs as the gases move through the upper airway. Injury to the tracheobronchial structures and pulmonary parenchyma is related to the toxins in the inhaled smoke and the ensuing host inflammatory response. Activation of the inflammatory cascade results in the recruitment of neutrophils and macrophages which propagate the injury. Cellular damage is perpetuated by those cells which further the inflammatory response which in turn leads to progressive pulmonary dysfunction. Altered surfactant release causes obstruction and collapse of distal airway segments. As part of the response to injury there is a marked and near immediate change in bronchial artery blood flow which can increase by up to twenty-fold. These changes in bronchial blood flow are also associated with marked alterations in vascular permeability within the lung and are thought to play an important role in the pathophysiologic response to inhalation injuries. The net effect is that extensive destruction and inflammation reduce pulmonary compliance and impair gas exchange resulting in altered pulmonary blood flow patterns and ventilation perfusion mismatches.

Asphyxiants: Carbon monoxide and cyanide gases are present in smoke and when inhaled are rapidly absorbed and cause systemic toxicity as well as impaired oxygen utilization and delivery. Carbon monoxide is an odorless, non-irritating gas that rapidly diffuses into the blood stream and rapidly binds to the iron moiety of the hemoglobin molecule. Carbon monoxide has a 240 times greater affinity for hemoglobin than does oxygen thus it easily displaces oxygen. Carbon monoxide directly impairs the ability of hemoglobin to deliver oxygen to the tissues. Carbon monoxide also binds to enzymes within the mitochondria involved in intracellular oxygen utilization and cellular energetics. Signs and symptoms of carbon monoxide poisoning are typically mild to none at all when carbon monoxide-hemoglobin (carboxyhemoglobin) levels are 10% or less. When carboxyhemoglobin levels are between 10% and 30%, symptoms are present and often manifested by headache and dizziness. Severe poisoning is seen in patients with carboxyhemoglobin levels of greater than 50% which may be associated with syncope, seizures and coma.

The diagnosis of carbon monoxide poisoning is made in a patient with burns on the basis of circumstances of injury, physical findings and the measurement of blood carboxy-hemoglobin
level. It is important to note that pulse oximetry values do not differentiate between carboxyhemoglobin and oxyhemoglobin. Patients with significant carbon monoxide intoxication can have elevated oxygen saturations but will not have satisfactory blood oxygen contents. The primary treatment modality for carbon monoxide intoxication is provision of increased levels of inspired oxygen. The carbon monoxide “half-life” will decrease from six to eight hours with room air to 40-80 minutes with 100% of FIO₂. Administration of oxygen in a hyperbaric chamber can further decrease the “half-life” to 15-30 minutes. In a recently reported randomized trial Weaver and colleagues found that hyperbaric oxygen therapy significantly benefitted patients with acute carbon monoxide poisoning. The utility of this in patients suffering major burns in association with carbon monoxide poisoning is yet to be demonstrated.

It is a significant practical question whether a patient can safely undergo treatment with hyperbaric oxygen therapy when there are other life-saving treatments that are needed. An approach that has worked well is to maintain the patient on 100% FIO₂ until carboxyhemoglobin levels are less than 15% and then to maintain this level of increased oxygen for an additional six hours at which time weaning of the FIO₂ can be initiated and conducted in accordance with standard criteria.

Cyanide poisoning which can occur in combination with carbon monoxide intoxication further disrupts normal cellular utilization of oxygen by binding to the cytochrome oxidase resulting in cellular lactic acid production and greater cellular dysfunction. Blood concentrations of cyanide greater than 0.5 mg/l are toxic. Treatment of cyanide poisoning includes the administration of oxygen as well as decontaminating agents such as amyl and sodium nitrates. These compounds induce the formation of methemoglobin, which can act as a scavenger of cyanide. Sodium thiosulphate which can be administered intravenously enhances the enzymatic detoxification of cyanide to thiocyanate but acts slowly. Hydroxycobalamin which acts more rapidly and has few side effects is the antidote of choice.

Smoke may also contain a variety of toxic compounds that cause or initiate further damage to the airway (Table IV). The composition of each fire is different adding to the difficulty in caring for these patients. Such additional products in smoke include acrolein, hydrogen chloride, phosgene, ammonia, nitric oxide and sulfur dioxide all of which are capable of causing significant injury.
**Airway Management:** The most critical factor in the initial assessment of a burn patient is the patency of the airway and the ability of the patient to maintain and protect the airway. Standard criteria should be used to determine the need for mechanical stabilization of the airway also keeping in mind the systemic response to a major burn and the local response to an airway injury which may combine to cause progressive airway swelling and edema that will impair air flow. In an adult trachea of 14 mm, one mm. of edema will result in a 25% reduction in cross-sectional area. A similar degree of swelling in a six mm trachea of a child will result in greater than a 50% reduction in cross-sectional area. Circumferential torso burns will further impair the ability of the patient to respire. Allowing airway compromise to proceed to a critical state before intubating the patient and stabilizing the airway is not appropriate care. The safest approach when there is concern about the airway, particularly in a patient needing transport for definitive care, is to perform early intubation.

Part of the initial management of the patient with inhalation injury should include a thorough evaluation of the airway including bronchoscopy. The clinical findings of an inhalation injury on bronchoscopy include airway edema, inflammation, increased bronchial secretions, presence of carbonaceous material which can diffusely carpet the airway, mucosal ulcerations and even endoluminal obliteration due to sloughing mucosa, mucous plugging, and cast formation (Fig. 5). Signs of gastric aspiration may also be evident. Repeat bronchoscopy can be performed for removal of debris and casts as well as surveillance for infection.61

Direct airway treatment has been attempted but with variable responses. Desai and colleagues conducted an open label trial of the use of aerosolized heparin and n-Acetylcysteine in children with inhalation injuries. The treated patients had an improved outcome compared to an untreated cohort.61 As of yet, a randomized prospective study has not yet been performed to confirm the utility of this approach particularly in adults. One noticeable affect with the empiric use of aerosolized heparin is the rapid clearing of particulate material and carbonaceous deposits from the airway. Inasmuch as the development of pneumonia in patients with inhalation injuries negatively impacts outcome, it is disappointing that prophylactic antibiotics have not been effective.62 As a practical matter, it is best to culture these patients early to identify the
organisms that have colonized their airways as a consequence of their injury and urgent airway manipulation. That information can guide therapy should the patient develop the signs and symptoms of early onset pneumonia. Steroids are not recommended in patients suffering inhalation injuries.63

**Mechanical Ventilation:** The critical feature in the management of patients suffering inhalation injuries is to minimize further damage to the airway and lung parenchyma while providing adequate gas exchange.64 A critical feature in the management of patients with inhalation injuries is to control airway pressures and thereby limit ventilation induced barotrauma65,49 It is important to recognize that lung damage is not homogenous but patchy in distribution and requires that the level of positive end expiratory pressure (PEEP) used to maximize airway recruitment be limited to avoid ventilator associated lung injury.66,67 In states of severe lung injury mechanical ventilation can lead to increases in shear forces and changes in pulmonary blood flow. This in association with reductions in elasticity and alterations in lung compliance results in further lung injury and ventilation perfusion abnormalities.68,69

Using a standard volume mode of ventilation does not represent the best management of the damaged airway. Inverse ratio ventilation provides a strategy one can use in an attempt to counteract these changes and allow reductions in the level of PEEP and respiratory pressures to improve oxygenation.70 Unfortunately, a clear advantage of inverse ratio ventilation over standard approaches has not been consistently shown.71 An alternative strategy is high frequency ventilation in which rapid respiratory rates and small tidal volumes are used to achieve adequate oxygenation and ventilation while minimizing barotrauma.72 The three major types of high frequency ventilation are high frequency interrupted flow positive pressure ventilation, high frequency jet ventilation and high frequency oscillation. High frequency interrupted flow positive pressure ventilation delivers small tidal volumes (4 ml/kg) at flow rates of 250 liters per min with a frequency of 100 breaths per minute. In this mode, expiration is passive and thus there is an increased risk of air trapping and over distention. High frequency jet ventilation employs small tidal volumes and high respiratory rates with the volumes determined by jet velocity and duration of flow. High frequency oscillation maintains lung volumes by applying a constant airway pressure but does not allow for patient triggered inspiratory flow. With this mode
of ventilation, inspiration and expiration are active processes and air trapping is reduced. Patient oxygenation is maintained by increasing the mean airway pressure until an adequate oxygen level is achieved while ventilation is achieved by oscillating airway pressure through electromagnetically driven pistons that deliver cyclic tidal volumes and facilitate ventilation. There has been limited experience with the use of extra-corporeal membrane oxygenation in the management of inhalation injuries in selected centers.

An approach that has worked well in many patients with inhalation injuries has been to identify promptly the presence of airway compromise and ensure that the patient is intubated with a properly sized endotracheal tube. Currently, it is our preference in the pediatric population to use a cuffed tube that may need to be inflated to achieve maximum ventilatory efficiency. In those patients who are seen to have signs of inhalation injury on bronchoscopy, there is aggressive management of retained secretions with the use of bronchodilators and mucolytic agents along with aerosolized heparin. Meticulous control of airway pressure is practiced with the early performance of torso escharotomies and prompt treatment of an abdominal compartment syndrome particularly in the burned child. Mean airway pressures are maintained at less than 32-34 cm of water and ready use is made of chemical paralysis of the patient with a low threshold for conversion to pressure controlled ventilation with titration of tidal volumes to lessen further the risk of ventilator associated barotrauma. This may require the acceptance of smaller than usual tidal volumes and permissive hypercapnia, which is acceptable as long as arterial blood pH is above 7.26 and the patient is hemodynamically stable. These approaches along with a tightly controlled fluid resuscitation will in most circumstances avoid the need for alternative ventilator strategies in the care of these patients. Others, including one of the authors (BAP) prefer to use high frequency interrupted flow positive pressure ventilation (HFIFPP) prophylactically. In all patients with inhalation injury HFIFPP ventilation is initiated on admission to minimize airway obstruction, maintain lung volume and reduce the risk of pneumonia.

OTHER ORGAN SYSTEM SUPPORT

Pain Control: The pain experienced by patients suffering from acute thermal injuries is a complex integration of the objective neurologic input from the damaged tissue and the patient’s fear and anxiety resulting from the traumatic event. The patient’s pain is compounded further by
wound care and the therapy required to maintain functional status. If the patient begins to perceive there is no escape from this situation, his/her fear and anxiety will be amplified and may be magnified further by the appearance and expression of concern by family and friends. Furthermore, as the burn begins to heal, there may be increased wound sensitivity during dressing changes and therapy sessions. This can further distress patients, as they perceive that instead of the pain improving with recovery it seems to be worsening. Pain is a fifth vital sign and it should be monitored, its level documented and treatment be properly planned. Appropriate therapeutic options must be available to provide patients with pain control.

In patients who are hospitalized for on-going care or require surgery, it is best to initiate long acting oral narcotic agents for background pain control and administer shorter acting narcotics either orally or parenterally, along with anxiolytic agents, for procedure related pain. For an acutely injured patient undergoing a procedure in the ICU or ward, intravenous morphine remains the mainstay for analgesia along with oral compounds such as hydrocodone, which is preferable to oxycodone, which has a greater propensity for abuse. Clonidine can also be added to the pain regimen in patients who are having an initial poor response to narcotics and anxiolytic agents. In patients who are intubated and being maintained on mechanical ventilation, the continuous administration of intravenous morphine and diazepam or propofol and fentanyl are two commonly used regimens to achieve pain control and sedation and prevent unplanned extubation. In the post-operative care of patients having skin graft procedures the most painful wound is often not the burn wound but the donor site. Jellish et al have reported that the treatment of the skin graft harvest site with local anesthetic agents significantly improved the patient's pain score post-operatively. Patient-controlled analgesia is also a very effective strategy in patients who understand and can manipulate the delivery system. An important factor leading to effective pain control is to have established protocols that all members of the team understand and can use safely. There must be flexibility in the medication regimen and the patients must be fully informed that every attempt will be made to provide them the greatest comfort within the context of safe and compassionate care.

**Neurological Deficits:** Immediately following burn injury the patient with an altered mental status needs to undergo a careful evaluation for injuries occurring during and prior to the time of
the fire. Additionally, the patient's pre-injury neurological status needs to be determined for any prior impairment. In patients who are obtunded the primary concern is CNS injury due to hypoxia and carbon monoxide poisoning. The use of alcohol or drugs in the time leading up to the burn injury can confound the assessment of the burn-injured victim. Testing for alcohol and drugs aids in the evaluation of such patients. This is particularly important since the impact of alcohol appears to significantly modify the patient's chance for survival. The patient, particularly an elderly patient, should also be evaluated for a primary CNS event that might have precipitated their injury such as a seizure, stroke or intracranial hemorrhage. The possibly of an assault must always be foremost in one's thoughts particularly in children when the history of the injury does not match the findings. It is not uncommon to find that a burn is a signal finding for child abuse. This can also be the case in adults where the initial event was an assault and the burn is an attempt to disguise the physical attack.

Patients presenting with agitation must be rapidly evaluated for hypoxia and treated. The initial management of a patient who has a deteriorating mental status is a review of the medications and medication doses that have been administered to determine whether reversal agents should be given. In the treatment of children the doses should be age and weight appropriate and carefully titrated to the patient's need. Later in the course of a burn patient's care, changes in mental status require a detailed review of medications for pain and sedation, measurement of serum electrolyte values particularly sodium, and evaluation for a septic process and the onset of renal or hepatic failure. Patients with impaired mental status either early or late must always be assessed for the status of their airway, their ability to maintain a patent airway and the need for tracheal intubation.

**Gastrointestinal Responses and Complications**

Impaired gastrointestinal motility and focal gastric mucosal ischemia occur in virtually all patients with burns involving more than 25% of the total body surface with the severity of change proportional to the extent of the burn. The resulting ileus necessitates nasogastric intubation to prevent emesis and aspiration. If the mucosa is unprotected by instillation of antacid or treatment with an H₂ histamine receptor antagonist, ischemic erosions in the mucosa may progress to frank ulceration with associated bleeding or even perforation. Sufficient antacid (typically 30 ml but
60 ml may be needed) should be instilled each hour to maintain the pH of the gastric contents above 5. At the present a histamine H₂ receptor antagonist (e.g. 400 mg of cimetadine given intravenously every 4 hours) or proton pump inhibitors are more commonly used for stress ulcer prophylaxis. When gastrointestinal motility returns, enteral feeding should be initiated and the antacid therapy, e.g. a 1200-2400 mg daily dose of cimetadine, can be added to enteral feeds. A randomized study comparing antacid prophylaxis and nonacid buffering sucralfate prophylaxis showed no difference in recovery of gram-negative organisms from the upper gastrointestinal tract, and no difference in the occurrence of gram negative pneumonia, but lesser gastric mucosal protection and a higher incidence of gram positive pneumonia in the sucralfate-treated group.79

WOUND CARE

Initial Wound Care: Initial wound care is focused on preventing further injury. Immediately upon removal of the burn victim from the site of injury attention should be given to removal of burning clothing, disrupting contact with metal objects that may retain heat, and cooling of any molten materials adherent to the skin surface. Attempted cooling of burn wounds must be done with caution as local vasoconstriction can impair wound blood flow and extend the depth of the injury. The use of surface cooling of the burn is limited to patients with small burns typically not requiring hospitalization. Hypothermia can rapidly occur in children as well as adults particularly elderly patients if they are placed in cool or wet dressings. Patients being prepared for transport or admitted for definitive care should be placed in sterile or clean dry dressings and be kept warm. Prolonged exposure of the burn victim’s wounds leads to cooling and further impairs the patient’s response to their injury. Items of clothing or jewelry that may impair circulation should be removed prior to the onset of burn wound edema to prevent further compromise of the circulation. In cases of chemical injury removal of contaminated clothing with copious water lavage of liquid chemicals and removal by brushing of powdered materials at the scene can limit the extent of the resultant burn injury. The ability to perform these maneuvers at the scene must be balanced against the patient’s associated injuries that would mandate immediate transport for the care of life threatening injuries. No attempt should be made at chemical neutralization of the suspected chemical agent, as such treatment would result in an exothermic reaction and cause additional tissue damage. The care provider must exercise extreme caution when working with victims of chemical injury to prevent self-contamination and personal injury. In all circumstances those individuals caring for burn patients should wear personal protection devices
After admission to the hospital and as soon as resuscitative measures have been instituted the patient should be bathed and the burn wounds cleansed with a detergent disinfectant. Chlorhexidine gluconate is a cleansing agent with an excellent antimicrobial spectrum. During cleansing of the wound the patient must not be allowed to become hypothermic. The treatment area and the cleansing fluids should be warm and the procedure should be done expeditiously. Materials that are densely adherent to the wound such as wax, tar, plastic and metal should be gently removed or allowed to separate during the course of subsequent dressing changes. Sloughing skin, devitalized tissue, and ruptured blisters should be gently trimmed from the wound. No formal attempt is made to remove the burned tissue during these wound-dressing debridements. Patients may experience considerable pain and apprehension during these dressing changes and should receive adequate pain medication. In the patient on whom blisters are present whether to remove them or allow them to remain is a matter of opinion. Blisters that are intact particularly thick blisters on the palm of a hand maybe left intact.\textsuperscript{60} Intact blisters must be closely monitored for signs of infection or rupture at which time they should be debrided and the wound treated. If a blister can be kept intact wound healing should be complete in less than three weeks. The notion that intact blisters are an effective biologic dressing has been called into question and some authors recommend removing all blisters and treating the burn as an open wound.\textsuperscript{21, 81, 82} Careful wound cleansing should be done at each dressing change with serial debridement of devitalized tissue performed as necessary. The wound should be monitored for signs of infection and change in depth from the initial assessment. It is not uncommon for the initial wound depth to have been under-estimated in children and young women. Additionally the extent of body surface involvement should be recalculated to ensure that the initial burn size determination was accurate.

**Topical Antimicrobial Therapy:** The burn injury sets in progress a series of events leading to impaired local and systemic immunity. The damaged skin surface can serve as the portal for microbial invasion if it becomes progressively colonized. As microbial numbers increase within the wound to levels of 100,000 organisms per gram of tissue an invasive wound infection and ultimately systemic sepsis may occur. Topically applied antimicrobial agents, which penetrate the burn eschar, are capable of achieving sufficient levels to control microbial proliferation within
the wound. Systemic antibiotics do not achieve sufficiently high concentrations in the wound to achieve therapeutic levels, as the eschar is in large part avascular. These concepts form the basis for the use of topical antimicrobial agents in the prophylactic treatment of the burn wound and as a part of the management of burn wound infections. Topical agents per se do not heal the wound but prevent local burn wound infection from destroying viable tissue in wounds capable of spontaneous healing. In burns that will require excision and grafting control of the burn wound microbial environment prevents the development of systemic sepsis secondary to invasive burn wound sepsis. The utility of topical agents is most clearly apparent in improving patient outcome in burns of greater than 30% TBSA. The need for the use of topical anti-microbial compounds in the management of small burns has never been demonstrated and these injuries can be effectively managed with a petroleum-based dressing. That being said it has become common practice to apply topical antimicrobial agents to even small burn wounds despite the fact that there are no data to support this approach. Topical antimicrobial burn wound agents include cream, ointment, and liquid based products that require daily to twice daily dressing changes and re-application. Also available are materials impregnated with antimicrobial compounds that typically are changed following a several day period of application.

Silver sulfadiazine, the most widely used agent, is available as a 1% suspension in a water-soluble micronized cream base. The cream is easily applied, causes little or no pain on application, and can be used open or as a closed dressing. As a closed dressing the cream can be directly applied to the wound as a continuous layer then covered over with a dressing or be impregnated into the dressing, which is then applied to the wound. At each dressing change the cream should be totally removed and not allowed to form a caseous layer that will obscure the wound bed. When silver sulfadiazine is used in the management of superficial partial thickness burns the dressing becomes discolored, the wound exudate appears infected and the wound develops a yellow-gray pseudoeschar that is easily removed. The ability of silver sulfadiazine to penetrate an eschar and prevent wound infectious complication in burns >40-50% TBSA is thought to be poor. However, wound cleansing with chlorhexidine as a part of the wound management along with silver sulfadiazine has proven to be a very effective combination in patients with larger burns. The most common toxic side effect of silver sulfadiazine is a transient leukopenia which when it does occur in up to 15% of treated patients, resolves spontaneously.
without discontinuation of the drug. The proposed mechanisms for this response have ranged from leukocyte margination in the wound (not drug related) to a direct cytotoxic effect of the drug on the bone marrow granulocyte and macrophage progenitor cells. Silver sulfadiazine is active against a wide range of microbes including S. aureus, E. coli, Klebsiella species, many but not all Pseudomonas aeruginosa, Proteus species, and Candida albicans.

Mafenide acetate was one of the first effective topical agents introduced for the management of the burn wound. It was initially available as Sulfamylon® Burn Cream (an 11.1% suspension in a vanishing cream base). It is commonly used on exposed wounds treated by the open technique though it is possible to use it under a light dressing. Mafenide acetate is highly effective against Gram-positive and Gram-negative organisms, but provides little antifungal activity. Mafenide acetate readily diffuses into the eschar and is the agent of choice for significant burns of the ears because it is also capable of penetrating cartilage. Drawbacks with the use of mafenide acetate include pain on application to partial thickness burns, and limited activity against methicillin-resistant S. aureus. Mafenide acetate also inhibits carbonic anhydrase which increases urinary loss of bicarbonate which may cause hyperchloremic acidosis and accentuate postburn hyperventilation. Fortunately, this affect is time limited because the kidney typically escapes from such inhibition in 8 to 10 days. Mafenide acetate has more recently become available as a 5% aqueous solution and is an excellent agent to use on freshly grafted wounds and is not associated with the problems found with the cream formulation.

Silver nitrate as a 0.5% solution has been available since the 1960s and is effective against Gram-positive and Gram-negative organisms. The silver moiety is deposited on the burn wound and does not penetrate the eschar to any great extent. Silver nitrate soak solution leaches sodium, potassium, chloride, and calcium from the wound in association with transeschar water absorption which can result in mineral deficits, alkalosis and water loading. Those side effects can be minimized by giving sodium and other mineral supplements and modifying fluid therapy. These problems and the labor required to use silver nitrate effectively limit its routine use currently and most see silver sulfadiazine as a highly acceptable alternative.
Silver impregnated dressings consisting of a polyethylene mesh coated with a nanocrystalline film of pure silver ions bonded to a flexible rayon-polyester sheet have recently become available for clinical use (Fig.6). When the fabric base is in contact with wound fluids the silver is released continuously and serves as the antimicrobial agent deposited onto the wound. The treatment interval with such a composite may extend up to several days depending on the fabrication design with dressing changes needed only once or twice per week. This approach approximates the therapeutic utility of silver nitrate and has not been associated with the problems found with silver nitrate. However, one must know that the wound has not become compromised during the extended treatment periods. It is not advisable to use this approach in the care of perineal wounds particularly in small children with frequent bowel movements or wounds with excessive drainage or difficulty in maintaining contact between the dressing and the burn wound surface. The effectiveness of this membrane in treating extensive full thickness burns is unconfirmed and at present it is used to treat partial thickness burns.

In superficial partial thickness burns the use of bacitracin ointment represents a satisfactory alternative particularly in patients with a known sulfa allergy. It may be used open especially with superficial facial burns or as a component of a closed dressing. Other topical agents include antibiotic combinations such as triple antibiotic ointment (neomycin, bacitracin zinc, and polymyxin B) and polysporin (bacitracin zinc and polymyxin B). In the case of methicillin-resistant staphylococci mupirocin represents a useful agent. These agents are also capable of being used with open or closed dressing techniques.

The application of topical antimicrobial agents to the burns of patients who will be transferred to a burn center may preclude the use of biological membrane dressings that must adhere to the wound surface to be effective. Additionally, upon admission to a burn center any previously placed dressing must be removed to permit the burn team to make a precise assessment of the extent of the burn and the depth of injury. In addition to burn wound evaluation the burned child must be inspected for any signs of abuse, which includes a detailed examination of the entire skin surface. Unless there will be an extended period of time before patient transfer to a burn center, placing the patient in a dry dressing, particularly if it is one with a non-adherent lining, and keeping the patient warm is the preferred initial management.
The sterilely gloved hand is used to apply the burn cream of choice in a thickness sufficient to obscure the surface of the burn (Fig. 7). The wounds are then either left exposed or covered by a light occlusive dressing. Twelve hours later the topical agent is reapplied to the entirely of the burn wound. If a dressing is used over the topical agent, it is removed prior to reapplication of the topical agent following which a new dressing is applied. To optimize antimicrobial coverage and minimize side effects, one can alternate topical agents, i.e. apply Sulfamylon® burn cream after the morning cleansing and apply Silvadene® burn cream in the evening. If 0.5% silver nitrate soaks are used for topical wound care (as may be necessary in a patient allergic to sulfonamides) they should be changed 2 or 3 times a day and kept moist between changes by infusing additional soak solution every 2 hours. All environmental surfaces and equipment as well as the clothing and exposed skin of attending personnel must be protected from contact with the silver nitrate solution which will cause dark brown discoloration of virtually anything with which it comes in contact.

Each day the topical agent is totally removed in the course of the daily cleansing and the entirety of the burn wound is examined to detect any signs of infection. If signs of infection are identified, a biopsy of the eschar and the underlying viable tissue should be obtained from the area of the wound suspected of harboring infection. The biopsy sample is subjected to histologic examination and should also be cultured to determine predominant organisms. Histologic confirmation of invasive infection (the presence of bacteria or fungi in viable tissue underlying or adjacent to the burned tissue) necessitates a change to Sulfamylon burn cream topical therapy (mafenide acetate can diffuse into nonviable tissue to limit microbial proliferation), subeschar antibiotic infusion, physiologic fine tuning of the patient, and prompt surgical excision of the infected tissue.

**Burn Wound Excision and Grafting:** Excision of the burned tissue and grafting is required for wounds that are full thickness in depth and is now considered to be the optimum management of wounds with a mixed depth of injury. Wounds that are capable of spontaneous closure within two-three weeks post-injury can be managed expectantly provided the cosmetic and functional outcomes will be acceptable. Wounds that will require a longer period of time to close, in general, develop significant scarring, painful or unstable scars, intense pruritus, and in areas of function