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Acute resuscitation and transfer management of burned and electrically injured patients

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PREHOSPITAL CARE

Optimal care of the thermally injured patient begins before the patient reaches the hospital and is usually provided by local fire or emergency medical services. Local treatment preferences, burn care protocols, and designation of appropriate receiving hospitals should be established to expedite prehospital burn care (1). Prehospital care should consist of stopping the burning process, conducting a primary and secondary survey to assess life-threatening injuries, and preparing for transport.

Initial priority is to remove the victim from the fire environment to a safe area. Once rescue is secured, the patient should have all burned clothing removed, including jewelry or other constricting objects. Melted polyester-type clothing should be cut free and any remaining cloth adherent to the wound left for later debridement.

Chemical burns require special attention. Dry chemicals should be brushed off the patient. When all visible chemical is removed, copious irrigation with water should then be undertaken. All other chemical burns should be immediately irrigated with water. Home showers or a garden hose can be quickly used. Chemical burns occurring in the workplace should be irrigated and decontaminated onsite prior to transport of the patient (2). Irrigation onsite should continue until the pain or burning sensation abates (3). However, because some chemicals, especially alkaline agents, may continue to irritate for hours, irrigation should continue en route to the hospital when feasible. Neutralization of acid or alkali burns with a corresponding base or acid produces additional heat that may further increase the depth of burn and is contraindicated. Pain produced by an eye burn causes most patients to keep their eyelids closed, and manual retraction of the eyelids is usually necessary

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to accomplish irrigation. Care should be taken not to contaminate the unaffected eye with irrigation runoff.

Once rescue is secured, a primary survey is undertaken. Guidelines as dictated by Advanced Trauma Life Support (4) and Advanced Burn Life Support (5) emphasize a rapid examination of the patient for life-threatening injuries in alphabet sequence: Airway, Breathing, Circulation (assessed by the presence or absence of distal pulses and by capillary refill), Disability (assessment of neurological deficits and stabilization of cervical spine if injury is suspected), and Exposure (removal of all clothing and jewelry, then cover with sheet and blanket). A primary survey should take ~30 s to perform. Any life-threatening abnormalities are corrected and supplemental oxygen delivered.

The secondary survey is a more detailed examination to detect additional traumatic injuries. Disregard for the possibility of multiple trauma may seriously jeopardize patient care. Common examples of multiple trauma with burn injury include vehicle crashes resulting in fires, electrical injuries that include a fall from a utility pole, or house fires in which the occupants jump from windows to escape. During the secondary survey, a rapid history is taken to include the mechanism of injury, history of impaired mentation and/or confinement in a burning environment, complaints of other associated injuries, any preexisting medical conditions, current medications, allergies, and history of tetanus immunization. Such information may not be available later if mental alertness fades or endotracheal intubation is required. Concomitant with the history, a rapid "trauma" physical exam is performed with special attention to deformities and localized tenderness. Vital signs, airway patency, and adequacy of breathing and circulation are reassessed at frequent intervals. Blood pressure should be measured (1).

The patient is next prepared for transportation. Transport of a burn patient should not be delayed by multiple attempts at i.v. access. It is not necessary to initiate i.v. fluid therapy in the field if the burn patient can arrive at the receiving hospital ≤30 to 45 min and if no other injury exists (6,7). If i.v. fluids are required prior to hospital arrival, two large-bore cannulas are inserted into peripheral veins and then sutured in place. Cannulas traversing burned skin are acceptable if necessary (3,4). Lactated Ringer's solution is then infused. For adolescent and older victims, a rate of 500 ml/h is suggested as an initial rate. For children (5–15 years), 250 ml/h is appropriate prehospital. To minimize transport delays and iatrogenic injury, the advanced burn life support course recommends that i.v. cannulation not be attempted in children <5 years of age (8).

Burn wounds are covered with clean dry sheets. The patient is then wrapped in thermal or other blankets to help maintain body temperature. Blisters should not be broken or debrided in the field. Application of ice packs or cold wet dressings to the burn wound may provide pain relief, but may result in systemic hypothermia when applied over a large body surface or for a prolonged interval. Thus, this treatment, if used at all, should be limited to small areas of the body (<5% body surface area) and only long enough to control pain, usually ≤10 min (6).

EMERGENCY DEPARTMENT CARE

The initial assessment and management of thermally injured patients proceeds in the same orderly manner as

TABLE 1. *Symptoms of carbon monoxide exposure*

Carboxyhemoglobin level (%)	Symptoms
0–15	None
15–20	Headache, confusion
20–40	Disorientation, nausea, visual impairment, lethargy
40–60	Hallucination, combativeness, delirium, coma, cardiovascular collapse
60+	Death

advocated for other trauma patients. The first priority is maintenance of the airway. Indications for endotracheal intubation include respiratory insufficiency and are usually associated with severe smoke inhalation or upper airway edema. Patients most likely to require endotracheal intubation include:

(a) Patients sustaining thermal injury while in a closed space who have evidence of smoke inhalation
(b) Patients with steam burns of the face and upper body
(c) Patients with severe facial and neck burns

Nasotracheal intubation is preferred for its more secured position. However, endotracheal tube size, usually a size ≥8 in adult males, should not be compromised as pulmonary toilet and possible bronchoscopy may be required.

Smoke inhalation is suspected in patients with facial burns, singed facial hair, and nasal vibrissae; intraoral burns or carbonaceous deposits in the oropharynx; or the history of being burned in a closed space. The diagnosis of inhalation injury is strongly suggested by the presence of carbonaceous sputum (not carbon-laden oral secretions). Fiberoptic bronchoscopic examination of the upper airway and tracheobronchial tree identifying presence of carbonaceous material, edema, erythema, or mucosal ulcerations confirms the diagnosis (9). Direct laryngoscopy may also be used to visualize the upper airway.

All patients with burns >20% of the body surface area or in whom smoke inhalation injury is suspected should have humidified oxygen administered during the resuscitative phase. If available, continuous t.c. pulse oximetric determination of oxygen saturation or serial arterial blood gas determinations should be utilized to assess the adequacy of oxygenation. Any discrepancy in oxygenation may signal carbon monoxide poisoning (10). Carbon monoxide binds strongly to hemoglobin, generating carboxyhemoglobin. Less hemoglobin is then available for oxygen binding, thereby reducing the oxygen-carrying capacity of the blood and impairing tissue oxygenation. In addition, carbon monoxide binds to myoglobin and to the terminal cytochrome oxidase, further impairing tissue oxygen utilization. Symptoms of carbon monoxide intoxication are related to the deficits in tissue oxygenation and correlate to the carboxyhemoglobin level (Table 1). The presence of carboxyhemoglobin or the chemical alteration of the cytochrome system by carbon monoxide does not effect the amount of dissolved oxygen in the blood; thus, the dissolved oxygen content of plasma as measured by the arterial oxygen tension (Po_2) will remain normal. However, the saturation of hemoglobin by oxygen may be markedly reduced as measured by the pulse oximeter. This discrepancy in Po_2 versus O_2 saturation in addition to measured levels of carboxyhemoglobin confirm the diagnosis. Carbon monoxide poisoning is treated by the administration of 100% oxygen by nonrebreathing mask or endotracheal tube to accelerate the dissociation

of carboxyhemoglobin. When breathing room air, the half-life of carboxyhemoglobin is ~300 min; however, if 100% high flow oxygen is administered, the half-life is reduced to ~100 min. One hundred percent oxygen should be administered until the measured level of circulating carboxyhemoglobin is <7%.

Hyperbaric oxygen therapy (2 to 3 atm) may further accelerate carbon monoxide displacement from hemoglobin. This therapy has been advocated for comatose patients with carbon monoxide poisoning. However, in the absence of any well-documented improvements in neurologic outcome as well as the difficulty in patient assessment and management imposed by the physical constraints of the hyperbaric chamber, the overall benefit of hyperbaric oxygen treatment is questionable.

Cyanide poisoning may also complicate emergency resuscitation. Cyanide may be produced by the combustion of common household materials such as nylon, wool, silk, polyurethane, or melamine (11). Consequently, trace levels of cyanide are demonstrable in smoke from house fires (12) and in the blood of those exposed to such smoke (13). On this basis, administration of cyanide antidotes to smoke inhalation victims has been proposed (11,13). This is almost always unnecessary, as prompt detoxification of cyanide occurs with adequate hepatic circulation and function (14). Rarely, a persistent metabolic acidosis unresponsive to fluid or oxygen administration will suggest the diagnosis of cyanide poisoning. Elevated blood cyanide levels in the absence of significant carboxyhemoglobinemia are extremely uncommon (14,15) and should prompt a reassessment of the presumed diagnosis. Metabolic acidosis should not be ascribed to cyanide poisoning until more common causes such as inadequate resuscitation or missed traumatic injuries have been ruled out. Blood cyanide levels can confirm the diagnosis, and treatment should then be initiated. Sodium thiosulfate promotes the conversion of cyanide to thiocyanate and is the initial treatment of choice. A half-dose may be repeated once if necessary. If blood cyanide levels are >3 mg/L, a chelating agent such as hydroxycobalamin may be added (16). Hydroxycobalamin is both safe and effective when given i.v. at a dose of 100 mg/kg. Unfortunately, this drug is available only as a 1 mg/ml concentration in the United States, requiring the administration of ~10 L of hydroxycobalamin to treat cyanide poisoning (14). Hydroxycobalamin is available in a 4-g dose in Europe. Methemoglobin-forming agents such as amyl nitrite, sodium nitrite, or dimethylaminophenol are also cyanide antidotes.

Once airway patency and ventilation are assured, fluid resuscitation takes priority. For severe burns (i.e., ≥20% total body surface area [TBSA] burn), two large-bore i.v. cannulas are preferred. Preference for placement include (a) a peripheral vein underlying unburned skin; (b) a peripheral vein underlying burned skin; (c) saphenous vein; (d) femoral vein; and lastly, (e) a central neck or subclavian vein. Along with the i.v. cannulation, a urethral catheter should be inserted, and the urine volume should be measured and recorded frequently. Since gastric paresis is common in patients with thermal injury involving ≥20% TBSA burn, a nasogastric tube should be inserted to minimize gastric distention, emesis, and aspiration. Furthermore, the ambient temperature of the room should be increased if possible, and the patient should be covered with blankets to prevent hypothermia. Electrocardiographic monitoring should be

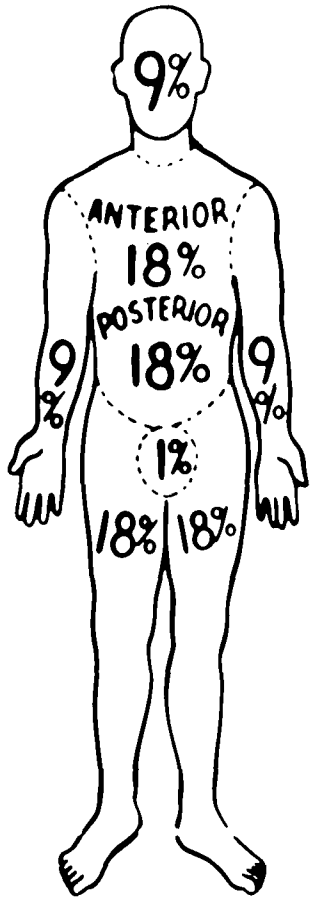
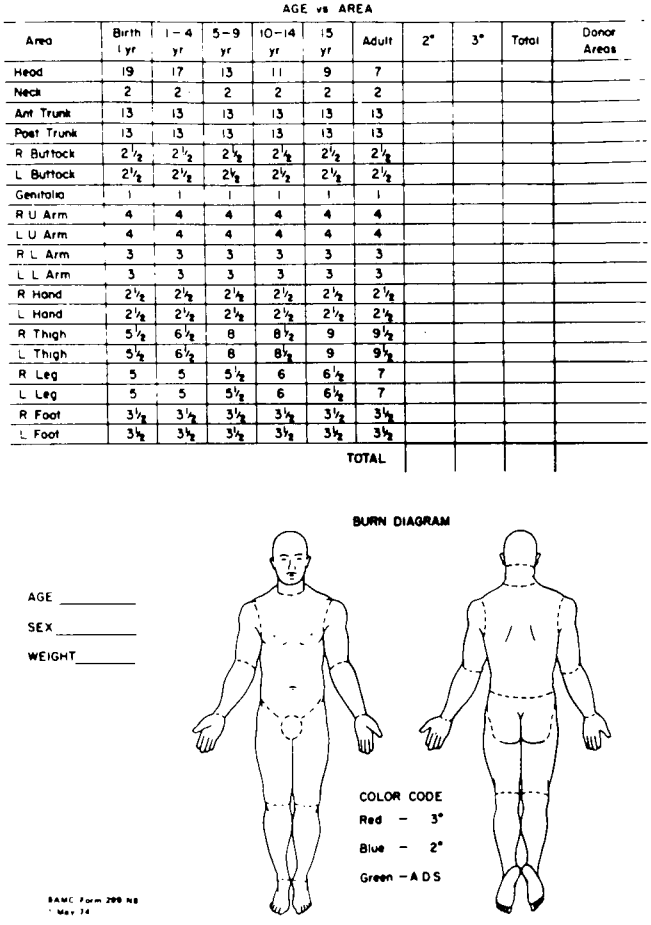


FIG. 1. Rule of nines, showing anatomical distribution of body surface area in the adult.

initiated in the emergency department in patients with high-voltage electric injury, burns >20% TBSA, suspected smoke inhalation injury, or preexisting cardiopulmonary disease.

Intravenous fluid resuscitation is initiated by infusing a physiologic salt solution (i.e., lactated Ringer's) with the initial rate tailored by the severity of injury. All resuscitation formulas in current use are based on the percent of the TBSA burned and patient weight (discussed herein). The TBSA burned can be quickly estimated using the "Rule of Nines," which assigns 9 or 18% of the TBSA to specific anatomic regions (Fig. 1). The surface area of small or irregularly shaped burns can also be easily estimated by equating the size of the patient's palm with 1% of his/her body surface area. Infants and children have a markedly different body surface area distribution due to their relatively larger head and smaller legs compared to adults (i.e., ≥10 years). The following calculation may be helpful in estimating the TBSA burned for children <10 years old: The surface area of the head and neck equals 18 minus the patient's age in years, and the body surface area of each lower extremity equals 13.5 plus 1/2 of the patient's age in years. Burn diagrams such as the Lund-Browder Burn Diagram (Fig. 2) (Lund CC, Browder MC. The estimation of burns. *SGO* 1944; 79:352) have been developed to more precisely determine the TBSA burned. The physical findings presented in Table 2 allow an initial determination of the depth of burn injury. Only the percentage of skin surface area involved in second and third degree burns is used to calculate fluid resuscitation requirements.

Typically, burns >20% of the TBSA require i.v. fluid resuscitation because ileus precludes sufficient oral fluid intake. For patients with small burns, liberal access to fruit juices, milk, or other electrolyte-containing fluids should be provided. If oral liquids are tolerated, then i.v. fluids may not be required. Excessive intake of water may result in hyponatremia and should be avoided. For patients requiring i.v. resuscitation, many formulas have been proposed (Table 3). A common theme for these formulas is that the volume of fluid required is dependent on the patient's size and extent of burn. However, any resuscitation formula serves only to guide the initiation of fluid therapy, and the actual amount of resuscitation fluid delivered is tailored to



AGE vs AREA

Area	Birth 1 yr	1–4 yr	5–9 yr	10–14 yr	15 yr	Adult	2°	3°	Total	Donor Areas
Head	19	17	13	11	9	7				
Neck	2	2	2	2	2	2				
Ant Trunk	13	13	13	13	13	13				
Post Trunk	13	13	13	13	13	13				
R Buttock	2½	2½	2½	2½	2½	2½				
L Buttock	2½	2½	2½	2½	2½	2½				
Genitalia	1	1	1	1	1	1				
R U Arm	4	4	4	4	4	4				
L U Arm	4	4	4	4	4	4				
R L Arm	3	3	3	3	3	3				
L L Arm	3	3	3	3	3	3				
R Hand	2½	2½	2½	2½	2½	2½				
L Hand	2½	2½	2½	2½	2½	2½				
R Thigh	5½	6½	8	8½	9	9½				
L Thigh	5½	6½	8	8½	9	9½				
R Leg	5	5	5½	6	6½	7				
L Leg	5	5	5½	6	6½	7				
R Foot	3½	3½	3½	3½	3½	3½				
L Foot	3½	3½	3½	3½	3½	3½				
						TOTAL				

FIG. 2. The use of a burn diagram permits more exact estimation of the extent of burn. Note that the surface areas of the head and lower extremities change significantly with age until age is >15 years.

each patient's physiologic response, usually best indexed by urine output. Frequent reassessment (at least hourly) and adjustment of the infusion rate is necessary to preserve adequate tissue perfusion and oxygen delivery. Failure to reevaluate the patient's response to resuscitation on a frequently scheduled basis may lead to either over- or under-resuscitation. Inadequate fluid administration may result in organ dysfunction or failure, whereas overzealous administration of i.v. fluids may result in pulmonary, cerebral, and/or excessive burn wound edema. Burn formulas provide only an estimate of the fluid resuscitation requirements, and blind adherence to any formula is doomed.

The composition of the resuscitation fluids also differs between formulas. In a clinical study evaluating resuscitation fluid composition, the total volume of fluid infused during the first 24 h postburn was less in the patients who received colloid-containing fluid as compared to those receiving only a crystalloid (17). However, no clinical benefit with colloid resuscitation was evident. Extravascular lung water in the crystalloid resuscitated patients was shown to remain essentially unchanged during the first postburn week. However, a pro-

TABLE 2. *Physical characteristics of burns*

	Partial thickness		Full thickness
	First degree	Second degree	Third degree
Mechanism	Exposure to sunlight, very brief exposure to hot liquid, flash, flame, or chemical agent	Limited exposure to hot liquid, flash, flame, or chemical agent	Prolonged exposure to flame, hot object, or chemical agent Contact with high-voltage electricity
Appearance	Red	Pink of mottled red	Pearly white, charred, translucent, or parchment-like Thrombosed vessels may be visible
Surface	Dry or very small blisters	Bullae or moist, weeping surface	Dry and inelastic
Sensation	Painful	Painful	Insensate surface

gressive increase in extravascular lung water was observed in those patients who received colloid. Thus, colloid-containing solutions should be used sparingly, especially in the early postburn resuscitation period. As capillary permeability normalizes and fluid requirements decrease (12–16 h postburn), the use of colloid may replete intravascular volume more efficiently without increasing tissue edema.

Resuscitation with hypertonic saline solutions has also been proposed as a means of reducing the required fluid volume (18,19). Although the volume of fluid administered when using hypertonic solutions is clearly less, the proposed benefits for fewer escharotomies and limited ileus have not been universally realized. Furthermore, hypernatremia (≥160 mEq/L) may be associated with significant cellular dehydration (20). Recently, hypertonic saline resuscitation has been associated with an increased incidence of acute tubular necrosis in animal studies of hypovolemic shock (21). Occasionally, some "volume sensitive" patients with reduced cardiac reserve may theoretically benefit from resuscitation protocols that

TABLE 3. *Commonly used resuscitation formulas (first 24 h)*[a]

Formula	Electrolyte-containing solution	Colloid-containing solution	5% Glucose in water
Evans	Normal saline, 1.0 ml/kg/% burn	1.0 ml/kg/% burn[b]	2000 ml
Parkland	Lactated Ringer's - 4 ml/kg/% burn	—	—
Hypertonic sodium solution	Volume of fluid containing 250 mEq of sodium/L to maintain hourly urinary output of 30 ml	—	—
Modified Brooke	Lactated Ringer's - 2 ml/kg/% burn	—	—
Consensus[c]	Lactated Ringer's - 2–4 ml/kg/% burn	—	—

[a]Formulas recommend that one-half of the calculated requirement be administered over the first 8 h following injury. The remaining half of the first 24-h resuscitation volume is delivered over the subsequent 16 h.

[b]Administered as blood, plasma, or a plasma expander.

[c]American Burn Association Advanced Burn Life Support Course.

minimize the fluid volume. However, invasive hemodynamic monitoring in such high-risk patients provides for the optimal fluid resuscitation regardless of fluid composition.

The authors recommend employing the modified Brooke formula for fluid resuscitation. Lactated Ringer's solution is preferred because of the more physiologic concentration of chloride ions. This solution is isotonic and closely matches the composition of extracellular fluid. The inclusion of glucose in the resuscitation fluid should be avoided because the large volumes in conjunction with stress-induced insulin resistance may result in flagrant hyperglycemia. Because glycosuria may misdirect subsequent fluid requirements, the presence of glucose in the urine should be checked frequently. As an exception, infants suffering large burns have a propensity to develop hypoglycemia. Serum glucose monitoring is important in this setting, and modest amounts of glucose should be included in their resuscitation fluid.

Once resuscitative measures are initiated, a complete history and physical examination should be performed with special attention to the possibility of other injuries. A history should emphasize the circumstances of the injury; the presence of preexisting disease, allergies, and medications; and the use of alcohol or illicit drugs prior to injury. Initial baseline laboratory data include analysis of arterial blood gases and pH, serum electrolytes, blood urea nitrogen, creatinine, glucose, and complete blood count. Tetanus prophylaxis for burn patients is identical to Advanced Trauma Life Support guidelines for other injuries.

In addition to the severity of burn injury, the presence of associated mechanical trauma may increase the resuscitation requirements beyond those predicted by standard formulas. Clearly, soft tissue trauma and bleeding from any injury increases the fluid required to establish adequate urine output. The presence of thermal injury, even if extensive, should not delay or alter the evaluation and subsequent therapy, including surgical intervention, of mechanical trauma.

Only after the previous measures have been initiated should attention turn to wound care. With burns of limited extent and for whom hospitalization is unnecessary (usually <5% partial-thickness burns), initial wound treatment can be performed in the emergency department. These wounds are cleansed with a mild surgical soap, and loose skin and bullae are debrided. After initial debridement, these burns may be covered with a petrolatum or topical antibiotic-impregnated fine mesh gauze covered by a bulky dressing. The wound should be cleaned frequently (usually twice daily) and wound care continued until complete healing. In patients with more extensive burns (>5%), any gross contamination of the wound should be removed and the wounds covered with a clean, dry sheet and blanket to await inspection by the surgeon. Debridement and the initiation of topical antimicrobial therapy can safely wait until referral to the definitive treatment facility.

Special concerns in electric injury

Tissue damage from electric injury (22) results from heat generated by the passage of electric current through the resistance of the body. In addition, these patients may have direct thermal injury caused by flash burns or by the ignition of clothing. The severity of injury is determined by the duration of contact, voltage and type of current (direct or alternating), and the path of current flow through the body.

The passage of electric current through the body may induce cardiac dysrhythmia. High-voltage injury (>1000 V) and lightning are often associated with asystolic cardiopulmonary arrest and require immediate cardiopulmonary resuscitation. Alternating current injuries more typically induce ventricular fibrillation. Although most cardiac arrests or dysrhythmias occur immediately after the injury, delayed dysrhythmias may occur. Therefore, patients should have continuous electrocardiographic monitoring for ≥12 h following a severe electric shock to allow prompt treatment should any dysrhythmia occur. Falls or tetanic skeletal muscle contractions may be associated with electric injuries. During the initial care of these patients, the spine should be immobilized until careful physical examination and/or appropriate cervical, thoracic, and/or lumbar radiographs exclude the presence of spinal fractures.

Extensive soft tissue damage from electric injury may occur. In addition to the obvious injury at the cutaneous contact sites, underlying tissues, especially muscle and occasionally organs along the path of the current, may be involved. This "iceberg" effect often results in resuscitation fluid requirements far exceeding those calculated based on the extent of cutaneous injury. With extensive muscle necrosis, hemochromogens are systemically liberated. The appearance of these pigments in the urine with subsequent precipitation on the renal tubules may cause acute renal failure. To minimize this complication, additional i.v. fluids should be administered at a rate sufficient to maintain an hourly urine output of 1 ml/kg body wt. If aggressive fluid resuscitation fails to sufficiently augment adequate urine output, an osmotic diuretic such as Mannitol may be helpful in increasing urine output and promoting clearance of the hemochromogen. With diuretic use, invasive hemodynamic monitoring of central venous or pulmonary artery pressures should be strongly considered to help guide further fluid management. The urine should be alkalized with i.v. sodium bicarbonate to reduce glomerular precipitation of the hemochromogen pigment in patients in whom pigment clearing is delayed.

Underlying damaged muscle may become edematous, resulting in increased compartmental pressures. Evidence of myoglobinuria, rigid musculature, or elevated measured tissue pressures (i.e., >35 mm Hg) indicate the need for fasciotomy. Muscle should be inspected, especially those deep and adjacent to bone. Muscle that is obviously necrotic should be debrided. Reinspection is frequently required as additional necrotic muscle becomes evident.

In-hospital monitoring and continued resuscitation

Because the objective of fluid resuscitation following burn injury is maintenance of tissue and organ perfusion and function, the adequacy of resuscitation can be monitored by several clinical indices of organ function. Adequate urine output indicating appropriate renal perfusion is the most convenient and reliable parameter for monitoring fluid resuscitation. A urinary output of 30 to 50 ml/h in adults or 1 ml urine/kg body wt/h in children indicates adequate resuscitation, in the absence of osmotically driven diuresis. Fluid infusion rates should be adjusted if the hourly urine output is below or above this desired goal. For patients requiring excessive fluids to maintain adequate urine output (i.e., ≥6 ml/kg body wt/% TBSA burn) or in whom prior cardiac or renal dysfunction

is evident, the use of a flow-directed pulmonary artery catheter should be strongly considered to help guide fluid management. Very rarely, patients manifest a markedly elevated systemic vascular resistance, a diminished cardiac output, and a pulmonary artery occlusion pressure that suggests adequate fluid resuscitation. Reduction of the systemic vascular resistance by administration of a short-acting afterload reducing agent, such as hydralazine or nitroprusside, may be beneficial (23,24). This therapy should be administered cautiously and only to patients who have received adequate fluid loading because the resulting vasodilation will precipitate hypotension in hypovolemic patients. Also in the setting of continued low cardiac output and elevated vascular resistance despite massive fluid resuscitation, dobutamine may be helpful for its inotropic action and effect as an afterload reducing agent.

Continuous monitoring of arterial blood pressure utilizing indwelling arterial cannulas is not routinely required in uncomplicated burn resuscitations. In patients with inhalation injury, or in those who do not respond as predicted to fluid resuscitation, distal extremity or femoral artery cannulation may be advisable.

Pulmonary dysfunction is a major morbidity in thermally injured patients, and adequacy of respiratory function must be continually reassessed. Tachypnea may indicate hypoxemia, restriction of chest wall motion due to constricting circumferential burns, or a compensatory response to metabolic acidosis. The evaluation of tachypnea includes chest auscultation, chest roentgenogram, and arterial blood gas analysis. Pulse oximetry can also be helpful. Decreases in the intensity of the pulsed signal may reflect inadequate distal perfusion due to constricting circumferential burn wounds or inadequate distal perfusion from underresuscitation. Low oxygen saturation, measured by pulse oximetry, may indicate inadequate alveolar oxygen diffusion due to severe smoke inhalation injury or pulmonary edema. Desaturation may also indicate carbon monoxide or cyanide poisoning.

Upper airway edema is not usually present immediately following burn injury, but develops as fluid resuscitation continues. If a patient with moderate-to-severe burns has evidence of upper airway inhalation injury, then the airway should be intubated prior to the onset of symptoms, unless the patient can be attended at all times by a person skilled in endotracheal intubation. In burn patients requiring endotracheal intubation and mechanical ventilation, end tidal carbon dioxide monitoring is useful to detect trends in the ventilation. This method of monitoring is particularly useful in pressure limited modes of mechanical ventilation that do not compensate for the progressive changes in pulmonary compliance commonly observed during resuscitation.

During resuscitation, edema forms beneath the inelastic eschar of full-thickness burns. If such burns occur circumferentially around an extremity, distal circulation may be compromised. Limbs can be salvaged with prompt escharotomy. A Doppler flow meter to detect pulsatile blood flow in the palmar arch and digital vessels in the upper extremities and pedal vessels in the lower extremities provides a reliable method for determining adequacy of distal perfusion. Absence or progressive diminution of pulsatile flow on sequential examination is an indication for escharotomy. Other clinical indicators of impaired extremity perfusion including distal cy-

anosis, impaired capillary refilling, deep tissue pain, and neurologic deficits. Fascial compartment pressures often exceed 30 mm Hg following circumferential extremity burns, and determining the need for escharotomy based on compartment pressures has been proposed (25).

Escharotomies can be performed without the use of general or local anesthesia since the burn eschar is insensate. The escharotomy incision should extend along the entire length of the full-thickness burn even across involved joints to ensure adequate release of vascular and neural compression (Fig. 3). Escharotomy incisions should only penetrate the eschar. When the incision is of sufficient depth, the underlying edematous tissue will suddenly expand. When performed at this level, blood loss is minimal since bleeding can be readily controlled by application of pressure or electrocoagulation. Fasciotomy is rarely required to restore blood flow in a thermally injured limb; however, in patients with extremely deep burns involving fascia and muscle or patients with associated traumatic or electric injuries, fasciotomies may be required to restore adequate limb circulation.

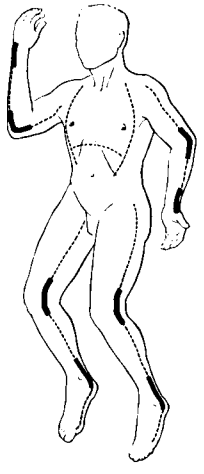


FIG. 3. The dashed lines show the preferred sites for escharotomy incisions. The solid segments of the lines emphasize the importance of extending the incisions across joints with full-thickness burns.

Circumferential or nearly circumferential truncal burns may result in restriction of chest wall movement as edema forms beneath the unyielding eschar. Patients may become restless, agitated, and tachypneic despite having an adequate airway. In mechanically ventilated patients, this airway restriction may manifest as a progressive increase in peak inspiratory pressure, a decrease in tidal volumes in pressure-controlled ventilation, and an increase in the exhaled end tidal carbon dioxide fraction. Escharotomy incisions along the anterior axillary lines and across the lower chest provide quick effective restoration of pulmonary compliance.

An alternative to the escharotomy incision employs the application of a collagenous enzyme (Travase) to the eschar (26). The lytic action of this enzyme produces a "chemical debridement" that softens the eschar and allows expansion of the underlying edematous tissues. To be effective, enzymatic debridement must be initiated early in the course of burn resuscitation. Application to the entirety of the wound or linear application along the midlateral and midmedial lines of the involved extremity have been successfully employed. Potential disadvantages include pain on application to second degree burns, inability to simultaneously apply topical antimicrobial agents, increased incidence of bacteremia or wound infection, bleeding, and potential injury to normal structures if application is prolonged. The use of enzymatic debridement should be reserved for burn centers familiar with this technique.

Aggressive nutritional support is essential in the management of patients

with extensive thermal injury. Early administration of nutrition by the enteral route is preferred to preserve enterohepatic delivery of substrates and maintain gut mucosal function and integrity. Animal studies have documented beneficial effects on immune function and substrate metabolism with early initiation of feedings (27). In theory, early gut nutrients inhibit bacterial translocation and minimize systemic inflammatory activation. However, the clinician must balance these theoretical benefits with the potential complication of aspiration in a patient with ileus. Placement of a transpyloric or jejunal feeding tube with simultaneous gastric decompression facilitate optimal management even in the early postinjury period.

After the first 24 h following burn injury, the movement of fluid into the interstitial compartment is greatly decreased. At this time, isotonic crystalloid fluids should be changed to 5% glucose solutions and the fluid volume reduced. The infusion rate is adjusted to maintain adequate urine output (0.5 ml/kg/h). Ideally, the volume of enteral nutrition can be quickly advanced and the i.v. fluids decreased accordingly.

Transfer and transportation

Patient transportation generally takes place in two phases: from fire scene to the closest hospital and from hospital to a specialized care facility (28). Stabilization at the closest hospital is not necessary if transport time from fire scene to the burn center will be <30 min (29). This time limit can be extended to ≤2 h if the patient is in the care of experienced paramedics, i.v. access and airway are secure, and communication to the receiving facility is established (29). In each emergency medical care system, designation of the appropriate receiving hospital and criteria for direct transportation to the burn center should be agreed on in advance. The American Burn Association has also established criteria for optimum treatment of burn patients, including criteria for transfer to a burn center. These criteria are listed in Table 4.

Burn patients may be transported by ground ambulance, helicopter, or fixed-wing aircraft. In addition to the condition of the patient, the mode of transportation (ambulance vs. helicopter) is dependent on such factors as distance, terrain, and prevailing weather. Concerns regarding the safety and cost of medivac helicopters in the transport of burn patients have been raised (30–32). The appropriate use of the helicopter is to "bring a higher level of medical expertise to the site of injury" and to insure that the patient is brought expeditiously to the most appropriate hospital (33). For burn patients, the use of a helicopter offers little advantage over the use of a ground ambulance if transport distance is <30 miles (29). Helicopter transfers may be efficacious if the distance is between 30 and 150 miles or if transport time is >30 min (34). Bear

TABLE 4. *American Burn Association burn center referral guidelines*

Any burn >10% TBSA in patients <10 and >50 years old
Burns involving >20% TBSA of any age
Full-thickness burns involving >5% TBSA
Significant burns of hands, face, feet, genitalia, perineum, or major joints
Significant electric injury
Significant chemical injury
Significant inhalation injury, concomitant mechanical trauma, significant preexisting medical disorders
Patients with special psychosocial or rehabilitative care needs

TBSA, total body surface area.

TABLE 5. *Physiologic effects of altitude*

Altitude (ft)	Barometric pressure (mm Hg)	P_AO_2 (alveolar) (mm Hg)	Po_2 (arterial) (mm Hg)	% Saturation
0 (sea level)	760	160	103	98
8,000	565	118	69	93
25,000	282	59	30	58

in mind that helicopter transport >100 miles is 400% more expensive than fixed-wing transport for a similar distance (35). Beyond 150 miles, patients are best transported by fixed-wing aircraft.

Aeromedical transportation poses several unique problems. Lack of space and proper lighting in most aircraft limits the ability to perform invasive procedures. For this reason, any anticipated escharotomy, airway, or vascular access procedure should be performed prior to flight (36). Aircraft also introduce the difficulties of noise, vibration, temperature variation, and changes in barometric pressure. The noise level of most aircraft render auscultation with a stethoscope difficult if not impossible (36). Also, mechanical ventilator alarms and leaks around the cuff of endotracheal tubes cannot be readily heard. Cardiac dysrhythmia should be controlled prior to flight because noise and vibration make in-flight cardiac monitoring difficult (28). Vibration may introduce motion artifact into electrocardiogram monitoring or cause internal cardiac pacemakers to malfunction (37). Ambient temperature within many aircrafts fluctuates depending on altitude and outside temperature (38). Burn patients are sensitive to decreases in ambient temperature, and frequent monitoring of patient core temperature (rectal probe or ear thermometer) is essential during flight.

Boyle's law states that if temperature is held constant, the volume of a gas is inverse to its pressure. Although oxygen content in room air remains at 21% regardless of altitude, oxygen availability decreases with increased altitude because the oxygen molecules are spread further apart. In clinical terms, with increased altitude elevation, there is a progressive decrement in the percentage of arterial blood oxygen saturation (Table 5). Normal individuals compensate for these changes with an increase in minute ventilation (39). An injured patient may lack the compensatory reserve and thus requires supplemental oxygen, cabin pressurization, or, occasionally, a planned descent to <10,000 feet altitude. Most commercial aircraft pressurize the cabin to an altitude of ~5,000 to 8,000 ft above sea level, resulting in an alveolar oxygen tension of 69 torr in a normal individual breathing room air (28,40).

A second consequence of Boyle's law is the tendency of an inflatable device or hollow viscus to expand with increasing altitude. For example, an endotracheal tube cuff with a pressure of 20 torr at sea level will have a pressure of 70 torr at 1,000 ft and 300 torr at 35,000 ft (40). Thus, endotracheal tube cuff pressure must be checked and corrected very frequently during flight. Likewise, air splints may present a problem if air is not removed as the aircraft ascends. A nonfunctional nasogastric tube can result in painful and potentially dangerous gastric distension as altitude increases.

Several medical conditions contrain-

dicate aeromedical transfer and are presented in Table 6. A patient must have sufficient respiratory reserve to tolerate the relative hypoxia of altitude or be on a level of ventilatory support that can be augmented to meet these increased demands. When feasible, patients who have undergone thoracic or abdominal surgical procedures should have aeromedical transportation delayed for 24 to 48 h to ensure adequate postoperative stabilization and to avoid altitude difficulties with air retained in body cavities (39). Patients with intracranial or intraocular air are at a tremendous risk of altitude-related complications.

For transportation from a referring hospital to a specialized care facility, early physician-to-physician contact with the burn center is preferable (Fig. 4). A burn center is defined as a hospital-based facility having the capability of caring for the thermally injured and staffed by personnel with expertise in general and plastic surgery, surgical critical care, burn nursing, occupational and physical therapy, nutrition, and burn research. At present, 138 hospitals in 45 states and the District of Columbia have self-designated expertise in burn care. All efforts should be directed at stabilization and prompt transfer to one of those centers.

TABLE 6. *Contraindications to aeromedical transportation*

Active hemorrhage, GI tract or otherwise
Temperature >39.5°C not controlled with antipyretics
Marginal respiratory reserve[a]
Uncontrolled cardiac dysrhythmia

[a]Institute of Surgical Research (ISR) criteria = >25 L minute ventilation.

PATHOPHYSIOLOGY OF BURN SHOCK

Organ system response to injury

Burn injury initiates a physiologic response in every organ system with the extent of response proportional to the size of the burn. Direct cellular injury by thermal energy is manifest by coagulation necrosis with the magnitude of tissue destruction determined by the temperature to which the tissue is exposed and the duration of contact. This tissue destruction results in a profound egress of plasma from the intravascular space into the tissues adjacent to the burn. The net loss of intravascular fluid volume is proportional to the severity of injury; thus, with extensive burns, hypovolemic shock may ensue (41).

All acute organ system responses to thermal injury are largely related to the diminished circulating blood volume either in attempt to compensate for hypovolemia or in dysfunction due to deficiency in end-organ perfusion. The immediate cardiovascular response to thermal injury is a depression in cardiac output with an elevated peripheral vascular resistance. Early reports postulated a myocardial depressive factor; however, such a factor has never been isolated. Recent clinical studies have demonstrated that in the absence of heart disease, the ventricular ejection fraction and velocity of myocardial fiber shortening are actually increased following thermal injury and that hypovolemia, as measured by decreased left ventricular end diastolic volume, results in the depressed cardiac output (42). With repletion of the plasma volume, cardiac output increases to even supranormal levels. This hyperdynamic state is characteristic of the hypermetabolic flow phase of thermal injury.

The alterations in pulmonary function postburn are similar to those seen

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PATIENT TRANSFER INFORMATION SHEET

Date and time of call_____

Referring MD_____Telephone_____

Hospital_____City_____State_____

PATIENT INFORMATION

Name_____SSAN_____Status:Active Duty_____
Retired_____
Age_____Sex_____Pre-Burn Weight_____ Dependent_____
VAB/BEC_____
Date of burn_____Cause_____ PHS_____
Civilian_____
Extent of burn_____3rd Degree_____

Areas burned_____

Inhalation injury_____Allergies_____

Associated injuries_____

Pre-existing diseases_____

TREATMENT CHECK-LIST

Resuscitation: Calculated need (2ml/Kg/% TBS)_____

Fluid in _____Urine Output_____

Airway_____Blood gases_____E - T Tube_____

Medications: Analgesics or sedatives_____Tetanus_____

Antibiotics_____Other Meds_____

Escharotomies: Arms_____Legs_____Chest_____

Wound Care: Wash and debride_____Topical Agent_____

Lab tests: HCT_____Electrolytes_____BS_____BUN_____

Request: Insert NG tube - Avoid general anesthesia or IM meds - Keep I & O

INFORMATION FOR FLIGHT PLAN

Burn Team_____Family to accompany patient_____

Location of nearest airport with jet traffic_____

Transportation for team at destination_____

FIG. 4. The use of a standard check-sheet facilitates assessment of the patient's physiologic status by the referring and receiving physicians.

after other forms of traumatic injury. Immediately postburn, minute ventilation usually increases. This initial hyperventilation may be in response to anxiety and pain but may also be in compensation to hypovolemia. Following resuscitation, respiratory rate and tidal volume progressively increase, resulting in a minute ventilation that may be 2 to 2½ times normal. This increase is proportional to the extent of burn and represents another manifestation of postinjury hypermetabolism. Pulmonary vascular resistance also increases immediately following the burn (43). The release of vasoactive amines and other mediators after thermal injury may be responsible. This increase in pulmonary vascular resistance may exert a protective effect during fluid resuscitation by decreasing pulmonary capillary hydrostatic pressure and thereby lowering the propensity for pulmonary edema. The accumulation of chest wall edema, exacerbated by the infusion of large volumes of resuscitation fluid, decreases total lung compliance and may promote atelectasis and hypoxemia. In the absence of inhalation injury, lung lymph flow studies have demonstrated no change in pulmonary capillary permeability after cutaneous thermal injury (44). However, overzealous fluid resuscitation may result in florid pulmonary edema. This complication can be exacerbated by any cardiac dysfunction and may become most severe between the third and sixth days postburn as the edema fluid is reabsorbed into the intravascular space.

In the immediate postburn period, glomerular filtration rate and renal blood

flow are reduced in proportion to any deficit in intravascular volume. Inadequate fluid resuscitation compromises renal perfusion and may lead to acute tubular necrosis and renal failure. Following successful resuscitation, a diuresis is often observed correlating to resorption of edema. This diuresis is associated with an increased renal plasma flow. However, blood volume measured by chromium^{51} blood cell labeling was only 81% of predicted values in a group of severely burned patients studied by Cioffi et al. (45). Plasma renin activity and antidiuretic hormone levels were also elevated consistent with a decreased blood volume. This phenomena may, in part, explain the propensity for tremendous sodium retention following thermal injury.

Gastrointestinal dysfunction also appears related to the magnitude of thermal injury. In patients with burns >25% of the total body surface, gastroparesis is commonly observed. Normal gastric motility usually returns by the third to fifth postburn day. Focal ischemic mucosal lesions of the stomach and duodenum may be observed as early as 3 to 5 h following burn and are present at an incidence approaching 100% in patients with burns >40% TBSA. Intestinal bacterial translocation following burn injury has been extensively studied in the laboratory, and increased intestinal permeability to low molecular weight sugars has been identified as a prodrome to the onset of infection (46). However, the clinical significance and therapeutic implications of these findings have not been fully determined.

As the magnitude of burn increases, so does the likelihood of early postburn hepatic dysfunction. An initial increase in the hepatic transaminase usually follows burns >50% TBSA. This is most likely due to a reduction in hepatic nutrient blood flow during the initial resuscitative phase. This detriment in hepatic perfusion may be exaggerated by splanchnic vasoconstriction. Following fluid resuscitation, hepatic enzymes promptly return to normal in most patients. Although the magnitude of initial enzyme derangements are not predictive of outcome, the early onset of jaundice is associated with a poor prognosis and is usually indicative of preexisting hepatic dysfunction. Hyperbilirubinemia and elevation of liver enzymes in a cholestatic pattern occurring later (>5 days) are most often associated with sepsis or multiorgan failure.

Mechanisms of edema formation

After thermal injury, the rate of edema formation into surrounding tissues is greatest in the first 6 to 8 h postburn. Edema formation continues for the next 24 h but at a lesser rate (47). In burns ≥20% TBSA, generalized edema involving noninjured skin, deeper tissues, and internal organs also occurs.

The increased translocation of fluid and plasma proteins into the interstitial compartment has been generally attributed to an increase in microvascular permeability. The direct effect of heat, the effects of various humoral factors liberated from injured tissue, and systemic cytokine liberation have all been implicated as mediators for the increased permeability. Studies aimed at investigating the mechanism of edema formation can be divided into those that explore the physical characteristics of the movement of fluid and proteins from the vascular to interstitial compartments and those that evaluate the biochemical mediators affecting this process.

Transcapillary fluid flux is controlled by physical factors outlined as Starling forces with the rate of transcapillary fluid

filtration (J_v) calculated from the following equation:

$$J_v = K_f[(P_c - P_i) - \sigma_d(\pi_c - \pi_i)]$$

where K_f = capillary filtration coefficient, P_c and P_i = capillary and interstitial hydrostatic pressures, respectively, π_c and π_i = the oncotic pressures in these compartments and (σ_d) = reflection coefficient:

(σ_d) describes the ability of the capillary membrane to selectively limit the passage of macromolecules. The limits of σ_d vary from 0 and 1, indicating complete permeability or impermeability to macromolecules, respectively. Lymphatic resorption and flow are also important determinants of edema formation.

The hydrostatic pressure in the interstitial space becomes strongly negative and has been shown to occur with maximal intensity during the first 30 min following injury (48). The duration and magnitude of the negative hydrostatic pressure change is proportional to the size of burn. Also in the immediate postburn period, capillary venular constriction results in a marked increase in capillary hydrostatic pressure (49). An early increase in the interstitial fluid colloid osmotic pressure has also been reported to occur immediately postburn (50). The magnitude of this effect reverses the normal transcapillary osmotic pressure gradient, resulting in a further net increase in edema formation. In moderate to extensive burns (i.e., >20% TBSA), intravascular protein depletion may occur secondary to interstitial and surface wound losses. When combined with the dilutional effect of crystalloid resuscitation, significant decreases in intravascular oncotic pressure often result.

Studies utilizing size selective macromolecules have shown that thermal injury to rodent skin consistently results in gaps between vascular endothelial cells ≥250 Å (51). Using pore-stripping analysis, burn injury in a canine paw scald burn model increased large pore radius by 10% and increased the relative number of large pores, resulting in a two-fold increase in large pore transcapillary volume flow (52). These findings may be of clinical importance when considering the effectiveness of oncotic agents used in resuscitation. Albumin and dextran 70 (53) are too small to have a significant oncotic effect across the large pores, whereas higher molecular weight dextrans and pentastarch (54) may be more effective.

Changes in vascular permeability and the subsequent edema formation have been attributed to cytokine and paracrine humoral factors. For example, the plasma concentration of histamine, a potent mediator for increasing vascular permeability, rises in proportion to burn area. Additionally, many inflammatory mediators including activated protease, prostaglandin, fibrin degradation products, leukotrienes, substance P, and bradykinin have also been demonstrated to increase microvascular permeability and edema formation following burn injury. Furthermore, specific mediator antagonists have been shown to decrease but not eliminate edema formation when administered prior to injury. This partial response with mediator blockage suggests a multifactorial etiology to postburn edema formation.

Leukocyte activation postburn results in the production and release of lysosomal enzymes and an increased activation of xanthine oxidase, complement byproduct activation and oxygen radicals, all of which have been shown experimentally to increase transcapillary fluid filtration and edema formation (55).

In animal studies, preburn neutrophil depletion has been reported to attenuate postburn lung injury; however, burn wound edema formation was not affected (56). These findings suggest that localized wound edema is largely dependent on local factors. Edema accumulation in uninjured tissues, which may be massive with extensive injury, is most likely due to decreases in serum oncotic pressure via either excessive protein loss or dilution following crystalloid resuscitation or simply as a consequence of the pressure effect of large infusion volumes.

Several approaches to reduce edema formation and restore circulatory integrity are being investigated. The free oxygen radical scavenger deferoxomine, when given concomitantly with resuscitation fluids, has been shown to reduce resuscitation fluid requirements, and thus intuitively lowers total body edema (57). Also, blockade of the classical and alternative complement pathways, by administration of a soluble complement receptor, has attenuated postburn edema formation in animals (58). Administration of osmotically active macromolecules, such as pentastarch (54), have been shown to decrease transcapillary fluid leak while maintaining circulatory integrity. Dextrans and hydroxyethyl starch have been shown to interfere with coagulation; therefore, their clinical usefulness is still in question.

Most thermally injured patients tolerate the edema formation that accompanies major fluid resuscitation, and interventions to limit transcapillary filtration of fluid and proteins may be unnecessary. Patients with massive burns and certain volume sensitive subpopulations would more likely benefit from effective resuscitation with lesser volumes of fluid, averting the complications of respiratory embarrassment, limb ischemia due to vascular compression, potential conversion of viable partial-thickness injury to full-thickness necrosis, and impairment of vital organ function. The frequency with which these patients and complications are encountered support further investigation to elucidate the causative mechanisms and identify means to reduce burn wound edema formation.

Though infrequently discussed, there may be a potential benefit to the development of burn wound edema and expansion of the interstitial space. Edema formation may dilute or delay if not prevent resorption of toxic substances and wound-derived mediators produced by, or generated in response to, coagulation necrosis of the skin. By limiting intravascular access of such agents, postburn organ dysfunction may be diminished (59). This hypothesis is more often discussed than investigated but should be considered in laboratory or clinical studies that propose to limit burn wound edema formation.

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