

was lowered on a vacuum mattress and further immobilized. A limited secondary survey was performed before take off, which confirmed the patient was now hemodynamically stable with good oxygenation. Bleeding from the tracheal tube and multiple hematomas of the chest wall suggested chest trauma. After additional monitoring (electrocardiogram, blood pressure, SpO₂, and EtCO₂) was initiated, the patient was transferred to the trauma center of the university hospital of Bern.

The initial hospital assessment showed systolic blood pressure of 130 mmHg and a pulse rate of 50 beats per minute. Both pupils were wide and did not react to light; the corneal reflex was negative. The initial computed tomography scan showed a severe cerebral edema with brainstem coning. A subdural hematoma of 2 cm with midline shift was noted as well. The patient died the following night from his severe head injury. The other patient made a good recovery.

CLINICAL ISSUES

Care of the Burn Patient: A Review

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Objective: To discuss burn patient management during the various phases of care in the prehospital environment, emergency department, operating room, and critical care unit, with emphasis on airway management.

Burned patients are probably the sickest patients a physician will ever see.¹ Major burns are associated with a high rate of multisystem failure and mortality.² Survival rates have improved over the past five decades due to improvement in many aspects of burn therapy, including emergency care, critical care, surgical care, anesthetic care, rehabilitative care, and, most importantly, the development of specialized burn centers operated according to guidelines from the American Burn Association (ABA).

Survival rates are still inversely affected by three risk factors: age >60 years, burn size >40% of total body surface area (TBSA), and the presence of inhalation injury.³ Predicted mortality is ~0.3% with no risk factors, 3% with one risk factor, 33% with two risk factors, and 90% with three risk factors. The relation between age and survival is biphasic, with mortality rates being lowest among patients 5 to 20 years old.⁴

Emergency Care of the Burn Patient

The burn patient is a trauma patient.⁵ The incidence and severity of inhalation-induced acute (or adult) respiratory distress syndrome (ARDS) is related to the number of breaths of smoke inhalation. At the scene, the priority is to isolate the

Summary

Over the past 30 years, Air Zermatt has developed a helicopter rescue service, that provides advanced trauma life support in even the most hostile and remote areas of the Swiss Alps. The service relies on teamwork and coordination among various dedicated professionals.

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patient from the heat source. Once rescued, the patient is provided with 100% oxygen and examined for associated trauma.

The “6 Cs” approach is applied for wound care: removing nonsticking clothing, cooling with water, cleaning with chlorhexidine, chemoprophylaxis, covering with a layer of gauze impregnated with petroleum jelly wrapped with absorbent gauze, and comforting (pain relief).⁶ Chemical wounds, particularly of the eyes, are irrigated immediately and continuously with water until arrival at a hospital.⁷ Neutralization of acids or alkalis is not attempted because it produces heat.⁸ Chemical burns caused by phosphorus, as in certain military burns, must be kept wet until complete surgical debridement.⁸ Burns caused by phenol are cleansed with polyethylene glycol in addition to prolonged irrigation with water. Burns caused by hydrofluoric acid are treated with topical calcium gluconate gel (2.5% q 2 hr) and calcium gluconate (1–3 g IV) to prevent a potentially lethal hypocalcemia. Esophageal alkali burns are treated subsequently with systemic steroids.⁹

At the hospital, the primary and secondary surveys of trauma are implemented, including toxicology screen and head computed tomography (CT) when indicated. Wound care continues by providing tetanus prophylaxis, analgesia such as morphine, topical antibiotics such as silver sulfadiazine, and elevating and cooling the burned area while protecting the patient from hypothermia.¹⁰

High-voltage (>1000 V) electrical burns cause deep tissue injury requiring escharotomy, fasciotomy, or amputation in 30% of cases.¹¹ Low-voltage (120–140 V) electrical burns cause minimal skin damage relative to the associated internal injury.¹² Lightning injuries cause all the complications of electrical injury, including fatal cardiac arrest.¹³ Continuous electrocardiographic monitoring and serial myocardial enzymes are indicated in electrical injury. Tetanic contractions may cause spinal cord injury, bone fracture, and rhabdomyolysis. The latter leads to hyperkalemia, hyperphosphatemia, hypocalcemia, and myoglobinuria. Myoglobinuria manifests as very dark “Coca-Cola-like” urine with no red blood cells on urine analysis. Treatment consists of lactated Ringer’s (LR) solution, with NaHCO₃ (50 mEq/L) and mannitol (12.5 g IV, every hour), with the goal of maintaining a urine output (UO) ≥ 2 ml/kg/hr and a urine pH ≥ 6.5.

Critical Care of the Burn Patient

Airway Management. Upper and lower airway injuries occur as a result of thermal or chemical insults. Airway edema occurs within seconds from exposure to heat, may take 4 to 5 hours to develop fully, may progress quickly to airway obstruction, and may take 4 to 5 days to resolve.¹⁴ Steroids do not reduce edema formation and increase the risk of infection. Indications for intubation include deep burn to the face or neck, decreased consciousness, shock, edema or blistering of the oropharynx, oronasopharyngeal soot or singed hairs, stridor, hoarseness, wheezing, bronchorrhea, respiratory distress, $\text{PaO}_2 < 60$, and $\text{PaCO}_2 > 55$ mmHg.

The intubation technique is based on the general condition and airway anatomy of the patient.¹⁵ Direct laryngoscopy and orotracheal intubation are suitable for most burned patients. A large-diameter tube is used to decrease resistance to gas flow and allow the passage of a suction catheter or fiberoptic bronchoscope. Fiberoptic bronchoscopy is used to assess subglottic injury and its resolution.¹⁶ Laryngeal mask airways (LMAs) can be used for emergency management of the airway; they have been used intraoperatively in pediatric burn patients.^{17,18} Airway exchange catheters are particularly dangerous in the burn patient because of airway edema.¹⁹ Tracheal extubation is performed after resolution of airway edema, which is assessed by testing for air leak around a deflated cuff and direct laryngoscopy. Tracheostomy performed through burned tissue is associated with an increased rate of infection. It can be performed through grafted tissue, 24 to 48 hours after grafting. Early conversion of an artificial airway to tracheostomy (before day 10) decreases the risk of subglottic stenosis.²⁰

Administration of succinylcholine to the burned patient may cause lethal hyperkalemia. The exact mechanism, onset, and duration of this response are not completely clear. The mechanism involves upregulation of extrajunctional acetylcholine receptors (AChRs) in burned and unaffected muscles.^{21,22} The same etiology is thought to be responsible for the resistance to nondepolarizing muscle relaxants. This process starts a few days after burn, peaks after few weeks, and lasts for several months.²¹ Resistance to metocurine has been reported 15 months after burn.²³ It is advisable to avoid the use of succinylcholine between 2 days and 2 years after burn injury.

Management of Inhalation Injury. The leading cause of death during the acute phase of burn is inhalation injury.²⁴ It causes more than 50% of burn deaths, increases mortality risk by sixfold, and is associated with a >70% incidence of respiratory failure.²⁵⁻²⁷ Thermal injury results in edema, sloughing, and ulceration of the mucosa. Laryngeal edema may develop rapidly toward airway obstruction.²⁸ Hot dry air is cooled by the moisture of the mucosa of the upper airways and causes less damage to lower airways. Hot steam, on the other hand, has a thermal capacity 4,000 times that of dry air and causes more damage to lower airways. Steam at 100°C is equivalent to dry air at 300°C. Thermal injury from dry air usually heals within a week. Steam injury takes longer to heal and is more prone to infectious complications.

Treatment consists of tracheal intubation, mechanical ventilation, PEEP, and bronchodilators.²⁸ A combination of heparin (5,000 units) and N-acetylcysteine (3 ml of a 20% solution) given as a nebulizer every 4 hours during the first 7 days after smoke inhalation decreases mortality and improves healing in children with inhalation injury.²⁹

Chemical airway injury results from reaction of smoke components with airway mucosa. Water-soluble gases, such as

ammonia (NH_3), sulfur dioxide (SO_2), and chlorine (Cl_2) form acids and alkalis, causing ulceration, edema, and damage to cilia. These gases cause predominantly upper airway symptoms such as laryngospasm and stridor. Lipid-soluble gases, such as nitrous oxide (N_2O), phosgene, and aldehydes cause cell membrane damage, surfactant inactivation, pulmonary edema, and impairment of gas exchange. Symptoms are manifestations of lower airway irritation, including wheezing, bronchorrhea, dyspnea, and hypoxia. Chemical injury may take 24 to 48 hours for full development. Treatment consists of tracheal intubation, mechanical ventilation, PEEP, pulmonary toilet, head elevation, and bronchodilators. Steroids are ineffective and may increase mortality and infectious morbidity.³⁰

Systemic injury due to absorption of carbon monoxide (CO) and cyanide (CN) leads to CO and CN poisoning, respectively. The leading cause of poisoning death in the United States is CO poisoning. The affinity of CO to Hb is 200 times that of O_2 .³¹ CO displaces O_2 from Hb and shifts the O_2Hb dissociation curve to the left. Binding to cytochrome C is not a major mechanism of CO toxicity. Symptoms appear at COHb levels >15%, and mortality is >50%.³² Diagnosis relies on measuring co-oximetric COHb and O_2Hb saturations. Treatment relies on the use of 100% oxygen and hyperbaric oxygen (HBO). Increasing FiO_2 5-fold, from 0.2 to 1.0, decreases the half-life ($t_{1/2}$) of COHb 5-fold, from 5 hours to 1 hour. Increasing the atmospheric pressure of $\text{FiO}_2=1.0$ 2.8-fold, from 1 atmosphere ($\text{PiO}_2=760$ mmHg) to 2.8 atmospheres ($\text{PiO}_2=2,128$), decreases the $t_{1/2}$ of COHb 2.8-fold, from 60 min to 22 min. HBO is indicated for the treatment of neurotoxicity of COHb.³³

Comatose patients with <30% COHb should be suspected of having CN toxicity. Normal CN levels are <0.1 mg/l; they become lethal at 1 mg/l.³² CN binds mitochondrial cytochrome oxidase and blocks its function, causing metabolic acidosis and high (>80%) mixed venous O_2 saturation (SvO_2). Treatment consists of 100% O_2 , CPR, and sodium thiosulfate (150 mg/kg IV, over 15 min).

Acute (adult) respiratory distress syndrome (ARDS) may develop due to direct pulmonary injury, systemic inflammation, or mechanical ventilation.³⁴ $\text{PaO}_2/\text{FiO}_2$ ratios (PFR) are used to quantify lung injury. Normal PFR is about 500. A PFR of 300 indicates acute lung injury (ALI); 200 indicates ARDS; and 150 indicates severe ARDS.^{35,36} Ventilator-induced lung injury (VILI) develops mainly through alveolar collapse and overdistention (volotrauma and barotrauma). Protective strategies against VILI include the use of tidal volume ≤ 6 ml/kg, plateau inspiratory pressure ≤ 35 cm H_2O , and PEEP above the lower inflection point on the ventilatory pressure-volume curve, permissive hypercapnia, and preferential use of pressure-limited ventilation.³⁷

Fluid Resuscitation. Fluid loss from burned areas begins immediately and lasts about 36 hours. Injury to the microvasculature and cell membranes is the main mechanism of fluid shifts and loss.¹⁴ Inhalation injury induces an oxidant injury, leading to increased fluid loss and fluid resuscitation requirements. Plasma loss may exceed 4 ml/kg/hr and sodium loss 0.5 mEq/kg/%TBSA. This rate of loss may lead to hypovolemic shock in adults with 15%TBSA burn or children with 10%TBSA burn. Both sodium and water need to be replaced to prevent shock.

The Parkland formula (LR at a rate of 4 ml/kg/TBSA%burn/24 hr, with 50% given during the first 8 hr) is based on the amount of fluid necessary to replace sodium loss.¹⁴ Children have higher fluid requirements and are more sensitive to excessive or inadequate fluid replacement; therefore, accurate TBSA assessment is mandatory in children.³⁸ The Evans formula (LR at

1 ml/kg/TBSA%burn + colloid at 1 ml/kg/TBSA%burn + D5W at 2 l/m²/24 hr) is more suitable for children and consists of LR, colloid, and D5W. Burns >50% TBSA are treated at 50%TBSA rates; and on day 2, 50% of day 1 requirements are used.⁷ After 48 hours, fluids are given for maintenance and replacement of evaporative losses. Fluids should be isotonic but not necessarily sodium containing. Evaporative losses are calculated on the basis of burn size and body surface area: evaporative losses = (25 ml+TBSA%burn) x (m²)/hr. Hypoalbuminemia increases edema. After 12 to 24 hours, if albumin is <2 g/dl, albumin 5% at 15 ml/hr or 25% at 3 ml/hr is given.³⁹

Inadequate fluid replacement leads to hypoperfusion and renal failure, while excessive fluid replacement leads to tissue edema and upgrading of burn depth. Monitoring includes heart rate (HR), blood pressure (BP), oxygen saturation, urinary output (UO), complete blood count, SMA-7, albumin, and lactate levels. Goals of resuscitation include HR<100, SBP>100, UO >0.5 ml/kg/hr for adults, UO >1 ml/kg/hr in children, UO >2 ml/kg/hr in the presence of myoglobinuria (with the addition of IV sodium bicarbonate to enhance myoglobin elimination), Hct <50%, Na <150 mEq/l, albumin >20 g/l, and urinary Na >40 mEq/l.⁴⁰ Pulmonary artery (PA) catheter is more sensitive in assessing the adequacy of fluid resuscitation. The ability to increase oxygen consumption in relation to burn size appears to distinguish survivors from nonsurvivors. Hypertonic lactated saline administration has been associated with increased mortality, and hypertonic saline administration has been associated with increased renal failure and mortality.^{41,42}

Nutritional Support. Burn injury produces a state of hypermetabolism and protein hypercatabolism that lasts as long as the skin is not covered.^{43,44} Caloric and protein requirements are increased 2- to 2.5-fold.^{45,46}

Caloric requirements in adults are calculated using the Curreri formula⁴⁷: caloric needs = (25 kcal/kg) + (40 kcal/TBSA%burn). The Galveston formula is more suitable for children: caloric needs = (1800 kcal/m²) + (1300 kcal/m² burned). Calories are supplied as 60% carbohydrate, 25% protein, and 15% fat.⁴⁸ Proteins are provided based on body weight (1.2–2 gm/kg/day) or the calorie/ nitrogen ratio (CNR) — a CNR of 150 for minor burns and 100 for major burns.^{49,50} A positive nitrogen balance of 0 to 4 g/day is the goal. Nitrogen balance is calculated based on measured urine urea nitrogen (UUN): nitrogen balance = [protein intake(g) x 16%] – [UUN+4]. Triglyceride levels are maintained below 250 mg/dL, measured 4 hours after cessation of lipid infusion. Water deficit or excess is calculated based on plasma sodium: H₂O deficit = 0.6 x (kg) x [(plasma Na/140) – 1]; H₂O excess = 0.6 x (kg) x [1 – (plasma Na/140)]. Copper, selenium, and zinc are involved in immune and antioxidative mechanisms and tissue repair.^{51,52} Vitamins C and E reduce the oxidative inflammatory response.⁵³

Enteral feeding is preferred over parenteral. It protects against stress ulcer and permeability to bacteria and toxins.⁵⁴ It is associated with less mortality and better T-cell helper/suppressor ratios than parenteral nutrition.^{55,56} It can be started as soon as the patient is hemodynamically stable.⁵⁷ Intolerance to enteral feeding is an early sign of sepsis and is associated with increased mortality.⁵⁸

Management of Infection. Infection is the leading cause of death in patients who survive the acute phase of burn injury.^{52,59,60} Burns suppress humoral and cellular specific immune functions and neutrophil and macrophage nonspecific immune functions.⁶¹ Burned skin produces a toxic lipid protein complex (LPC) (cutaneous burn toxin [CBT]) that is highly immunosuppressive.⁶² Both burns and infection increase

bowel permeability, facilitating translocation of bacteria and absorption of endotoxins.⁶³

Tracheal intubation is the most significant risk factor for the development of nosocomial infection, particularly pneumonia, burn wound infection, and bacteremia.⁶⁴ The most common sources of infection are the lungs, wound, IV catheters, blood, urinary tract, gall bladder, and pancreas. In children, the most common infections are burn wound infection and catheter-associated septicemia.⁶⁵ In these patients, changing intravenous catheters every 3 days is justifiable.

Wound biopsy allows quantification of the organism count (>100,000/g), staging of the invasive process, and identification of histopathologic changes characteristic of bacterial, fungal, or viral infection. Bacterial or fungal wound infections require surgical removal of infected tissue and appropriate topical and systemic antibiotics. Systemic antibiotics are used only for the treatment of infection or preoperative prophylaxis. Use of broad-spectrum antibiotics and delay in wound closure increase the risk of *Candida* infection. Treatment of fungal infection requires surgical removal of infected tissue, wound closure, and systemic amphotericin B.⁶⁶ All patients should receive tetanus prophylaxis.

Surgical Care of the Burn Patient

Assessment and Categorization. The severity of burn injury is categorized according to size, depth, and complicating injuries or illnesses (Table 1). Burn size is estimated using the rule of nines or the Lund and Browder chart. The rule of nines divides the body into 11 areas of 9% each and assigns the remaining 1% to the perineum. The 11 areas are the head, each arm, the chest, the abdomen, the upper back, the lower back, and each of the anterior and posterior aspects of the lower extremities. The Lund and Browder chart assigns fixed percentages to the torso and upper extremities while it assigns to the head and lower extremities variable percentages according to age.^{67,68}

Treatment. Full-thickness burns are treated with excision and grafting. Early excision and grafting decreases morbidity and mortality. Burns to the face and other functional areas have priority for treatment.

Cultured autologous epithelium (CAE), cultured epithelial allografts (CEA), human cadaver allograft skin (HCAS), cultured skin substitutes (CSS), and biosynthetic skin substitutes (BSS) are used to overcome limitations of autografts or when autografts are not feasible due to patient condition. In addition, anabolic hormones may be used to enhance wound healing.⁶⁹

Anesthetic Care of the Burn Patient

Anesthetic management aims at maintaining homeostasis in the face of hypermetabolism and massive blood loss.⁷⁰ Hypermetabolism increases basal body temperature (BBT) by 0.03°C/1% TBSA burned and increases CO₂ production. The expected BBT of a patient with 50%TBSA is 38.5°C. Ambient temperature is maintained above 28°C (83°F). Intravenous fluids and inhaled gases are warmed. Hypothermia impairs platelet function and exacerbates thrombocytopenic coagulopathy. Increased CO₂ production is counteracted by mild hyperventilation.

Blood loss is reduced by early excision; avoidance of intraoperative hypothermia and hypertension; the use of tourniquets; and the use of topical thrombin, topical fibrin glue, topical epinephrine, subcutaneous epinephrine, and intravenous triglycyl-lysine vasopressin (TGLVP).⁷¹ Transfusion requirements may be reduced by acute hemodilution or pre-

Table 1. Degrees of Burns, Clinical Manifestations, and Treatment

Category	Tissues Affected	Clinical Manifestation	Prognosis	Wound Care
First degree	Superficial epidermis	Painful erythema	Heals in 2–4 days	Cleaning Antibiotic dressing
Second degree (superficial)	Epidermis Basal membrane Superficial dermis	Painful erythema Blistering Blanching with pressure	Heals in 10–14 days	Cleaning Antibiotic dressing Elevate wound
Second degree (deep)	Epidermis Basal membrane Superficial dermis	Painful erythema Little/no blisters No blanching with pressure	Heals poorly; if heals, takes 3–4 weeks	Antibiotic dressing Elevate wound Excision, grafting
Third degree	Epidermis Basal membrane Dermis	Painless Blisterless	No spontaneous resolution	Excision, grafting Antibiotic dressing Elevate wound Escharotomy/ fasciotomy

operative administration of erythropoietin. Massive transfusion may lead to hypocalcemia at infusion rates >50 ml/min, particularly in patients with liver disease or immature liver function such as infants and small children.

Intraoperative topical lidocaine spray (2%) applied to harvest sites reduces postoperative narcotic requirements. Clonidine (3 mcg/kg) and meperidine (0.4 mg/kg) are effective therapy for postoperative shivering. Dimenhydrinate (0.5 mg/kg) and ondansetron (0.1 mg/kg) are effective for the prevention and treatment of postoperative nausea and vomiting.^{72,73}

Postoperative and Post-injury Analgesic Care of the Burn Patient

Analgesia attenuates the stress response and its hypermetabolic consequences. Opioids are the most common analgesics used for baseline and procedural pain. Ketamine is used for procedural pain. Acetaminophen is used for minor baseline pain.⁷⁴ Methadone is useful for baseline pain and to reestablish sensitivity to opioids if tolerance develops. Patient-controlled analgesia (PCA) is safe and effective in addressing the individual variations in analgesic requirements.⁷⁵

Treatment of anxiety and depression enhances analgesia. Antidepressants are used for the treatment of depression, acute stress disorder, and chronic baseline pain. Distraction provided by virtual reality enhances analgesia for procedural pain. Hypnosis enhances analgesia for procedural and baseline pain. Treatment of posttraumatic stress disorder requires pharmacotherapy, psychotherapy, and behavioral therapy.

Psychological and Physical Rehabilitative Care of the Burn Patient

Depression, anxiety, and phobias may develop in children. Mothers of burned children may have a higher rate of psychological sequelae than their children. Burns in children may be the result of individual or familial psychosocial problems. A comprehensive approach of care with appropriate follow-up is essential.⁷⁶ Burn rehabilitation units are efficacious in decreasing the length of stay and improving the quality of recovery.⁷⁷

Conclusion

Burn injury leads to the greatest disruption in the physiological, emotional, and social aspects of the patient's life. The outcome from burn is dependent upon a comprehensive therapeutic approach that involves every aspect of care. Survival rates have been increasing over the past few decades, with a satisfying quality of life in most cases.

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