Part X: Trauma

Chapter 73: Burns

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Severe burns produce devastating physical and psychological effects. Management involves a wide spectrum of activities ranging from initial resuscitation to eventual surgery and rehabilitation. Burn patients are most effectively treated by specially trained staff in an environment controlled in temperature, humidity and perhaps by laminar flow isolation. This chapter will limit its scope to resuscitation and early care of burn patient.

Pathophysiology

Burns may be inflicted by heat (wet or dry), chemicals, electricity or radiation. The severity and extent of injury is usually classified as:

1. *First degree burn* which involves only the epithelial layer. It is often very painful, but resolves with no residual scarring.

2. *Second degree burn* which involves epithelium and to a varying degree of dermis. Pain and scarring vary according to the depth of the dermal injury.

3. *Third degree burn* which involves the full skin thickness. It is usually painless due to destruction of cutaneous innervation and will lead to scarring.

Cardiovascular and Circulatory Effects

There is a marked, immediate increase of vascular permeability in the area of burn. Transvascular leakage becomes generalized in burns involving greater than 20% of total body surface area (TBSA). The transvascular fluid is similar to plasma in protein and ionic content. Sequestration and oedema formation is maximal in the first 24 hours. The increased vascular permeability is probably due to the direct effect of heat on the vascular tissue, and to vasoactive substances such as the leukotrienes, prostaglandins, oxygen radicals and histamine that are released from the burned tissues.

Cardiac output falls significantly due to reduced plasma volume. The contribution of a circulating myocardial depressant factor to the reduction in cardiac output remains controversial. Burn shock is most likely to occur in the first 48 hours in patients with more than 20% TBSA burn, leading to hypoperfusion of vital organs.

Red cells loss due to haemolysis and intravascular coagulation is likely to occur in patients with extensive burns (over 20% TBSA) and may be as high as 1% of red cell mass per 1% of full skin thickness burn. The increased red cell destruction is thought to be due to some agents in plasma which modify red cells making them more liable to destruction.

Coagulopathy may occur in the resuscitation phase. Apart from dilution, there is also a rapid activation of clotting factors leading to shortening of clotting times and acute

reduction of various clotting factors including prothrombin, Factors V, VIII and IX. Disseminated intravascular coagulation (DIC) is common in extensive burns. Intravascular platelet aggregation with thrombocytopenia and thrombocytopathy may necessitate platelet transfusion. Thereafter a phase of normal coagulation parameters is usually present. A hypercoagulable state may develop 2-4 weeks post burn.

Respiratory Effects

Pulmonary dysfunction is a major cause of mortality and morbidity. The incidence and causes are related to numerous factors. Thermal injury ni a closed environment may not only cause pulmonary inhalation injury, but also intoxication from combustion products. Of these, the important ones are carbon monoxide and cyanide. Upper airway damage is due to heat and inhalation of hot particles or noxious chemicals. The pathophysiology of parenchymal lund damage is still uncertain. It may be caused directly by thermal injury and chemical irritation or indirectly by fluid overloading, secondary infection, the adult respiratory distress syndrome, and pulmonary embolism.

The work of breathing increases significantly from increased airway resistance and reduced lung compliance. Arterial hypoxaemia with hypocarbia is the usual finding on blood gas analysis. These changes are most marked during the 10 to 14 days following burns. Bronchopneumonia is usually caused by airborne organisms rather than by haematogenous spread from the infected burn wound.

Metabolic Effects

Increase in metabolic rate associated with nitrogen loss following thermal injury, is greater than after any other forms of trauma. There is a proportion increase in oxygen consumption. The stress response is manifested by persistent hyperpyrexia, tachycardia, hyperventilation and hyperglycaemia. The mediators of the response are humoral; primarily an increased release of catecholamines, along with other anti-insulin hormones are responsible for the glucose intolerance noted during the early postburn period. Plasma insulin level is low immediately after thermal injury, but it is usually followed by a prolonged "insulin resistance" phase. Hypermetabolism increases with cooling, pain and sepsis. Hence efforts to limit the metabolism are directed to raising the environmental temperature, reducing evaporative loss by covering the burn areas with heterograft, and dressing the wound with pain-free dressing.

Immunological Effects

Both immune and nonspecific inflammatory host defense systems are generally depressed in severe burn injuries. Complement and immunoglobulin levels are initially depressed, but swiftly return to normal. A plasma inhibitor of chemotaxis is probably responsible for the depression of chemotaxis. Despite advances in burn wound care and in topical and systemic antibiotics, sepsis still accounts for more than 50% of the mortality in burns.

Other Systemic Effects

Renal failure, as oliguric and non-oliguric failure, may occur as a complication of renal hypoperfusion, haemoglobinuria, myoglobinuria or septicaemia. It is associated with a high mortality even after dialytic therapy. There is progressive azotaemia, acidosis and hyperkalaemia although the symptoms is non-oliguric failure are less prominent. After thermal injury, acute ulceration of the stomach or duodenum, known as Curling's ulcer, has been shown to occur in approximately 11% of the total burn population. Major bleeding episodes from this ulceration may require surgical intervention.

Management

Calculation of the area of skin with second or third degree burn is made in the adult by the "Rule of 9". Each arm carries 9% of body skin. Front and back of the trunk are 18% each, head 9%, legs 18% each and perineum 1%. At the time of admission body weight is measured or estimated.

1. Fluid Resuscitation

Intravenous fluid resuscitation is indicated in adults if the burn involved more than 20% TBSA or 15% with inhalation injury. There is disagreement regarding the quantity and choice of fluids to administer. Hypertonic salt solutions have been shown to achieve the same resuscitation with less volumes than isotonic saline, but serious side effects (eg, hypernatraemia, hyperosmolality, and intracellular dehydration) are possible. Colloids (as albumin or plasma) cause concern, because of the increase in vascular permeability induced by burns, with consequent extravasation of plasma proteins. However, more recent studies suggest that this increased vascular permeability is probably transient and significant only in the first 3-6 hours post burn. Hence, withholding colloidal proteins for too long after the injury could be detrimental, with possible decreased oncotic pressure, hypovolaemia, and peripheral and pulmonary oedema. Convincing, consistent clinical evidence of the best type of fluid to use are yet unavailable, but a mixture of Ringer's lactate with albumin may be superior. Consensus conference recommendations for supportive therapy in burn care are:

(a) In the initial 24 hours postburn, only balanced salt solutions (eg, Ringer's lactate) is given at a rate of 2-4 mL/kg/% burn, the higher volume for larger burns. Half of the estimated quantity is to be given in the first 8 hours.

(b) Colloids are administered as required after the initial 24 hours, when the capillary leak has most probably been sealed. Recent work (see above) tend to favour earlier use of colloids, eg, after the first 6 hours. Various formulae have been recommended (Table 1), but they serve only as quidelines, and the quantity and composition of the resuscitation fluids should be titrated against individual patient response. The use of glucose containing solutions is probably inadvisable, because of an early glucose intolerance in the immediate postburn period.

Sodium requirement is about 0.5 mmol/kg/% burn. However, frequent monitoring of patient response and biochemical investigations are necessary to adjust the fluid replacement regimen. Blood transfusions are indicated in patients with massive haemolysis or blood loss.

Haematocrit between 0.30-0.35 is optimal for oxygen carrying capacity. If haemolysis or muscle damage is significant, mannitol is given to produce a urine output of 1-2 mL/kg/h to facilitate elimination of nephrotoxic haem pigments.

Table 1. *Various Fluid Resuscitation Formulae in Adult Burns*

0-24 hours

Formula	**Crystalloids**	**Colloid**
Evans	Lactated Ringer's 1.0 mL/kg/% burn Dextrose water 2000 mL	1.0 ml/kg/% burn
Parkland	Lactated Ringer's 4.0 mL/kg/% burn	None
Brooke	Lactated Ringer's 1.5 mL/kg/% burn Dextrose water 2000 mL	0.5 ml/kg/% burn
Modified	Lactated Ringer's 2 mL/kg/% burn	None

24-48 hours

Formula	**Crystalloids**	**Colloid**
Evans	Lactated Ringer's 0.5 mL/kg/% burn Dextrose water 2000 mL	0.5 ml/kg/% burn
Parkland	None	20-60% of calculated plasma volume
Brooke	Lactated Ringer's 0.75 mL/kg/% burn Dextrose water 2000 mL	0.25 ml/kg/% burn
Modified burn.	None	0.3-0.5 mL/kg/%

2. Respiration

Careful assessment of patency of the upper airway with a high index of suspicion is vital. Upper airway obstruction may develop suddenly and catastrophically. If any doubt exists, the airway should be secured immediately with an endotracheal tube. Tracheostomy is associated with high mortality and morbidity, but may occasionally be the only option. Intubation and ventilatory support, with or without positive end expiratory pressure are required if respiratory failure and/or the adult respiratory distress syndrome (ARDS) develops. Suxamethonium should be avoided in the first 60 postburn days, as severe hyperkalaemia may follow its administration. These patients are relatively insensitive to non-depolarizing drugs and may require large doses for paralysis. The lungs are particularly sensitive to barotrauma

and may rupture easily with excessive inspiratory pressure.

Carbon monoxide poisoning should be suspected if patients show signs of mental disturbances. Oxygen should be administered immediately by a device capable of delivering 100%. (See Chapter 18, Oxygen Therapy.) This will decrease the half life of COHb from 4 hours on air to about 80 minutes. Blood COHb level can be measured by co-oximeter. Symptoms appearing at a COHb level of 15% include headache, nausea and angina pain in patients with ischaemic heart disease. At 25% COHb, ST depression on ECG may appear and sensorium is depressed. Arterial PO_2 may be normal during significant CO poisoning. A valuable diagnostic clue is the measured oxyhaemoglobin saturation being much lower than expected in relation to PaO_2. Cyanide poisoning is very dangerous, and unfortunately difficult to detect. Anticyanide chemotherapy includes sodium nitrate, sodium thiosulfate and hydroxycobalamine.

3. Electrolyte Disturbances

Electrolyte disturbances are commonly encountered following the initial resuscitation phase. Hypernatraemia is usually due to salt loading and inadequate replacement of insensible fluid losses. Salt restriction and dextrose in water will correct this abnormality. Hyponatraemia and hypokalaemia may have arisen from frequent bathing of burned patients in water rather than in an isotonic solution. Therapy with a mixture of sodium and potassium chlorides will restore the electrolyte concentrations. Hyperkalaemia may be severe in the acute phase due to tissue and red cell destruction. Dextrose and insulin may be required if hyperkalaemia is marked. Later renal losses of potassium may be high and potassium supplement with 80-200 mmol/day is then indicated. Hypocalcaemia is usually due to albumin depletion, and correction of hyponatraemia is often all that is required.

4. Acid Base Status

Acidosis develops quickly (within hours) after severe thermal injury exceeding 30% TBSA. It has both metabolic and respiratory components. The metabolic part arises from the products of heat-damaged tissues and relative hypoxia, while respiratory acidosis is most commonly due to inhalation of smoke. Prompt correction is desirable because acidosis reduces cardiac output, increases total systemic vascular resistance, decreases oxygen carrying capacity of haemoglobin, and reduces renal blood flow.

5. Nutritional Support

Assessment of injury and nutrition should be made within 48 hours of admission. Nutritional support should be commenced when resuscitative phase is complete, usually within 72 hours. The oral and nasogastric route is favoured. In the presence of impaired gastrointestinal function, total parenteral nutrition (TPN) is then indicated. There are many formulae to calculate caloric and protein requirement. The Curreri formula is simple and applicable, where daily energy requirements:

25 kcal (105 kJ)/kg + 40 kcal (168 kJ)/% TBSA.

There is little reason to increase caloric intake beyond that calculated for a 50%

TBSA. A calorie-to-nitrogen ratio of 100:1 and amino-acid solutions rich in the branched-chain aminoacids have been recommended for facilitating anabolism. A daily protein regime of 1.5 to 2.5 g/kg is given. The energy requirement is provided by glucose and lipids. Insulin may be required to control hyperglycaemia. (See Chapter 82, Parenteral Nutrition.)

6. The Burn Wound

Early excision and grafting is widely accepted for the treatment of burns, because it minimizes infection and hastens wound healing. Excision is carried out only after fluid resuscitation and the patient's condition is stable, usually 24-36 hours post burn. However, mortality is high in patients with extremes of age and more than 60% body surface burn. Contributing factors include massive blood loss and hypothermia. The excised wound must be grafted or temporarily covered using human skin (ie, autograft, allograft), pigskin (xenograft), collagen sheet, or synthetic membranes.

Topical antimicrobial therapy after wound cleaning and the deroofing of blisters, may lower the rate of infection, but not by itself, the treatment of choice.

(a) *Silver sulphadiazine* (SSD) 1% is the agent most widely used. Dermal hypersensitivity reactions and transient leucopenia occur in up to 5% of patients.

(b) *Mafenide acetate* 10% is effective, but can produce pain on application, and may cause metabolic acidosis by carbonic anhydrase inhibition.

(c) *Silver nitrate solution* 0.5% is effective and safe but being hypotonic, may cause hyponatraemia. Methaemoglobinaemia is occasionally encountered.

7. Systemic Antimicrobial Therapy

Systemic antibiotics are valuable in burn patients when properly used. However, injudicious use may be harmful either through direct toxicity or by contributing to the emergence of resistant strains of micro-organisms. General guidelines and principles for systemic antibiotic use are:

(a) The burned patient, despite all efforts, will be exposed to micro-organisms, and no single agent or combination of agents can destroy all organisms.

(b) Treatment is indicated after identification of the responsible organism.

(c) Appropriate antimicrobial agents should be chosen.

(d) Once chosen, the antibiotic should be used for a long enough period to achieve clinical effect (usually 5-7 days), but not long enough to allow for emergence of opportunistic or resistant organisms.

(e) Dosage must be adjusted based on serum concentration, because the pathophysiological changes occurring in burn patients may have a profound effect on the pharmacokinetics of the drugs administered.

(f) Routine prophylactic administration of penicillin in the immediate postburn period is no longer recommended.

(g) In general, prophylactic systemic antibiotics are indicated in only a few clinical situations. These include immediate perioperative periods associated with excision and autografting, and possibly in the early phases of burns in children. Choice of agent is based on wound culture or prior knowledge of the bacterial colonization pattern in the burn unit. Gram positive coverage using first generation cephalosporin is commonly used in autografting.

(h) Constant review of the agents and route of administration is mandatory.

The penetration of systemic antibiotic into burn eschar is unreliable because this aspect is not yet fully studied. *Staphylococcus aureus,* Group A *Streptococci, Pseudomonas, Klebsiella,* and *Escherichia coli* are common wound pathogens. Early burn excision and removal of infection nidus, nutritional support, and avoidance of invasive monitoring are important factors to be observed in controlling clinical sepsis.

Use of immunomodulation therapy such as *Corynebacterium parvum* vaccine, fibronectin replacement therapy, cryoprecipitate infusion are currently under investigation.

8. Tetanus Toxoid

Tetanus toxoid is given on admission if indicated by immune status. Patients not previously immunized should receive 250 units of tetanus human immune globulin and the first of a series of active immunization with tetanus toxoid (See Chapter 45, Tetanus.)

9. Analgesia

A partial thickness burn is often very painful. Immersion or showering of the affected area with cool water reduces the extent of thermal damage and provides pain relief. A small dose of continuous intravenous infusion of pethidine or morphine titrated against the patient's response is the method of choice to relieve pain. Ketamine is a useful anaesthetic and analgesic agent for burn patients.

10. Renal Failure

Renal failure may require dialytic therapy. Early dialysis, using the criteria of a blood urea nitrogen in excess of 6.6 mmol/L or a plasma creatinine concentration exceeding 180 micromol/L, regardless of volume or quality of the urine has been advocated. Bacterial infection is a major hazard, and many dialysed patients die from the results of infection rather than renal failure.

Investigations and Monitoring

The following recommendations are modified according to the severity of the burn.

Cardiovascular Function

Monitoring should include measurement of pulse, arterial blood pressure, central venous pressure and continuous ECG. Pulmonary capillary wedge pressure and cardiac output estimations are indicated only in difficult cases, as the complications of insertion of Swan-Ganz catheters are greater in the burn patient than in the critically ill, non-burn patient. Urine output and measurement of acid-base balance are useful in monitoring the perfusion.

Respiratory Function

This is assessed clinically with the aid of chest X-ray, pulse oximetry, arterial blood gases and carboxyhaemoglobin levels. The upper respiratory tract can be evaluated by fibreoptic bronchoscopy. Xenon 133 lung scanning is used to identify areas of air trapping caused by small airway obstructions. Pulmonary function tests may be useful in predicting the extent of damage.

Metabolic and Fluid Balance

These are monitored by daily weighing and strict fluid balance charting. A urinary catheter is indicated when burns in excess of 20% BSA are present. Daily serum electrolyte and osmolality estimations are indicated in the early post-burn stage. Blood sugar, serum proteins and parameters of hepatic and renal function, should be assessed as indicated. Haemoglobin, packed cell volume, white cell, platelet counts and clotting profile should be closely followed.

Bacteriological Studies

Bacteriological studies of wound swabs and tissue biopsies are performed with isolation, quantification, and determination of antibiotic sensitivity of colonizing organisms. Sputum and urine should be cultured regularly.

Tissue Status

Tissue status is constantly observed. Viability is estimated by clinical observation. Ultrasonic flow meters and xenon flow studies have been reported to be useful.

Prognosis

Serious burns constitute a devastating injury. Burns of more than 20% TBSA plus severe smoke inhalation are fatal in 50-80% of patients. Major contributing factors to the improved survival of burn patients are adequate nutritional support, control of sepsis and early skin grafting.