Lesson 262: PreAnesthetic Assessment of the Patient With Burn Injuries (Part 1)

**PREANESTHETIC ASSESSMENT**

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A small percentage of patients—usually those who have large, deep burns, are at the extremes of age, have an inhalation injury, or have preexisting medical conditions—will fail to respond to conventional resuscitation. Although some burn centers use plasma exchange in this subset of patients to drastically reduce the fluid needed to maintain hemodynamic stability, the technique is not widely used. Several formulas for fluid resuscitation are listed in Table 1. It is important to note that certain patients have additional fluid requirements: those with inhalation injuries, electrical burns, or associated trauma; those in whom resuscitation is delayed; and children. Although several variables (including blood pressure, heart rate, urine output, and central venous pressure) have been used to assess the adequacy of fluid replacement (Table 2), the single best criterion is the output of urine at a rate of 30 mL per hour in an adult (0.5 mL/kg per hour) and of 1 mL/kg per hour in a child.6,8 The size of a wound directly affects decisions about fluid resuscitation, nutritional support, and surgical intervention; thus, thermal injuries must be accurately evaluated and classified. Burns are classified according to the TBSA of injured tissue, depth of the wound, and presence or absence of an inhalation injury. In addition, these parameters assist the physician in determining the severity and prognosis of the injury and the disposition of the patient. The rule of nines method provides a good estimate of the TBSA affected in an adult with a burn injury.5,11 In children, the Lund and Browder chart offers the most accurate method for evaluating the burned surface area because it takes into account changes brought about by growth.6 Table 3 outlines the criteria for classifying burns. Table 4 outlines a system for grading the severity of burns and determining the disposition of the patient.

### Pathophysiology of Thermal Injuries

**Mediators Involved in Local and Systemic Effects**

Mediators released from a wound after a burn injury contribute to local inflammation and edema. The mediators include oxygen-derived free radicals, arachidonic acid metabolites, and complement proteins.14 In minor burns, the inflammatory process is confined to the wound itself; however, after major thermal injuries, local injury triggers the release of mediators that circulate to produce a systemic response characterized by hypermetabolism, immune suppression, and the systemic inflammatory response syndrome (Figure). The circulating mediators appear to be the primary cause of hypermetabolism, and the manifestations of the systemic inflammatory response syndrome are identical to those seen in sepsis. Thus, antibiotic use should be curtailed when the signs of systemic infection are absent.15 Cytokines seem to be the primary mediators of systemic inflammation. An endotoxin is often present several days after a thermal injury, even in the absence of infection.15,16 The concentration of endotoxin is proportional to burn size, and a high concentration predicts the onset of multiple organ failure and death.16 Elevated concentrations of nitric oxide may also contribute to the hemodynamic and immunologic changes seen after a burn injury.17 Table 5 lists the pathophysiologic effects of major burn injuries.

**Cardiovascular Effects**

Because thermal injuries can have profound effects on the systemic circulation, hemodynamic management is a major component of perioperative care. It is critical for the anesthesiologist to evaluate both hemodynamic adequacy and the fluid resuscitation requirements of the patient (Tables 1 and 2). Cardiac output, mixed venous oxygen saturation, cardiac and pulmonary filling pressures, and oxygen delivery are all especially important parameters of hemodynamic status. In addition to the patient’s fluid requirements, a determination of the hemoglobin level and the need for vasopressors or inotropic drugs is essential during preanesthetic preparation.11

The term "burn shock" is used to describe the hypovolemic response that can occur immediately after a thermal injury as a result of alterations in microvascular permeability of both injured and normal tissue.18 Such alterations shift protein-rich fluid from the intravascular compartment to the interstitium.19,20 Other mechanisms that contribute to the fluid flux include an increase in intravascular hydrostatic pressure, a decrease in interstitial fluid hydrostatic pressure, and an increase in interstitial osmotic pressure.21 Extravascular protein and fluid leakage cause a washout of the interstitial space and markedly increase lymph flow, which results in edema during the first 24 to 48 hours after injury.21 Increased permeability in the unburned tissue persists for less than 24 hours; however, in burned tissue this permeability remains high for longer than 72 hours.9

Cardiac output is depressed after a burn, independently of the intravascular volume.19,22,23 The presence of circulating humoral factors, a reduced responsiveness to catecholamines, and a decreased coronary blood flow diminish the contractility of cardiac tissue. The blunted cardiovascular response to both endogenous and exogenous catecholamines is caused by a reduced affinity of adrenergic receptors and a decreased production of second-messenger

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**Table 1. Fluid Resuscitation in Adult Burn Patients**

<table>
<thead>
<tr>
<th>Colloid formulas</th>
<th>Electrolyte</th>
<th>Colloid</th>
<th>DSW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evans</td>
<td>NS at 1.0 mL/kg*</td>
<td>1.0 mL/kg*</td>
<td>2,000 mL/24 h</td>
</tr>
<tr>
<td>Brooke</td>
<td>LR at 1.5 mL/kg*</td>
<td>0.5 mL/kg</td>
<td>2,000 mL/24 h</td>
</tr>
<tr>
<td>Slater</td>
<td>LR at 2 L/24 h</td>
<td>FFP</td>
<td>75 mL/kg per 24 h</td>
</tr>
</tbody>
</table>

**Table 2. Criteria for Adequate Fluid Resuscitation**

- Normalization of blood pressure
- Urine output, 1-2 mL/kg per hour
- Serum lactate, <2 mmol/L
- Base deficit, less than –5
- Gastric intramucosal pH, >7.32
- Central venous pressure, <3 mm Hg
- Cardiac index, 4.5 L/min per square meter
- Oxygen delivery index, 600 mL/min per square meter

Adapted from Woodson et al.11

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*Multiplied by the percentage of TBSA burned.

DSW, 5% dextrose in water; FFP, fresh frozen plasma; LR, lactated Ringer’s solution; Na, sodium; NaHCO₃, sodium bicarbonate; NS, normal saline; TBSA, total body surface area

Adapted from Monafo6 and Woodson et al.11

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The kidneys are especially vulnerable to injury in burn patients. The incidence of acute renal failure after a burn injury has been variously reported to be between 0.5% and 38%, and its development depends mostly on the severity of the burn and the presence of lung injury that leads to edema and a decrease in the volume of circulating blood. Acute renal failure is an indicator of a poor prognosis; the associated mortality rate is as high as 85%.

Early acute renal failure, defined as occurring within 5 days after the burn injury, is usually due to hypotension and myoglobinuria. Increased levels of circulating catecholamines, angiotensin, vasopressin, and aldosterone lead to systemic vasoconstriction, further contributing to renal insufficiency.

Late acute renal failure occurs 5 days after the burn injury and is caused primarily by sepsis, with a small number of cases caused by nephrotoxic drugs. Factors that lower the incidence of acute renal failure and its associated mortality are appropriate fluid resuscitation, early wound excision, and the prevention of infection. The glomerular filtration rate increases 3 to 7 days after thermal injury as a result of increased cardiac output and a hypermetabolic state. The pharmacokinetics of renal excretion is altered; therefore, it is important to appropriately adjust the dosages of certain antibiotics and other medications to compensate for the elevated clearance rate.

**Hepatic Effects**

In burn patients, liver damage may develop as a result of hypoxemia, hypoperfusion immediately after the burn injury, and the inhalation or absorption of chemical toxins. Later, liver dysfunction may result from drug toxicity, sepsis, or blood transfusion. Some studies have suggested that the hypermetabolic phase of a burn injury increases hepatic blood flow, protein synthesis and breakdown, and gluconeogenesis. A sustained increase in blood flow causes drug to be delivered to the liver at a faster rate, and this, combined with drug-induced enzyme induction, may result in a decreased half-life of the drug.

**Hematologic Complications**

The hematocrit increases because of hemoconcentration secondary to the movement of fluid into the interstitium and the decreased oncotic pressure of plasma. The hematocrit remains elevated despite the administration of large volumes of resuscitative fluid during the first 48 hours and therefore cannot be used as a meaningful indicator of adequate resuscitation. Anemia of burn injury develops during the first few weeks as a result of bleeding from wounds, frequent sampling of blood for laboratory tests, surgical blood loss, and the shortened half-life of erythrocytes. This latter phenomenon is attributed to red blood cell damage sustained during the thermal injury, in addition to circulating factors. The responses to exogenous and endogenous erythropoietin are conflicting because whereas the bone marrow response can be reticulocytosis, the response is not proportional to the elevated levels of erythropoietin found in burn anemia.

In patients with moderate or severe burns injuries, thrombocytopenia often develops during resuscitation. Some of the decrease is caused by hemodilution, but a larger effect is the result of increased platelet aggregation and the trapping of platelets in skin and smoke-damaged lungs. Bleeding due to thrombocytopenia is rare and limited during surgery; a platelet transfusion is rarely required. If the onset of thrombocytopenia is sudden, sepsis may be the cause. The thrombocyte level returns to normal by the end of the first week but is followed by an elevated count approximately 10 to 14 days after the injury that may persist for several months. Both thrombotic and fibrinolytic mechanisms are initiated after major burn injuries. Clotting factor levels are lowered by dilution and consumption as blood coagulates in the skin.

**Gastrointestinal Complications**

The development of gastric and intestinal ileus can immediately diminish gastrointestinal function after a thermal injury. The stomach should be adequately decompressed by the placement of a nasogastric tube. The administration of appropriate prophylaxis for excessive gastric acid soon after passage of the tube should minimize aspiration of the gastric contents. Gastrointestinal function is usually restored within 48 to 72 hours after a burn injury when generalized edema diminishes. To reduce a hypermetabolic response and prevent gluconeogenesis and stress ulceration, enteral feeding should be commenced at this time. Early enteral feeding provides the additional advantage of minimizing muscle catabolism and reducing bacterial translocation through the intestinal mucosa.

Stress ulcers, also known as Curling ulcers, are life-threatening complications. In studies of ICU patients, including adult and pediatric burn patients, the administration of usual doses of cimetidine provided inadequate protection against increased gastric acidity—most likely caused by altered pharmacokinetics and pharmacodynamics of the antacid. Frequent feedings as tolerated, and the generous use of antacids combined with larger, more frequent doses.
of histamine-receptor antagonists, are all beneficial in the prevention of stress ulceration.

**Calcium Homeostasis**

Many burn victims have abnormally low levels of ionized calcium. Aberrations of calcium metabolism in both the acute and recovery phases can persist for up to 7 weeks after the injury. Hypophosphatemia and hypermagnesemia usually abate during the latter stage of recovery from an acute injury. The reciprocal metabolism of calcium and inorganic phosphate is not evident in patients with major thermal injury; therefore, calcium supplementation is crucial, especially during extensive surgical procedures, when low calcium levels can cause such adverse effects as dysrhythmias, hypotension, and heart failure. A study by Coté et al on calcium dosing found that frequent small boluses are safer and more effective than less frequent large boluses.

**Inhalation Injury**

Injuries from smoke inhalation are associated with a mortality rate below 10% if the injury is isolated; however, when an inhalation injury is associated with any cutaneous burn, the rate of mortality is doubled. Inhalation injuries result from the harmful effects of smoke on the upper and lower respiratory tract. Products of combustion, including ammonia, nitrogen dioxide, sulfur dioxide, and chlorine, all combine with water in the respiratory tract to produce strong acids and alkalis. These products induce bronchospasm, edema, and ulceration of the mucous membranes, and they can deeply penetrate the respiratory tract to cause damage of the alveolar membranes, impairment of local defenses, and a reduction of surfactant activity. Inhalation of such compounds causes necrosis of the tracheal and bronchial epithelium, resulting in a partial or complete obstruction of the airway and removing an important barrier to infection.

Aldehydes such as acrolein are produced by the combustion of cotton, wood, and various other synthetic fibers. Even at very low concentrations, these compounds weaken ciliary function and damage the respiratory mucosa, resulting in pulmonary edema and sloughing. Physiologically, capillary permeability and airway resistance increase, and lung compliance and lung volume decrease. A progressive worsening of ventilation and perfusion occurs as pulmonary shunting increases.

The likelihood of an inhalation injury can be assessed based on certain historical and physical findings. Persons involved in closed-space fires or entrapped in a house or automobile are usually at the highest risk. Respiratory distress, carbonaceous sputum, and facial burns are all classic indications of an inhalation injury. The partial pressure of oxygen, in addition to the oxygen saturation and carboxyhemoglobin levels, should be determined by arterial blood gas analysis. Chest radiography, radionuclide lung scans, and fiber-optic bronchoscopy further aid in the diagnosis.

Most upper airway problems necessitate early intubation of the trachea. Smoke inhalation resulting in severe bronchospasm, alveolar damage, or pulmonary edema requires assisted ventilation. Although overexposure to carbon monoxide (CO) from smoke inhalation requires specific treatment (discussed in the next section), ventilatory support and intensive care are the main components in managing injuries due to smoke inhalation.

**Carbon Monoxide Poisoning**

Exposure to CO is one of the most frequent—and immediate—causes of mortality from smoke inhalation injuries and must be treated promptly. CO is produced by the incomplete combustion of carbon-containing compounds (eg, wood, coal, and gasoline) and has an affinity for hemoglobin that is 250 times greater than that of oxygen. Consequently, CO exerts its toxic effects by displacing oxygen and decreasing the oxygen-carrying capacity of hemoglobin. CO also shifts the oxygen dissociation curve to the left, reducing the unloading of oxygen to tissues. By binding to cytochrome oxidase, CO impairs the activity of several intracellular enzymes, resulting in hypoxia and metabolic acidosis.

CO poisoning can be diagnosed easily in the emergency room based on the clinical neurologic findings and measurement of the carboxyhemoglobin concentration. At carboxyhemoglobin levels below 20%, patients experience headaches, tinnitus, and nausea. At levels between 20% and 40%, weakness and drowsiness develop, and at carboxyhemoglobin concentrations above 40%, severe neurologic dysfunction and coma. At levels between 55% and 70%, cardiac dysrhythmia and brain damage are often fatal. Standard pulse oximeters do not differentiate between oxyhemoglobin and carboxyhemoglobin and so cannot be used to monitor oxygenation because when carboxyhemoglobin is present they overestimate the oxygen saturation. However, transcutaneous oxygen analyzers are useful. In addition, the first noninvasive carboxyhemoglobin monitor was recently introduced by Masimo Corporation.

CO poisoning is treated by administering 100% oxygen; the half-life of carboxyhemoglobin when 100% oxygen is breathed is decreased by a power of 4 compared with the half-life when room air is breathed. The elimination of CO depends primarily on alveolar oxygen pressure rather than alveolar ventilation; therefore, oxygen should be administered to burn patients as soon as possible. Although the utility of a hyperbaric oxygen chamber for the treatment of CO poisoning has not been clearly established in humans, it may help patients who are comatose and those whose carboxyhemoglobin level is higher than 30% at time of admission or whose TBSA with burns is greater than 40%.

Hyperbaric oxygen therapy should be considered only if it causes no delay in the treatment of other life-threatening problems, such as a compromised airway or hemodynamic instability.
In addition, tumor necrosis factor (TNF), early: burn shock, hypovolemia, impaired cardiac contractility.

Early: decreased renal blood flow and function, myoglobinuria.

Direct effects: Early: obstruction of upper airway, smoke inhalation, asphyxia.
Late: restriction of chest wall with thoracic burns.

Indirect effects: Early: effects of inflammatory mediators, complications of resuscitation (pulmonary edema).
Late: complications of ventilation (O₂ toxicity, barotrauma, infection), complications of intubation (laryngeal damage, tracheal stenosis, fistula).

Metabolic: Increased metabolic rate, increased CO₂ production and O₂ utilization, impaired thermoregulation.

Coagulation and hematologic: Early: hemoconcentration, hemolysis, activation of thrombotic and fibrinolytic systems.
Late: anemia.

Renal: Early: decreased renal blood flow and function, myoglobinuria.
Late: increased renal blood flow, variable drug clearance.

Immunologic and infectious: Impaired immune function (burn wound sepsis, pneumonia), endotoxemia, multiple organ failure.

Adapted from MacLennan et al.¹⁹

### Table 5. Pathophysiologic Effects of Major Burns

<table>
<thead>
<tr>
<th>System</th>
<th>Early: Burn Shock</th>
<th>Late: Burn Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Burn shock, hypovolemia, impaired cardiac contractility</td>
<td>Increased cardiac output, hypertension, tachycardia</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Direct effects: Obstruction of upper airway, smoke inhalation, asphyxia</td>
<td>Early: restriction of chest wall with thoracic burns</td>
</tr>
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<td>Impaired immune function (burn wound sepsis, pneumonia), endotoxemia, multiple organ failure</td>
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Adapted from MacLennan et al.¹⁹

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### Pharmacologic Considerations

A major thermal injury and its treatment result in physiologic alterations that can modify the body’s response to drugs. Both the pharmacokinetic and pharmacodynamic determinants of drug response are altered. Hence, to avoid toxicity or decreased efficacy of drugs in burn patients, different dosages may be required. Specific dosage guidelines cannot be formulated because of the complex nature of the pathophysiologic changes, interpatient variations in the nature and extent of burns, and the dynamic nature of the resuscitation and recovery phases of injury.⁴⁴

The cardiovascular response to thermal injury has 2 distinct phases that separately affect pharmacokinetic parameters. A loss of fluid from the vascular space decreases cardiac output and perfusion of the kidneys and liver—organs responsible for most drug elimination. Fluid resuscitation during this phase dilutes the protein concentration in plasma and expands the extravascular space; this can increase the patient’s sensitivity to many drugs by prolonging their action for 24 to 48 hours after injury.⁷ In addition, a decrease in cardiac output accelerates the rate of alveolar accumulation of inhalation agents, possibly resulting in exaggerated hypotension during the induction of general anesthesia.⁴⁴

A hypermetabolic and hyperdynamic circulatory phase, which develops approximately 48 to 72 hours after injury, results in elevated cardiac output, oxygen consumption, and core body temperature. Increased renal and hepatic blood flow, along with increased activity of some drug-metabolizing enzymes, may enhance the clearance of certain drugs, so that higher doses are needed.¹¹

The altered concentration of plasma proteins after large burn injuries can affect drug response because many anesthetics are highly protein-bound. Drug effects and elimination are related to the unbound fraction that is available for glomerular filtration, receptor interaction, or enzymatic metabolism.¹¹ Albumin and α₁-glycoprotein, the 2 major drug-binding proteins in the plasma, are affected in opposite ways by thermal injury. Albumin binds mostly acidic and neutral drugs (eg, diazepam and thioental), and its concentration is decreased after injury. α₁-Acid glycoprotein binds alkaline drugs (eg, propanolol, lidocaine, and imipramine) and doubles in concentration after a burn incident. Hence, changes in drug binding, response, and clearance depend on which protein has the higher affinity for the drug involved.⁴⁴

Clearance, the most significant determinant of the dose required for maintenance of anesthesia, can influence the patient’s response to drugs administered by infusion or repeated bolus injection during anesthesia. Drug clearance is affected by the following 4 factors: metabolism, protein binding, renal excretion, and novel excretion pathways (eg, drug loss in wound exudate). These factors all undergo modification, often to an extent requiring an adjustment in drug dosage. Such complex changes make it difficult to establish protocols for most drugs administered to burn patients. The key principle of effective drug therapy for these patients is to monitor the response and titrate the dose to effect.¹¹

The administration of muscle relaxants to burn patients is the component of their anesthetic management with the most profound and clinically significant effects. Fortunately, the altered responses to muscle relaxants are more easily predicted than those to other classes of drugs. Patients with large burn injuries exhibit an increased sensitivity to succinylcholine and an exaggerated hyperkalemic response—which can induce cardiac arrest; thus, according to various researchers, succinylcholine should not be given to burn

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![Figure. Mediator responses to burn injury.](image)

TNF, tumor necrosis factor

Adapted from MacLennan et al.¹⁹
patients for the period from 24 hours to 21 days after the injury. In the absence of solid evidence for a safe period of succinylcholine use, many clinicians avoid administering it to acute burn patients whenever possible. In contrast to the exaggerated response to succinylcholine, a marked tolerance to most nondepolarizing muscle relaxants is characteristic of burn patients, and larger doses are required to achieve the desired effects. The only exception is mivacurium, which retains its efficacy at standard doses in burn patients.

References

8. All of the following are characteristics of the systemic inflammatory response syndrome in burn patients, except:
10. Monafo WW. Initial management of burns.