Preanesthetic Assessment of the Patient With Burn Injuries

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PROFESSIONAL GAPS:
Management of burn patients is a problem frequently encountered by anesthesiologists. While fluid resuscitation protocols have been available for decades, more recent evidence has indicated complications with massive fluid overload. Many anesthesiologists are not aware of several mechanisms that contribute to pain in these patients. An understanding of this pathophysiology aids in selecting appropriate analgesics.

TARGET AUDIENCE
Anesthesiologists

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LEARNING OBJECTIVES
At the completion of this activity, the reader should be able to:
1. Define the types of burn injuries
2. Understand the pathophysiology of burn injury
3. Describe the mechanism of burn pain
4. Classify burn injury severity
5. Discuss the preoperative systemic changes with burn injuries
6. Recognize the importance of airway assessment and its management
7. Calculate fluid resuscitation needs and list hemodynamic goals
8. Describe the different analgesic options for burn pain control
9. Identify opioid tolerance versus opioid-induced hyperalgesia
10. Manage opioid-induced hyperalgesia

PREANESTHETIC ASSESSMENT
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CASE

A 31-year-old man with unknown past medical conditions was admitted after sustaining a 70% body surface area (BSA) burn from a flaming fire. The burned area included head, torso, bilateral upper extremities, left lower extremity, and medial portion of the right lower extremity. His trachea was intubated in the field and invasive lines were placed in the intensive care unit (ICU). Fluid resuscitation was given as per protocol along with midazolam 1 mg per hour and fentanyl 1 mcg/kg per hour infusions, which were started for sedation and analgesia, respectively.

After 3 days, the patient was scheduled for debridement of his anterior and posterior torso with split-thickness skin grafts. Before coming to the operating room (OR), the patient developed hypotension, for which he received 1 L of normal saline, 2 units of packed red blood cells (RBCs), and 500 mL of albumin 5%. Albumin infusion was then started at 100 mL per hour. Vasopressin infusion was administered at a rate of 0.04 units per minute. His vital signs were as follows: blood pressure (BP), 77-89/39-50 mm Hg; pulse, 130 bpm; arterial oxygen saturation, 99%; respiratory rate (RR), 20 breaths per minute; central venous pressure, 4-7 mm Hg; and body temperature, 38.4°C. His lungs were mechanically ventilated with the following parameters: tidal volume, 600 mL/kg; RR, 20; positive end-expiratory pressure (PEEP), 5 cm H₂O; and fraction of inspired oxygen, 0.4%.

Introduction

Each year, burn injuries affect approximately a half-million people in the United States; and of these, up to 40,000 people require hospitalization.¹ Over the past 30 years, the mortality rate associated with severe burn injuries has been drastically reduced, mostly by improved access to emergency medical care, targeted resuscitation, and advanced ventilation techniques. In addition, a better understanding of the pathophysiology of inhalation injury, infection control, nutritional support, early excision and grafting, and focus on the hypermetabolic response, have all contributed to improved care of burn injuries.¹

Mechanism and Pathophysiology

The initial instantaneous pain with a burn injury results from stimulation of skin nociceptors such as thermoreceptors that respond to heat, mechanoreceptors that respond to mechanical distortion, and chemical stimulants, both exogenous—such as hydrofluoric acid—and endogenous, such as inflammatory mediators.² These inflammatory mediators include histamine, prostaglandins, thromboxane, bradykinin, serotonin, catecholamines, platelet aggregation factor, angiotensin II, vasopressin, oxygen radicals, and corticotropin-releasing factor. They cause surrounding tissue hypoperfusion and capillary vasoconstriction that leads to disruption of deep skin structures.² As a result, these local responses cause systemic changes.

Furthermore, nerve endings that are not damaged but are exposed generate pain. These immediate pain sensations are elicited by unmyelinated C and thinly myelinated A delta primary afferent neurons that synapse with the neurons in the dorsal horn of the spinal cord.²

The characteristics of primary and secondary hyperalgesia differ. In primary hyperalgesia, there is hyperalgesia to both mechanical and heat stimuli, whereas in secondary hyperalgesia, there is hyperalgesia to mechanical but not heat stimuli. This dichotomy suggests that the neural mechanisms of primary and secondary hyperalgesia differ. In primary hyperalgesia, there is an inflammatory response that releases inflammatory mediators that sensitize the active nociceptors, which causes the skin to become sensitive to mechanical stimuli, such as touching, rubbing, and heat. In secondary hyperalgesia, the repeated peripheral stimulation of nociceptive afferent fibers renders the surrounding unburned areas sensitive to mechanical stimuli but not heat.²

Burn Severity Classifications

Size, depth, and location of burn wounds are the 3 key determinants of their overall severity. First-degree or superficial burns are localized within the epidermis and heal quickly without scarring or pigmentation changes due to preservation of skin barrier functions (eg, sunburn). Second-degree or partial-thickness burns occur along the epidermis with varying extension to the dermis.³ There can be 2 types of second-degree burns: superficial partial and deep partial. Superficial partial-thickness burns pose little risk for scar formation but can be hyperemic, edematous with blistering, and are usually painful. Deep partial-thickness burns are associated more with scar formation but are less painful. Third-degree or full-thickness burns affect the epidermis with complete involvement of the dermis, leaving a waxy, white, or leathery appearance. There is usually no pain because of nerve destruction.³ As the regeneration capabilities are lost, wound healing can occur through peripheral granulation and contraction, leading to an increased incidence of infection and scar formation. As a result, surgical intervention is required for excision and grafting. Fourth-degree burns involve the epidermal and dermal layers and extend beyond the fascia to muscles, tendons, and bones.³

The severity of burn injuries can be further characterized as minor, moderate, and major. The estimation of dermal involvement is recorded as a percentage of total BSA (TBSA). Minor injuries involve superficial burns to less than 15% of TBSA. Moderate injuries involve 15% to 25% of TBSA in adults and 10% to 20% of TBSA in children.⁴ Major burn injuries that result in partial-thickness burns affect more than 25% of TBSA. In addition, major burn injuries include inhalation injury, electrical burns, and complicated burn injury in patients with comorbid conditions. They are also characterized as burns that result in functional or cosmetic impairment to the face, eyes, ears, hands, feet or perineum.
Preoperative Systemic Assessment and Management

The perioperative period begins with a complete history and full physical assessment of the patient. The burn injury should be classified according to percentage of TBSA involved based on the Lund-Browder chart (Figure); in addition, the depth of the burn wounds and presence of inhalation injury should be noted.

Respiratory Assessment

Further assessment of the pulmonary system is extremely important. The indications for immediate tracheal intubation for patients with major burn injury include burns affecting more than 40% of TBSA, signs of impending airway obstruction, prolonged transport time, and any evidence of smoke inhalation. Inhalation injury is more likely if the burn occurred in an enclosed space. It is important to frequently reevaluate the airway and respiratory status of the patient because of development of airway edema and complications that may arise from superimposed smoke inhalation, as well as onset of ventilator-associated pneumonia. Inhalation of smoke, flame, and gases can cause injury and irritation to the lungs via direct thermal injury, chemical irritation, and/or systemic toxicity. These situations can cause an inflammatory process that may lead to bronchospasm, bronchitis, impairment of ciliary function, distal airway obstruction, and atelectasis. Hypoventilation and loss of hypoxic pulmonary vasoconstriction result in pulmonary ventilation-perfusion mismatch.

Carbon monoxide (CO) exposure and cyanide poisoning are 2 common complications of smoke inhalation. CO shifts the oxygen dissociation curve to the left. Patients can present with nausea, headache, and confusion. With CO levels greater than 55%, cardiac dysrhythmias and severe neurologic impairment become prominent. Clinical suspicion and use of CO oximetry to obtain accurate measurements are indicated. Cyanide impairs mitochondrial oxygen consumption, altering the normal cellular aerobic metabolism to an anaerobic state, producing lactic acidosis. At a level of 50 parts per million (ppm), patients present with dizziness, headache, tachypnea, and tachycardia. At 100 ppm, lethargy, seizures, and respiratory failure ensue.

Bronchoscopy is the main modality perioperatively that detects ongoing injury and the need to remove any obstructive debris, and obtain cultures. Patients with circumferential burns to the chest have a restrictive lung defect and may need emergent escharotomy. Cuffed endotracheal tubes support ventilation and oxygenation, guaranteeing high minute ventilation and PEEP. Aerosolized albuterol and epinephrine are the 2 pharmacologic treatments that help in bronchodilation and vasoconstriction, while reducing inflammation.

Cardiovascular Assessment

Major cardiovascular changes can occur in patients with significant burn injuries. Patients are initially hypovolemic owing secondarily to extensive fluid redistribution from increased vascular permeability and hypoproteinemia. Physiologically, there is slowed isovolemic relaxation, impaired contractility, and decreased diastolic compliance of the left ventricle, manifested mainly by a decrease in cardiac output. Decreased myocardial contractility is possibly related to release of tumor necrosis factor-alpha. These changes, coupled with fluid loss from the burn wound, result in systemic hypotension and end-organ hypoperfusion. Therefore, compensatory increments in heart rate and peripheral vascular resistance are crucial as well as maintenance of baseline hemodynamic parameters and loading conditions.

Renal Assessment

With burn injuries, kidney function may be affected by hypotension and low cardiac output. Acute renal dysfunction results from hypoperfusion, intravascular depletion, renovasoconstriction, myoglobin-induced tubular damage, and mechanical obstruction secondary to cast formation from hemolysis. Nephrotoxins (ie, antibiotics) should be avoided to prevent further renal dysfunction.

Figure. Lund-Browder Chart.
A tool useful in the management of burns to estimate the total body surface area affected.
Gastrointestinal Assessment

Gastrointestinal (GI) dysfunction is common in burn patients, resulting in complications such as acute colonic pseudo-obstruction (ACPO), sepsis, and abdominal compartment syndrome (ACS). Management of GI function in the setting of burns is complex and challenging. Moreover, there are no current generally accepted guidelines that direct management and improve outcomes.

Factors such as opioid analgesia, electrolyte imbalances (including hypokalemia), prolonged immobility, abdominal trauma, sepsis, and surgery, which can result in reduced GI motility and constipation, all contribute to complications in the care of burn patients. ACPO should be considered in the assessment of burn patients with constipation. A diagnosis of ACPO is made in the presence of abdominal distention with large bowel dilatation on abdominal x-ray. Management of ACPO is initially conservative with supportive measures such as nasogastric suction, discontinuation of opioids and correction of electrolyte imbalances.9

Moreover, bacterial translocation occurs as a result of mucosal barrier dysfunction and may lead to sepsis. The role of bacterial translocation as the cause of sepsis and multiorgan failure in major burns is controversial. There is strong evidence that selective digestive tract decontamination reduces mortality and infectious episodes in major burns.9

Burn patients with ACS develop features including increased intra-abdominal pressure, sepsis, oliguria, hyperventilation, and hypotension. Patients with large burn areas (>40% of TBSA) have an increased risk for developing ACS. This is thought to arise from a burn-related systemic inflammatory response proportional to the burn size and to the large fluid volumes used in resuscitation. The high morbidity and mortality associated with ACS highlights the importance of prevention, early diagnosis, and treatment. A recent systematic review by Strang et al reported a prevalence of 64.7% to 74.5% and a mean mortality rate of 74.8%.10

Patients identified as being at risk for developing ACS should have intra-abdominal pressure monitoring. Fluid resuscitation should be precisely tailored in order to avoid overload and development of intra-abdominal hypertension. Intra-abdominal hypertension (>12 mm Hg) must be diagnosed early to avoid progression to ACS.

Recent guidelines recommended a stepwise approach to manage intra-abdominal hypertension or ACS. The first line and mainstay of treatment is reduction in fluid resuscitation and diuresis to reduce intestinal edema. Goal-directed fluid resuscitation using colloids or hypertonic saline has been advocated as a possible means of achieving reductions in infused fluid volumes. Other noninvasive measures that can be used include inotropic support to increase cardiac output, positive pressure ventilation to improve oxygenation and ventilation, nasogastric decompression, rectal decompression, sedation, analgesia, and neuromuscular blockade.9

Hepatic Assessment

Hepatic dysfunction is a key contributor to mortality in burn patients and its development should be closely monitored. The extent of hepatocellular damage is influenced by hepatic blood flow that is compromised in the early burn phase secondary to blood loss, large-volume fluid shifts, and vasodilation. The duration and severity of hepatic dysfunction depends on the ability to recover hepatic function. Overall, recovery involves proper wound closure, infection control and nutritional support.9 As a physiologic response to burn injuries, stress hormone release (ie, glucagon and glucocorticoids) and insulin resistance are common. As a result of these changes, hyperglycemia may occur; thus, serum glucose should be controlled perioperatively to improve wound healing and decrease infection risk. Electrolytes should be monitored and replaced, as hyponatremia and hypophosphatemia are common during the early post-burn period.9

Fluid Management

Edema, a high-protein fluid, produced after a burn contributes significantly to burn wound depth conversion. Without fluid resuscitation, this volume peaks on day 1 and on day 2 after fluids are given.11 As the loss of intravascular fluids can be catastrophic, fluid resuscitation is vital. However, excessive crystalloids can further contribute to edema formation. The Parkland formula (used for patients aged 50 years or older) has been used for initial post-burn resuscitation using isotonic crystalloid based on body weight and burn size (4 mL/kg/%TBSA burn). For the first 8 hours, one-half of the volume should be used, with the second half given over the next 16 hours.7 Following this, maintenance volume should be continued with the addition of colloid boluses as needed in the second 24 hours to keep urine output at least 0.5 mL/kg per hour for adults and 1 cc/kg per hour for pediatric patients. However, as noted above, excessive fluids cause ACS and increased mortality.

The National Institutes of Health workshop on fluid resuscitation for burn patients agreed on 2 important guidelines: 1) the least amount of fluid necessary to maintain adequate organ perfusion should be given, and 2), the volume infused should be continually titrated to avoid both under- and over-resuscitation. Titration of fluids to maintain renal perfusion for urinary output of 0.5 mL/kg per hour is considered adequate for adults, and 1 cc/kg per hour is an appropriate target for pediatric patients.8
Colloids and albumin have been used both as volume expanders and to reverse hypoproteinemia. Studies suggest that albumin during acute resuscitation achieves adequate goals for total overall volume requirement, with maintenance of intravascular volume and cardiac output along with decreased overall edema. Extravascular lung water may increase. In another study of 46 children with significant burns, early albumin infusion, compared with use of the standard Parkland formula, reduced the need for crystalloid infusion with fewer cases of fluid creep and shorter hospital stays. Furthermore, a meta-analysis suggested that albumin can improve outcomes during burn shock resuscitation.

**Intraoperative Management**

**Airway**

Intraoperative management starts with assessment of the airway for tracheal intubation. During the acute post-injury phase, oxygen demand is increased, which can be complicated by edema of the face and upper airway. During the recovery phase, face and neck contractures present an important challenge for airway management. Therefore, a key assessment is to determine the patency and soft tissue compliance of the airway. It is important to palpate the neck and submandibular space to identify any tightness that might limit displacement of the tongue and soft tissues into the submandibular area, thus making laryngoscopy a challenge. Laryngoscopy can be useful in assessing tongue mobility and visualizing the pharynx. A flexible fiber-optic scope can be used to assess the epiglottis and surrounding tissues if patency of the hypopharynx is unclear. If the perioperative examination reveals a concern for upper airway patency, such as severe tightness of the lips or intraoral and/or pharyngeal tissue swelling, then an awake fiber-optic tracheal intubation is indicated as long as the patient is cooperative. If the patient is uncooperative, then a gentle IV or inhalational induction that preserves spontaneous ventilation helps in the advancement of the fiber-optic scope. If the airway exam reveals moderate tightness of the lips, intraoral or pharyngeal tissue swelling, and/or if there is moderate restriction due to contractures, then a gentle post-induction test of mask ventilation is indicated before administering a neuromuscular blocker. Laryngoscopy or video laryngoscopy can be used for both intubating and diagnostic purposes in these circumstances. Patients who are severely burned may require tracheostomy for long-term mechanical ventilation. The indications for tracheostomy in burn patients after 10 days of hospitalization include failure to wean off the ventilator, failed extubation, and/or expected prolonged mechanical ventilation.

**Ventilation**

Intraoperative ventilation of burn patients can be difficult due to increased airway reactivity, retained secretions, acute lung injury, and acute respiratory distress syndrome (ARDS). ARDS occurs commonly in burn patients, so it is important to maintain plateau pressures lower than 35 cm H₂O while using sufficient positive end-expiratory airway pressure during mechanical ventilation. Low tidal volume with permissive hypercapnia is important in the care of ARDS patients.

Bronchospasm is also frequently seen in burn patients, and aggressive bronchodilator therapy with beta-2 agonists (eg, albuterol) is recommended. Furthermore, an alveolar recruitment maneuver (ARM), defined as the reopening of collapsed lung tissue by increased airway inspiratory pressure, may be helpful for re-expansion of atelectasis and oxygenation. The most common approach for a recruitment maneuver is sustained inflation by continuous positive airway pressure, with pressures varying from 30 to 40 cm H₂O for 30 to 90 seconds.

**Intravenous Access and Monitoring**

Vascular access may be challenging because of difficult direct visualization of the burned skin, which may be broken, bleeding, and/or scarred. Thus, placing vascular access in any intact skin, if possible, is important. Moreover, ultrasound guidance to obtain vascular access is helpful. It is preferable to use large-bore IV access in a patient with severe burns as access for rapid volume replacement. Interosseous access may be needed in patients with restricted access. Ultimately, central cannulation is more often necessary for secured vascular access to allow rapid resuscitation. However, there is a risk for increased complications or infections due to the pathophysiologic destruction of the glycocalyx (ie, the sugar-protein that coats all healthy vascular endothelium and is an integral part of the vascular barrier). Thus, extra caution adhering to full sterile technique should be used for all central-access cannulation.

Monitors may be difficult to place or adhere to the patient’s skin due to burn wounds (and associated ointments used). Electrocardiographic monitoring may require needle electrode insertion. Invasive BP monitoring is usually needed for continuous measurements.

**Temperature**

Burn patients can lose up to 1°C every 15 minutes; thus, intraoperative measurement of body temperature is crucial. Maintaining normothermia in burn patients is challenging, especially because neutral temperature for burn patients is reset by the hypothalamus to 38.5°C. The pathophysiology of the hyperthermic response in major burn injury is poorly understood. It could be secondary to an infective etiology or a metabolic response to systemic inflammation. Regardless of the reason, sustained hyperthermia above 40°C can culminate in cellular injury and death.

Some methods that can be used to minimize intraoperative heat loss include increasing the temperature in the OR to higher than 35°C, use of convective warming devices, warmed IV fluids, minimizing skin surface exposure, and covering nonoperative extremities in plastic wrap to prevent radiative heat loss. Temperature can be monitored via either an esophageal or rectal approach for core body temperature assessment.

**Medications**

Induction of anesthesia can be achieved by a multimodal approach. Propofol is a common induction agent that can be slowly titrated as
needed during induction. For neuromuscular relaxation, succinylcholine can be used for tracheal intubation; however, it should be avoided within 24 hours of a burn given its increased risk for hyperkalemia and dysrhythmias. Nondepolarizing neuromuscular agents are good alternatives for intubation and maintenance without the risk for hyperkalemia.

Opioids are the mainstay of pain control intraoperatively. However, other nonopioid adjuvants can be used if appropriate, including low-dose ketamine infusion, clonidine, local anesthetic infusion, or dexmedetomidine. Moreover, ketamine is a good sedative analgesic because it maintains airway reflexes along with heart rate and BP. It can be used for hemodynamically compromised adults and pediatric patients, but care must be taken in those who already function at maximum sympathetic response because sudden cardiovascular decompensation may result after administering ketamine from the effect of sympathetic response reduction. Ketamine also has advantages as a continuous infusion at subanesthetic or anesthetic doses when used for surgical procedures (and in the ICU) because it reduces opioid consumption. Additionally, it can be effective as an adjuvant medication for pain that is poorly controlled by opioids.

There is no single best anesthetic agent for maintenance of general anesthesia. Nitrous oxide is a better inhaled agent, as it has less effect on BP. Overall, a multimodal approach is the most important technique, taking into consideration the severity of burns, hemodynamic values, and duration of opioid use prior to surgery.

Postoperative Management

Pain control is imperative in burn patients and can be divided into four different categories: rest pain, breakthrough pain, procedural pain, and psychogenic pain. Opioids, due to their potency, are the mainstay and include morphine, hydromorphone, and fentanyl. Morphine is the most commonly used agent, but rapid tolerance is an undesired consequence. Longer-acting opioids such as methadone, ketamine, dextromethorphan, and clonidine can be used to reverse the acute tolerance effects of opioids. In fact, it has been shown that N-methyl-D-aspartate (or NMDA) receptor antagonists, like ketamine, have a preventive role in developing morphine tolerance. This specifically is applicable to burn patients who are commonly treated with fentanyl as a first-line agent and then morphine for breakthrough pain. When subsequent doses of morphine are given after large doses of fentanyl, tolerance to morphine’s analgesic effect develops. In these situations, a single dose of ketamine (ie, at subanalggesic doses) has been shown to prevent acute morphine tolerance. After the use of opioid infusions, ketamine can attenuate the development of acute tolerance to analgesia. Overall, ketamine has the potential to reduce opioid consumption even in subanalggesic doses.

Methadone has much better efficacy in burn patients who are opioid tolerant and have developed chronic neuropathic pain, because of its bioavailability and longer duration of action. Fentanyl can be used as an adjunct for procedural burn care activities, as it provides better pain control by rapid onset of action and quick redistribution. Regional anesthesia is ideal for decreasing postoperative pain, particularly in reconstructive procedures at skin graft donor sites. The technique also helps to decrease total opioid use. The overall goal is to control postoperative and long-term burn pain with a multimodal approach to prevent opioid tolerance or opioid-induced hyperalgesia (OIH).

Burn patients can require long hospital stays and should be reasessed daily for tracheal extubation, especially in patients who have received large doses of opioids or benzodiazepines and intraoperative fluid volumes. The main treatment priorities during this period are pain management and procedural sedation. Depression must also be addressed. Attempting to wean patients off high-dose infusions of opioids and benzodiazepines poses a key challenge postoperatively, as opioid tolerance and OIH may develop, with the latter more difficult to diagnose.

Opioid Tolerance Versus Opioid-Induced Hyperalgesia

Typically, opioid tolerance to pain is unchanged from original pain and improves as opioid doses are increased and worsens with decreased

| Table. Comparison of Features of Opioid Tolerance and Opioid-Induced Hyperalgesia |
|---------------------------------|---------------------------------|-------------------------|
| **Features**                    | **Opioid Tolerance**            | **Opioid-Induced Hyperalgesia** |
| Pain characteristics           | Typically unchanged from original pain | More diffuse, other locations, whole body allodynia, hyperalgesia |
| Pain sensitivity               | Unchanged                       | Decreased               |
| Expected response to increase in opioid dose | Pain improves | Pain worsens |
| Expected response to decrease in opioid dose | Pain worsens | Pain improves |

Adapted from reference 18.
Management of the Case

After placement of standard monitors and instituting controlled ventilation, a low-dose ketamine infusion was started at a rate of 0.15 mg/kg per hour for analgesia. The patient could not tolerate even small doses of inhalation agents due to hypotension, so additional midazolam boluses were given to provide amnesia. Rocuronium was used for paralysis, and albumin 5% and vasopressin infusions were continued. Two units of packed RBCs were given to compensate for blood loss during debridement. Ketamine controlled pain well and fentanyl was not needed. Throughout the procedure, BP improved to 110-120/60s mm Hg and heart rate decreased to 110s bpm.

After conclusion of the procedure, the patient was transferred, still intubated, to the ICU and the low-dose ketamine infusion was continued in the postoperative period. The ketamine infusion was continued for 3 to 4 days postoperatively as an adjuvant analgesic agent. Finally, the patient’s trachea was extubated and further care was managed by the primary intensive care team.

Conclusion

Managing burn patients poses many key challenges to the anesthesiologist in the acute and intraoperative care periods. It is important to first understand the mechanisms and characteristics of burn pain, which can then be applied to provide proper perioperative care. Perioperative care requires a full assessment of the patient systematically, with key attention to airway anatomy and resuscitation. Anticipation of hemodynamic and airway management, as well as the key role of pain management, are vital in guiding surgical care and decreasing morbidity and mortality in this patient population.

References


Post-Test:

1. Which of the following is not an indication for immediate tracheal intubation?
   a. Burns of total body surface area (TBSA) of less than 20%
   b. Signs of impending airway obstruction
   c. Prolonged transport time
   d. Any evidence of smoke inhalation injury

2. Burn patients are initially hypovolemic due to extensive redistribution of fluid and ________.
   a. liver failure
   b. acute kidney injury
   c. hypoproteinemia
   d. hypotension

3. The key assessment to make before managing the airway of a burn victim is to determine the patency and ________ compliance of the airway.
   a. tracheal
   b. pharyngeal
   c. soft tissue
   d. intraoral

4. Which of the following is least likely to contribute to difficult intraoperative ventilation in burn patients?
   a. Acute respiratory distress syndrome (ARDS)
   b. Retained secretions
   c. Decreased airway reactivity
   d. Acute lung injury

5. Which of the following is a sedative that best maintains airway reflexes and hemodynamic stability?
   a. Succinylcholine
   b. Ketamine
   c. Fentanyl
   d. Nitrous oxide

6. Which of the following is true of opioid-induced hyperalgesia?
   a. Pain improves with increased opioid dose.
   b. Pain is produced in one location and is less diffuse.
   c. Pain response usually worsens with increased opioid dose.
   d. Pain sensitivity increases.

7. All of the following will increase in the immediate post-burn period except:
   a. Fluid requirements
   b. Systemic vascular resistance
   c. Cardiac output
   d. Vascular permeability

8. Major burns can result in all of the following except:
   a. Thrombocytopenia
   b. Polycythemia
   c. Disseminated intravascular coagulation
   d. Septicemia

9. Which of the following is contraindicated 24 to 48 hours after a burn?
   a. Rocuronium
   b. Propofol
   c. Ketamine
   d. Succinylcholine

10. What is the percentage of TBSA in an adult patient with burns to anterior surfaces of the right upper and lower extremities?
    a. 4.5%
    b. 13.5%
    c. 18%
    d. 22.5%