Anesthesia for burn patients

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INTRODUCTION — Anesthesia providers may be involved in the management of burn patients throughout the perioperative period, including preoperative airway management, intraoperative anesthetic care, postoperative intensive care, and management of postoperative pain. This topic reviews these aspects of the anesthetic management of burn patients. Other issues in the management of burn patients are reviewed separately. (See "Overview of the management of the severely burned patient" and "Emergency care of moderate and severe thermal burns in adults" and "Treatment of minor thermal burns").

PREANESTHESIA CONSULTATION: KEY CONSIDERATIONS

Burn injury

**Burn extent and severity** — An estimate of the percent total body surface area (TBSA) burned and the location and severity of the burn is essential since these factors affect many aspects of anesthetic care, including airway management, fluid management, drug dosing, and temperature regulation. Calculation of TBSA is based on the Lund-Browder chart (figure 1). A major burn injury is a partial-thickness burn greater than 25 percent TBSA in adults, or greater than 20 percent in children younger than 10 years old or adults older than 40 years old (table 1). Major burn injury is also defined as full-thickness burns involving more than 10 percent TBSA. High-voltage burns, smoke inhalation injury, and burns to the face, eyes, ears, hands, feet, or perineum are also defined as major burns. Major burns may cause an intense physiologic and inflammatory response that has a direct impact on the subsequent hospital course. (See "Overview of the management of the severely burned patient" and "Classification of burns").

**Fluid resuscitation** — The first 48 hours after the thermal insult is the "resuscitative phase" or "ebb phase" of the burn injury. A systemic inflammatory response occurs during this period, with loss of plasma fluid, electrolytes, and proteins due to increased capillary permeability [1-3]. Effective fluid resuscitation is guided by formulas (eg, Parkland formula) targeted to achieve the American Burn Association (ABA)-recommended urinary output of 0.5 to 1 mL/kg per hour as the resuscitative endpoint [4]. Excessive intravenous fluids administration (eg, "fluid creep") can put the patient at risk of developing pulmonary edema, abdominal compartment syndrome, or compromised circulation of extremities. (See "Emergency care of moderate and severe thermal burns in adults", section on "Fluid resuscitation").

**Cardiovascular** — Cardiac output (CO) is low, in an "ebb" state, during the resuscitative phase immediately following a severe burn [3,5] (see "Complications and long-term outcomes of a severe burn", section on "Cardiac failure"). CO is reduced up to 60 percent from baseline due to hypovolemia from permeability-induced plasma loss, reduced myocardial response to catecholamines, myocardial ischemia due to decreased coronary blood flow, and increased systemic vascular resistance due to elevated vasopressin levels [6,7]. Initial under-resuscitation with fluid therapy, followed by over-resuscitation, leads to pulmonary edema and right heart failure.

At 72 to 96 hours postburn, during the recovery phase, cardiovascular changes evolve into a "flow" state. The hyperdynamic and hypermetabolic responses during this phase include increased CO, tachycardia, increased myocardial oxygen consumption, and decreased systemic vascular resistance [3].
Prevention of inadequate end-organ perfusion in burn-induced cardiogenic shock may involve invasive monitoring (eg, direct intraarterial blood pressure or central venous pressure monitoring) or goal-directed fluid resuscitation to maintain optimal hemodynamics [8]. Also, we use respiratory variation in the arterial waveform as a guide to volume and vasoactive therapy [3]. (See 'Monitoring' below and "Intraoperative fluid management", section on 'Goal-directed fluid therapy' and "Complications and long-term outcomes of a severe burn").

A nonselective beta blocker to block catecholamine receptors is used to treat cardiac dysfunction in the ebb state, as well as the hyperdynamic response occurring in the flow state [9,10]. (See "Complications and long-term outcomes of a severe burn", section on 'Cardiac failure'.)

**Pulmonary** — Immediately after a burn injury, systemic inflammatory processes lead to pulmonary hypertension and disruption of the pulmonary capillary alveolar membrane. This occurs in a setting of hypoproteinemia and reduced plasma oncotic pressure due to aggressive crystalloid resuscitation (see 'Fluid resuscitation' above). Thus, patients often have increased extravascular lung water and impaired gas exchange. Also, bronchospasm is common and may require aggressive bronchodilator therapy [11].

**Smoke inhalation** — Frequently, burn patients present with smoke inhalation, particularly if the fire was in a closed space [3]. Singing of facial or nasal hair, evidence of oropharyngeal carbonaceous deposits, or carboxyhemoglobin (COHb) levels >10 percent are highly predictive of inhalation injury confirmed by bronchoscopy. Patients with burn injury and concomitant smoke inhalation have increased fluid resuscitation requirements and a higher likelihood of pulmonary complications and mortality, compared with those having isolated burn injury [12,13].

The inflammatory response produced by chemicals in the smoke induces bronchospasm, ciliary dysfunction, increased transvascular fluid flux (pulmonary edema), and thickening of secretions with concomitant atelectasis. These changes result in hypoventilation, loss of hypoxic pulmonary vasoconstriction, and increased pulmonary shunting with ventilation-perfusion mismatch [14]. Systemic and respiratory effects of chemicals in smoke (carbon monoxide, cyanide) are discussed in detail elsewhere. (See "Emergency care of moderate and severe thermal burns in adults" and "Smoke inhalation").

**Late pulmonary sequelae** — Pulmonary failure due to decreased oxygenation may occur either immediately after burn injury, or a few days later, due to progression of the inhalation injury and/or development of acute respiratory distress syndrome (ARDS) [3,15]. (See "Complications and long-term outcomes of a severe burn", section on 'Pulmonary failure').

A restrictive lung defect with impaired ventilation can result from severe chest wall edema due to circumferential burns to the chest wall or from scar contraction after excision and grafting of the chest and abdomen [3]. Patients may develop hypoventilation and respiratory failure due to loss of chest wall elasticity and decrease in effective lung volumes. Intubation and mechanical ventilation and/or chest wall escharotomies may be necessary, depending on the degree of compromise [16].

**Metabolic and nutritional** — Blood glucose should be monitored throughout the perioperative period in burn patients because of their peripheral insulin resistance, elevated protein and lipid catabolism, increased energy expenditure, and stimulated synthesis of acute phase proteins.

Burn patients require aggressive nutritional support, often with enteral tube feedings, due to elevated catabolism. In contrast to most surgical patients, in whom enteral nutrition is withheld before and during surgery, nutritional supplementation (enteral, parenteral) can be continued throughout the perioperative period in burn patients to improve long-term outcomes, including reduction in caloric deficit, need for exogenous albumin supplementation, and incidence of wound infections [3,17,18]. This decision is made on a case-by-case basis.

The hypermetabolic response to a burn injury can persist for three years following a severe burn. (See "Hypermetabolic
Pharmacology — Burn patients have altered pharmacokinetics and pharmacodynamics for many medications, including commonly used anesthetics, analgesics, and muscle relaxants [3,19-21] (see 'Induction of general anesthesia' below and 'Maintenance of general anesthesia' below). Causes include changes in plasma protein concentrations, alterations in specific drug receptors, and cardiovascular factors. In particular, reduction in plasma albumin levels increases the volume of distribution and the available free fraction of any medication that binds to albumin (e.g., benzodiazepines). Albumin levels are decreased because of plasma protein loss through endovascular “leaking,” as well as protein dilution resulting from crystalloid resuscitation (see 'Fluid resuscitation' above). In severely burned patients, shock-induced decreases in cerebral, hepatic, and renal blood flow will affect drug pharmacology [22].

ANESTHETIC MANAGEMENT — Burn patients undergo multiple surgical procedures at the hospital bedside and in the operating room that require anesthetic management. (See "Emergency care of moderate and severe thermal burns in adults" and "Principles of burn reconstruction: Overview of surgical procedures".)

Early excision and wound coverage are crucial aspects of the treatment of thermally injured patients. The burn wound is excised either at a dermal level (e.g., tangential excision, for partial-thickness burns) or deep to the subcutaneous tissue (e.g., fascial excision, for full-thickness burns), depending on the depth of the burn. Then, the excised areas are covered, ideally with split-skin autografts. When there is insufficient donor skin, other coverage is used (e.g., allografts, xenografts, synthetic skin products, and cultured epithelial autografts) [23,24]. Blood loss during burn excision operations can be extensive [3]. (See 'Intraoperative blood transfusion' below.)

Burn dressing changes are painful procedures that usually occur in the hospital bed. Depending on the complexity of the wound, the age of the patient, and the anticipated procedural pain intensity, these patients may require moderate to deep sedation, and occasionally general anesthesia, outside of the operating room setting.

Small burns (<5 percent total body surface area [TBSA]) that do not involve joints or vital organs can generally be treated on an outpatient basis. (See "Treatment of minor thermal burns".)

Further details regarding burn reconstruction procedures are reviewed elsewhere. (See "Principles of burn reconstruction: Extremities and regional nodal basins" and "Principles of burn reconstruction: Face, scalp, and neck" and "Principles of burn reconstruction: The female breast" and "Principles of burn reconstruction: Perineum and genitalia".)

Monitoring — Standard American Society of Anesthesiologists (ASA) monitoring is recommended in all patients receiving procedural sedation, or regional or general anesthesia [25]. However, the burn patient may present challenges in the use of standard monitors. For example, burns on the torso and upper extremities prevent adhesive electrocardiogram lead placement. In these cases, needle electrodes or surgical stapling of the electrodes may be necessary. Available sites for pulse oximeter probe application may be limited in patients with extensive burns to the face, neck, extremities, and/or planned skin graft harvests in unburned extremities. Thus, probes for multiple sites should be immediately available. Also, carboxy-hemoglobinemia in patients with smoke inhalation and carbon monoxide poisoning may falsely elevate pulse oximetry values. In these cases, a co-oximeter may be necessary to obtain an accurate level of oxyhemoglobin. (See "Pulse oximetry", section on 'Abnormal hemoglobins' and "Carbon monoxide poisoning", section on 'Diagnosis'.)

Placement of a noninvasive blood pressure cuff may not be possible in a patient with significant edema or generalized burns to the extremities. In these cases, an intraarterial catheter may be necessary. When no other options are available, the catheter may be placed into burned or newly grafted skin. An intraarterial catheter provides moment-to-moment measurement of arterial blood pressure and allows arterial pressure waveform analysis to guide fluid therapy and calculate stroke volume [8]. (See "Intraoperative fluid management", section on 'Dynamic parameters'.)
The effectiveness of perfusion is monitored using a urinary catheter, and often a central venous catheter and/or cardiac output monitor, in the severely burned patient.

Transesophageal echocardiography (TEE) may provide valuable information regarding ventricular filling (ie, hypovolemia or fluid overload) and both left and right ventricular function [26]. Emergency use of intraoperative or perioperative TEE is indicated to determine the cause of any unexplained persistent or life-threatening hemodynamic instability (“rescue echo”) when equipment and expertise are available [27].

**Temperature management** — Hypothermia is prevented in the operating room by using convective warming devices (ie, forced air warmers), administering warmed intravenous fluids, maintaining ambient temperature ≥25°C, and minimizing the body surface area that is exposed.

Hypothermia has been associated with increased risk of coagulopathy, infection including sepsis, and mortality in surgical populations, including burn patients [3,28-34]. Burn patients are at high risk for perioperative hypothermia because of accelerated heat loss through injured skin, exacerbated by prolonged exposure of a large surface area to ambient temperature, as well as administration of cold intravenous fluids. Severely burned patients can lose up to 1°C every 15 minutes when exposed to ambient temperature.

**General anesthesia**

**Airway management** — Stridor, hoarseness, or dysphagia indicate impending airway obstruction and an urgent need for endotracheal intubation. Once massive edema of the airway has developed, emergency intubation may be extremely difficult. (See "Emergency care of moderate and severe thermal burns in adults", section on 'Airway management'.)

The patient should be evaluated for clinical evidence suggesting difficulty with laryngoscopy. Signs of probable inhalation injury and potential anatomic challenges for laryngoscopy due to oropharyngeal tissue injury or edema include burns around the mouth, deep facial or neck burns, singed hair in the nares, blistering or edema of the oropharynx, persistent cough or wheezing, carbonaceous sputum, or abnormalities in arterial blood gases (ie, hypoxia or hypercapnia). Ongoing fluid resuscitation may exacerbate laryngeal swelling. Mouth opening and neck mobility may be restricted due to pain, edema, burn eschars, or contractures. Tightness of the submandibular space can limit displacement of the tongue, making laryngoscopy difficult. A tongue depressor or laryngoscope blade may be used to evaluate the degree of mouth-opening and appearance of the oropharyngeal tissue.

Bag-mask ventilation of patients with burns to the face may be challenging due to burn dressings that prevent a good seal around the mask. It is advisable to use a two-person bag-mask ventilation technique in these situations. Removal of the burn dressing may be necessary, but extreme care to prevent further damage to the burnt area is necessary.

When upper airway patency is a concern, a preanesthetic fiberoptic evaluation may identify specific abnormalities and facilitate airway management planning (algorithm 1) [35]. Awake fiberoptic tracheal intubation is performed if patency of the upper airway appears to be compromised in a patient who can cooperate. (See "Flexible scope intubation for anesthesia".)

If the patient cannot cooperate, we attempt to preserve spontaneous ventilation (with or without bag-mask assistance) during induction of anesthesia (see "Emergency airway management in the adult with direct airway trauma" and "Management of the difficult airway for general anesthesia"). Preserving spontaneous ventilation ensures gas exchange and may facilitate advancement of the fiberoptic scope into the trachea. After intubation and induction of anesthesia, we test ability to ventilate before injecting a neuromuscular blocking agent. Succinylcholine may be used as part of a rapid sequence intubation during the first 48 hours following a severe burn, but we avoid succinylcholine administration after that because of the risk of severe hyperkalemia [3]. (See 'Neuromuscular blocking agents' below.)

Securing the endotracheal tube is challenging in patients with burns to the face. Techniques to achieve adequate fixation
of the tube include placement of interdental wires [36] or surgical sutures around the teeth to secure to the tube at a desired depth [37]. Circummandibular, nasomaxillary, or transseptal sutures can also be used to secure the endotracheal tube in patients with facial burns, achieving reliable tube fixation with minimal obstruction of the surgical field [38].

Whether to perform a tracheostomy in severely burned patients is controversial. The decision, including timing, is made on a patient-by-patient basis. (See "Complications and long-term outcomes of a severe burn", section on 'Pulmonary failure'.)

**Induction of general anesthesia** — All anesthetic induction agents should be administered cautiously (ie, slow administration of a reduced dose) during the initial resuscitative phase immediately following a severe burn injury because they may precipitate hypotension in the setting of intravascular volume depletion [39]. However, other factors may be more significant for development of hypotension (eg, burn severity or concurrent sepsis) [40].

**Induction agents** — Dosing and hemodynamic effects of induction agents depend on the phase of the burn injury [3].

- **Propofol** – During the resuscitative (ebb) phase immediately after burn injury, the induction dose of propofol is reduced to avoid hypotension due to further cardiovascular depression and reduction in systemic vascular resistance [39,41,42]. However, during the recovery (flow) phase three to four days after the injury, burn patients may require larger bolus doses or increased infusion rates of propofol compared with other patients in order to attain and maintain therapeutic plasma propofol concentrations [43,44].

  Propofol pharmacokinetics are determined by the interaction between cardiac output, hepatic clearance, and renal clearance, as well as protein binding [43]. Despite a burn-induced hepatic ischemic-reperfusion injury, hepatic clearance continues to account for the majority of total propofol clearance during this hyperdynamic flow state. Since reduction in albumin plasma levels increases the free propofol fraction, hepatic and renal clearance are increased. These pharmacokinetic changes result in plasma propofol concentrations that are lower for a given dosing regimen during the hyperdynamic recovery (flow) phase, possibly necessitating an increase in dose [44].

- **Etomidate** – In patients with cardiovascular compromise, induction of anesthesia with etomidate results in few hemodynamic changes, compared with propofol [39,40,42,45]. However, in the setting of hypovolemic shock (eg, the resuscitative phase of a burn injury), administration of etomidate or any other induction agent may result in hypotension.

  Also, a significant cautionary drawback to the use of etomidate is its association with transient acute adrenal insufficiency [46-49] and possibly increased risk of mortality in both critically ill patients [48] and those not critically ill [50]. However, this finding is not consistent [51], and there are no studies examining safety of etomidate specifically in burn patients.

- **Ketamine** – Anesthetic induction with ketamine usually results in significant increases in mean arterial pressure, heart rate, and plasma epinephrine levels due to centrally mediated sympathetic nervous system stimulation [42]. However, this stimulatory effect depends upon the presence of adequate sympathetic reserve. In patients who have maximally activated their sympathetic response (eg, burn patients with hypovolemic shock), administration of ketamine may result in hypotension due to its mild direct myocardial depressant effect [3].

  Beneficial effects of ketamine that are useful in burn patients include potent analgesic efficacy, bronchodilation, and maintenance of hemodynamic stability and airway reflexes [3,52]. Also, ketamine exhibits a complex interaction with opioid receptors that can benefit burn patients with opioid tolerance [53]. This interaction is mediated by N-methyl-D-aspartate (NMDA) receptor blockade, which reduces release of glutamate.

Ketamine is often administered to burn patients outside of the operating room during dressing changes,
hydrotherapy, and other procedures. Also, ketamine-based total intravenous anesthesia (TIVA) has been used to manage critically ill burn patients requiring specialized perioperative mechanical ventilation precluding use of an anesthesia machine and inhalation anesthetic agents [54].

Side effects of ketamine include nausea and vomiting, hallucinations, mood alteration, vivid dreams, and emergence delirium, which persist during anesthetic recovery.

**Neuromuscular blocking agents**

- **Succinylcholine** may be used as part of a rapid sequence intubation during the first 48 hours following a severe burn, but not thereafter for at least one year [3,55]. Administration of succinylcholine to burn patients after this time carries the risk of acute severe hyperkalemia and life-threatening arrhythmias [3]. This is due to upregulation of immature extrajunctional nicotinic acetylcholine receptor (nAChR) subunits caused by inflammation and local denervation of muscle [55,56]. Although there are no reports of hyperkalemia at or before 72 hours after a burn, upregulation of nAChR receptors has been demonstrated as soon as 24 hours after injury in the alpha-7 and gamma gene subunits [56], and risk is increased in burn patients with infection, sepsis, or immobilization [55].

- Resistance to all nondepolarizing neuromuscular blocking agents (NMBAs) develops in patients with burns >30 percent of TBSA after approximately one week and peaks five to six weeks after injury. Increasing the dose can partly overcome this resistance [21,57-60]. Also, recovery time from the block is shortened in burned patients.

This resistance to all nondepolarizing NMBAs is caused in part by the upregulation of nAChR subunits, as described above [55,56]. Additional reasons include increased acute phase alpha 1-glycoprotein, with decreased binding of NMBAs to free fraction glycoprotein [57]. Also, in the later hyperdynamic (flow) stage of the burn injury, enhanced renal elimination may occur because of decreased protein binding and increased glomerular filtration [21,58]. Resistance to atracurium and cisatracurium also occurs and is primarily due to the upregulation of nAChRs rather than altered protein binding (since these drugs are metabolized in the plasma through organ-independent Hofmann elimination) [59].

- **Opioids** — It is often prudent to reduce doses of opioids during the acute resuscitative (ebb) phase immediately after burn injury because metabolic clearance may be impaired by liver dysfunction [61].

In the subsequent hyperdynamic stage of the burn injury (flow phase), the requirement for opioids such as fentanyl is increased. In part, this is due to increased volume of distribution caused by protein dilution with decreased protein binding [3,20]. However, opioid tolerance, characterized by increasingly poor analgesic response to standard doses of analgesics, is another important factor that makes pain management challenging during all phases of burn care [62]. Tolerance may develop in burn patients after only two weeks of uninterrupted opioid use, particularly in patients with preexisting opioid abuse.

Furthermore, it is not uncommon for burn-injured patients to manifest an extreme opioid tolerance, with opioid requirements that far exceed usual textbook recommendations. This is due to opioid-induced hyperalgesia (OIH), an aberrant response to opioids that manifests as enhanced pain sensitivity. Unlike tolerance, which can be overcome by escalating the dose of opioid, OIH worsens with increasing doses of opioids, complicating the management of pain in burn patients.

As noted above, the use of NMDA receptor antagonists (eg, ketamine) may produce analgesic relief in patients with OIH [53,62,63]. (See "Burn pain: Principles of pharmacologic and nonpharmacologic management" and 'Induction agents' above.)

**Maintenance of general anesthesia** — Maintenance of anesthesia for burn surgery can be achieved effectively with
various techniques, including a “balanced” technique employing an inhalation anesthetic, opioid, and muscle relaxant, or a TIVA technique. A retrospective study comparing ketamine-based TIVA with inhalation anesthesia in patients undergoing debridement and grafting of burn injuries noted no differences between the techniques in use of vasopressors or in overall outcome [54].

Due to expense, we reserve use of TIVA for patients who require special ventilator parameters (eg, high-frequency ventilation). When we do select a TIVA technique, we prefer continuous infusions of propofol, ketamine, and an opioid (eg, fentanyl or remifentanil), in combination with muscle relaxant.

Doses of anesthetic agents required to maintain appropriate anesthetic depth depend on the pathophysiologic state of the patient. During the acute resuscitative (ebb) phase immediately after burn injury, we decrease the dose of intravenous and/or inhalational anesthetic agents, and we use benzodiazepines and/or ketamine as adjuvants to decrease risk of intraoperative awareness.

Regional anesthesia — The use of local and regional anesthesia techniques not only provides intraoperative anesthesia, but also may improve postoperative analgesia and decrease the requirement for opioids, thus promoting rehabilitation [3]. Regional anesthesia is a particularly useful technique for intraoperative excision of small and/or localized burns.

However, the volume of local anesthesia that can be infiltrated without reaching toxic doses is limited. The use of regional anesthesia as the sole anesthetic for surgery in burn patients is usually limited due to the location and size of the burn injury, use of multiple distant donor sites for skin harvesting, and/or associated painful traumatic injuries.

Donor sites are sometimes more painful than the grafted burn wound itself. Subcutaneous tumescent infiltration of local anesthetics for cutaneous surgery may provide adequate surgical anesthesia, with less blood loss from the graft donor sites [64-66]. In one feasibility study (n = 8 patients), postoperative pain was decreased by continuous infusion of subcutaneous bupivacaine into the donor sites [67].

Hemodynamic goals — The hemodynamic goals are to maintain end-organ perfusion and prevent burn shock [3]. Although the best endpoints for adequacy of resuscitation in major burn injury have not been conclusively determined, the American Burn Association (ABA) primarily recommends maintaining urine output at 0.5 mL/kg per hour for adults and 1 mL/kg per hour for children. (See "Overview of the management of the severely burned patient", section on 'Acute management in the ICU' and "Emergency care of moderate and severe thermal burns in children", section on 'Monitoring fluid status'.)

Intraoperative fluid management — Standard intraoperative fluid administration is adjusted for the magnitude of burn excision (large excisions incur more blood loss and require more volume replacement), the depth of burn (partial-thickness burn excisions involve more blood loss than full-thickness burn excisions), the specific hemostatic techniques employed (eg, topical epinephrine), and the surgeon's use of tumescent fluid administration.

If the patient undergoes surgical debridement or escharotomies during the resuscitative phase, replacement of intraoperative volume losses are added to formula-guided resuscitative fluid volume for burn patients (eg, the Parkland formula). (See "Emergency care of moderate and severe thermal burns in adults", section on 'Estimating initial fluid requirements'.) Important factors guiding fluid management include acuity of blood loss, hemoglobin measurements, development of hypoxemia, and clinical assessment of perfusion (urine output, cardiac output, base deficit, serum lactate).

Fluid repletion is carefully optimized to avoid under- or over-resuscitation, both of which may lead to further complications in the postoperative period. Although it may be helpful to use automated technology to obtain dynamic indices of fluid responsiveness (eg, cardiac output, stroke volume variation, pulse pressure variation, or systolic pressure variation), further study is needed before widespread implementation can be recommended in burn patients. (See
Intraoperative blood transfusion — Intraoperative blood loss during burn surgery can be large. For each 1 percent of burn wound excised or autograft harvested, it is estimated that 2.6 percent of an adult patient's total blood volume is lost (3.4 percent in children) [68,69].

When intraoperative transfusion is necessary in a hemodynamically stable patient, we use a restrictive transfusion threshold (hemoglobin approximately 7 g/dL [hematocrit approximately 21 percent]), with a ratio of 1:1 for transfusion of red blood cells (RBCs) and fresh frozen plasma (FFP). The Transfusion Requirements in Critical Care (TRICC) trial and the ASA transfusion guidelines have acknowledged the potential benefits of this restrictive transfusion threshold; additional transfusions are associated with higher rate of infectious complications and higher mortality [70,71]. Burn centers in the United States have reported that their hemoglobin transfusion threshold is 8 g/dL for patients with >20 percent TBSA burns [72].

With massive blood loss (ie, >50 percent of blood volume), a unit of FFP is administered for every 1 to 4 units of transfused packed RBCs [73]. (See "Massive blood transfusion".)

Techniques to minimize transfusion — Multiple techniques are used to minimize intraoperative bleeding, including application of topical thrombin, use of compressive devices (tourniquets), staged procedures, and topical or subcutaneous injection of vasoconstrictors (epinephrine, vasopressin analogs, or phenylephrine) [74]. A combination of these techniques may be used to decrease blood loss and transfusion requirements [75].

Topical epinephrine is the most commonly used technique to limit blood loss in both burn excision and donor skin harvest sites. The anesthesiologist should monitor for potential systemic effects of absorbed epinephrine (eg, increased blood pressure, heart rate, or serum glucose) after topical application or subcutaneous injection [76,77]. One study of topical epinephrine use during skin grafting reported increased serum epinephrine and lactate levels, a higher lactate–pyruvate ratio, and higher heart rate in burn patients compared with other patients needing skin grafts [76]. However, other studies report no difference in catecholamine levels or hemodynamic parameters in burn patients receiving epinephrine solutions [78,79]. Phenylephrine (5 mcg/mL) has also been used for subcutaneous infiltration of skin donor sites and was effective in reducing blood loss without systemic effects [80]. Despite these efforts to decrease intraoperative hemorrhage, burn excision is often accompanied by significant blood loss.

One method to reduce intraoperative allogeneic transfusion is the use of intraoperative acute normovolemic hemodilution (ANH), with subsequent autologous blood transfusion. In one study, patients with <20 percent TBSA burn who received intraoperative ANH had reduced allogeneic transfusion requirements. Although total blood volume loss was unchanged, ANH significantly reduced the actual loss of RBCs, coagulation factors, and platelets, presumably because the lost blood was hemodiluted blood [81]. The risks of this technique in burn patients, such as bacteremia, have not been clearly determined. (See "Surgical blood conservation: Intraoperative hemodilution".)

We do not recommend administration of activated recombinant factor VII (rFVIIa) to reduce transfusion until its safety and effectiveness have been established in this setting. Although the use of rFVIIa was associated with a 60 percent reduction in the number of blood products transfused in one study of patients with burn injuries [82], this use of rFVIIa is off-label and has been associated with increased risk of thrombosis [83].

POSTOPERATIVE MANAGEMENT — Postoperative mechanical ventilation is usually indicated for patients who have sustained inhalational burn injuries or who have developed acute respiratory distress syndrome (ARDS). The role of extracorporeal membrane oxygenation for severe respiratory failure in patients with burns and smoke inhalation is unclear and requires further study [84].

Patients who are at increased risk of ongoing bleeding or frequent return to the operating room for additional surgery are
generally not extubated [3]. Another advantage of keeping the patient intubated and well-sedated is minimization of the risk of postoperative motion and graft disruption. This is particularly important with delicate sheet grafting to the face and neck. If postoperative extubation is planned, we often use adjunctive analgesic techniques (eg, administration of ketamine or infiltration of local anesthetics) to reduce opioid requirements, since burn patients are at risk for development of opioid tolerance.

Continuous pain management and intermittent sedation for bedside procedures are treatment priorities in the postoperative period [3]. The management of pain in burn patients is reviewed separately. (See "Burn pain: Principles of pharmacologic and nonpharmacologic management".)

Other therapeutic interventions, including nutritional support, deep venous thrombosis prophylaxis, beta blocker administration, and temperature modulation, are resumed early in the postoperative period. (See "Complications and long-term outcomes of a severe burn".)

SUMMARY AND RECOMMENDATIONS

- Anesthesia providers may be involved in preoperative airway management, intraoperative anesthetic care, or postoperative pain management or intensive care for burn patients. (See 'Introduction' above.)

- The preanesthesia consultation focuses on (see 'Preanesthesia consultation: Key considerations' above):
  - Estimating the percent total body surface area (TBSA) burned and burn severity (see 'Burn extent and severity' above)
  - Assessing adequacy of fluid resuscitation (see 'Fluid resuscitation' above)
  - Assessing cardiovascular changes including (see 'Cardiovascular' above):
    - The “ebb” stage with low cardiac output during the resuscitative phase immediately following the burn injury
    - The “flow” stage with hyperdynamic and hypermetabolic hemodynamics (eg, increased cardiac output, tachycardia, increased myocardial oxygen consumption) during the recovery phase 72 to 96 hours after the burn injury
  - Assessing degree of pulmonary dysfunction due to the burn injury and/or smoke inhalation, including bronchospasm, hypoxemia or hypercarbia, acute respiratory distress syndrome (ARDS), or a restrictive lung defect from circumferential chest burns (see 'Pulmonary' above)
  - Checking blood glucose and continuing perioperative nutritional support (see 'Metabolic and nutritional' above)

- Standard American Society of Anesthesiologists (ASA) monitoring may require selection of alternative monitoring sites (eg, blood pressure cuff and/or intraarterial catheter placement site), equipment (eg, pulse oximetry probes appropriate for multiple sites, co-oximeter for carbon monoxide poisoning), and techniques (eg, electrocardiogram leads stapled in place). (See 'Monitoring' above.)

Effectiveness of perfusion is monitored by urine output (optimal output is 0.5 mL/kg per hour) and, in severe burn cases, cardiac output.

- Impending airway obstruction requires urgent endotracheal intubation, particularly if stridor, hoarseness, or dysphagia is present. Indications of a potentially difficult airway due to oropharyngeal injury or edema include mouth or nasal burns, blistering or edema of the oropharynx, persistent cough or wheezing, or hypoxemia or
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REFERENCES

2. Kumar P, D’Souza J, Bhaskara KG, Bharadwaj S. Serum protein level in conjunction with serum albumin/globulin hypercarbia. (See 'Airway management' above.)

In patients with facial burns, the endotracheal tube may be secured with interdental, circummandibular, nasomaxillary, or transseptal sutures. (See 'Airway management' above.)

Sucoinylcholine, a depolarizing neuromuscular blocking agent (N MBA), may be used to facilitate rapid sequence intubation during the first 48 hours following a severe burn injury, but not thereafter, because of the risk of severe hyperkalemia. (See 'Induction of general anesthesia' above.)

Resistance to all nondepolarizing NMBAs develops after approximately one week and peaks at five to six weeks after injury. Increasing dose partly overcomes this resistance. (See 'Neuromuscular blocking agents' above.)

During the resuscitative (ebb) phase immediately after burn injury, the doses of anesthetic induction agents, especially propofol, are reduced to avoid hypotension. During the recovery (flow) phase three to four days after the burn injury, larger doses of propofol are necessary to attain or maintain therapeutic plasma concentrations. (See 'Induction of general anesthesia' above.)

Other induction agent options include:

- Etomidate, which causes little hemodynamic change but is associated with acute adrenal insufficiency.
- Ketamine, which has beneficial properties, including potent analgesia, bronchodilation, and maintenance of hemodynamic stability and airway reflexes.

During the resuscitative (ebb) phase, opioid doses are reduced because metabolic clearance may be impaired by liver dysfunction. During the recovery (flow) phase, opioid requirement is increased due to both increased volume of distribution and opioid tolerance. Some burn patients develop opioid-induced hyperalgesia (OIH), an aberrant response to opioids manifesting as enhanced pain sensitivity. (See 'Induction of general anesthesia' above.)

Local and regional anesthesia techniques provide intraoperative anesthesia and may improve postoperative analgesia, thus decreasing opioid requirement. However, the size of the burn injury and multiple distant donor skin harvest sites limit their use. (See 'Regional anesthesia' above.)

Perioperative hypothermia is prevented by maintaining operating room temperature ≥25°C, using convective warming devices, administering warmed intravenous fluids, and minimizing exposed body surface area. (See 'Temperature management' above.)

During surgical debridement or escharotomies, fluid and blood to replace losses are added to formula-guided resuscitative fluid volume. Factors guiding fluid management include assessment of blood loss (eg, hemoglobin measurements), perfusion (urine output, cardiac output, base deficit, serum lactate), and oxygenation. (See 'Intraoperative fluid management' above.)

We use a restrictive transfusion threshold (hemoglobin 7 to 8 g/dL) in hemodynamically stable patients without massive blood loss. (See 'Intraoperative blood transfusion' above.)

http://www.uptodate.com/contents/anesthesia-for-burn-patients?to...Key=ANEST%2F94580&elapsedTimeMs=6&view=print&displayedView=full


27. American Society of Anesthesiologists and Society of Cardiovascular Anesthesiologists Task Force on


76. Papp AA, Uusaro AV, Ruokonen ET. The effects of topical epinephrine on haemodynamics and markers of tissue


Topic 94580 Version 5.0
Modified Lund-Browder chart

Numbers refer to the percent body surface area burned.

Graphic 71190 Version 3.0
# Burn center referral criteria*

<table>
<thead>
<tr>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partial-thickness burns greater than 10% of TBSA</td>
</tr>
<tr>
<td>Burns that involve the face, hands, feet, genitalia, perineum, or major joints</td>
</tr>
<tr>
<td>Third-degree burns in any age group</td>
</tr>
<tr>
<td>Electrical burns, including lightening injury</td>
</tr>
<tr>
<td>Chemical burns</td>
</tr>
<tr>
<td>Inhalation injury</td>
</tr>
<tr>
<td>Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality</td>
</tr>
<tr>
<td>Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk for morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be stabilized initially in a trauma center before being transferred to a burn unit. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.</td>
</tr>
<tr>
<td>Burned children in hospitals without qualified personnel or equipment for the care of children</td>
</tr>
<tr>
<td>Burn injury in patients who will require special social, emotional, or rehabilitative intervention</td>
</tr>
</tbody>
</table>

TBSA: total body surface area.

* A burn center may treat adults, children, or both. Burn injuries that should be referred to a burn center include any of the criteria listed.

Intraoperative management of the airway of burn patients

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Graphic 98577 Version 1.0
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