

## Anesthesia for Patients with Burns: An Updated Review for Anesthesiologists and Nursing

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### Abstract

*Burn injuries affect nearly 500,000 individuals annually, with significant implications for anesthesia management. The unique pathophysiology of burns, including airway management, hemodynamic instability, and altered metabolic responses, poses challenges for anesthesiologists. Inhalation injuries, particularly those affecting the upper airway, and systemic complications, complicate anesthesia care for burn patients. This review aims to update anesthesiologists and nursing professionals on the critical aspects of anesthetic management in burn patients, emphasizing airway management, fluid resuscitation, ventilation strategies, and addressing inhalation injuries. The article provides a comprehensive review of the pathophysiological changes that occur in burn patients, with a focus on the distinct phases of burn shock and hypermetabolic response. It discusses the anatomy, physiology, and systemic effects of burn injuries, as well as the anesthetic considerations for managing these complex cases, including airway management, ventilation strategies, and the handling of inhalation injuries. The review identifies key challenges, such as airway obstruction due to supraglottic edema, the need for careful fluid management during the burn shock phase, and the importance of optimizing ventilation strategies to avoid complications like ARDS. The article emphasizes the role of fiberoptic intubation in managing difficult airways and highlights the systemic effects of inhalation injuries, such as hypoxia and acidosis. Effective anesthesia management for burn patients requires a thorough understanding of their unique pathophysiological conditions and careful planning. The use of appropriate anesthetic agents, careful airway management, and vigilant monitoring of systemic effects are essential in ensuring optimal outcomes. Multidisciplinary collaboration between anesthesiologists, nurses, and other healthcare professionals is critical in managing burn patients effectively.*

**Keywords:** Burn Injuries, Anesthesia, Airway Management, Inhalation Injuries, Hypermetabolic Phase, Fluid Resuscitation, Ventilation, Fiberoptic Intubation.

### Introduction

Annually, nearly 500,000 individuals require medical attention for burn-related injuries. In these cases, the anesthesiologist is pivotal in addressing complex issues, including airway management, hemodynamic stabilization, intravascular access, thermoregulation, and pulmonary support. The airway in severely burned patients is especially vulnerable, and traditional anesthesia techniques may not suffice, demanding meticulous management to maintain proper ventilation. Additionally, the intricate pathophysiological

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alterations associated with burns present significant challenges in intraoperative fluid resuscitation, the selection of appropriate induction agents, and the formulation of effective ventilation strategies. In this context, the anesthesiologist is integral in optimizing the overall care for these critically injured patients [1][2].

### *Anatomy and Physiology*

Major burns cause direct tissue damage and distort anatomical structures, complicating traditional airway management, monitoring, and hemodynamic access. Additionally, a cytokine-mediated inflammatory response is triggered, leading to local and systemic pathophysiological effects. This response can be divided into two distinct phases: the burn shock (ebb) phase and the hypermetabolic (flow) phase. The inflammatory response begins within minutes of tissue injury, resulting in the sensitization and irritation of pain fibers [1][3]. The burn shock or ebb phase occurs within the first 24 to 48 hours following a severe burn, defined as second- and third-degree burns covering 20% or more of the total body surface area (TBSA). This phase is characterized by reduced end-organ perfusion and diminished cardiac output, primarily due to intravascular volume loss. The condition represents a distributive shock state, with fluid extravasation causing edema in both burned and unburned tissues. Hemoconcentration of red blood cells occurs as a result of significant fluid shifts into adjacent tissues. This edematous state can obscure the glottic opening during direct laryngoscopy. The reduction in cardiac output is exacerbated by intravascular volume loss, direct myocardial depression, and increased systemic vascular resistance. A surge in catecholamines and antidiuretic hormone during this phase leads to vasoconstriction and reduced blood flow. Despite aggressive fluid resuscitation, only partial compensation is achievable due to disruptions in the cellular transmembrane ionic gradient and reduced sodium ATPase activity, which may persist for days post-injury [1][2][4].

The hypermetabolic or flow phase begins 48 to 72 hours after the initial injury and is marked by increased oxygen consumption and carbon dioxide production. Cardiac output improves compared to the burn shock phase, enhancing end-organ perfusion. During this phase, cardiac output may rise to 2–3 times the normal range due to tachycardia and decreased systemic vascular resistance, mimicking sepsis. Improved blood flow to all organs occurs as vascular resistance decreases, though arteriovenous shunting may increase venous oxygen saturation. Resorption of edema can lead to pulmonary edema, exacerbating damage from inhalation injuries. Excessive fluid resuscitation during the burn shock phase may complicate ventilator weaning intraoperatively as fluid is resorbed. The hypermetabolic phase is also characterized by the release of catabolic hormones and insulin resistance, leading to protein catabolism, such as muscle wasting, and hyperglycemia. Elevated glucocorticoid levels and inflammatory cytokines increase the caloric demands of severely burned patients. This phase can persist for up to two years [1][2][4]. When burns affect 25% to 30% of the TBSA, the inflammatory response induces systemic physiological alterations in burn patients [1][5].

The initial pathophysiological changes during the burn shock (ebb) phase can be summarized by the organ system as follows. In the cardiovascular system, increased capillary membrane permeability leads to the loss of intravascular proteins into the interstitial compartment. Vasoconstriction occurs due to elevated systemic vascular resistance, while decreased myocardial contractility results in reduced cardiac index, stroke volume, and blood pressure. Metabolic acidosis develops due to decreased kidney perfusion, leading to systemic hypotension and reduced end-organ perfusion. In the pulmonary system, pulmonary edema and increased susceptibility to bronchospasm due to bronchorrhea are observed. These changes can potentially lead to the development of adult respiratory distress syndrome (ARDS). The integumentary system experiences fluid loss through compromised skin, with generalized edema occurring when burns exceed 25% TBSA. Circumferential burns of the chest, abdomen, or limbs may cause compartment syndrome, and impaired thermoregulation further complicates the clinical picture. These changes necessitate aggressive fluid resuscitation and careful management of circumferential burns. In the immunologic system, a downregulated immune response increases susceptibility to infections. These systemic alterations highlight the complexity of managing burn patients and underscore the importance of a multidisciplinary approach to care [1][5].

## *Indications*

### *Inhalation Injuries*

Inhalation injuries associated with burns significantly increase morbidity and mortality. Patients with severe burns and inhalation injuries require 30% to 50% more fluid resuscitation compared to those without inhalation injuries. Chest radiographs may initially appear normal until complications such as infection or atelectasis develop. Clinically, inhalation injuries may take several days to manifest [1]. These injuries can be categorized into three subclassifications: supraglottic, subglottic, and systemic.

### *Supraglottic Injuries*

Thermal injury is the most common type of supraglottic inhalation injury. The primary anesthetic concern with supraglottic injuries is the upper airway edema, which can obstruct the airway and complicate intubation. Exposure to direct heat or steam may cause swelling of structures such as the face, tongue, epiglottis, and glottis, making mask ventilation and glottic visualization more challenging. This issue may worsen as the burn shock phase progresses. Supraglottic injuries can be assessed through physical examination and tools like fiberoptic bronchoscopy to directly evaluate the oropharynx and vocal cords for edema. Patients exhibiting mental status changes, stridor, hoarseness, or mucosal edema should undergo early intubation to prevent airway complications and respiratory arrest. The subglottic structures are typically protected from thermal injuries due to the glottis's reflexive closure and the upper airway's ability to dissipate heat. Supraglottic swelling usually resolves within 3 to 6 days. The optimal anesthetic approach for patients with significant supraglottic injuries involves maximizing the use of local anesthetics and minimizing general anesthesia, employing a fiberoptic technique. This approach allows adequate time to assess and navigate a difficult airway while maintaining spontaneous respiration, thereby reducing the risk of patient harm and airway manipulation [1][6].

### *Subglottic Injuries*

Subglottic injuries are often caused by the inhalation of noxious chemicals and irritants, leading to direct damage and inflammation. This results in airway mucosal hyperemia, potential bronchospasm, and mucosal sloughing. The dysfunction of the mucociliary escalator can lead to bacterial accumulation and debris, increasing the risk of pneumonia in the days following the injury. Inflammatory processes may also impair hypoxic pulmonary vasoconstriction, causing right-to-left shunting and hypoxia. Alveolar collapse can occur due to loss of surfactant production or debris plugging. Intermittent bronchospasm and airway edema further limit oxygenation by impeding oxygen delivery to the alveoli. These findings may be evident in ventilated patients through a prolonged expiratory phase and elevated peak inspiratory pressures. Cast formation from mucus sloughing can obstruct already narrowed airways. Intraoperative bronchoscopy can be used to remove casts, improving airway pressures and oxygenation. Widespread cast formation redistributes tidal volumes to unobstructed airways, increasing pulmonary pressures in healthy lung tissue and contributing to barotrauma. This can lead to acute respiratory distress syndrome (ARDS) and pneumothorax. ARDS typically develops within a week of the inciting event and is not attributable to congestive heart failure or hypervolemia. It is classified based on PaO<sub>2</sub>/FiO<sub>2</sub> ratios: mild ARDS (200-300), moderate ARDS (100-200), and severe ARDS (<100). While subglottic injuries are primarily diagnosed clinically, bronchoscopy can provide additional insights. These injuries are particularly relevant to anesthesiologists due to the need for lung-protective ventilation strategies, such as low tidal volumes (4-6 mL/kg) and appropriately titrated positive end-expiratory pressure, to minimize barotrauma [1][6][7].

### *Systemic Effects*

Inhalation injuries can have systemic consequences, including hypoxia, acidosis, and systemic inflammation, leading to pulmonary edema. Carbon monoxide and cyanide, which are soluble in blood, can directly damage tissues and impair oxygenation. Intraoperative monitoring of these systemic effects can be achieved through arterial blood gas analysis and laboratory studies when necessary. Carbon monoxide interferes with hemoglobin's oxygen-carrying capacity, having a higher affinity for hemoglobin than oxygen. Toxicity is

suspected when carboxyhemoglobin levels exceed 15%. Cyanide, produced by the combustion of nitrogenous materials, interacts with mitochondrial cytochrome systems, inhibiting oxygen utilization. This disrupts oxidative phosphorylation, preventing the conversion of pyruvate to adenosine triphosphate (ATP) and forcing cells to rely on anaerobic metabolism. Cyanide toxicity is characterized by an anion gap metabolic acidosis despite adequate oxygen delivery. Persistent intraoperative hypoxia and acidosis may indicate carbon monoxide or cyanide toxicity, necessitating prompt intervention [1][6].

### *Carbon Monoxide Poisoning*

Pulse oximetry is unreliable for accurately measuring oxygenation levels in cases of carbon monoxide poisoning because it cannot distinguish between oxyhemoglobin and carboxyhemoglobin. To assess the severity of carbon monoxide poisoning, laboratory tests are required to measure carboxyhemoglobin levels in the blood. In time-sensitive situations, a carbon monoxide oximeter may be used, although such devices are not commonly available in modern hospitals. Carbon monoxide has an affinity for hemoglobin that is approximately 230 to 270 times greater than that of oxygen, causing a leftward shift in the oxygen-hemoglobin dissociation curve. This shift reduces oxygen delivery to tissues, leading to symptoms such as mental confusion, agitation, nausea, dizziness, and headaches, all of which are indicative of carbon monoxide poisoning. Immediate administration of 100% oxygen is essential for any patient suspected of carbon monoxide poisoning. Normobaric 100% oxygen can reduce the half-life of carbon monoxide from 240 to 320 minutes to 40 to 80 minutes, significantly accelerating recovery. Once the carboxyhemoglobin level falls below 10%, it is safe to discontinue 100% oxygen administration. However, patients should be monitored for approximately 24 hours to ensure the absence of respiratory complications. Patients with carboxyhemoglobin levels exceeding 20% should be considered for intubation and mechanical ventilation to further expedite carbon monoxide elimination and improve tissue oxygenation. For mechanically ventilated patients with carbon monoxide poisoning, 100% FiO<sub>2</sub> should be administered until carboxyhemoglobin levels normalize to facilitate the dissociation of carbon monoxide from hemoglobin. In severe cases, hyperbaric oxygen therapy should also be considered as a treatment option [1][6][7][8].

### *Preparation*

#### *Airway Management*

When managing a burn patient requiring anesthesia in an acute setting, a thorough airway assessment is essential to identify any signs of airway edema that could complicate intubation. Burn contractures may reduce mandibular mobility, while glottic edema from airway swelling can make laryngoscopy challenging. In cases of suspected difficult airways due to thermal injury to supraglottic structures, fiberoptic intubation is the most widely accepted and safest approach. This technique involves the application of topical anesthetics and minimal general anesthesia. If fiberoptic intubation is chosen, ketamine-induced sedation or anesthesia can create optimal intubation conditions by maintaining pharyngeal muscle tone. Ketamine is advantageous due to its ability to preserve hemodynamic stability, hypercapnia responses, and reduced airway resistance, making it an ideal agent for patients with supraglottic swelling. Conversely, anesthetic agents such as propofol and paralytic agents, which disrupt airway tone, may exacerbate airway obstruction and complicate intubation [1][3].

#### *Intravascular Access*

Severe burn injuries often complicate intravenous access due to damage to common access sites such as the neck, limbs, and groin. Additionally, edema from the burn shock phase or fluid creep resulting from excessive fluid resuscitation can further hinder vascular access. Delayed fluid resuscitation beyond two hours post-injury is associated with increased mortality, underscoring the critical importance of establishing vascular access promptly. When conventional methods of intravascular access prove difficult, intraosseous access can be highly beneficial. Intraosseous access is typically faster than peripheral intravenous or central venous line placement and has a higher success rate on the first attempt. However, intraosseous access is more likely to cause insertion and infusion pain compared to conventional methods. Despite its advantages, it remains underutilized. To prevent failure, proper familiarity with the device is crucial, as improper

placement is the most common cause of failure. The proximal tibia is the most frequently used site for intraosseous needle placement, though the proximal humerus is equally effective. The proximal tibia is particularly advantageous as it does not interfere with cardiopulmonary resuscitation [2][9][10].

### *Thermoregulation*

Severe burn patients lose their primary barrier to heat retention, making hypothermia a significant anesthetic consideration. Evaporative heat and water loss from burn wounds directly contribute to heat loss. Additionally, the cerebral mechanisms that regulate thermoregulation become dysregulated in severe burns. The critical temperature, which triggers physiological responses such as shivering, vasoconstriction, and cellular non-shivering thermogenesis, is lowered in burn patients. When combined with general anesthesia, which redistributes heat from the core to the periphery and inhibits central thermoregulatory control, burn patients are at a heightened risk of hypothermia. Effective strategies to maintain optimal thermoregulation include forced-air warming, fluid warming, and increasing the operating room temperature. Accurate temperature monitoring, using distal esophageal or rectal temperature probes, is essential for the perioperative management of severely burned patients [4][11][12].

### *Technique or Treatment*

#### *Fluid Resuscitation*

Proper fluid resuscitation is critical for burns involving more than 15% of the total body surface area (TBSA) to prevent complications associated with the burn shock phase. Without early and aggressive fluid resuscitation, severe burns (>15% TBSA) can lead to hypovolemic shock due to intravascular volume depletion caused by fluid shifts and increased capillary permeability. Delaying fluid resuscitation is beyond two hours after the initial injury is associated with a significant increase in mortality. It is important to note that only second- and third-degree burns are included in TBSA calculations, as first-degree burns are excluded. Adequate intravascular volume repletion helps mitigate complications such as tissue hypoperfusion and reflexive vasoconstriction. For burns involving less than 15% TBSA, oral fluids or a maintenance intravenous fluid rate of 1.5 times normal may suffice [1][2]. The preferred crystalloid for fluid resuscitation is lactated Ringer solution, as large volumes of normal saline (0.9%) can exacerbate metabolic acidosis. Patients with severe burns are already predisposed to metabolic acidosis due to reduced kidney perfusion during the burn shock phase. While several formulas exist to calculate the required fluid volume, clinicians must prioritize clinical markers of adequate resuscitation, such as urine output, fractional excretion of sodium, BUN-to-creatinine ratio, echocardiogram findings, and arterial blood gas analysis. Key indicators include a urine output goal of 0.5 to 1 mL/kg/h, a fractional excretion of sodium <1% suggesting hypovolemia, a BUN-to-creatinine ratio >20 indicating hypovolemia, and a base deficit <5 on arterial blood gas suggesting hypoperfusion in the absence of carbon monoxide poisoning [1][2].

Excessive fluid administration, known as "fluid creep," can occur when clinicians fail to monitor physiological indicators closely, particularly urine output. Fluid creep leads to excessive tissue and pulmonary edema, complicating the patient's condition. Pulmonary edema can result in respiratory distress, necessitating tracheal intubation or increasing the risk of pneumonia. Additionally, fluid creep can exacerbate airway edema, further complicating airway management. This phenomenon often arises from miscalculations of fluid resuscitation volumes in the first 24 hours post-burn. Clinicians must also account for intravenous medications such as antibiotics, analgesics, and sedatives when calculating fluid requirements. While the role of colloids remains undefined, they may serve as adjunct therapy to prevent hypervolemia [2]. The Parkland formula is the most widely used method for estimating fluid resuscitation needs in burn patients. It calculates the total fluid volume required over the first 24 hours, with half of the volume administered in the first 8 hours. The formula is as follows:

*Parkland Formula:* 4 mL x kg x percentage of TBSA burned

*Example:* A 75 kg male with a 30% TBSA burn

- Calculate the total amount of lactated Ringer solution for the first 24 hours:  
 $4 \text{ mL} \times 75 \text{ kg} \times 30 = 9,000 \text{ mL}$
- Calculate the amount of fluid to administer in the first 8 hours:  
Half of the total volume =  $9,000 \text{ mL} / 2 = 4,500 \text{ mL}$
- Administer the remaining 4,500 mL over the next 16 hours.

This approach ensures adequate fluid resuscitation while minimizing the risk of complications such as fluid creep [1].

#### *Estimation of TBSA*

The Wallace Rule of Nines is a widely used method for estimating the total body surface area (TBSA) affected by burns, valued for its simplicity and practicality in clinical settings. For smaller burns, clinicians may use the Rule of Palms, where the patient's palm size represents approximately 1% of TBSA. It is important to note that only second- and third-degree burns are included in TBSA calculations for fluid resuscitation or clinical diagnosis, as first-degree burns are excluded. However, variability in body surface area due to factors such as gender, age, and body mass index (BMI) can complicate the accuracy of these estimations. For instance, individuals with a higher BMI often have an overestimated TBSA calculation. Advances in technology, such as 3D body scanning, may offer more precise methods for determining TBSA in the future. Accurate TBSA estimation is critical for determining the need for transfer to specialized burn centers and guiding fluid resuscitation goals. Anesthesiologists must communicate effectively with other healthcare professionals when managing severely burned patients in the operating room to ensure a clear understanding of fluid management strategies during the perioperative period. Given the prevalence of miscalculations and the adverse effects of fluid creep, anesthesiologists should be proficient in estimating TBSA during initial assessments to administer fluids appropriately [13][14].

#### *Neuromuscular Blockade*

A significant concern in burn patients is the upregulation of acetylcholine receptors following a burn injury, which can lead to life-threatening hyperkalemia after the administration of succinylcholine. In healthy individuals, succinylcholine typically causes a minimal increase in serum potassium (0.5 mEq/L), but in burn patients, this response is exaggerated. This heightened susceptibility to hyperkalemia is likely due to changes in the nicotinic acetylcholine receptor (nAChR) subunits, with evidence suggesting upregulation of alpha7 and gamma subunit genes. These changes result in an increased number of acetylcholine receptors distributed throughout the body, some of which may have abnormal electrophysiological interactions with succinylcholine, leading to significant hyperkalemia. This receptor upregulation does not occur immediately but develops approximately 24 to 48 hours after the initial burn injury. In contrast, resistance to non-depolarizing neuromuscular blockers develops more rapidly, often within the same 24-48 hour window. As a result, burn patients may require higher-than-usual doses of non-depolarizing neuromuscular blockers, such as rocuronium, to achieve adequate intubating conditions. A dose of 1.2 mg/kg of rocuronium can provide satisfactory intubating conditions in burn patients [15][16][17].

#### *Complications*

##### *Infection*

Severe burns compromise the immune system due to elevated cytokine inflammatory markers, increasing the risk of infection. Burn wounds are often colonized initially by gram-positive organisms such as *Staphylococcus aureus* and *Staphylococcus epidermidis*. Over time, gram-negative organisms like *Pseudomonas aeruginosa* and *Escherichia coli* may also colonize the wounds. Systemic antibiotic therapy is not routinely indicated for perioperative management of these colonizations, although thorough wound cleansing with soap, water, normal saline, or chlorhexidine is recommended. Topical antibiotic therapy is generally

sufficient for perioperative wound care in the early stages of burn management. Collaboration between surgeons and anesthesiologists is essential to minimize the overuse of antibiotics in burn patients [18][19][20].

### *Clinical Significance*

Anesthesia for burn patients is a highly specialized and challenging field that requires a thorough understanding of burn pathophysiology to optimize clinical outcomes. Managing severe head and neck burns, in particular, demands careful attention to potential airway difficulties to avoid the critical "can't ventilate, can't intubate" scenario. A conservative approach, using minimal anesthesia to maintain patient comfort and spontaneous ventilation while performing fiberoptic intubation, is often the safest strategy. Understanding the hemodynamic changes associated with burns is crucial for effective intraoperative fluid resuscitation, ensuring patients are stabilized before transfer to intensive care. Additionally, addressing thermoregulatory changes in burn patients under anesthesia can help prevent complications such as wound infections, impaired coagulation, and perioperative shivering. Anesthesiologists play a pivotal role in the perioperative care of burn patients, and a comprehensive understanding of the unique challenges in treating this population can significantly reduce morbidity and mortality [1][2].

### *Enhancing Healthcare Team Outcomes*

Approximately 500,000 individuals present to emergency departments annually with burns, though only a subset require critical care [1]. In severe cases, timely intervention is essential, as airway swelling can rapidly progress to a life-threatening situation. Anesthesiologists, with their expertise in physiology, pharmacology, airway management, and critical care, are integral to managing these cases. However, optimal outcomes depend on effective communication and coordination among all members of the interprofessional care team. Severely burned patients present complex challenges, including difficult airways, smoke inhalation injuries, and unique hemodynamic considerations. A multidisciplinary approach involving anesthesiologists, surgeons, critical care physicians, and nursing staff is essential for optimizing care. For example, a retrospective study on the Difficult Airway Response Team (DART), comprising anesthesiologists, otolaryngologists, and trauma surgeons, demonstrated that a team-based approach significantly improved airway management outcomes, reducing the need for emergency cricothyrotomy [21]. Applying such a collaborative model to burn patients with difficult airways can enhance safety and reduce complications.

The Parkland Formula is a valuable tool for estimating fluid resuscitation needs in burn patients. However, relying solely on this formula can lead to errors, such as overlooking fluid volumes from intravenous medications. Both under-resuscitation and over-resuscitation (fluid creep) are associated with adverse outcomes, including increased morbidity, mortality, and higher rates of infection and intubation [1][2]. A retrospective review found that burn patients often require significantly more fluid than calculated by the Parkland Formula, highlighting the importance of individualized fluid management [22]. Effective communication among healthcare team members is critical to preventing fluid creep and its associated complications. Nursing staff play a vital role in accurately recording intake and output and providing feedback on fluid volumes from medications such as antibiotics, sedatives, and electrolyte replacements. Collaboration between anesthesiologists, critical care physicians, and surgeons ensures that fluid delivery is adjusted to avoid compromising surgical sites or respiratory function. By fostering open communication and teamwork, healthcare providers can improve outcomes for burn patients without the need for new therapies [1][2][21][22][23].

### *Role of Nursing Protocols*

Nursing protocols play a critical role in the management of burn patients, ensuring standardized, evidence-based care that addresses the complex and multifaceted needs of this population. Burn injuries require meticulous attention to detail, from initial assessment and resuscitation to long-term rehabilitation, and nurses are at the forefront of delivering this care. Well-defined nursing protocols help streamline processes, reduce variability in practice, and improve patient outcomes.

### *Initial Assessment and Triage*

Nursing protocols guide the initial assessment and triage of burn patients, ensuring timely and accurate evaluation of burn severity, total body surface area (TBSA) involvement, and associated injuries. Nurses are trained to use tools such as the Rule of Nines or the Rule of Palms to estimate TBSA, which is critical for determining fluid resuscitation needs and the necessity of transfer to a specialized burn center. Early recognition of inhalation injuries, airway compromise, or signs of shock is also emphasized in nursing protocols, enabling prompt intervention and reducing the risk of complications.

### *Fluid Resuscitation and Monitoring*

Nurses play a central role in administering and monitoring fluid resuscitation, a cornerstone of burn management. Protocols provide clear guidelines for calculating fluid requirements using formulas like the Parkland Formula and adjusting infusion rates based on clinical indicators such as urine output, vital signs, and laboratory values. Nurses are responsible for maintaining accurate intake and output records, identifying signs of fluid creep, and communicating with the medical team to optimize fluid balance. This vigilance helps prevent complications such as pulmonary edema, compartment syndrome, and inadequate tissue perfusion.

### *Wound Care and Infection Prevention*

Burn wound care is a core component of nursing protocols, focusing on preventing infection, promoting healing, and minimizing scarring. Nurses are trained in proper wound cleansing techniques, the application of topical antimicrobial agents, and the use of advanced dressings. Protocols also emphasize the importance of aseptic techniques during wound care to reduce the risk of colonization by pathogens such as *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Regular wound assessments and documentation are essential for tracking progress and identifying early signs of infection.

### *Pain Management*

Effective pain management is a priority in burn care, and nursing protocols provide guidelines for assessing pain levels and administering analgesics. Nurses are trained to use both pharmacological and non-pharmacological interventions, such as distraction techniques or relaxation therapy, to address the intense pain associated with burn injuries and procedures like dressing changes. Protocols also emphasize the importance of reassessing pain levels and adjusting treatment plans to ensure patient comfort.

### *Thermoregulation and Environmental Control*

Burn patients are at high risk of hypothermia due to the loss of skin barrier function and evaporative heat loss. Nursing protocols include measures to maintain normothermia, such as using forced-air warming devices, warming intravenous fluids, and maintaining a warm ambient temperature in patient rooms. These interventions help prevent complications such as impaired wound healing, coagulopathy, and increased metabolic demand.

### *Patient and Family Education*

Nursing protocols emphasize the importance of patient and family education throughout the burn recovery process. Nurses provide guidance on wound care, pain management, nutrition, and rehabilitation exercises, empowering patients and families to participate actively in care. Education also addresses psychological and emotional support, helping patients cope with the long-term impact of burn injuries.

### *Interprofessional Collaboration*

Nursing protocols highlight the importance of collaboration with other healthcare professionals, including physicians, physical therapists, dietitians, and psychologists. Nurses serve as a bridge between the patient

and the multidisciplinary team, ensuring that care is coordinated and comprehensive. Effective communication and teamwork are essential for addressing the complex needs of burn patients and achieving optimal outcomes. In conclusion, nursing protocols are indispensable in burn care, providing a structured framework for delivering high-quality, patient-centered care. By adhering to these protocols, nurses can effectively manage the unique challenges of burn injuries, reduce complications, and support patients throughout their recovery journey.

## Conclusion

Anesthesia management in burn patients involves navigating a series of complex and dynamic challenges related to the burn injury's unique pathophysiology. Anesthesiologists play a critical role in ensuring optimal outcomes by addressing issues related to airway management, hemodynamic stability, and ventilation, particularly in severe burn cases. The burn shock phase, characterized by volume loss and organ hypoperfusion, requires careful fluid resuscitation, which must be balanced to avoid complications such as pulmonary edema. The hypermetabolic phase presents additional challenges, with heightened oxygen consumption, hyperglycemia, and catabolic processes, demanding continued monitoring and adjustment of anesthesia strategies. Airway management remains one of the most critical aspects of burn anesthesia, particularly in cases involving significant supraglottic injuries. The swelling of the upper airway, if left unaddressed, can lead to difficulties in intubation and ventilatory support. Fiberoptic intubation is widely accepted as the most effective technique in these situations, as it allows for controlled airway assessment and management. Additionally, the use of ketamine for induction is beneficial in preserving airway tone and maintaining hemodynamic stability during intubation, particularly in cases of airway swelling. Inhalation injuries further complicate anesthesia care, often leading to systemic effects such as carbon monoxide toxicity and acute respiratory distress syndrome (ARDS). Monitoring for these conditions, including the use of arterial blood gas analysis and specific toxicological tests, is essential for early detection and intervention. As inhalation injuries can increase fluid requirements by up to 50%, careful attention to fluid management remains essential throughout the perioperative period. Ultimately, the key to successful anesthesia management in burn patients lies in the coordination of a multidisciplinary team. This includes anesthesiologists, nurses, and other healthcare providers who can collaborate on decision-making and ensure the patient's complex needs are met. A tailored approach to each individual's injury severity and comorbid conditions, alongside vigilant monitoring and timely interventions, is necessary to navigate the multifaceted challenges that arise during anesthesia care for burn patients.

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## التخدير للمرضى المصابين بالحروق: مراجعة محدثة لأطباء التخدير والمرضى

### الملخص:

**الخلفية:** تؤثر إصابات الحروق على ما يقرب من 500,000 شخص سنويًا، مع نداعيات كبيرة على إدارة التخدير. إن الفسيولوجيا المرضية الفريدة للحروق، بما في ذلك إدارة المجاري التنفسية، وعدم الاستقرار الديناميكي الدموي، والاستجابات الأيضية المتغيرة، تخلق تحديات لأطباء التخدير. وتؤدي الإصابات الناتجة عن الاستنشاق، وخاصة التي تؤثر على المجاري التنفسية العلوية، والمضاعفات الجهازية، إلى تعقيد رعاية التخدير لمرضى الحروق.

**الهدف:** تهدف هذه المراجعة إلى تحديث أطباء التخدير والمرضى حول الجوانب الحيوية لإدارة التخدير في مرضى الحروق، مع التركيز على إدارة المجاري التنفسية، الإنعاش السوائي، استراتيجيات التهوية، ومعالجة الإصابات الناتجة عن الاستنشاق.

**الطرق:** يقدم المقال مراجعة شاملة للتغيرات الفسيولوجية المرضية التي تحدث في مرضى الحروق، مع التركيز على المراحل المتميزة من صدمة الحرق والاستجابة الأيضية المفرطة. يناقش المقال تشريح وفسيولوجيا وتأثيرات الإصابات الحرقية على الجسم، بالإضافة إلى الاعتبارات التخديرية لإدارة هذه الحالات المعقدة، بما في ذلك إدارة المجاري التنفسية، استراتيجيات التهوية، والتعامل مع الإصابات الناتجة عن الاستنشاق.

**النتائج:** تحدد المراجعة التحديات الرئيسية، مثل انسداد المجاري التنفسية بسبب الوذمة فوق الحنجرة، والحاجة إلى إدارة السوائل بحذر أثناء مرحلة صدمة الحرق، وأهمية تحسين استراتيجيات التهوية لتجنب المضاعفات مثل متلازمة الضائقة التنفسية الحادة (ARDS). يسلط المقال الضوء على دور التنبيب باستخدام الألياف الضوئية في إدارة المجاري التنفسية الصعبة ويبرز التأثيرات الجهازية لإصابات الاستنشاق مثل نقص الأوكسجين والحمض.

**الخلاصة:** يتطلب إدارة التخدير الفعالة لمرضى الحروق فهماً شاملاً لحالاتهم الفسيولوجية المرضية الفريدة وتخطيطاً دقيقاً. إن استخدام عوامل التخدير المناسبة، وإدارة المجاري التنفسية بحذر، والمراقبة الدقيقة للتأثيرات الجهازية أمر ضروري لضمان نتائج مثالية. التعاون متعدد التخصصات بين أطباء التخدير والمرضى وغيرهم من المتخصصين في الرعاية الصحية أمر بالغ الأهمية لإدارة مرضى الحروق بفعالية.

**الكلمات المفتاحية:** إصابات الحروق، التخدير، إدارة المجاري التنفسية، إصابات الاستنشاق، المرحلة الأيضية المفرطة، الإنعاش السوائي، التهوية، التنبيب باستخدام الألياف الضوئية.