**Abstract:**
This overview is to provide the reader with an understanding of the unique pathophysiologic changes occurring in the pediatric burn patient that increases their risk of infection. In addition, thermal injury induces a persistent inflammatory and hypermetabolic state in the burn-injured child. This makes the identification and diagnosis of infection, and consequently, sepsis challenging. The diagnostic approach to infection in the burn patient is reviewed as well as preventative strategies to reduce the risk of sepsis in this susceptible population.

**Keywords:**
pediatric; burns; thermal injury; infection; sepsis

*Department of Pediatrics, Division of Pediatric Critical Care, Northwestern University Feinberg School of Medicine, Ann and Robert H Lurie Children's Hospital of Chicago, Chicago, IL; †Division of Pediatric Surgery, Ann and Robert H Lurie Children's Hospital of Chicago, Chicago, IL.
Reprint requests and correspondence: Sifrance Tran, MD, Department of Pediatrics, Division of Pediatric Critical Care, Northwestern University Feinberg School of Medicine, Ann and Robert H Lurie Children's Hospital of Chicago, 225 E Chicago Ave, Box No. 205, Chicago, IL 60611.
stran@luriechildrens.org

1522-8401


# Burn Sepsis in Children

**Sifrance Tran, MD*, Anthony C. Chin, MD†**

In the United States, burn injuries account for 450 000 medical visits annually, and an estimated 40 000 require hospitalization.[1] Up to one-third of burn unit admissions involve children.[2] The most common mechanism of burn-related injury in children below the age of 5 years is scald burns, whereas older children typically have flame injuries. In 2011, more than 136 000 children ages 19 years and below were seen in the emergency department for thermal injuries.

Because of the significant morbidity and mortality associated with burns, considerable resources have been dedicated to the specialty to improve outcomes in burn patients.[3] With advances in resuscitation and antimicrobial topical agents, early surgical wound management, improvements in ventilator management, improved understanding of the burn-induced hypermetabolic response, and increased utilization of specialized burn centers, the death rate of children from fires and burns decreased by from 1.2 to 0.6 per 100 000 from 1999 to 2010.[3-5] Furthermore, over this period, the burn size that was associated with increased risk of mortality at a specialized burn center has increased from 40% total body surface area to more than 60% total body surface area.[3]

Despite reduction in mortality over the past decade, burn injury remains the fifth leading cause of unintentional child injury-related death.[2] Death from burns occurs at 3 different time intervals after initial injury. Immediate death typically occurs within the first few hours from burn shock; early death in the following days is due to respiratory failure; and late death in the subsequent weeks is secondary to complications of sepsis and multiple organ failure. Advancements in burn management and critical care have markedly improved survival from immediate and early deaths. However, with improved survival, sepsis has paradoxically become the leading cause of pediatric death in the burn unit.[3,6,7]

In this article, the pathogenesis of infection after burn injury, current challenges in diagnosing sepsis in the burn population, and strategies to limit infection in the burn patient will be reviewed.

## PATHOGENESIS OF INFECTION

Because of the dramatic anatomical and immunologic changes that occur after severe burn injury, children who have significant burns are at increased risk for infectious complications.[8] Anatomically, the skin, respiratory tract, and digestive tract have normal defense mechanisms to exogenous infectious agents. With thermal injury, each of these systems may be disrupted, exposing the patient to infectious pathogens and sepsis.

### Burn Wound Infection

Thermal destruction of the epidermis leads to loss of a significant physical barrier, and consequently, open wounds with exposed subcutaneous tissue, cartilage, or bone are inherently susceptible to contamination. Although burn wounds are sterilized after initial thermal injury, they are protein-rich environments that are ultimately colonized by endogenous microorganisms. Within a few hours, surviving Gram-positive bacteria deep within hair follicles or sweat glands, such as *Staphylococcus aureus* and S epidermidis, rapidly colonize the wound. By 5 to 7 days, other microbes from the environment, including gram-negative bacteria, fungi, or viruses, eventually also inhabit these wounds from exposure or cross-contamination. Deep burn wounds with eschar are avascular. Tissue avascularity results in limited migration of host immune cells to the affected area. With restricted delivery of the host immune response, severe burn wounds are further predisposed for microorganism overproliferation, infection, and invasion.[9]

### Respiratory Tract Infection

Inhalational injury of the lower respiratory tract is rarely due to heated air because the upper airway efficiently dissipates heat, and the vocal cords reflexively close when exposed to low levels of heat. Smoke and inhaled toxins are the primary offenders that induce damage to the lower airway. Inhaled chemical toxins result in direct cell epithelial cellular damage of the respiratory mucosa and loss of mucociliary movement. With the mucociliary action destroyed, excretion of mucus and pathogens is profoundly reduced. Large and small airways may become obstructed from bronchial edema, bronchospasm, and debris from mucosal sloughing. Chemical irritants from smoke also induce an inflammatory reaction in the pulmonary parenchyma that disrupts surfactant synthesis. Further alveolar collapse and decreased pulmonary compliance result from surfactant loss. The combination of all these airway changes and need for prolonged ventilator support predisposes severe burn patients to pulmonary infectious complications.[6,9-11]

### Gut Bacterial Translocation

Normally, the gastrointestinal mucosal layer, normal microbial gut flora, and host immune system all function together as a protective barrier to confine pathogens to the intestinal lumen. With major thermal injury, this delicate balance is disrupted, predisposing burn patients to bacterial translocation. In the burn shock state, resultant mesenteric vasoconstriction and splanchnic hypoperfusion lead to intestinal ischemia and mucosal injury. Damage to the mucosa results in increased gut permeability to macromolecules. Intestinal permeability reaches a peak at 5 hours after initial burn injury and returns to baseline by 24 hours.[12,13] Thermal injury also disturbs the normal ecosystem of gut microflora by promoting pathogenic bacterial proliferation. Postulated mechanisms of bacterial overgrowth include intestinal stasis, starvation, and antibiotic exposure. In addition, burn patients experience a state of immunosuppression, characterized by a decrease in splenic and peripheral T-cell proliferation and macrophage dysfunction. These pathophysiologic factors compromise the natural gut barrier and allow the passage of endotoxins and microorganisms across the intestinal wall into the systemic circulation, causing systemic inflammation, distant organ injury, and sepsis. The most common translocating bacteria are Gram-negative anaerobes, particularly *Escherichia coli, Proteus*, and *Enterobacter* species.[12]

### Device-Related Infections

Other potential routes of infectious exposure include central venous catheters (CVCs), arterial catheters, bladder catheters, nasogastric feeding tubes, and endotracheal tubes.[6,14] Although these tools are necessary supportive devices to manage severely injured children in the burn unit, prolonged use offers additional portals of entry for exogenous infectious agents.

### Burn-Induced Immunosuppression

Compounding the numerous risks for infection in burn patients, thermal injury also has immunosuppressive effects. Although the exact sequence of events that result in immunosuppression remains to be elucidated, there are several postulated mechanisms for postburn immune dysfunction.[6,14] One of the prevailing theories implicates hyperactive