# The Physiologic Basis of Burn Shock and the Need for Aggressive Fluid Resuscitation

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**KEYWORDS**

• Burn • Thermal injury • Shock • Resuscitation • Inflammation • SIRS • Edema • Hypovolemia

**KEY POINTS**

- The inflammatory responses to burn injury cause multiorgan failure and early death without adequate resuscitation.
- Inflammatory mediator's effects on endothelial and smooth muscle cells result in leakage of fluid from the intravascular to extravascular space at the site of the burned tissue, and systemically in all organs leading to hypovolemic shock.
- Resuscitation causes edema, which contributes to morbidity and mortality in the thermally injured patient.
- Reactive oxygen species produced by injured tissue contributes to the inflammatory response.
- Nitric oxide production after injury potentiates endothelial leak, contributing to hypotension and poor organ perfusion.

## INTRODUCTION

Burn trauma in the current age of medical care still portends a 3% to 8% mortality. Of patients who die from their burn injuries, 58% of deaths occur in the first 72 hours after injury, indicating death from the initial burn shock.[1] Significant thermal injury incites an inflammatory response, which distinguishes burns from other trauma. Since World War I, human and animal studies have brought us closer to understanding the unprecedented inflammatory reaction caused by burn injury. Rapid and extensive fluid shifts in burned and nonburned tissue result in progressive hypovolemia, peripheral edema, multiorgan failure, and death.[2–4] Capillary leak both locally and systemically causes complications from edema and fluid overload.[5] Research efforts have been focused

Disclosure Statement: Nothing to disclose.
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Crit Care Clin ■ (2016) ■–■
http://dx.doi.org/10.1016/j.ccc.2016.06.001 criticalcare.theclinics.com
0749-0704/16/$ – see front matter 

on resuscitation to ameliorate burn shock, multiorgan failure, and the surge of inflammatory mediators to improve morbidity and mortality. The inflammatory mediators of burned skin trigger a systemic response that alters cardiovascular function, including macrocirculation and microcirculation, and pulmonary, hepatic, renal, gastrointestinal, and endocrine function. This article focuses on the current understanding of the pathophysiology of burn shock, the inflammatory response, and the direction of research and targeted therapies to improve resuscitation, morbidity, and mortality. Despite improvements in critical care and burn survival, a recent study showed that the greatest burden of mortality from burn is still burn shock and failure of initial resuscitation.[1] Of those who died of thermal injury, 55% died within 72 hours, 36% died of burn shock defined as acute functional hypoperfusion, and 28% died of subsequent multiorgan failure or sepsis.

## PROGRESSION OF BURN SHOCK AND MULTIORGAN DYSFUNCTION

Shock is defined as inadequate oxygen delivery to meet the metabolic demands of the tissues; this leads to organ dysfunction and eventually failure. Mortalities increase with the number of failing organ systems. The development of multiorgan dysfunction syndrome (MODS) due to sepsis is well established; it is also clear that systemic inflammatory responses can lead to MODS in the absence of infection.[6]

The physiology of burn injury is well recognized as a model of sterile shock and a clear cause for the development of MODS, distinguishing systemic inflammatory response syndrome (SIRS) from sepsis.[7] A study of thermally injured patients was performed evaluating the severity of organ failure with outcomes. An organ failure scale was applied to 529 burn patients; the scale ranged from 0 to 6 (0, no organ dysfunction; 6, organ failure) for each of 6 organ systems. Of the 93.7% of patients who survived, the mean organ failure score was 3.2 compared with 23.1 in nonsurvivors. A mortality of approximately 50% correlated with an organ failure score of greater than 14.[8] In the pediatric burn population, a study was conducted to assess the incidence of multiple organ failure and outcomes. Denver 2 scores were used to assess the degree of organ dysfunction. Increased burn size and depth of burn were correlated with increased incidence of organ failure as well as increased incidence of infection and sepsis. Respiratory failure was most common, followed by cardiac, hepatic, and renal failure. Although the incidence of hepatic or renal failure may be less, this resulted in mortality more frequently.[9] Lactic acidosis and elevated base deficit (BD) levels are well-established markers of inadequate tissue oxygenation and progressive organ dysfunction and portend worse outcomes in the critically ill. In a study of 38 severely burned patients with mean total burn surface area (TBSA) of 36% $\pm$ 15%, a BD less than $-6$ mmol/L in the first 24 hours after burn correlated with increased incidence of acute respiratory distress syndrome (ARDS) and MODS.[10] Another study evaluating BD and lactate levels during burn resuscitation confirmed increased mortality with increased BD and lactate derangement. As well, they noted that normalization of BD within 24 hours after burn injury correlated with improved outcomes compared with those with prolonged elevated BD.[11] These findings have been corroborated in other studies in burn patients.[12,13] Therefore, it can be concluded that increased tissue hypoxia may represent underresuscitation and result in increased organ dysfunction and higher mortalities.

## BURN EDEMA AND HYPOVOLEMIA

Burn shock results from the loss of intravascular fluid, because proteins and plasma are sequestered in burned and nonburned tissues. Observed fluid shifts triggered