Acute Fluid Management of Large Burns

Pathophysiology, Monitoring, and Resuscitation

Justin Gillenwater, MD, MS, Warren Garner, MD*

KEYWORDS

• Fluid resuscitation • Burn shock and burn edema • Colloid • Crystalloid

KEY POINTS

- The systemic inflammatory response caused by large burns requires resuscitation to maintain tissue perfusion.
- While the endpoints of resuscitation are still debated, the mean arterial pressure and hourly urine output remain standard markers for adequate perfusion in most patients.
- Newer technologies may lead to improvements in guided resuscitation, but their use is not yet widespread. More research is required to validate their use in patients with burns.

INTRODUCTION

This article discusses the underlying mechanistic reasons for fluid resuscitation after a burn injury and a suggested algorithm for achieving restoration of normal fluid stasis. Like many important medical treatments, there is much information, consensus on general points, and some remaining controversies. The single most important suggestion is that each patient and injury is unique. The best result is achieved by individualizing treatment to each specific patient.

PATHOPHYSIOLOGY

Overview

The response to burn injury occurs on a local and systemic level. Large burns (>20%) result in release of inflammatory mediators from damaged tissue that can exert their effects on the body as a whole. Predictable alterations of major organ systems are the result, leading to hypovolemic shock in the short term and multiple organ system dysfunction in the subacute setting.

In the first 24 hours after a massive burn, increased vascular permeability leads to migration of water into the interstitium. This decreases the intravascular fluid volume and necessitates replacement to maintain perfusion. If intravascular losses are not replaced, there is generalized and organ-specific hypoperfusion. Cardiac output (CO) is suppressed because of fluid shifts and changes in systemic vascular resistance as catecholamine release occurs on a large scale. Gastrointestinal and renal systems are the first to evidence dysfunction but eventually all organ systems are affected. Electrolyte imbalances are common and intercellular ion shifts occur from cellular death in thermally damaged tissues. Endocrine function is depressed, insulin and cortisol requirements increase, and hyperglycemia is common. The initial massive inflammatory response is followed by a period of immunosuppression. An effective therapeutic response requires knowing

Division of Plastic Surgery, Department of Surgery, Keck School of Medicine, University of Southern California, 1510 San Pablo Street, Suite 415, Los Angeles, CA 90033, USA
* Corresponding author.
E-mail address: wgarner@med.usc.edu

Clin Plastic Surg ■ (2017) ■–■
http://dx.doi.org/10.1016/j.cps.2017.02.008

the physiologic changes that occur locally and systemically.

Local Response: Burn Wound Edema and Tissue Loss

Thermal injury to the skin results in cell death through coagulation, protein denaturation, and cell rupture. Cellular injury and death cause the release of many inflammatory mediators. Histamine, bradykinin, and prostaglandins act locally to promote tissue edema by altering connections in the basement membrane and increasing endothelial cell permeability. This results in transudation of large, osmotically active intravascular proteins out of the capillaries and into the burned tissues. Plasma oncotic pressure is reduced and water is leaked from the microvascular capillary circulation into burned tissue.[1,2] Additionally, interstitial hydrostatic pressure is increased as integrins are broken and cell-to-cell adhesion is disrupted. This leads to exposure of hydrophilic proteoglycans, which further drive water into the interstitial space. The sum total of these interactions leads to profound and immediate edema in burn-injured tissue.

In addition, within thermally damaged tissue, the release of cytokines, such as interleukin-1, interleukin-8, and tumor necrosis factor-α, attracts leukocytes to the wound. Neutrophil degranulation results in release of proteases and reactive oxygen species. Although in small burns this serves as a useful microbicide, in large burns these are cytotoxic to normal tissue. Complement activation occurs, which furthers disruption of dermal microvasculature and perpetuates local tissue ischemia and necrosis.[3,4]

Systemic Response: Burn Shock and Burn Edema

Burns greater than 20% of total body surface area cause a system-wide inflammatory response. The large volume release of inflammatory mediators and cytokines into the circulation leads to leaky microvasculature, vasodilation, and decreased CO. Simplistically, the local response overwhelms the microenvironment and becomes systemic.

As in burned tissue, capillary integrity becomes compromised systemically. Low-flow state coupled with osmotic pressure generated by transudate of proteins and electrolytes results in a profound efflux of intravascular volume into the interstitial space. Hematocrit increases as intravascular volume rapidly decreases. Changes in cell membrane integrity cause additional sequestration of fluid within the cellular space. The result is rapid onset total body edema, with maximal fluid shifts occurring at around 12 hours postburn.[5,6]

In contrast to burned tissue, capillary integrity in nonburned tissue returns to near normal within 24 hours, and transudation of colloids out of the vascular space decreases. However, water continues to collect in the interstitial space in nonburned tissue even after capillary integrity has been restored because of loss of normal oncotic gradient. The loss of plasma proteins into burned tissue is significant enough to decrease vascular oncotic pressure, resulting in ongoing third spacing of water.

Burn shock is multifactorial because of the interplay between loss of intravascular volume, cardiac dysfunction, and vascular changes. Although hypovolemia is common early, vasodilatation also develops, caused by large volume release of inflammatory mediators. Cardiac dysfunction is common in large burns.[7] This can be a primary cardiac dysfunction as a result of massive cytokine release or decreased circulating blood volume from serum loss.[8] These changes in preload, contractility, and after-load can alone or in combination result in low CO and hypoperfusion. The kidney is most vulnerable to damage from burn shock.[9,10] Increased blood viscosity from the elevated hematocrit and myoglobinemia from deeper tissue damage coupled with intravascular volume loss lead to poor perfusion and acute renal failure. Furthermore, injured tissue within the “zone of stasis” dies.[11]

RESUSCITATION

The mainstay of treatment of acute burn shock is providing supportive care with fluid resuscitation until vascular permeability is restored and interstitial fluid losses are minimized. The goal is to maintain end organ perfusion while limiting fluid overload. Overresuscitation has undesirable sequelae, such as conversion of partial-thickness burns to full thickness, pulmonary edema, and abdominal compartment syndrome. There is ongoing debate as to the optimal fluid used for resuscitation, the timing of fluid administration, and the volume of fluid to administer. Similarly, precise end points of resuscitation are controversial. However, two guiding principles are clear. First, resuscitation should involve the least amount of fluid necessary to provide organ perfusion. Second, the resuscitation should be continuously adjusted to prevent overresuscitation and underresuscitation.[3]

Two determinants that guide initial efforts at resuscitation are the size of the burn and the size of the person burned. The larger the burn, the larger the person, the more fluid needed to resuscitate.[12] Multiple formulas have been advocated using these two variables, of which the Parkland