Hemodynamic Monitoring in Sepsis

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KEYWORDS

• Hemodynamic • Monitoring • Sepsis • Goal-directed
• Intensive care • Physiologic

The hemodynamics of sepsis (ie, blood flow and tissue perfusion) is complicated because of the phasic nature of sepsis. Cardiovascular derangements lead to development of tissue hypoperfusion.[1] Tissue hypoperfusion is an important factor in the development of multiple organ failure. Therefore, recognition of sepsis-induced tissue hypoperfusion and timely clinical intervention to prevent and correct this are fundamental aspects of managing patients with sepsis and septic shock. Hemodynamic monitoring plays a key role in the management of the critically ill and is used to identify hemodynamic instability and its cause and to monitor the response to therapy. However, the utility of many forms of hemodynamic monitoring that are commonly used in management of sepsis and septic shock remain controversial and unproven.[2]

RATIONALE FOR HEMODYNAMIC MONITORING

There are an increasing number of different technological advances available that allow monitoring and assessment of a wide range physiologic variables[3]; however, most intensive care (ICU) monitors display only blood pressure (BP), heart rate (HR), and oxygen saturation by pulse oximetry (SpO2). These monitors serve to alert the patient's caregivers to vital signs that require further attention but are not sufficiently sensitive to drive treatment protocols. For example, blood pressure alone is not sufficient in identifying the presence or absence of tissue hypoperfusion in patients with sepsis; patients with sepsis-induced hypoperfusion can present with normal blood pressures.[4,5] It is therefore important to monitor other signs that are indicative of tissue hypoperfusion and hemodynamic instability.[6] Because the primary goal of the cardiovascular system is to supply adequate amounts of oxygen to meet the metabolic demands of the body, calculation of systemic oxygen delivery (DO2) and oxygen consumption (VO2), identifying tissue ischemia (usually monitored by mixed venous

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oxygen saturation [SvO2]) as well as measures of ventricular performance (stroke work) are required to comprehensively understand an individual patient's pathophysiology. Several important points must be remembered about monitoring hemodynamics. First, currently available hemodynamic monitoring only assesses global circulatory status but does not assess the microcirculation or individual organ function.[7,8] This is particularly relevant in sepsis where even with restoration of adequate blood pressure and normal or supranormal cardiac output, signs of tissue hypoperfusion may persist.[9] This may be related to maldistribution of blood flow at the regional (splanchnic, mesenteric, and renal) or microvascular level and/or a cellular inability to use oxygen despite adequate oxygen delivery (cytopathic hypoxia).[10,11] Second, hemodynamic monitoring cannot alone improve outcomes. Therefore, it must be coupled to a treatment that definitively improves outcome. Validation of therapeutic goals in clinical trials has only recently been shown for hemodynamic monitoring–driven protocolized resuscitation in selected high-risk patient groups.[12] The time point during the course of disease when monitoring is applied will also have profound effects on outcome. For example, preoperative optimization of cardiovascular status[13] and emergency department early goal-directed therapy in septic shock[12] reduces morbidity, whereas the same monitoring and treatment applied after injury in unstable patients with existing shock-induced organ injury does not improve outcome.[14–16]

HEMODYNAMIC CHANGES IN SEPSIS

The hemodynamic profile of severe sepsis and septic shock is initially characterized by components of hypovolemic, cardiogenic, and distributive shock.[17] In the early phases of sepsis, increased capillary leak and decreased vasomotor tone will result in a decrease in venous return to the heart.[18] This will result in a decrease in cardiac output.[19] The normal hemodynamic response would be increased sympathetic tone resulting in tachycardia and restoration of mean arterial blood pressure (MAP) toward normal values by reducing unstressed circulatory blood and increased arterial vasomotor tone. Complete restoration in arterial vasomotor tone does not typically occur in sepsis because of a loss of vascular responsiveness.[20] Therefore, normotension can be preserved only by a significant increase in cardiac output and, importantly, the presence of normotension does not ensure hemodynamic stability.[21] This is further compounded by global systemic vasodilation of the resistance vessels of various organ beds further impairing autoregulated blood flow induced by hypotension. The host's ability to respond to the septic insult with an increase in cardiac output is further impeded by cytokines released secondary to the inflammatory response to sepsis directly causing myocardial depression.[18] The end result of these changes is a decrease in stroke volume and ejection fraction.[22]

Fluid therapy will modify this hemodynamic profile.[23,24] Fluid administration can increase venous return, compensating for the increased capillary leak and increased venous capacitance. In the early stages of sepsis, before fluid therapy, patients may present with decreased cardiac output. Fluid therapy will usually result in a hyperdynamic state with a high normal or elevated cardiac output. Rapid restoration of fluid deficits not only modulates inflammation[25–30] but also decreases the need for vasopressor therapy and inotrope support by restoring cardiac output.[20] After adequate restoration of left ventricular preload, hypotension—if present—is dependent on the degree of decreased systemic vascular resistance (SVR) and on impairment of contractility. It is this chronology that forms the basis for the protocol used by Rivers