Endpoints of Resuscitation

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KEYWORDS

• Shock • Resuscitation • Hypoperfusion • Oxygen demand

KEY POINTS

- The state of shock, regardless of cause, essentially consists of oxygen supply not meeting tissue metabolic demands.
- Shock can progress along a continuum of compensatory processes, starting with catecholamine-based vasoconstriction, progressing to tachycardia and moderate hypotension, and terminating with loss of vascular autoregulation, cellular hypoxia, and severe metabolic acidosis.
- Various endpoints of resuscitation are in clinical use and can be broadly divided into 2 groups: hemodynamic markers, which provide global information regarding hemodynamic status, and perfusion endpoints, which can be useful indicators of oxygenation at the cellular level.
- Despite the availability of multiple endpoints of resuscitation in clinical use, there is no one single data point or laboratory value that can be used to identify the patient who has been completely resuscitated from the shock state, as each value or technique has its own benefits and limitations.
- At this point, global markers of oxygenation and perfusion, such as central venous oxygen saturation, base deficit, and lactate, are practical and easily available indices that are useful as supplements to experienced clinical assessments during resuscitation.

INTRODUCTION

Definition of Shock

Shock is broadly defined as the abnormal physiologic state in which inadequate tissue oxygenation does not meet metabolic demands. As early as 1872, shock was described by Gross[1] as a "manifestation of the crude unhinging of the machinery of life" as well as "a peripheral circulatory failure, resulting from a discrepancy in the size of the vascular bed and the volume of the intravascular fluid" by Blalock in 1937.[2]

In an attempt to better define the shock state, the 2006 International Consensus Conference on hemodynamic monitoring in shock defined it as a "failure to deliver

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and/or utilize adequate amounts of oxygen."[3] Using degrees of derangements in physiologic parameters such as heart rate, blood pressure, and urine output to classify hypovolemic shock into 4 stages, the American College of Surgeons Advanced Trauma Life Support manual broadly defines shock as "an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation.[4]

Although multiple causes of shock exist, including hypovolemic/traumatic, cardiogenic, septic, and neurogenic, all shock states have the common abnormality of oxygen supply not meeting tissue metabolic demands.

Compensated and Uncompensated Shock

The body's response to shock involves several stages of compensation. These compensatory stages proceed sequentially, starting with a phase of catecholamine-mediated vasoconstriction during which blood is diverted toward the heart and brain and away from other organs and tissues. The combination of catecholamines and fluid shifts from extracellular regions to the intravascular compartment helps to maintain blood pressure during these early stages of shock. Although patients may have lost up to 15% of blood volume in the setting of bleeding, they may still exhibit a normal pulse, blood pressure, and other physiologic parameters because of these compensatory mechanisms.

As shock progresses, the patient will then enter a state of partial compensation exhibited by mild to moderate hypotension, tachycardia, and a decreased pulse pressure. As the metabolic needs of tissues are not met, an imbalance between oxygen delivery (DO_2) and oxygen utilization occurs, resulting in oxygen debt. Because of this lack of oxygen, tissues activate anaerobic metabolism to produce energy, and anaerobic byproducts such as lactate are released. Ultimately, this is followed by a state of decompensation, or uncompensated shock, in which autoregulation of vascular beds fails, blood flow becomes pressure dependent, and cellular hypoxia leads to significant metabolic acidosis.

Resuscitation

Resuscitation from shock involves the restoration of normal physiology, particularly at the cellular level. Despite the achievement of normal physiologic values, persistent hypoxemia, lactic acidosis, and anaerobic metabolism may still exist. Physiologic parameters, which are typically used to monitor hemodynamic and oxygenation status, such as heart rate, blood pressure, urine output, and blood gases, can be normal in the setting of tissue hypoxia and cannot be used to rule out imbalances between oxygen supply and demand.[5] Dysoxia and capillary flow abnormalities may well be present despite normalization of these traditional markers. Physiologic markers, or endpoints of resuscitation, have therefore been sought to better guide resuscitative efforts in the setting of shock.

Endpoints of Resuscitation

Various endpoints have been identified to better guide resuscitation in the setting of shock. These endpoints can be broadly divided into 2 groups based on the information they provide. Hemodynamic markers, such as arterial blood pressure, central venous pressure (CVP), mixed and central venous oxygen saturation (SvO_2), arterial pulse waveform analysis, and values obtained from echocardiography, can provide global information regarding hemodynamic status. Perfusion endpoints including lactate, base deficit (BD), near-infrared spectroscopy (NIRS), and gastric or sublingual tonometry can be useful indicators of oxygenation at the cellular level (**Table 1**).