Shock and Monitoring

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SHOCK CLASSIFICATION

The most appropriate definition of shock should focus on the concept of inadequate cellular oxygenation affecting multiple organ systems. Inadequate delivery of oxygen to the tissues leads to "dysoxia". Anaerobic metabolism becomes the only source of energy for the cells. Lactate is produced, leading to a systemic lactic acidosis. Multiple inflammatory cascades become activated, leading to multiple organ system dysfunction syndrome (MODS) and death.

Beyond vascular integrity, the first, and most important, component of the cardiovascular system is intravascular volume. The next key component is the heart. Cardiac output is determined by preload, heart rate, contractility, and afterload. Regarding the vasculature, arterioles contribute the most to vascular resistance. Increase or decrease in arteriolar tone can impact blood pressure as well as regional distribution of blood flow. Autoregulation of blood flow allows organs to maintain normal blood flow over a wide range of mean arterial pressures, typically 60 to 130 mm Hg. Autoregulation may be impaired in a variety of disease states, including chronic hypertension or sepsis.

Shock can be hypodynamic, with low cardiac output and narrow pulse pressure, or hyperdynamic, with a low systemic vascular resistance, widened pulse pressure, and high cardiac output. The differential diagnosis for a hypodynamic shock state includes hypovolemia (exemplified by hemorrhagic shock), cardiogenic shock (as in an acute myocardial infarction), and obstructive shock (e.g., pulmonary embolism, cardiac tamponade, and tension pneumothorax). Examples of hyperdynamic shock, also referred to as distributive or vasodilatory shock, include sepsis, anaphylaxis, adrenal crisis, liver failure, neurogenic shock, and medications.

Shock represents a state in which tissue oxygen demands are not met by oxygen delivery. Most commonly, disturbances in the oxygen supply/demand ratio are caused by changes in oxygen delivery, i.e., hemoglobin concentration, oxygen saturation, or cardiac output. As oxygen delivery decreases, the tissue extraction ratio increases (from a normal level of 20%, up to 60%) to maintain normal oxygen consumption. The point of critical oxygen delivery is when oxygen consumption becomes supply dependent, leading to "dysoxia", lactic acidosis, oxygen debt, organ dysfunction and death.

Cytopathic hypoxia refers to a state in which cells have adequate oxygen delivery but are unable to appropriately utilize this oxygen because of impaired mitochondrial oxygen utilization.[1, 2]

During ischemia, ATP is converted to hypoxanthine. At the same time, xanthine dehydrogenase is converted to xanthine oxidase. With reperfusion, oxygen becomes available to the cells. Oxygen free radicals are then formed and lipid mediators are released, causing additional tissue injury. [3]

INITIAL MANAGEMENT

The initial approach to any patient in shock should follow the ABC's; airway, breathing, and circulation need to be addressed. Fluid resuscitation, inotropic and/or inotropic support may be needed. Simultaneous with the initial resuscitation, definitive therapy should be sought. For example, control of bleeding, cardiac revascularization, or septic source control.

MONITORING

Once initiated, therapy for shock must be titrated utilizing appropriate monitoring. Assuring adequate intravascular volume (preload) should always be the first step in resuscitation. Central venous pressure and pulmonary artery occlusion pressure are poor predictors of fluid responsiveness. Functional hemodynamic monitoring approaches take advantage of changes in intra-thoracic pressure caused by positive pressure ventilation. As patients become more hypovolemic, pulse pressure variability increases.[4] Similarly, echocardiography can demonstrate variability in the diameter of the venae cava.

A separate, but effective, approach involves observing the hemodynamic response to passive leg raising.[5]

Adequacy of oxygen delivery to tissues can be assessed by measurement of mixed venous oxygen saturation, which decreases as oxygen delivery decreases or oxygen demands increase. Inadequate oxygen delivery also leads to anaerobic metabolism, which can be monitored via base deficit or lactate levels.

Optimal management of the patient in shock requires rapid initiation of resuscitation, often simultaneous with diagnostic studies. Additional interventions may be needed to manage the underlying cause of shock. Use of appropriate monitoring can help guide therapy.

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