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MODERN APPROACHES TO THE PATHOPHYSIOLOGY AND CLASSIFICATION OF SHOCK IN CRITICAL CARE

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The word “shock” is derived from the French “choc,” meaning a clash or collision between two opposing forces. Historically, numerous definitions of shock have been proposed. The current understanding is largely based on Alfred Blalock’s theory, which defines shock as a disorder of blood volume.

Global hypoperfusion leads to an imbalance between oxygen supply and demand, resulting in insufficient ATP synthesis, anaerobic respiration, and lactic acidosis. Therefore, shock is a life-threatening medical emergency characterized by inadequate delivery of oxygen-rich blood to tissues and organs. Shock leads to a dangerous drop in blood pressure and reduced blood flow. The underlying pathophysiological mechanism of shock is widespread circulatory failure resulting in inadequate tissue perfusion and oxygen delivery.

Shock can be classified into four main types: hypovolemic, cardiogenic, obstructive, and distributive. The distributive category includes septic, anaphylactic, and neurogenic shock. Among these, septic shock is the most common in intensive care units, followed by cardiogenic and hypovolemic shock. When oxygen supply becomes inadequate, the body initiates several adaptive mechanisms to maintain perfusion pressure and oxygen delivery. This compensatory period is known as compensated shock, during which early clinical signs may be detected. The initial response to hypoperfusion involves activation of the baroreceptor reflex and the renin–angiotensin–aldosterone system. Aldosterone acts on the principal cells of the renal collecting tubules to increase sodium reabsorption, resulting in fluid retention and improved cardiac output. When compensatory mechanisms fail and oxygen delivery (DO_2) declines beyond a critical threshold, shock progresses to the uncompensated stage. Prolonged hypoxia and anaerobic metabolism cause rapid clinical deterioration. The final stage, termed irreversible or refractory shock, is typically associated with multiple organ dysfunction syndrome (MODS) and carries a mortality rate of 96–99%. A common mechanism underlying MODS is ischemia–reperfusion injury, which contributes significantly to mortality among intensive care patients.

Recent advancements in understanding shock pathogenesis emphasize the critical role of microcirculatory dysfunction and endothelial injury**,** even when traditional "macro" measures like blood pressure appear stable. This has led to the development of new diagnostic and therapeutic strategies focused on the cellular and microvascular levels.

Keywords: shock, hypoperfusion, oxygen supply, hypoxia, intensive care