AV, Atrioventricular; *BP*, blood pressure; *CHD*, congenital heart disease; *CVP*, central venous pressure; *SVT*, supraventricular tachycardia.

Pathophysiology

A healthy child's circulatory system is able to transport oxygen and metabolic substrates to body tissues, which require a constant source for these essential needs. The cardiac output and distribution to the various body tissues can change rapidly in response to intrinsic (myocardial and intravascular) or extrinsic (neuronal) control mechanisms. In shock states, these mechanisms are altered or challenged.

Reduced blood flow, as in hypovolemic shock, causes diminished venous return to the heart, low CVP, low cardiac output, and hypotension. Vasomotor centers in the medulla are signaled, causing a compensatory increase in the force and rate of cardiac contraction and constriction of arterioles and veins, thereby increasing peripheral vascular resistance. Simultaneously, the lowered blood volume leads to the release of large amounts of catecholamines, antidiuretic hormone, adrenocorticosteroids, and aldosterone in an effort to conserve body fluids. This causes reduced blood flow to the skin, kidneys, muscles, and viscera to shunt the available blood to the brain and heart. Consequently, the skin feels cold and clammy, there is poor capillary filling, and glomerular filtration rate and urinary output are significantly reduced.

As a result of impaired perfusion, oxygen is depleted in the tissue cells, causing them to revert to anaerobic metabolism, producing lactic acidosis. The acidosis places an extra burden on the lungs as they attempt to compensate for the metabolic acidosis by increasing the respiratory rate to remove excess carbon dioxide. Prolonged vasoconstriction results in fatigue and atony of the peripheral arterioles, which leads to vessel dilation. Venules, which are less sensitive to vasodilator substances, remain constricted for a time, causing massive pooling in the capillary and venular beds, which further depletes blood volume.

Complications of shock create further hazards. CNS hypoperfusion may eventually lead to cerebral edema, cortical infarction, or intraventricular hemorrhage. Renal hypoperfusion