Vasopressor and Inotropic Support If intravascular volume status has been optimized with volume resuscitation but hypotension and inadequate tissue perfusion persist, then vasopressor and inotropic support should be initiated. The use of vasopressors and inotropes must be tailored to the primary physiologic disturbance. The clinician must understand the receptor selectivity of various agents and that for some agents the selectivity may be dose-dependent. In patients with distributive shock, the aim is to increase the SVR. Norepinephrine is the first-choice vasopressor, with potent α_1 and β_1 adrenergic effects. The α_1 causes vasoconstriction while β_1 has positive inotropic and chronotropic effects. At high doses, epinephrine has a similar profile (at lower doses the β effects predominate) but is associated with tachyarrhythmia, myocardial ischemia, decreased splanchnic blood flow, pulmonary hypertension, and acidosis. In distributive shock, vasopressin deficiency may be present. Vasopressin acts on the vasopressin receptor to reverse vasodilation and redistribute flow to the splanchnic circulation. In a randomized trial in patients with septic shock, the addition of low-dose vasopressin did not reduce all-cause 28-day mortality compared to norepinephrine. Vasopressin is safe and has a role as a second agent for hypotension in septic shock. Dopamine does not have a role as a first-line agent in distributive shock. A randomized control study in patients with all-cause circulatory shock did not show a survival benefit but did reveal an increase in adverse events (arrhythmia). In this study, the subgroup of patients with cardiogenic shock had increased mortality. For patients with cardiogenic shock, dobutamine is the first-line agent; it is a synthetic catecholamine with primarily β-mediated effects and minimal α adrenergic effects. The β_1 effect is manifest in increased inotropy and the β_2 effect leads to vasodilation with decreased afterload; it can be used with norepinephrine in patients with mixed distributive and cardiogenic shock.

■ OXYGENATION AND VENTILATION SUPPORT

In addition to the cellular hypoxia caused by circulatory failure, patients with shock may present with hypoxemia. For patients with