signs of shock may safely receive, and usually benefit greatly from, a 20-mL/kg bolus of an isotonic crystalloid over 5 to 15 minutes. This dose may be repeated until a response is noted. Colloids contain larger molecules that may stay in the intravascular space longer than crystalloid solutions and exert oncotic pressure, drawing fluid out of the tissues into the vascular compartment. However, long-term risks of colloids may exceed benefits. Care must be exercised in treating cardiogenic shock with volume expansion because the ventricular filling pressures may rise without improvement of the cardiac performance. Carefully monitoring cardiac output or central venous pressure guides safe volume replacement.

Cardiovascular Support

In an effort to improve cardiac output after volume resuscitation or when further volume replacement may be dangerous, a variety of inotropic and vasodilator drugs may be useful (Table 40-2). Therapy is directed first at increasing myocardial contractility, then at decreasing left ventricular afterload. The hemodynamic status of the patient dictates the choice of the agent.

Therapy may be initiated with dopamine at 3 to 15 mcg/kg/min; however, epinephrine or norepinephrine may be preferable in patients with decompensated shock. In addition to improving contractility, certain catecholamines cause an increase in systemic vascular resistance. The addition of a vasodilator drug may improve cardiac performance by decreasing the resistance against which the heart must pump (afterload). Afterload reduction may be achieved with dobutamine, milrinone, amrinone, nitroprusside, nitroglycerin, and angiotensin-converting enzyme inhibitors. The use of these drugs may be particularly important in late shock, when vasoconstriction is prominent.

Respiratory Support

The lung is a target organ for inflammatory mediators in shock and SIRS. Respiratory failure may develop rapidly and become progressive. Intervention requires endotracheal intubation and mechanical ventilation accompanied by the use of supplemental oxygen and PEEP. Care must be taken with the process of intubation, because a child with compensated shock may suddenly decompensate on administration of sedative medications that reduce systemic vascular resistance. Severe cardiopulmonary failure may be managed with inhaled nitric oxide and, if necessary, extracorporeal membrane oxygenation.

Renal Salvage

Poor cardiac output accompanied by decreased renal blood flow may cause prerenal azotemia and oliguria/anuria. Severe hypotension may produce **acute tubular necrosis** and **acute renal failure.** Prerenal azotemia is corrected when blood volume deficits are replaced or myocardial contractility is improved, but acute tubular necrosis does not improve immediately when shock is corrected. Prerenal azotemia is associated with a serum BUN-to-creatinine ratio of greater than 10:1 and a urine sodium level less than 20 mEq/L; acute tubular necrosis has a BUN-to-creatinine ratio of 10:1 or less and a urine sodium level between 40 and 60 mEq/L (see Chapter 165). Aggressive fluid replacement is often necessary to improve oliguria associated with prerenal azotemia. Because the management of shock requires administering large volumes of fluid, maintaining urine output greatly facilitates patient management.

Prevention of acute tubular necrosis and the subsequent complications associated with acute renal failure (hyperkalemia, acidosis, hypocalcemia, fluid overload) is important. The use of pharmacologic agents to augment urine output is indicated when the intravascular volume has been replaced. The use of loop diuretics, such as furosemide, or combinations of a loop diuretic and a thiazide agent may enhance urine output. Infusion of low-dose dopamine, which produces renal artery vasodilation, also may improve urine output. Nevertheless, if hyperkalemia, refractory acidosis, hypervolemia, or altered mental status associated with uremia occurs, dialysis or hemofiltration should be initiated.

COMPLICATIONS

Shock results in impairment of tissue perfusion and oxygenation and activation of inflammation and cytokine pathways. The major complication of shock is multiple organ system failure, defined as the dysfunction of more than one organ, including respiratory failure, renal failure, liver dysfunction, coagulation abnormalities, or cerebral dysfunction. Patients with shock and multiple organ failure have a higher mortality rate and, for survivors, a longer hospital stay.

PROGNOSIS

Early recognition and **goal-directed intervention** in patients with shock improve survival. However, delays in treatment of hypotension increase the incidence of multiple organ failure and mortality. Goal-directed therapy focused on maintaining mixed venous oxygen saturation may improve survival.

Table 40-2 Medications Used to Improve Cardiac Output

	POSITIVE INOTROPE	POSITIVE CHRONOTROPE	DIRECT PRESSOR	VASOCONSTRICTOR	VASODILATOR
Dopamine	++	+	±	++ (high dose)	+ (low dose)*
Dobutamine	++	±	–	–	+
Epinephrine	+++	+++	+++	++ (high dose)	+ (low dose)†
Norepinephrine	+++	+++	+++	+++	–
Milrinone	+	-	-	-	+

*Primarily splanchnic and renal in low doses (3–5 mcg/kg/min).
†Low dose (<0.3 mcg/kg/min).