- *Neurogenic shock* results from inadequate sympathetic nervous system (SNS) stimulation. The SNS normally maintains sufficient vascular tone (ie, a small amount of vasoconstriction) to support adequate blood circulation. Neurogenic shock may occur with depression of the vasomotor center in the brain or decreased sympathetic outflow to blood vessels.
- *Septic shock* can result from almost any organism that gains access to the bloodstream but is most often associated with gram-negative and gram-positive bacterial infections and fungi.

It is important to know the etiology of shock because management varies among the types. The types of shock, with their causes and symptoms, are summarized in Table 54–1.

ANTISHOCK DRUGS

Drugs used in the management of shock are primarily the adrenergic drugs, which are discussed more extensively in Chapter 18. In this chapter, the drugs are discussed only in relation to their use in hypotension and shock. In these conditions, drugs with alpha-adrenergic activity (eg, norepinephrine, phenylephrine) are used to increase peripheral vascular resistance and raise blood pressure. Drugs with beta-adrenergic activity (eg, dobutamine, isoproterenol) are used to increase myocardial contractility and heart rate, which in turn raises blood pressure. Some drugs have both alpha- and beta-adrenergic activity (eg, dopamine, epinephrine). In many cases, a combination of drugs is used, depending on the type of shock and the client's response to treatment. In an emergency, the drugs may be used to maintain adequate perfusion of vital organs until sufficient fluid volume is replaced and circulation is restored.

Adrenergic drugs with beta activity may be relatively contraindicated in shock states precipitated or complicated by cardiac dysrhythmias. Beta-stimulating drugs also should be used cautiously in cardiogenic shock after myocardial infarction because increased contractility and heart rate will increase myocardial oxygen consumption and extend the area of infarction.

Individual drugs are described in the following section; indications for use and dosage ranges are listed in Drugs at a Glance: Drugs Used for Hypotension and Shock.

TABLE 54–1 **Types of Shock**

Types of Shock	Possible Causes	Clinical Manifestations
Hypovolemic	Trauma Gastrointestinal bleed Ruptured aneurysms Third spacing Dehydration	Hypotension Tachycardia Cool, clammy skin Diaphoresis Pallor Oliguria
Cardiogenic	Acute myocardial infarction Cardiac surgery Dysrhythmias Cardiomyopathy	Signs and symptoms of heart failure Signs and symptoms of decreased cardiac output
Distributive		
Neurogenic	Spinal cord damage Spinal anesthesia Severe pain Drugs	Hypotension Bradycardia Warm, dry skin
Septic	Infection (eg, urinary tract, upper respiratory infections) Invasive procedures	Hypotension Cool or warm, dry skin Hypothermia or hyperthermia
Anaphylactic	Contrast dyes Drugs Insect bites Foods	Hypotension Hives Bronchospasm

INDIVIDUAL DRUGS

Dopamine is a naturally occurring catecholamine that functions as a neurotransmitter. Dopamine exerts its actions by stimulating alpha, beta, or dopaminergic receptors, depending on the dose being used. In addition, dopamine acts indirectly by releasing norepinephrine from sympathetic nerve endings and the adrenal glands. Peripheral dopamine receptors are located in splanchnic and renal vascular beds. At low doses (0.5 to 10 mcg/kg/min), dopamine selectively stimulates dopaminergic receptors that may increase renal blood flow and glomerular filtration rate (GFR). It has long been accepted that stimulation of dopamine receptors by low doses of exogenous dopamine produces vasodilation in the renal circulation and increases urine output. More recent studies indicate that low-dose dopamine enhances renal function only when cardiac function is improved. At doses greater than 3 mcg/kg/min, dopamine binds to beta and alpha receptors and the selectivity of dopaminergic receptors is lost beyond 10 mcg/kg/min. At doses that stimulate beta receptors (3 to 20 mcg/kg/min), there is an increase in heart rate, myocardial contractility, and blood pressure. At the highest doses (20 to 50 mcg/kg/min), beta activity remains, but increasing alpha stimulation (vasoconstriction) may overcome its actions.

Dopamine is useful in hypovolemic and cardiogenic shock. Adequate fluid therapy is necessary for the maximal pressor effect of dopamine. Acidosis decreases the effectiveness of dopamine.

Dobutamine is a synthetic catecholamine developed to provide less vascular activity than dopamine. It acts mainly on $beta_1$ receptors in the heart to increase the force of myocardial contraction with a minimal increase in heart rate. Dobutamine also may increase blood pressure with large doses. It is less likely to cause tachycardia, dysrhythmias, and increased myocardial oxygen demand than dopamine and isoproterenol. It is most useful in cases of shock that require increased cardiac output without the need for blood pressure support. It is recommended for short-term use only. It may be used with dopamine to augment the $beta_1$ activity that is sometimes overridden by alpha effects when dopamine is used alone at doses greater than 10 mcg/kg/min.

Dobutamine has a short plasma half-life and therefore must be administered by continuous IV infusion. A loading