

## Diagnosis and Management of Shock

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### OBJECTIVES

1. Identify the four major categories of shock and outline management principles.
3. Discuss goals of fluid resuscitation in shock.
4. Describe the physiologic effects of vasopressors and inotropic agents.
5. Understand the concepts of oxygen supply and demand in managing shock.
6. Discuss the differential diagnosis of oliguria.

### I. DEFINITIONS

Shock is not a single disease entity, but rather the common end result of a variety of causes. Therefore, it will be assumed that the diagnosis and treatment of that primary cause will be vigorously pursued. Shock is characterized by blood flow that is inadequate to meet tissue oxygen demand, as discussed in Chapter 6. Prompt recognition of hypotension and hypoperfusion (inadequate organ blood flow) is essential for timely treatment and improved outcome. In adults, a systolic blood pressure <90 mm Hg, a mean arterial pressure <60 mm Hg, or a decrease in systolic blood pressure of >40 mm Hg from the patient's baseline pressure constitutes significant hypotension. However, hypoperfusion may be present in the absence of significant hypotension. Shock is hypotension with associated hypoperfusion abnormalities. Evidence of hypoperfusion includes mental status changes, oliguria, or lactic acidosis. Hypoperfusion may lead to organ dysfunction or death. Management should be directed towards correcting hypoperfusion, not hypotension, as the primary endpoint.

### II. SHOCK

There are four major categories of shock: cardiogenic, hypovolemic, distributive, and obstructive. A careful history and physical examination often provide information that is helpful in determining the likely cause of shock. Although not mandated as a management tool, data gathered by means of a pulmonary artery catheter allow classification and understanding of the etiology of shock through distinctive hemodynamic profiles (Table 7-1). Left ventricular filling pressure is assessed by pulmonary artery occlusion pressure (PAOP). Cardiac output and mean arterial pressure (MAP) (see Chapter 6) are used to calculate systemic vascular resistance (SVR). Knowledge of these hemodynamic profiles is helpful in the diagnosis and management of shock.

#### A. Cardiogenic Shock

In cardiogenic shock, forward blood flow is inadequate because of a primary defect in cardiac function. The typical hemodynamic pattern shows decreased cardiac output, high left ventricular filling pressures, increased SVR, and decreased left ventricular stroke work (LVSW).

Table 7-1. Classification of shock by hemodynamic profiles

Type of Shock	Pulmonary Artery Occlusion Pressure	Cardiac Output	Systemic Vascular Resistance
Cardiogenic shock	↑	↓	↑
Hypovolemic shock	↓	↓	↑
Distributive shock	↓ or nl	↑ or nl or ↓	↓
Obstructive shock			
Cardiac tamponade	↑	↓	↑
Pulmonary embolus	↓ or nl*	↓	↑

\* May be technically difficult to obtain; nl, normal.

**B. Hypovolemic Shock**

Hypovolemic shock occurs when intravascular volume is depleted as a result of hemorrhage, vomiting, diarrhea, dehydration, or third-space losses. The hemodynamic findings in hypovolemic shock are decreased cardiac output, decreased left ventricular filling pressure, and an increased SVR.

**C. Distributive Shock**

In distributive shock, several different causes result in a loss of peripheral vascular tone with the effect of relative hypovolemia. The hemodynamic profile is usually characterized by a normal or increased cardiac output with a low SVR and low to normal left ventricular filling pressures. The most common form of distributive shock is septic shock. Other forms of distributive shock include anaphylactic shock, acute adrenal insufficiency, and neurogenic shock.

**D. Obstructive Shock**

The common feature in obstructive shock is the impedance of adequate cardiac filling. The hemodynamic profile is characterized by decreased cardiac output, increased SVR, and variable left ventricular filling pressures, depending on the etiology. In cardiac tamponade, the pressures of the right heart chambers, the pulmonary artery, and the left heart chambers equilibrate in diastole. A drop of >10 mm Hg in systolic blood pressure during inspiration (pulsus paradoxus) is an important clinical finding in patients with suspected cardiac tamponade. Other forms of obstructive shock include tension pneumothorax and massive pulmonary embolus.

*Note: The increased SVR present in cardiogenic, hemorrhagic, and obstructive shock is the body's attempt to maintain blood pressure (perfusion pressure) by increasing arteriolar tone.*

***For Pediatric Patients***

In hypovolemic children, blood pressure is maintained longer than it is in adults, and blood pressure is not a good indicator of the state of perfusion. The child's capillary refill and extremity temperature are much more reliable indicators of hypovolemia, since these parameters may become abnormal much earlier than blood pressure in the child with shock.

Obstructive shock in infants is commonly caused by congenital lesions that interfere with outflow from the left ventricle, such as coarctation of the aorta or interrupted aortic arch. These infants develop signs of shock when the ductus arteriosus closes, thus interfering with the delivery of cardiac output to the distal aorta.

### **III. PRINCIPLES OF MANAGEMENT**

The overall goal of shock management is to increase oxygen delivery to the tissues in order to correct the existing perfusion deficit. The treatment of shock centers on increasing cardiac output and optimizing the oxygen content of blood. An increase in blood pressure is beneficial if it translates into improved perfusion of the periphery. These goals are usually accomplished with some combination of adjusting preload, increasing cardiac contractility, optimizing SVR, and assuring adequate oxygenation of sufficient hemoglobin. Pulse abnormalities, such as bradycardia or other abnormal rhythms, may cause or contribute to hypotension and must be treated. A urinary catheter should be inserted to monitor urine output as an indicator of renal perfusion. Clinical and laboratory data should be collected with the goal of assessing adequacy and improvement of perfusion. Specific treatment should be directed to correcting the causative defect.

#### **A. Cardiogenic Shock**

The primary goal in treating cardiogenic shock is to improve myocardial function. Arrhythmias should be treated promptly. Diastolic dysfunction during myocardial ischemia may decrease ventricular compliance and elevate the left ventricular end-diastolic pressure (LVEDP) and PAOP, falsely indicating adequate preload. Therefore, a cautious trial of fluid administration may be warranted. The elevated SVR may also impair cardiac output if it is a primary hemodynamic alteration as occurs in chronic congestive heart failure. Often in acute cardiogenic shock, the SVR is secondarily elevated to maintain vascular perfusion pressure. Treatment aimed primarily at reducing afterload with a vasodilator, such as nitroprusside, should be initiated very cautiously and only in patients with minimal hypotension. Intravenous inotropes, such as dobutamine (or dopexamine, which is available in some countries), are indicated to augment myocardial contractility in the presence of normal or slightly decreased blood pressure and may have a secondary beneficial effect of decreasing SVR. The driving pressure for coronary artery perfusion is aortic diastolic pressure (coronary artery perfusion occurs primarily during diastole). A low aortic diastolic pressure may be cautiously increased with vasopressors. However, when blood pressure is severely decreased in cardiogenic shock, initial therapy with a single agent that has inotropic and vasopressor effects (e.g., norepinephrine or high-dose dopamine) is indicated. The addition of dobutamine may be considered after blood pressure stabilizes, with the goal of decreasing vasopressor therapy. If moderate hypotension is not responsive to initial therapy, consultation should be obtained for consideration of intra-aortic balloon counterpulsation, left/right ventricular assist devices, etc.

When cardiac failure is characterized by low cardiac output, normal or elevated blood pressure, and hypoxemia due to high pulmonary capillary pressure, preload and afterload reduction are helpful in improving hypoxemia. High pulmonary capillary pressure is diagnosed clinically or approximated by the PAOP if a pulmonary artery catheter is in place. Preload reduction is accomplished with loop diuretics (furosemide or bumetanide) and venodilators (nitroglycerin and morphine). Afterload reduction is accomplished with arterial vasodilators (angiotensin-converting enzyme inhibitors or, occasionally, nitroprusside). Afterload and preload reduction should be avoided in cardiac failure when hypotension is present. If the blood pressure can be increased to normal levels with inotropes, then the cautious addition of afterload and preload reduction is feasible in the presence of low cardiac output or high pulmonary capillary pressure, respectively.

### **B. Hypovolemic Shock**

Restoration of intravascular volume and prevention of further volume loss are the treatment goals for hypovolemic shock. Therapy of hypovolemic shock should be targeted to re-establish normal blood pressure, pulse, and organ perfusion. For initial resuscitation, either colloid or crystalloid fluids are effective if given in sufficient volume. Subsequently, the fluid that is used should replace the fluid that has been lost. For example, blood products may be needed to replace blood loss (see also Chapter 9), and crystalloid should be used for vomiting and dehydration. For hypotension, the crystalloid choice is normal saline or lactated Ringer's solution because of the osmolality needed to restore intravascular volume. In large-volume resuscitation, however, normal saline infusion may produce hyperchloremic metabolic acidosis. Dextrose 5% in water does not offer any expansion of intravascular volume, since it is quickly distributed throughout body fluid compartments and should not be used to treat hypovolemic shock. For the same reasons, 0.45% saline is also less effective than 0.9% saline for volume expansion. Colloid solutions (5% albumin and hetastarch) offer the most efficient intravascular volume expansion, but at increased cost and with no documented survival benefit.

### **C. Distributive Shock**

The initial approach to the patient with septic shock is restoration and maintenance of adequate intravascular volume. Volume expansion is usually initiated with isotonic crystalloid solutions. Prompt institution of appropriate antibiotics is essential, as is a search for a focus of infection requiring surgical intervention. Vasodilation and diffuse capillary leak are common in septic shock, and fluid requirements may be very large. Fluid input and output volumes are therefore not helpful in determining aggressiveness of volume resuscitation, since these volumes do not reflect fluid movement into the extravascular space. If the patient with septic shock remains hypotensive despite adequate fluid resuscitation, inotropes and/or vasopressors are necessary. Unless the MAP is <60 mm Hg, inotropes are typically the initial choice. Inotropes generally raise cardiac output and blood pressure, but the increase in cardiac output is more predictable. Either dobutamine (5 to 20  $\mu\text{g}/\text{kg}/\text{min}$ ) or dopamine (5 to 10  $\mu\text{g}/\text{kg}/\text{min}$ ) in this circumstance may be titrated to maintain adequate organ perfusion. An initial MAP <60 mm Hg may require initiation of vasopressor therapy until fluid resuscitation and inotropic therapy are optimized. For combination inotrope and vasopressor effect, dopamine is often initiated at 5  $\mu\text{g}/\text{kg}/\text{min}$  and is increased, if necessary, to 15 to 20  $\mu\text{g}/\text{kg}/\text{min}$ . Norepinephrine at an initial infusion rate of 0.05  $\mu\text{g}/\text{kg}/\text{min}$  is also effective for increasing blood pressure in septic shock. The routine addition of low doses of dopamine to norepinephrine is not recommended but may increase renal blood flow in some patients. No specific agent or combination of agents is known to be superior in the management of septic shock. The goal is not to achieve a specific blood pressure, but to maintain organ perfusion, especially renal and splanchnic beds. Also recall (Chapter

6) that an elevated PAOP may not indicate adequate preload because of decreased left ventricular compliance in sepsis. Inotropic support (dobutamine) should be administered if preload is adequate.

Anaphylactic shock is treated with subcutaneous epinephrine and volume resuscitation. In circumstances of very low blood pressure and poor peripheral perfusion, titrated intravenous epinephrine is indicated. Acute adrenal insufficiency is treated with volume therapy, intravenous corticosteroids, and vasopressors, if needed (see Chapter 12). See Chapter 9 for management of neurogenic shock.

***For Pediatric Patients***

In children, epinephrine may be used in preference to norepinephrine as a vasopressor agent since it increases heart rate, a major determinant of cardiac output in the pediatric patient.

**D. Obstructive Shock**

In the patient with obstructive shock, relief of the obstruction is the treatment of choice. If cardiac tamponade is present, pericardiocentesis may be lifesaving (Appendix 12). Tension pneumothorax must be treated promptly. Maintenance of intravascular volume is vitally important in patients with all forms of obstructive shock. Fluid resuscitation may improve the patient's cardiac output and hypotension temporarily. Diuretics should be avoided. Inotropes or vasopressors have a minimal role in the management of obstructive shock, and these agents provide only temporary improvement, if any.

***For Pediatric Patients***

In neonates and infants with suspected obstructive left heart lesions (metabolic acidosis, absent or poor femoral pulses, absent or poor urine output), prostaglandin infusion should be initiated rapidly to reopen the ductus arteriosus and restore perfusion to the distal aorta. The usual dose is 0.05 to 0.1  $\mu\text{g}/\text{kg}/\text{min}$  as a continuous infusion. The side effects of PGE1 infusion include periodic breathing, apnea, and peripheral vasodilation, so the clinician must be prepared to support ventilation and administer additional fluids. Once the ductus has reopened, it is critical to avoid hyperventilation and hyperoxia. Both of these conditions will lead to preferential pulmonary blood flow through the ductus and will worsen systemic shock and distal perfusion.

**IV. FLUID THERAPY**

The initial therapy for most forms of shock is replacement of intravascular volume. Physical examination may provide valuable information about the intravascular volume status. Diffuse or dependent crackles, as well as distended neck veins, suggest high left ventricular filling pressures, unless acute respiratory distress syndrome or diffuse pneumonia is present. Clear lung fields and flat neck veins suggest a need for additional fluid resuscitation in the hypotensive patient. When preload is inadequate, the nature and degree of fluid deficit must be determined to identify the necessary type of fluid replacement.

## *Fundamental Critical Care Support*

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Intravascular volume deficiency in the patient who is not anemic may be replenished with either crystalloid or colloid solutions. Crystalloid solutions include lactated Ringer's and normal saline. Colloid solutions include hetastarch, albumin (5% dissolved in normal saline), and gelatins (not available in the U.S.). Crystalloids and colloids appear to be equally effective when infused to physiologic endpoints. In the adult, 1 L crystalloid (lactated Ringer's solution or normal saline) in titrated boluses of 250 to 500 mL or 250 mL of colloid may be given initially and repeated as necessary while closely monitoring appropriate parameters. Crystalloids are less expensive than colloids and typically accomplish the same goals. Systematic reviews suggest that crystalloid resuscitation is associated with a lower mortality in trauma patients and that albumin may increase mortality in critically ill patients.

In addition to crystalloid or colloid, packed red blood cells are indicated to increase oxygen-carrying capacity in the patient with significant bleeding or anemia. In many critically ill patients, a hemoglobin level of 7 to 9 g/dL may be adequate. Fresh frozen plasma should only be used for correction of a coagulopathy and not for volume replacement. Priorities in the administration of fluids are resuscitation and then replacement of ongoing losses. As the patient's clinical course continues, the solution that most closely approximates the patient's losses should be used with serum electrolytes guiding therapy.

The first target in fluid resuscitation is correction of hypotension. Once hypotension is corrected, further fluid resuscitation will decrease elevated heart rate and correct hypoperfusion abnormalities, thus achieving the true endpoint of resuscitation. It is important to monitor urine output to judge adequacy of resuscitation. Acceptable targets to gauge correction of hypoperfusion abnormalities include adequate urine output (0.5 to 1 mL/kg/hr), return to baseline mental status, and correction of lactic acidosis. The potential deleterious effect of overly aggressive fluid resuscitation is deterioration of oxygenation owing to an increase in pulmonary capillary pressure resulting in pulmonary edema. Therefore, frequent auscultation of the chest for the presence of crackles and monitoring of PaO<sub>2</sub> or oxyhemoglobin saturation by pulse oximetry should be performed during fluid resuscitation. In the absence of a pulmonary artery catheter, volume therapy can be administered vigilantly in patients with persistent hypotension or hypoperfusion until a significant drop in arterial oxygenation is noted or until hypotension and hypoperfusion are corrected. This approach to volume therapy presents minimal risk in patients with adequate oxygenation.

### ***For Pediatric Patients***

Although hepatomegaly can be a sign of fluid overload in the pediatric patient, it must be viewed with caution. Disease processes common to children (e.g., asthma, respiratory syncytial virus, pneumonitis) can cause lung hyperinflation and displacement of the liver. Other signs of volume overload should also be considered in the evaluation of these patients. If a child with an enlarged liver fails to respond to initial fluid administration, radiologic examination of the chest may help to evaluate heart size. Rales and crackles may occur late in children in the process of developing congestive failure, and a gallop may be difficult to discern because of the rapid heart rate in an infant.

In children, 20 mL/kg of crystalloid may be given initially and repeated as necessary. After 60 mL/kg of crystalloid has been administered, inotropic/vasopressor support should be considered.

## **V. VASOPRESSORS AND INOTROPIC AGENTS**

A variety of vasopressors and inotropic agents are available for the acute management of the patient with shock. The physician should be familiar with the agents most commonly used. Table 7-2 provides usual concentrations and preparation for pediatric patients.

### **A. Dopamine**

Dopamine is one of the more frequently used inotropic/vasopressor agents. Although dose response varies greatly among patients, some generalization can be helpful with regard to dose and expected effect. At low rates of infusion (2 to 3  $\mu\text{g}/\text{kg}/\text{min}$ ), dopamine has modest inotropic and chronotropic effects. At this dose range, dopamine acts on the dopaminergic receptors in the kidney and may increase renal blood flow and urine output. At intermediate rates of infusion (4 to 10  $\mu\text{g}/\text{kg}/\text{min}$ ), dopamine has primarily inotropic effects and loses its effect on the kidney. At higher infusion rates ( $\geq 10$   $\mu\text{g}/\text{kg}/\text{min}$ ), dopamine has significant  $\alpha$ -agonist effects that produce dose-related vasoconstriction. At infusion rates  $\geq 25$   $\mu\text{g}/\text{kg}/\text{min}$ , dopamine usually offers no advantage over norepinephrine, which may offer greater vasopressor effect. Potential adverse effects include arrhythmias and tachycardia.

### **B. Dobutamine**

Dobutamine is a  $\beta$ -adrenergic agonist. Used in doses of 5 to 20  $\mu\text{g}/\text{kg}/\text{min}$ , dobutamine is a potent inotrope and is usually associated with an increase in cardiac output. Arterial blood pressure may remain unchanged, decrease, or increase slightly. Dobutamine must be introduced with care in the hypotensive patient. In the face of inadequate intravascular volume replacement, blood pressure can drop precipitously, and tachycardia may be problematic. This agent has variable chronotropic effects.

### **C. Norepinephrine**

Norepinephrine is a potent  $\alpha$ -adrenergic vasopressor agent. Norepinephrine also has  $\beta$ -adrenergic, inotropic, and chronotropic effects. In adults, the infusion rate of norepinephrine starts at 0.05  $\mu\text{g}/\text{kg}/\text{min}$  and is titrated to desired effects. Norepinephrine is frequently used in combination with lower doses of dopamine to preserve renal blood flow, with unproven clinical impact.

### **D. Epinephrine**

Epinephrine has both  $\alpha$ - and  $\beta$ -adrenergic effects. Epinephrine is a potent inotrope and chronotrope. Doses initially start at 0.1  $\mu\text{g}/\text{kg}/\text{min}$  and can be titrated to desired effects. The epinephrine-induced increase in myocardial oxygen consumption may limit the use of epinephrine in adults, especially in the presence of coronary artery disease. The same is true to a lesser degree with all inotropes and vasopressors, which may increase myocardial oxygen demand above supply and precipitate myocardial ischemia.

*For Pediatric Patients*

Epinephrine is an excellent drug for inotropic and perfusion support in children.

**Table 7-2. Preparation of catecholamine infusions in infants and children**

Drug	Preparation*	Special Instructions
Isoproterenol Epinephrine Norepinephrine	0.6 × body weight (kg) is the mg dose added to make 100 mL	1 mL/hr delivers 0.1 µg/kg/min
Dopamine Dobutamine	6 × body weight (kg) is the mg dose added to make 100 mL	1 mL/hr delivers 1 µg/kg/min

\*This provides a starting dose only; the subsequent concentration can be adjusted to fluid tolerance. According to the above formulas, the amount of drug needed to make 100 mL of fluid to deliver the desired dose at the slow infusion rate of 1 mL/hr may necessitate a high volume of medication. If the volume of medication needed is unusually large (generally a problem only with isoproterenol), it is possible to deplete the available drug supply. To reduce the volume of drug needed, decrease the amount mixed by a factor of 10, and increase the hourly infusion rate by a factor of 10. For example, according to the formula above, a 20-kg child would require 12.0 mg (20 x 0.6) or 60 mL (0.2 mg/mL) of isoproterenol. Instead, use 1.2 mg (6 mL) and infuse the final solution at 10 mL/hr (instead of 1 mL/hr). Adapted from Textbook of Pediatric Advanced Life Support, Dallas, American Heart Association, 1994.

**VI. SHOCK AS A MISMATCH BETWEEN TISSUE OXYGEN DEMANDS AND TISSUE OXYGEN SUPPLY**

During shock, tissue demands for oxygen exceed the supply of oxygen to the tissues. This pathophysiologic condition can occur through several mechanisms. In cardiogenic shock, primary cardiac pump failure is the dominant abnormality. In hypovolemic shock, pump function may be satisfactory, but intravascular volume is insufficient. In septic shock, cardiac output may greatly exceed normal values, but oxygen extraction is deranged and as vasodilation occurs, a “relative” decrease in intravascular volume also may occur. Obstructive shock implicates an extracardiac source of obstruction to cardiac filling, such as cardiac tamponade or tension pneumothorax. With this concept in mind, shock treatment can be formulated by decreasing the demand for oxygen or by increasing oxygen supply. Useful clinical interventions to decrease oxygen demand include intubation of the patient to help support the work of breathing, sedation, analgesia, and treatment of fever. As outlined in Chapter 6, oxygen delivery can be increased by increasing cardiac output, hemoglobin concentration, or oxyhemoglobin saturation. Increasing hemoglobin concentration in the anemic patient is one of the most efficient ways of improving oxygen delivery. For example, increasing the hemoglobin concentration from 7 to 9 g/dL increases oxygen delivery by almost 30%, even if cardiac output remains constant. On the other hand, increasing PaO<sub>2</sub> from 60 to 90 torr (8.0 to 12.0 kPa) only increases the SaO<sub>2</sub> from 88% to 95%. Once the PaO<sub>2</sub> has been raised to a range of 60 to 70 torr (8.0 to 9.3 kPa), little additional benefit is gained by raising the PaO<sub>2</sub> further. The resulting increase in oxygen delivery is only 7%.

Adequacy of cardiac output is best judged by cardiac index (cardiac output/body surface area [BSA]). In the critically ill patient, the goal is not to achieve a normal cardiac index, but to reach an optimal cardiac index for that patient's condition. The same applies for oxygen delivery. Cardiac index and oxygen delivery should be targeted to reverse hypotension, hypoperfusion abnormalities, and shock. For the patient with cardiogenic shock, a cardiac index of 2.5 L/min/m<sup>2</sup> may be satisfactory. For the patient with trauma or sepsis, a cardiac index  $\geq 4.5$  L/min/m<sup>2</sup> may be required. To optimize cardiac index in the hypotensive patient, a PAOP of 12 to 15 mm Hg is a useful clinical goal. Patients with poor left ventricular function and decreased compliance may need a higher PAOP level. Inotropes may be added to improve contractility if necessary. Vasopressors may be necessary with low mean arterial pressure or SVR. Attempts to increase cardiac index and/or oxygen delivery should cease when all abnormalities are reversed or when all efforts prove counterproductive.

## VII. OLIGURIA

Oliguria, defined as urine output  $< 0.5$  mL/kg/hr for  $> 2$  hours, is an important marker of hypoperfusion. Oliguria may also be due to inherent renal injury or postrenal obstruction. These two circumstances prevent the use of urine output as a target of adequate resuscitation of shock. Causes of oliguria are categorized as prerenal, renal, and postrenal (Table 7-3).

**For Pediatric Patients**

For children, urine output  $< 1$  mL/kg/hr defines oliguria. In infants  $< 2$  years old, urine output  $< 2$  mL/kg/hr defines oliguria.

**Table 7-3. Differential diagnosis of oliguria**

### Prerenal

- Decreased cardiac output (e.g., volume depletion, cardiac failure, tamponade)
- Redistribution of blood flow (distributive shock) with peripheral vasodilation and/or shunting

### Renal

- Glomerular disease (glomerulonephritis)
- Vascular disease (e.g., vasculitis)
- Interstitial disease (e.g., antibiotics)
- Renal tubular disease
- Ischemia
- Nephrotoxic drugs

### Postrenal (Obstructive)

- Bilateral ureteral obstruction
- Urethral stricture
- Bladder outlet obstruction
- Urinary catheter obstruction

In the critically ill patient, assessment of volume status by physical examination is often difficult, and invasive hemodynamic monitoring can be of great help. For the patient with oliguria, additional laboratory screening can help to assess the ability of the kidney to manage sodium and water. Some laboratory tests of renal function are shown in Table 7-4. Results of these tests should be obtained before the administration of diuretics.

**Table 7-4. Laboratory tests to distinguish prerenal conditions from acute tubular necrosis (ATN)**

Laboratory Test	Prerenal	ATN
Blood urea nitrogen/creatinine ratio	>20	10–20
Urine specific gravity	>1.020	>1.010
Urine osmolality (mOsm/L)	>500	<350
Urinary sodium (mmol/L)	<20	>40
Fractional excretion of sodium* (%)	<1	>2

\*Fractional excretion of sodium (FENa) =  $(\text{urine sodium} \div \text{serum sodium}) \div (\text{urine creatinine} \div \text{serum creatinine}) \times 100$ .

### VIII. MANAGEMENT OF ACUTE RENAL INSUFFICIENCY

Shock-induced hypoperfusion may lead to renal insufficiency or failure, as may other direct renal insults associated with critical illness. Management of acute renal insufficiency should focus on correcting the underlying cause, if possible. Adequate intravascular volume should be maintained. Urinary tract obstruction, although uncommon, is easily treated and must be considered. The diagnosis of obstruction is based on findings of dilation of the kidney or urine collecting system on ultrasound. Careful monitoring of the urine output is best accomplished with the use of a urinary catheter. Volume resuscitation has been used to maintain urine output and prevent renal failure before and after the administration of radiocontrast agents or in the presence of major soft-tissue injury. Loop diuretics have also been used in the treatment of patients with oliguria. Although urine output may increase after diuretic administration, this increase may not translate into improved glomerular filtration. Intravascular volume must be optimized before loop diuretics are used. Lower doses of dopamine, although not proven effective, have been used in attempts to increase renal blood flow. Once oliguric acute renal failure is confirmed, fluids should be restricted to the replacement of ongoing losses (including insensible losses). In disease states associated with ongoing loss of intravascular volume, fluid administration is necessary to maintain adequate left ventricular preload. These losses may be substantial, as in pancreatitis, severe sepsis, and large open wounds.

Doses of drugs that are renally excreted should be adjusted, and nephrotoxic drugs should be avoided if possible. The electrolyte and acid-base status of the patient with acute renal failure must be closely monitored. Special attention should be paid to potassium, phosphate, calcium, and bicarbonate concentrations. Hyperkalemia usually can be managed medically until dialysis is available (see Chapter 12). Dialysis or hemofiltration should be instituted for the management of uremia, volume overload, hyperkalemia, or persistent acidemia. Dialysis may be achieved through hemodialysis, peritoneal dialysis, continuous arteriovenous hemofiltration with dialysis, or continuous veno-venous hemofiltration with dialysis.

**KEY POINTS: DIAGNOSIS AND MANAGEMENT OF SHOCK**

1. Shock is characterized by organ blood flow that is inadequate to meet tissue oxygen demands.
2. The four major categories of shock with characteristic hemodynamic patterns are cardiogenic, hypovolemic, distributive, and obstructive.
3. The treatment of shock centers on optimizing the oxygen content of blood and increasing the cardiac output with some combination of fluid resuscitation, increasing cardiac contractility with inotropes, and raising the systemic vascular resistance with vasopressors. The endpoint of resuscitation is adequate tissue perfusion.
4. Intravenous inotropes are indicated to improve myocardial contractility in cardiogenic shock, but an agent with vasopressor and inotropic effects is required initially when significant hypotension is present.
5. Therapy of hypovolemic shock should be targeted to re-establish normal blood pressure, pulse, and organ perfusion, usually by replacement of lost fluid with fluid of the same type (crystalloid, colloid, blood).
6. The initial approach to the patient with septic shock is restoration and maintenance of adequate intravascular volume.
7. Maintenance of intravascular volume is vital in patients with obstructive shock.
8. Oliguria may result from prerenal, renal, or postrenal (obstructive) causes. Appropriate history, hemodynamic evaluation, laboratory and urine testing, and radiographic examination may be necessary to diagnose a specific cause and direct therapy.
9. Intravascular volume must be optimized before loop diuretics are used in patients with oliguria.

***For Pediatric Patients***

1. In hypovolemic children, blood pressure is maintained longer than in adults, and blood pressure is not a good indicator of the state of perfusion.
2. In children, epinephrine may be used in preference to norepinephrine as an inotropic/vasopressor agent.
3. In children with shock, 20 mL/kg of crystalloid or 10 mL/kg of colloid may be given initially and repeated as necessary. After 60 mL/kg of crystalloid has been administered, inotropic/vasopressor support should be considered.
4. For children, a urine output  $<1$  mL/kg/hr defines oliguria. In infants  $<2$  years old, a urine output  $<2$  mL/kg/hr defines oliguria.

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