Caring for patients receiving vasopressors and inotropes in the ICU

Vigilant monitoring will maximize outcomes.

By Sonya M. Grigsby, DNP, APRN, AGACNP-BC

**Learning Objectives**

1. Identify receptors related to cardiovascular physiology.
2. Differentiate the effects of vasopressors and inotropes.
3. Discuss nursing care of patients receiving vasopressors and inotropes in the ICU.

Shock requires early recognition and quick action to prevent organ failure. (See *Types of shock.*) After initial I.V. fluid resuscitation, pharmacologic agents—such as vasopressors and inotropes—are used in critical care settings as supportive therapies to improve myocardial contractility, heart rate, and vascular resistance in patients with low cardiac output.

When critical care nurses understand shock pathophysiology and hemodynamic monitoring, they can effectively and safely titrate (increase or decrease an infusion rate for therapeutic effect) these medications using the lowest possible dose to avoid adverse effects.

**Cardiovascular physiology**

Shock disrupts cardiovascular physiology, particularly blood pressure, so understanding normal function is important. Short-term blood pressure (BP) regulation is controlled by the autonomic nervous system (ANS) via baroreceptors in the aortic arch and carotid sinus. The ANS regulates the heart, secretory glands, and smooth muscles via activation of...
Types of shock

Shock is a decline in tissue perfusion and oxygen delivery, leading to cellular dysfunction and death. Organ hypoperfusion results from decreased circulating volume (hypovolemic shock), decreased cardiac output (cardiogenic and obstructive), and vasodilation (distributive).

<table>
<thead>
<tr>
<th>Types of shock</th>
<th>Common causes</th>
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<tbody>
<tr>
<td>Hypovolemic</td>
<td>• Hemorrhage</td>
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<td>• Emesis</td>
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<td>• Diarrhea</td>
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<td>• Burns</td>
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<td>• Third spacing (too much fluid moving from the</td>
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<td>intravascular space into the interstitial</td>
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<td>space)</td>
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<td>• Polyuria</td>
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<tr>
<td>Cardiogenic</td>
<td>• Myocardial infarction</td>
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<td>• Cardiomyopathy</td>
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<td>• Valvular diseases</td>
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<td></td>
<td>• Arrhythmias</td>
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<td></td>
<td>• Myocarditis</td>
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<tr>
<td>Obstructive</td>
<td>• Tension pneumothorax</td>
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<td></td>
<td>• Pulmonary embolism</td>
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<td>• Air embolism</td>
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<td></td>
<td>• Cardiac tamponade</td>
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<td></td>
<td>• Aortic dissection</td>
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<td>Distributive</td>
<td>• Anaphylaxis</td>
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<td></td>
<td>• Neurologic condition (such as spinal cord trauma)</td>
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<td></td>
<td>• Sepsis</td>
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The sympathetic and parasympathetic systems. Body processes activate the ANS in response to information produced by internal and external stimuli, including blood pressure, body temperature, heart rate, respiratory rate, metabolism, urination, defecation, digestion, and sexual response via chemical and neurotransmitter signaling.

The sympathetic system prepares the body for stressful or emergent situations, resulting in increased heart rate and contractility, vasoconstriction, and norepinephrine and epinephrine release, which increase BP. The parasympathetic system conserves and restores body processes during normal conditions, resulting in reduced heart rate, cardiac output, and BP.

**Receptors**

Adrenergic receptors (which affect the sympathetic nervous system) within the heart and vascular system facilitate the hemodynamic effects of vasopressors via the interaction of catecholamines and synthetic medications such as norepinephrine and epinephrine. Adrenergic receptors are classified as alpha (α1, α2) or beta (β1, β2), and their location determines their physiologic response. Other receptors are vasopressin (V1 and V2; this article focuses on V1) and dopamine. (See Receptor physiology.)

**Adrenergic receptors.** α1 receptors are located within the veins, arterioles, and many capillary beds. When stimulated, they produce vasoconstriction. α2 receptors also are located on the nerve terminals of the peripheral nervous system and function to inhibit transmitter release.

Beta receptors are found on the arterioles of the heart, smooth muscle tissues, and the lungs where they bind to circulating norepinephrine and epinephrine. β1, the dominant receptor found in the heart and kidney, enhances myocardial contractility and relaxation when it’s activated. It also increases heart rate and cardiac output. Systemic vasodilation may offset increased cardiac output, resulting in decreased arterial pressures. β1 receptor activation in the kidneys induces the release of renin into the blood, promoting angiotensin synthesis and elevating BP. β2 receptors, which are located in the arterioles of the heart, smooth muscle tissues, and lungs, help mediate many essential processes. Although β2 receptors cause vasodilation, their effect on cardiac output is less than that of β1. In addition, β2 receptors have a significant impact on smooth muscles in the airway and cause bronchodilation.

**Dopamine receptors.** Site-specific dopamine receptors also exert cardiovascular actions. Small doses of dopamine result in vasodilation. As activation increases, vasoconstriction occurs via stimulation of α1 receptors and inhibition of norepinephrine release. Dopamine receptor activation within the kidney vasculature leads to enhanced renal perfusion.

**Vasopressin receptors.** Arginine vasopressin, an antidiuretic hormone, is synthesized in the hypothalamus and controls the body’s osmotic balance, regulates BP, and influences water reabsorption in the kidneys. V1 receptors are found in vascular smooth muscles. Their stimulation results in vasoconstriction, primarily in the splanchnic, renal, and hepatic arteriole vasculature. Hypovolemia and decreased arterial blood volume stimulate vasopressin release via baroreceptors in the carotid sinuses and aortic arch, resulting in increased water retention that causes vascular smooth muscle constriction.
Supportive pharmacologic therapies
Vasopressors and inotropes are commonly used in the ICU to manage shock and support the cardiovascular system. (To view a detailed list of common vasopressors and inotropes, visit myamericannurse.com/?p=71893.)

**Vasopressors.** Vasopressors are I.V. medications that produce arteriole vasoconstriction via positive inotropic or chronotropic effects that lead to increased systemic vascular resistance and BP. Common vasopressors are norepinephrine, epinephrine, phenylephrine, and vasopressin. Indications include a decrease in systolic pressure of more than 30 mmHg from baseline or mean arterial pressure (MAP) less than 60 mmHg resulting in end-organ dysfunction. (MAP, the average pressure in the arteries during one cardiac cycle, is the best indicator of organ perfusion.)

Vasopressors work on specific adrenergic receptors by activating endogenous or synthetic hormones to restore normal arterial pressure and organ perfusion during times of physical or emotional stress.

**Inotropes.** Inotropes, which are crucial to resuscitating patients with cardiogenic shock and heart failure, can exude positive or negative effects on the heart. Positive inotropic agents, which include dopamine and dobutamine, increase myocardial contractility, boosting cardiac output. These agents have been used to treat heart failure, but variability in conditions and heart rate may alter their effects. Adverse reactions associated with positive inotropic agents include arrhythmias, induced myocardial ischemia, and hypotension. Negative inotropic agents, which include beta blockers and calcium blockers (such as metoprolol, diltiazem, and verapamil), decrease the heart’s workload by reducing the force of muscle contraction; they are outside the scope of this article.

**Hemodynamic monitoring and medication administration**
Hemodynamic monitoring involves using invasive and noninvasive methods to gather information about a critically ill patient’s cardiovascular functioning for diagnosis and treatment guidance. Hemodynamic instability can be the result of hypovolemia or cardiac or vasomotor dysfunction, which can lead to organ dysfunction, multi-organ failure, or death. Noninvasive monitoring includes manual BP, electrocardiogram, temperature, respiratory rate, pulse oximetry, and urine output.

Invasive monitoring includes inserting an arterial line for continuous BP measurement and arterial blood gas analysis, and central venous catheterization or pulmonary artery catheters (PACs) for advanced hemodynamic monitoring to guide fluid resuscitation and the need for vasopressors or inotropic infusions.

Both invasive and noninvasive monitoring are helpful for assessing patients’ responses to medications, so that titration is effective. Here is a closer look at invasive techniques used for monitoring and medication administration.

**Arterial catheterization**
Vasopressor and inotropic drugs act quickly, so invasive BP monitoring, typically by arterial catheterization, may be required to safely titrate vasopressor and inotrope infusions. Arterial catheterization also provides access for diagnostic laboratory blood testing.

A trained provider places the arterial line using sterile technique. Radial and femoral

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**Receptor physiology**
Adrenergic (alpha and beta), dopamine, and vasopressin receptors exert actions on specific organs.

<table>
<thead>
<tr>
<th>Receptors</th>
<th>Location</th>
<th>Action</th>
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<tbody>
<tr>
<td>Adrenergic α1</td>
<td>Smooth muscle, Vascular walls, Heart</td>
<td>Vasoconstriction, Increased force of heart contraction</td>
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<tr>
<td>Adrenergic α2</td>
<td>Central nervous system, Arterioles, Pancreas</td>
<td>Sedation, Analgesia, Vasodilation, Inhibition of insulin release</td>
</tr>
<tr>
<td>Adrenergic β1</td>
<td>Cardiac muscle, Sinus node, Atrioventricular junction</td>
<td>Increased force and rate of heart contraction</td>
</tr>
<tr>
<td>Adrenergic β2</td>
<td>Skeletal vascular, Bronchial smooth muscle, Liver, Cell membranes</td>
<td>Vasodilation, Bronchodilation, Hepatic glycogenolysis</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Kidney, Splanchnic organs, Heart, Cerebral vascular beds</td>
<td>Vasodilation, Higher receptor activation causes vasoconstriction by inhibiting norepinephrine release</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Vascular smooth muscle</td>
<td>Vasoconstriction</td>
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sites are the most common, but other locations include the brachial, axillary, and dorsalis pedis artery. As the critical care nurse, you're responsible for arterial transducer setup (including leveling) line patency, hemodynamic response assessment using arterial waveform interpretation, and insertion site care. Transducer setup requires tight tubing and catheter connections, a pressure bag set at 300 mmHg to maintain system patency, and a transducer leveled to the phlebostatic axis. The phlebostatic axis is located at the convergence of a vertical line drawn from the fourth intercostal space to the right of the sternum midway from the anterior to the posterior chest. Mark the site to ensure consistency for accurate pressure readings. Transducer leveling should be performed every shift or whenever a data discrepancy occurs.

The arterial line is a continuous measurement of heart rate, BP, systemic perfusion, MAP, pulse pressure, and pulse pressure variation. Arterial waveforms represent left ventricular pressure during systole and consist of three components: systolic phase (steep rise in arterial pressure from the lowest point to the peak), dicrotic notch (closure of the aortic valve), and the diastolic phase (lowest point).

The Square Wave Test ensures accuracy of the hemodynamic values. It's performed by opening the continuous flush valve to a high-pressure signal, invoking transducer vibration from a “fast flush.” If the waveform is adequately dampened, only two oscillations follow the flush with a subsequent clear arterial waveform and a visible dicrotic notch.

Waveform dampening also can result from kinked or obstructed cannulas, loose connections, and air bubbles within the tubing or transducer, resulting in false low or high readings. Inaccurate and misinterpreted waveforms can affect infusion titrations and clinical endpoints.

Arterial line complications include severe blood loss, local or systemic infections, arterial thrombosis, air embolism, and accidental medication injection. (See Arterial line complications and nursing considerations.)

Central venous catheterization
Central venous catheterization (CVC) allows for central venous access using one of three options: the internal jugular vein, common femoral vein, or subclavian vein. CVC is used for multiple infusions, if peripheral access can’t be obtained, and to administer medications that may be caustic to tissues when given via peripherally inserted catheters. Vasoactive infusions through a peripherally inserted venous catheter may cause severe tissue injury and necrosis with infiltration. Controversy exists over the absolute need for a central venous catheter instead of a peripherally inserted one to administer vasopressor and inotrope infusions. Considerations include drug concentration, infusion rate, and duration. However, the absence of central access shouldn’t hinder the initiation of these drugs, as failing to initiate therapy promptly could result in detrimental effects to the patient. If central access isn’t available, initiate the infusion at the lowest rate and concentration necessary to achieve directed hemodynamic goals, and carefully monitor the peripheral site.

If infiltration is suspected, immediately discontinue the infusion, aspirate any residual fluid from the catheter, irrigate the line with saline, elevate the affected area, apply warm or cold compresses per protocol, and notify the pharmacy and provider. Pharmacologic treatment for infiltration of vasopressors and inotropes includes subcutaneous administration of 5 to 10 mg of phentolamine mesylate in 10 mL of saline around the infiltration within 12 hours of extravasation to prevent local ischemia.

To prevent intravascular catheter-related in-
Infections, the Centers for Disease Control and Prevention recommends using aseptic technique when changing dressings and prompt removal when the catheter is no longer needed.

**Pulmonary artery catheterization**

PAC (Swan-Ganz catheter) has been the gold standard for cardiac output monitoring for nearly 50 years, primarily in cardiac surgery patients. The PAC is inserted through a central vein (femoral, jugular, antecubital, or brachial) and advanced through the right side of the heart to the pulmonary artery. It’s used to measure intrathoracic intravascular pressures in the right atrium (central venous pressure), pulmonary artery, and pulmonary veins (pulmonary wedge pressure). A PAC is particularly helpful in patients with right ventricular dysfunction or pulmonary arterial hypertension.

Routine PAC site care requires following organization policy for sterile dressing changes and documenting catheter placement and signs and symptoms of site infection. The catheter’s sterile sleeve integrity must be maintained to allow catheter movement without contamination. In addition to site care, monitor for dysrhythmias, check waveform and hemodynamic values, oversee patient activity and positioning, and remove the catheter as ordered.

Assessing patients with a PAC is essential for preventing and identifying potential complications, including balloon rupture, dysrhythmias, air embolism, pulmonary thromboembolism, pulmonary artery rupture, and infarction. Review daily chest imaging to check catheter position, and zero and level transducers after repositioning. Spontaneous balloon wedging is life-threatening and requires immediate intervention. Ensure the wedging syringe isn’t full of air or accidentally inflated and that the balloon is deflated. (See PAC ports.)

**Vasopressor and inotropic infusion titration**

When the initial vasopressor or inotrope has been selected based on the underlying cause of shock, titrate the dose to achieve sufficient blood pressure or end-organ perfusion indicated by the MAP, as ordered by the provider. The most common target for vasopressor and inotropic titration is a MAP value of 60 mmHg to 65 mmHg.

Continuous vasopressor/inotropic infusions must be administered via infusion pump to prevent interrupted flow, unexpected changes in flow rates, and undesired boluses. Vasopressors have a short half-life and duration of action, so they require continuous infusion and rapid titration, usually every 5 to 15 minutes. You want to find a balance between hemodynamic stability and adequate perfusion while using the minimum dose needed. When hemodynamic stability has been achieved, begin weaning vasopressors to facilitate the lowest dose required to maintain the desired effects.

Your familiarity with organizational and unit titration policies will help ensure patient safety and reduce medication error and adverse event risks. Smart pumps can help reduce medication errors, but you must calculate and verify infusions before administration and confirm minimum and maximum doses.

**Knowledge and competency**

Hemodynamic monitoring and vasopressor and inotrope administration and titration are essential to ensuring sufficient tissue oxygenation and end-organ perfusion. Your knowledge of anatomy, physiology, and pharmacology, along with competent assessment skills, can ensure a safe outcome for your patients.

To view a list of references, visit myamericannurse.com/?p=71893.

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**PAC ports**

Critical care nurses should be familiar with the pulmonary artery catheter (PAC) port colors and their associated purposes.

<table>
<thead>
<tr>
<th>Port</th>
<th>Purpose</th>
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<th>Purpose</th>
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</thead>
<tbody>
<tr>
<td>Yellow (PA distal)</td>
<td>• Measures pulmonary artery/pulmonary artery wedge pressures</td>
<td>• Can be used to draw specimens for mixed venous gases</td>
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<tr>
<td>White (proximal infusion)</td>
<td>• Infusion/injection of I.V. fluids and medications</td>
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<tr>
<td>Blue (proximal injectate)</td>
<td>• Measures central venous pressure</td>
<td>• Can be used for infusion/injection of I.V. fluids and medications</td>
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<tr>
<td>Red (wedge)</td>
<td>• Measures pulmonary artery wedge pressure</td>
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<td></td>
</tr>
<tr>
<td>Yellow/red cap (thermistor)</td>
<td>• Measures temperature at tip of pulmonary artery</td>
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Please mark the correct answer online.

1. Effects of the parasympathetic nervous system include
   a. increased contractility.
   b. vasoconstriction.
   c. reduced heart rate.
   d. higher blood pressure.

2. Vasopressin receptors are located in
   a. splanchnic organs.
   b. cerebral vascular beds.
   c. vascular smooth muscle.
   d. bronchial smooth muscle.

3. When activated, which receptor type causes inhibition of insulin release?
   a. Adrenergic α2
   b. Adrenergic β2
   c. Dopamine
   d. Vasopressin

4. When activated, which receptor type causes bronchodilation?
   a. Adrenergic α2
   b. Adrenergic β2
   c. Dopamine
   d. Vasopressin

5. An example of a positive inotrope is
   a. diltiazem.
   b. metoprolol.
   c. verapamil.
   d. dopamine.

6. Indications for use of a vasopressor in a patient experiencing shock include
   a. decrease in systolic pressure of more than 30 mmHg from baseline.
   b. decrease in systolic pressure of more than 40 mmHg from baseline.
   c. mean arterial pressure less than 80 mmHg.
   d. mean arterial pressure less than 90 mmHg.

7. The phlebostatic axis is located at the convergence of a vertical line drawn from the
   a. fifth intercostal space to the right of the sternum midway from the anterior to the posterior chest.
   b. fourth intercostal space to the right of the sternum midway from the anterior to the posterior chest.
   c. fourth intercostal space to the left of the sternum midway from the anterior to the posterior chest.
   d. third intercostal space to the right of the sternum one-third away from the anterior to the posterior chest.

8. The dicrotic notch in an arterial waveform represents
   a. closure of the mitral valve.
   b. closure of the aortic valve.
   c. opening of the pulmonary valve.
   d. opening of the tricuspid valve.

9. Which of the following statements about arterial waveform dampening is correct?
   a. In the Square Wave Test, it should be followed by three oscillations.
   b. In the Square Wave Test, it should be followed by four oscillations.
   c. It can be caused by air bubbles within the tubing or transducer.
   d. It does not affect drug infusion titrations or clinical endpoints.

10. You suspect infiltration in a patient who is receiving a dopamine infusion through a central venous catheter. Which is an appropriate step?
    a. Monitor the site for 4 hours to determine severity, then flush with saline.
    b. If the infusion is in the femoral vein, place the leg in a dependent position.
    c. As ordered, administer 10 to 20 mg of phentolamine mesylate in 10 mL of saline around the infiltration.
    d. As ordered, administer 5 to 10 mg of phentolamine mesylate in 10 mL of saline around the infiltration.

11. Which port of a pulmonary artery catheter is used to measure pulmonary artery pressure?
    a. Injectate
    b. Thermistor
    c. Distal
    d. Proximal

12. At which infusion rate does dopamine produce vasoconstriction?
    a. More than 10 mcg/kg/min
    b. More than 7 mcg/kg/min
    c. 1 to 3 mcg/kg/min
    d. 4 to 6 mcg/kg/min

13. An inotropic agent that is a first drug of choice for cardiogenic shock is
    a. milrinone.
    b. dobutamine.
    c. epinephrine.
    d. vasopressin.

14. Mesenteric ischemia is a possible adverse effect for which drug?
    a. Dobutamine
    b. Dopamine
    c. Phenylephrine
    d. Vasopressin