Early recognition of cardiogenic shock

By Laci M. Ludlum, BSN, and Penny A. Sauer, PhD, RN, CCRN-K, CNE

Cardiogenic shock occurs when the heart can’t pump enough blood to meet the body’s demands. There are several causes of cardiogenic shock, but it most frequently happens immediately following an acute myocardial infarction (AMI). After an AMI, the heart muscle becomes stunned, which causes hypotension, decreased cardiac output, and decreased tissue perfusion. Even with proper medical treatment, patients with cardiogenic shock have 40% to 50% mortality. It’s critical for nurses to be able to recognize the early symptoms of cardiogenic shock so patients have the best chance of surviving.

This article reviews the pathophysiology of cardiogenic shock, its signs and symptoms, diagnostic tests, and treatments.

Pathophysiology

Besides AMI, cardiogenic shock may occur from myocardial contusion, dysrhythmia, cardiac surgery, or constrictive pericarditis. All of these conditions prevent adequate blood flow, oxygen, and nutrients from reaching the cardiac muscle cells. Without adequate blood flow, the cardiac muscle cells become ischemic; if blood flow isn’t restored, the muscle cells die, causing necrosis of the muscle. The death or injury of myocardial cells causes the heart muscle to become weak and unable to pump effectively (see Picturing cardiogenic shock).

When the heart is unable to pump effectively, the rest of the body is in danger of not receiving adequate blood, oxygen, and nutrients. Cardiac output is the amount of blood that’s pumped out of the heart in liters per minute. As cardiac output decreases, the body activates compensatory mechanisms to help overcome the damage caused to the heart during the AMI. The major compensatory mechanisms include the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS). Although these mechanisms are meant to help support perfusion, they’re sometimes counterproductive.

The sympathetic nervous system is the first responder when the body detects a decrease in peripheral perfusion. It releases hormones that cause vasoconstriction, increasing the heart rate and cardiac contractility. This compensatory mechanism is designed to increase cardiac output.
Although the sympathetic nervous system is attempting to help the body by increasing the heart rate, it’s actually causing more damage to the heart and decreasing perfusion further.

When the kidneys sense a decrease in perfusion, the RAAS is activated, which increases BP as a result of renin and angiotensin secretion. Aldosterone is released, causing the kidneys to retain sodium and water, and an increase in intravascular volume. Although the body is trying to help with perfusion, this excess blood causes the heart to be negatively affected. With the extra volume, the heart has to work harder so it can pump out the extra blood, thus causing more strain.

These compensatory mechanisms are often effective in other shock states; however, in cardiogenic shock, the problem originates in the heart, which causes some unique challenges. After an AMI, the myocardial cells are dead or injured, resulting in decreased BP and cardiac output. The hormones released by the sympathetic nervous system lead to an increase in heart rate and afterload, which causes the cardiac muscles to work harder, increasing the cardiac demand for oxygen and nutrients. This can cause more damage to the heart muscle or increase the area of damage, ultimately decreasing the heart’s ability to pump. The RAAS causes similar challenges: An increase in BP will lead to increased afterload, resulting in the heart muscle working harder to overcome the resistance of the increased afterload. Aldosterone causes the kidneys to hold on to sodium and retain water to increase blood volume (increased preload), which
increases the work of the heart with the increased volume that needs to be moved.

**Signs and symptoms**
The signs and symptoms of cardiogenic shock occur because the heart muscle is unable to pump enough blood to meet the body’s metabolic needs. The symptoms are similar to heart failure because of the excess fluid that the heart can’t manage (see Assessment findings).

Findings on physical assessment include:
- blue-tinged lips, nailbeds, and skin
- decreased systolic BP
- decreased mean arterial pressure (MAP)
- narrow pulse pressure (difference between systolic and diastolic pressure)
- decreased urine output
- elevated preload
- neurologic changes, such as confusion or decreased level of consciousness (LOC).

These symptoms are ominous in a patient who’s recently had an AMI, signaling that the tissue and cells aren’t receiving the blood flow, oxygen, and nutrients they need.

**Diagnosis**
Cardiogenic shock is diagnosed through the patient’s signs and symptoms, as well as multiple tests. However, the main symptom that helps diagnose cardiogenic shock is BP. When a patient is in cardiogenic shock, he or she will have very low BP.

An ECG will show if there’s damage to the heart that isn’t allowing it to function correctly and, in turn, causing the patient to go into cardiogenic shock.

A chest X-ray will show whether the heart is enlarged from the strain of having to pump harder to get an adequate amount of blood out into the body.

The last common test that’s done to help diagnose cardiogenic shock is an arterial blood gas (ABG) test. An ABG is drawn from an artery and will show how much oxygen is in the blood. If a patient

### Assessment findings

<table>
<thead>
<tr>
<th></th>
<th>Normal assessment findings</th>
<th>Assessment findings with cardiogenic shock</th>
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</thead>
<tbody>
<tr>
<td>BP</td>
<td>90/60 to 120/80 mm Hg</td>
<td>Decreased systolic BP due to poor stroke volume</td>
</tr>
<tr>
<td>MAP</td>
<td>70 to 110 mm Hg (minimum of 60 mm Hg is needed to properly perfuse the vital organs)</td>
<td>Decreased MAP due to poor stroke volume</td>
</tr>
<tr>
<td>Heart rate</td>
<td>60 to 100 beats/minute</td>
<td>Increased heart rate due to the heart trying to pump blood out into the body</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>30 to 50 mm Hg</td>
<td>Becomes narrowed (the number gap between systolic and diastolic BP becomes smaller)</td>
</tr>
<tr>
<td>Skin color and warmth</td>
<td>Warm, dry, and a capillary refill of less than 3 s</td>
<td>Cool, clammy, and a delayed capillary refill (greater than 3 s)</td>
</tr>
<tr>
<td>Urine output</td>
<td>30 mL/h</td>
<td>Decreased urine output due to lower renal perfusion and increased release of aldosterone</td>
</tr>
<tr>
<td>Preload</td>
<td>Normal central venous pressure range: 2 to 6 mm Hg</td>
<td>Elevated preload indicated by an increase in central venous pressure</td>
</tr>
<tr>
<td>Afterload</td>
<td>Normal systemic vascular resistance range: 800 to 1,200 dynes/s/cm²</td>
<td>Elevated afterload indicated by an increase in systemic vascular resistance</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>4 to 8 L/min</td>
<td>Decreased cardiac output due to the myocardium being injured and unable to effectively pump blood</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Normal LOC</td>
<td>Altered mental state due to decreased cerebral perfusion</td>
</tr>
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</table>
is in cardiogenic shock, the body won’t be properly oxygenated.

**Treatment**
The primary goals of treatment are to address whatever caused the heart to weaken and restore perfusion to meet the metabolic needs of the body’s cells. Treating the cause of cardiogenic shock varies, but the most common cause is AMI for which the priority need is to reestablish blood flow to the cardiac muscle. Often, the muscle tissue is “stunned” after an AMI and requires support to maintain cardiac output. Treatments are also aimed at improving cardiac output without increasing the workload of the cardiac muscle. These goals aren’t easily achieved. Medications that increase cardiac output do so by increasing the heart rate and increasing contractility, which increases the workload of the heart and requires more oxygen. On the other hand, the medications that decrease workload and oxygen consumption also decrease cardiac output.

In addition to managing cardiac output, cardiac workload, and oxygen consumption, the patient’s fluid status needs to be carefully managed. With decreased cardiac output, pressure increases in the lungs and right side of the heart; both the preload and the afterload are elevated. Remember that the kidneys released aldosterone, which decreased sodium excretion and increased fluid retention, complicating fluid balance. This is a difficult situation that requires thorough assessments and communication between team members to provide the best care for the patient.

**Pharmacologic**
The medications used to treat cardiogenic shock include vasopressors and inotropic agents.

Vasopressors include the medications dopamine, dobutamine, epinephrine, and norepinephrine. These medications cause vasoconstriction, which increases MAP. Unfortunately, vasopressors increase the heart rate and the amount of oxygen that the cardiac cells need. A little vasoconstriction of the arteries may be beneficial in the treatment of cardiogenic shock. However, the increase in heart rate and myocardial oxygen consumption can cause more damage to the cardiac heart muscle. Out of the previously stated vasopressors, dopamine is more likely to cause an increased heart rate compared with other vasopressor medications, such as dobutamine.

Inotropes are a class of medications that increase myocardial contractility, thus improving cardiac output. Milrinone is a medication with both inotropic and vasodilatory properties. It’s often used to treat heart failure and may also be used in the treatment of cardiogenic shock. Vasodilatory action widens the blood vessels, resulting in decreased BP, MAP, afterload, and preload. The combination of increased contractility and decreased afterload allows for an increase in cardiac output without the significant increase in workload or oxygen demand. This combination also promotes the diuresis of excess fluids. However, milrinone treatment is often limited by low BP, which can further complicate treatment.

Due to the fact that both vasopressors and inotropes have positive effects on the heart during cardiogenic shock but can also cause heart damage, these drugs should be given cautiously. The current clinical guidelines recommend using the lowest possible dosage to obtain optimal patient outcomes.

**Devices**
Although pharmacologic methods are the initial choice for the treatment of cardiogenic shock, mechanical treatment is often required. The intra-aortic balloon pump (IABP) is one form of mechanical treatment to help patients with cardiogenic shock; however, IABP use hasn’t greatly impacted mortality. It’s still widely used around the world, with approximately 50,000 being implanted per year. An IABP is contraindicated in patients with aortic
stenosis, aortic aneurysm, aortic dissection, and aortic regurgitation.

The IABP is a catheter-mounted balloon that increases blood flow to the coronary arteries by inflating the balloon on the catheter during diastole, or when the heart is relaxing and the coronary arteries are filling with blood (see A closer look at the IABP). The inflation of the balloon during diastole displaces blood volume into the descending aorta while at the same time increasing aortic pressure.

The balloon at the end of the catheter can help support a patient’s heart by inflating and deflating on every heartbeat, every other heartbeat, or every third heartbeat. The setting for the IABP is determined by the healthcare provider who assesses how much support the heart truly needs. However, as a patient’s cardiogenic shock worsens, the IABP will be set to assist the heart on every beat to provide the optimum amount of support.

The second effect that the IABP has on the heart is that it helps reduce afterload, which is achieved during deflation of the balloon on the catheter. The overall desired effect is increased myocardial oxygen supply with a reduction in the workload of the left ventricle. The hope is that there will be less stress on the heart while at the same time the heart is being properly perfused, so it can continue to pump blood out into the body.

Insertion of an IABP is through the femoral artery; the patient must be on I.V. anticoagulants during treatment. Monitor the catheter site for bleeding, as well as the balloon for displacement.

**Heart pump**

A small heart pump can be placed in the left ventricle to assist pumping function. This device is used to help with decreased perfusion by assisting the heart in pushing blood out through the aorta and allowing the coronary arteries to be perfused, along with the vital organs.

The pump is inserted through a catheter in the femoral artery. During the insertion process and after the pump has been placed, the patient will be on a continuous heparin drip, which can cause the potential for a bleed. Similar to the IABP, monitor the patient’s incision site for bleeding since a major artery is used for placement.

**Ventricular assist device**

The ventricular assist device (VAD) is a mechanical pump that helps with heart function, as well as assisting with blood flow from the heart. It’s frequently used as a bridge to heart transplant, but it can also be utilized to help support the patient’s heart while it recovers from damage or as a final treatment for cardiogenic shock or severe heart failure.

The VAD also uses a tube to carry blood away from the heart and out into the patient’s blood vessels. For this device.
to be implanted and attached to the patient’s heart, cardiac surgery is required using general anesthesia. Patients with a VAD require anticoagulants to prevent clots. Once the VAD is in place, it helps assist with perfusion of the vital organs until the heart can resume adequate function or until a new heart is transplanted.

No time to waste
Cardiogenic shock is a major complication associated with AMI. Nurses need to be aware of the causes of cardiogenic shock and able to recognize early symptoms. Besides the heart, cardiogenic shock affects many parts of the body due to the fact that when the heart is damaged, it’s unable to properly perfuse the major organs. With how much of an effect cardiogenic shock has on the body, the need for education about this condition is crucial.

REFERENCES

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