

# Take a rapid treatment approach to cardiogenic

## **Cardiac failure with cardiogenic shock continues to be an ongoing clinical problem.**

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Cardiogenic shock is a major and frequently fatal complication of a variety of acute and chronic disorders. It is the failure of the heart muscle to effectively pump blood forward, thus a failure to maintain adequate tissue perfusion. Cardiac failure with cardiogenic shock continues to be an ongoing clinical problem. The management of this condition requires a rapid and well-organized treatment approach.<sup>1</sup>

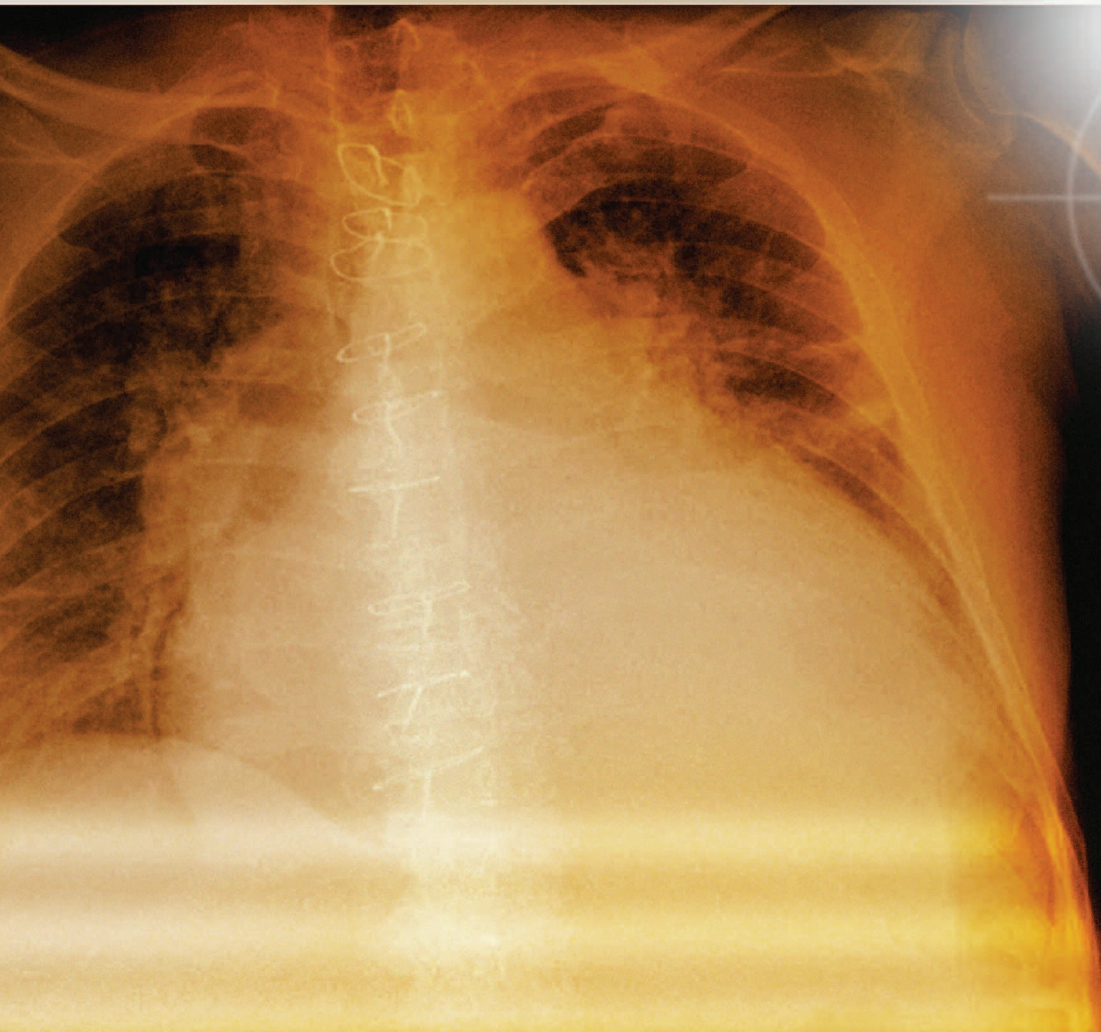
The most common cause of cardiogenic shock is acute myocardial infarction (AMI). Timely recognition of cardiogenic shock is essential to provide appropriate interventions.

The incidence of cardiogenic shock ranges from 5% to 10% in patients with AMI. Several multicenter fibrinolytics trials in Europe report a prevalence rate of approximately 7% for cardiogenic shock following AMI.<sup>1</sup> The mortality rate from cardiogenic shock is approximately 50%; recent studies have reported comparable in-hospital mortality rates in the range of 56% to 67%. With the initiation of fibrinolytics, improved interventional procedures, and better medical therapies for heart failure, the mortality rates from cardiogenic shock are expected to continue to decline.<sup>1</sup> We'll use this case study to present signs and symptoms of cardiogenic shock.



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# shock



## Case study

Mr. B., a 48-year-old man, was in a business meeting when he developed severe substernal chest pain, diaphoresis, nausea, and shortness of breath. His colleague called 911 and he was promptly transported to the emergency department. On arrival, Mr. B. continues to complain of chest pain and is noted to be diaphoretic and pale, with cool and mottled extremities. His initial vital signs are heart rate 110 beats/minute, blood pressure 85/50, respiratory rate 28 breaths/minute and labored, and temperature 97.1° F orally. Mr. B. initially is given supplemental oxygen via 100% non-rebreather mask; the cardiac monitor is applied, which shows sinus tachycardia; and I.V. access is obtained. An initial 12-lead electrocardiogram indicates that Mr. B. is having an anteroseptal wall MI. (See **ST-elevations in leads V<sub>1</sub>-V<sub>4</sub> suggest acute anteroseptal wall myocardial infarction.**) His presenting signs and symptoms suggest that he is in cardiogenic shock, a life-threatening complication of AMI associated with high mortality.

Mr. B. needs aggressive treatment to survive this condition.

## Definitions

Following cardiogenic shock, oxygen cannot be delivered to tissues.<sup>2</sup> Many conditions can lead to cardiogenic shock. (See **Other conditions that can lead to cardiogenic shock.**)

In AMI, heart muscle (myocardium) dies. Areas of dead myocardium lead to diminished contractility, resulting in reduced ejection fraction (percentage of blood present in the ventricle at end-diastole that is pumped out with each heart beat) and reduced cardiac output. This reduced ventricular emptying increases pressure within the ventricles, resulting in dilation of the ventricles and eventually failure of one or both ventricles, causing hypotension or congestive heart failure.<sup>3</sup> Hemodynamic measurements reveal persistent hypotension, low cardiac output, and high filling pressures.<sup>4</sup>

## Signs and symptoms

Clinical signs and symptoms that are associated with cardiogenic

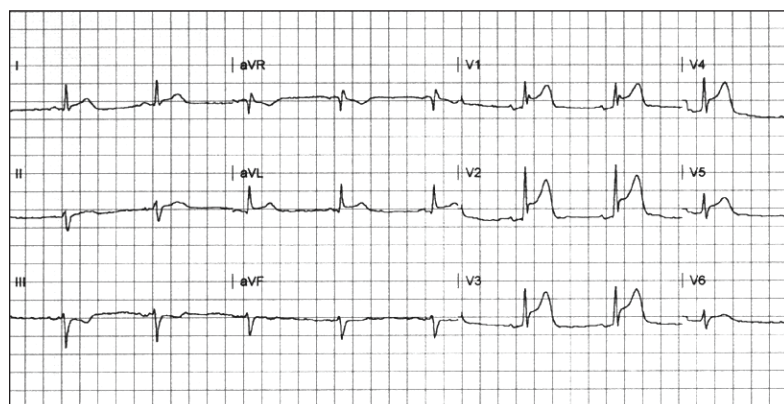
## Other conditions that can lead to cardiogenic shock

- Congestive heart failure
- Cardiomyopathy
- Dysrhythmias
- Cardiac tamponade
- Severe valvular dysfunction
- Papillary muscle rupture
- Acute pulmonary embolism
- Tension pneumothorax
- Ventricular septal defect
- Aortic dissection
- Myocarditis
- Drug overdose
- Cardiac or chest trauma
- Severe electrolyte imbalance (hypocalcemia, hypophosphatemia)
- Endocarditis

Source: Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States*. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2006.

shock include jugular vein distension, a pathological S<sub>3</sub> or ventricular gallop, and pulmonary edema. Cardiogenic shock is also defined as sustained hypotension (systolic blood pressure less than 90 mm Hg for longer than 30 minutes) and evidence of tissue hypoperfusion with adequate left ventricular filling pressure.<sup>4</sup> Tissue hypoperfusion can be defined or exhibited by such signs as cool extremities; oliguria (urine output less than 30 mL/hour or less than 0.5 mL/kg/hour); or both.<sup>3</sup> Occult tissue hypoperfusion can also be detected by drawing a serum lactic acid level. While a patient may not exhibit low blood pressure initially, a lactic acid value greater than 4 mmol/L can detect organ dysfunction at the cellular level even before the patient becomes hypotensive.<sup>5</sup> This measurement can be a very valuable

## ST-elevations in leads V<sub>1</sub>-V<sub>4</sub> suggest acute anteroseptal wall myocardial infarction (ASMI)



tool in any type of shock assessment.

Cardiogenic shock signs and symptoms can be related to inadequate cardiac output or related to venous congestion. Inadequate cardiac output leads to hypoperfusion of major organs. Signs of hypoperfusion may present as altered mental status or decreased urine output. Backup of blood into the lungs can be auscultated as pulmonary crackles. As the body attempts to improve oxygenation, the respiratory rate increases. This measurement is verified on arterial blood gas results as hypocapnia and alkalosis ( $\text{PaCO}_2$  less than 35 or pH greater than 7.45). As shock progresses, the respiratory system continues to fail and the kidneys also fail as a result of hypoperfusion. Hypercapnia and acidosis ensue and are evident on subsequent arterial blood gas measurements.<sup>2</sup>

The skin becomes cool, pale, and clammy as blood is shunted away from the periphery to the vital organs. As blood is shunted away from the skeletal muscles, wasting and lactic acid buildup occur. The shunting of blood away from the gastrointestinal tract causes bowel sounds to decrease and can eventually progress to absent bowel sounds or paralytic ileus.<sup>2</sup> Breathing may become labored as a result of pulmonary congestion as evidenced by coarse crackles or wheezing. Tachycardia, hypotension, diaphoresis, and poor peripheral pulses may also be present.<sup>3</sup>

In our case, Mr. B.'s initial presentation reveals signs of the early compensatory phase of shock. Prompt aggressive med-

ical treatment should occur if Mr. B. is to survive.

In the state of shock, the sympathetic nervous system is responding to a failing heart. Stimulation of the renin-angiotensin-aldosterone system leads to vasoconstriction and sodium and water retention to maintain blood pressure. Cardiac output needs to be maintained sufficiently to perfuse organs. Heart rate and stroke volume (amount of blood pumped out with each ventricular contraction or the difference between the end-diastolic and end-systolic volumes) increase in response to the failing left ventricle.

### **The hemodynamic principles of cardiogenic shock**

A pulmonary artery catheter and arterial line are important tools used in assessing hemodynamics. The pulmonary artery catheter is used to measure the pressures within the heart and the cardiac output. An arterial line is used to monitor blood pressure continuously and can be used to monitor laboratory values, including arterial blood gases.

Cardiac output, measured in liters per minute, is defined as the amount of blood pumped out of the heart per minute. Normal cardiac output values are 4 to 8 L/minute; in cardiogenic shock this value can drop significantly. Cardiac index is a better way of measuring cardiac output based on a person's height and weight or body surface area. A normal cardiac index is 2.5 to 4 L/minute.<sup>2</sup> In a cardiogenic shock state, the body responds by increasing the heart rate or stroke volume or both in an attempt to keep the cardiac

output and index within the normal range. However, the increase in heart rate also increases the oxygen demand of already damaged heart muscle. In addition, the increase in heart rate decreases diastolic filling time, which negatively impacts cardiac output even further. The pump continues to fail and cannot keep pace with the increase in volume.

**Preload:** The degree of stretch and pressure in the myocardium that is produced by blood volume in the ventricles at the end of diastole is termed preload.<sup>2</sup> The central venous pressure or the right atrial pressure reflects right-sided heart preload. The pulmonary artery wedge pressure reflects left-sided heart preload. In a cardiogenic shock state, one or more of these pressures may be greatly decreased or elevated depending on the type of AMI.

**Afterload:** Afterload is defined as the resistance against which the ventricles have to pump.<sup>2</sup> The components of afterload include systemic vascular resistance, which is the resistance that the left ventricle must pump against, and the pulmonary vascular resistance, which the right ventricle must pump against. Normally, the systemic vascular resistance value ranges from 800 to 1200 dynes/second/cm<sup>5</sup> and the pulmonary vascular resistance value is less than 250 dynes/second/cm<sup>5</sup>. In shock, these values may be significantly elevated because of constriction of the vasculature as the body attempts to respond to sympathetic nervous system stimulation. Mean arterial blood pressure (MAP) is a measurement of end-organ perfusion (normal MAP, 60 to 110 mm Hg). In

shock, a value of less than 60 mm Hg is not adequate to maintain organ perfusion. Mean arterial blood pressure is directly affected by cardiac output. As the cardiac output falls, so does MAP. (See **Normal and cardiogenic shock hemodynamic values.**) Continuously monitoring these values helps clinicians determine adequacy of current interventions.

**Contractility:** Cardiac contractility is the ability of the myocardium to expand and contract to pump blood. It can be indirectly measured by the right and left ventricle stroke work indexes. Poor contractility directly affects cardiac output and decreases stroke volume. In cardiogenic shock, contractility can be manipulated by using a positive inotropic agent. To apply appropriate therapy, it is important for the clinician to recognize changes in hemodynamic values quickly and to have a clear understanding of each of these parameters in rela-

tion to the patient's clinical presentation and how they are affected in the shock state.

### Available treatments

See **The hemodynamic goals of I.V. drug therapy** and **Treatments and nursing considerations** for outlines on specific interventions, benefits, and goals of cardiogenic shock therapy.

Mr. B.'s best chance of recovery relies on rapid percutaneous or surgical revascularization, but there are pharmacologic measures that can optimize his cardiac output to buy time while awaiting revascularization. It is clear that Mr. B. is demonstrating signs of poor cardiac output. He has mottled skin, cool extremities, is hard to arouse, and has poor urinary output. Positive inotropic medication may be needed to increase contractility resulting in increased cardiac output. Inotropes must be used cautiously in patients because they lead to increased myocardial oxy-

### Normal and cardiogenic shock hemodynamic values

Normal	Cardiogenic shock
Mean arterial pressure 60 to 110 mm Hg	<60 mm Hg
Heart rate 60 to 100 bpm	>110 bpm
Cardiac output 4 to 8 L/minute	<4 L/minute
Cardiac index 2.5 to 4 L/minute	<2.2 L/minute
Central venous pressure/right atrial pressure 2 to 8 mm Hg	>12 mm Hg
Pulmonary artery wedge pressure 8 to 12 mm Hg	>20 mm Hg
Systemic vascular resistance 800 to 1200 dynes/second/cm <sup>5</sup>	>1200 dynes/second/cm <sup>5</sup>
Pulmonary vascular resistance 20 to 130 dynes/second/cm <sup>5</sup>	>250 dynes/second/cm <sup>5</sup>

Source: The Pocket Doctor. Hemodynamic table. Available at: <http://homepage.mac.com/lawhusick/pds/id44.htm>. Accessed May 6, 2008.

### Treatments and nursing considerations

Supplemental oxygen/  
ventilatory support

Antiarrhythmics

Inotropes

Fibrinolytics

Vasodilators and diuretics

Intra-aortic balloon pump

I.V. fluids

Primary PCI/early revascularization

Vasopressors

Glucose control

Ventricular assist device

Coronary artery bypass graft surgery/  
early surgical revascularization

Pain management

#### REFERENCE

1. Holcomb SS. Helping your patient conquer cardiogenic shock. *Nursing*. 2002;32(9):32cc1-32cc6.

Oxygen administration helps improve tissue oxygenation. Pulmonary congestion is present in up to 64% of patients in cardiogenic shock. Supplemental oxygen is an American Heart Association (AHA) Class I recommendation for those patients.<sup>1</sup> Mechanical ventilation may be required for respiratory failure caused by shock state and pulmonary edema.

**Goal:** Titrate supplemental O<sub>2</sub> to keep SaO<sub>2</sub> > 90%, PaO<sub>2</sub> > 60 mm Hg

Cardiogenic shock patients are at risk for life-threatening cardiac rhythm disturbances, such as atrial fibrillation, supraventricular tachycardia, and ventricular fibrillation, which can further decrease cardiac output (CO) and increase myocardial oxygen demand. Antiarrhythmics help suppress dysrhythmias and slow down heart rate.

**Goal:** Lower heart rate and suppress dysrhythmia

I.V. medications, such as dobutamine and milrinone, can increase cardiac contractility, thereby increasing CO and cardiac index (CI), although this may cause tachydysrhythmias and hypotension.

**Goal:** CO >4 L/min, CI >2.1 L/min

AHA Class I recommendation for all patients with acute ST-elevation myocardial infarction (STEMI) whose symptoms started ≤ 12 hours before presenting and who do not have contraindications, although primary percutaneous coronary intervention (PCI) is preferred.<sup>1</sup>

**Goal:** Reperfusion of injured area

Vasodilators, such as nitroglycerin, and diuretics, such as furosemide, can help to decrease preload if too high. Can only be used if patient has adequate blood pressure and adequate kidney function.

**Goal:** Decrease preload and pulmonary congestion

Mechanical-assist device placed in aorta that increases coronary artery and end organ perfusion and decreases afterload, which improves CO and decreases myocardial oxygen demand.

**Goal:** Increased perfusion to organs, decreased myocardial workload

Indicated if preload is too low to help maintain adequate CO and blood pressure. In patients with right ventricular involvement, aggressive fluid administration must be given to increase CO.

**Goal:** Increase preload if low, maintain normal BP

AHA Class I recommendation for all patients with STEMI whose symptoms started ≤12 hours before presenting.<sup>1</sup>

This treatment is preferable to fibrinolytic therapy in patients with cardiogenic shock, because studies have shown decreased mortality.<sup>1</sup> Also recommended in patients who develop cardiogenic shock within 36 hours of myocardial infarction.

**Goal:** Reperfusion of injured area and correction of underlying cause of pump failure

In severely compromised patients with persistent hypotension, I.V. vasopressors, such as dopamine, norepinephrine, and epinephrine, can be used to increase afterload and blood pressure. Patient must have adequate volume status before initiating vasopressors. These medications may increase heart rate and myocardial oxygen demand.

**Goal:** Mean arterial pressure >60 mm Hg

Tight glycemic control has been shown to decrease mortality in critically ill patients. Increased stress and medications can cause hyperglycemia in both diabetics and nondiabetics. Close monitoring of glucose levels and intensive insulin therapy can improve patient outcomes.

**Goal:** Blood sugars consistently <110 mg/dL

Implantable mechanical ventricular assist device is used in severely compromised patients for whom other therapies have failed. Can be used as a bridge to cardiac transplant.

**Goal:** Provide left ventricular support while awaiting transplant

Revascularization treatment of choice when patient is not a candidate for PCI.

**Goal:** Reperfusion of injured and ischemic areas and correction of underlying cause of pump failure

Morphine is treatment of choice for pain not relieved by nitroglycerin.

**Goal:** Decrease pain but also can decrease blood pressure by decreasing preload

gen demand. Increasing contractility and cardiac output in an ischemic heart may increase the incidence of a fatal dysrhythmia.<sup>6</sup> Examples of inotropic drugs include dopamine, dobutamine, and milrinone.

Pulmonary congestion or edema may result in hypoxemia, which can further increase oxygen demand. Mechanical ventilation may be necessary to provide adequate oxygenation. It can also help decrease the work of breathing. Patients who are intubated receive I.V. sedation, which can also aid in decreasing metabolic demands by decreasing anxiety level. Sedation must also be used cautiously, so as not to cause a further drop in blood pressure. Additional considerations include defibrillators and temporary pacing equipment, to treat dysrhythmias such as ventricular tachycardia, ventricular fibrillation, or heart block.

In the treatment of cardiogenic shock, a drug that treats one variable often undermines another. For example, diuretics may be used to decrease preload or lessen pulmonary edema. As a

result, hypotension may occur, necessitating the use of vasopressors or I.V. fluids to increase preload. In patients with right ventricular dysfunction, aggressive fluid therapy is often given to increase cardiac output. Diuretics in this case are contraindicated.

Mr. B. is tachycardic to compensate for his decreased stroke volume and hypotension. It is important to avoid an extremely high heart rate because of the increase in oxygen demand; however, in the early stages of AMI it is important to use such medications as beta-blockers to slow the compensatory increase in heart rate if there are no signs or symptoms of acute heart failure.

Antiarrhythmic medications such as amiodarone or lidocaine may be indicated to treat life-threatening ventricular dysrhythmias.

Although decreasing systemic vascular resistance and providing inotropic support are important in the management of cardiogenic shock, maintenance of adequate MAP to prevent end-organ damage is vital.<sup>7</sup> Norepinephrine may be added to the

medication regimen to increase MAP, but it may have a negative effect on cardiac output. In cardiogenic shock its use is mainly limited to combination therapy in severe hypotension.<sup>8</sup> No data exist on improving outcomes of patients with cardiogenic shock treated by any catecholamine; however, by monitoring urinary output and calculating cardiac output, assumptions are made that these drugs are supporting the patient's organs and are "buying time" before revascularization and the return of adequate pump function.<sup>6</sup> Any patient admitted with a diagnosis of acute coronary syndromes, including patients in cardiogenic shock, is treated with I.V. anticoagulation (heparin) and aspirin to reduce the progression of the infarct, unless contraindicated.

In one study, experts found there was no reduction of in-hospital or long-term mortality for patients treated with fibrinolytics.<sup>9</sup> If there is a possibility that a patient will undergo percutaneous or surgical revascularization, the use of fibrinolytics is not recommended. The aforementioned

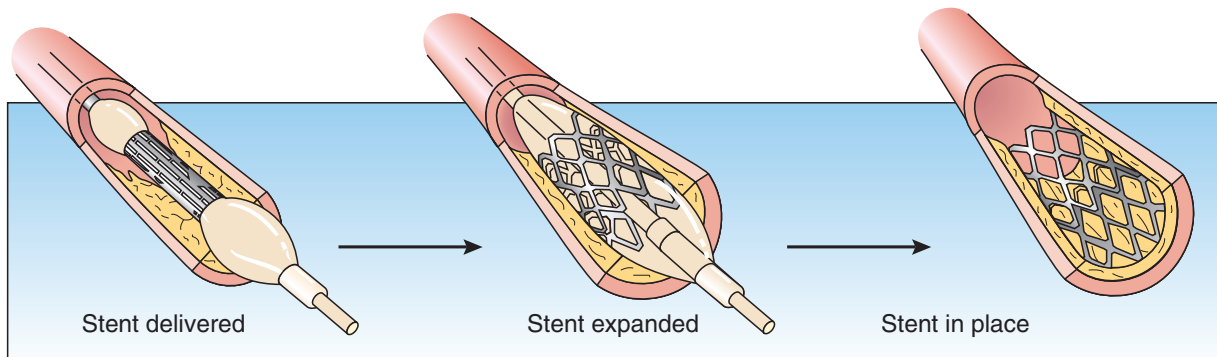
## Hemodynamic effects of drugs

### Hemodynamic values

	Vasopressors: Epinephrine, dopamine, norepinephrine	Inotropes: Dopamine, dobutamine, milrinone	Vasodilators: Nitroglycerin, nitroprusside, diuretics
Central venous pressure	—	—	↓
Systemic vascular resistance	↑	↑	↓
Pulmonary artery wedge pressure	—	—	↓
Pulmonary vascular resistance	↑	—	↓
Contractility	—	↑	—
Mean arterial pressure	↑	↑	↓
Cardiac output/cardiac index	↑	↑	—
Heart rate	↑	↑	—

Source: Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med.* 2004;32(3):858-873.

## Intracoronary stent deployment



tioned study, however, noted an improvement in hospital mortality with the use of the glycoprotein IIb/IIIa inhibitor abciximab (ReoPro).<sup>9</sup> It has recently been found to reduce mortality from 40% to 50% down to 18% to 26% in cardiogenic shock treated with stent implantation.<sup>10</sup>

### Percutaneous coronary intervention

To prevent or limit damage to Mr. B's heart muscle, immediate reperfusion of the heart muscle is vital. Percutaneous coronary intervention (PCI) describes a nonsurgical coronary revascularization procedure that relieves the narrowing or obstruction of the coronary artery or arteries to allow more blood and oxygen to be delivered to the heart muscle. In this procedure, a small balloon-tipped catheter inserted into the femoral or brachial artery is advanced to the site of narrowing in the coronary artery. The balloon is then inflated to displace the narrowed lumen of the artery. An intracoronary stent can also be deployed during this procedure.

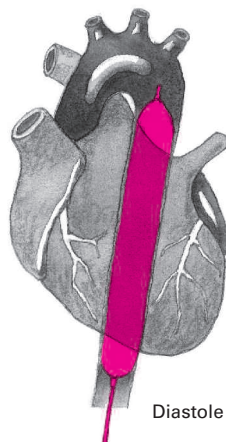
Several studies and randomized trials have demonstrated the superiority of percutaneous transluminal coronary angioplasty

(PTCA) for early reperfusion of patients with AMI, resulting in improved patency rate and clinical outcomes.<sup>11-13</sup> Percutaneous transluminal coronary angioplasty is the preferred reperfusion strategy and is widely used in many institutions in the United States.<sup>11</sup> The American College of Cardiology and the American Heart Association have revised the 2003 PTCA Guidelines, aiming to provide reperfusion of the infarct artery within 90 minutes after arrival to the hospital. These new guidelines also assist in decision making regarding PCI, ensuring patient safety and improving patient quality of care.<sup>4</sup>

PTCA does not offer absolute

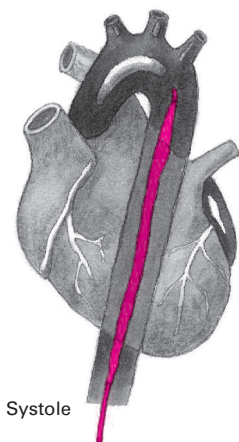
safety to the patient. Emergent and late complications have also been identified with this intervention. One of the most serious complications is abrupt coronary artery occlusion or acute restenosis. This could be a result of tearing of the inner lining of the artery, blood clotting at the balloon site, and constriction of the artery at the balloon site. The introduction of a drug-eluting stent has decreased the incidence of abrupt closures by eliminating the problem of flow-limiting arterial dissection, elastic recoil, and spasm of the artery, enhancing the safety and efficacy of PTCA.<sup>14</sup> The stent remains permanently in place in the artery. (See

### The ins and outs of the IABP



Diastole

The IABP rapidly shuttles helium gas in and out of the balloon, which is located in the descending aorta. The balloon is inflated at the onset of cardiac diastole and deflated at the onset of systole.



Systole

**Intracoronary stent deployment.)<sup>9</sup>**

The use of PTCA and stenting improves oxygenation to the heart muscle, decreasing the ensuing complication of an AMI, which is cardiogenic shock.<sup>15</sup>

**Intra-aortic balloon pump**

The use of an intra-aortic balloon pump (IABP) improves coronary artery perfusion and reduces afterload. This mechanical device consists of a 34- to 40-mL balloon

catheter, which is placed percutaneously into the patient's aorta just distal to the origin of the subclavian artery and above the renal artery branches.<sup>16</sup> The IABP operates by using counterpulsation therapy. The IABP inflates during ventricular diastole (increasing coronary artery perfusion) and deflates during ventricular systole (decreasing afterload or the resistance against which the heart has to pump).<sup>11</sup>

(See **The ins and outs of the IABP.**) The use of an IABP improves the patient's cardiac output and ejection fraction by increasing coronary artery perfusion, and ultimately increases MAP and end-organ perfusion. The IABP also helps decrease the heart rate (which decreases oxygen consumption) and pulmonary artery pressures, especially pulmonary artery diastolic and wedge pressures, decreasing

**Diagnostic tools**

Tool	Benefit and goal
12-lead ECG	Identifies type and location of myocardial ischemia, injury, or infarct. <b>Goal:</b> Identify patients with ST-segment elevation myocardial infarction, non-ST-elevation myocardial infarction, or ischemia; normal ECG does not rule out MI
Chest X-ray	Used to diagnose pulmonary vascular congestion and pulmonary edema, which occurs in up to 64% of cardiogenic shock patients. <sup>2</sup> Also used to confirm placement of central venous access devices, pulmonary artery catheters, endotracheal tubes, and intra-aortic balloon pump. <b>Goal:</b> Prompt identification and treatment of pulmonary edema and placement of catheters and tubes
Laboratory work	Cardiac markers such as troponins can help establish diagnosis of AMI (acute myocardial infarction). Chemistry panel, complete blood cell count, and coagulation studies should be done and abnormalities treated. A brain natriuretic peptide level can indicate heart failure and help to differentiate cardiogenic shock from other forms of shock. Lactate, creatinine, and liver function tests can help determine effects of shock on other organs. <b>Goal:</b> Prompt identification and treatment of abnormal laboratory values
Pulmonary artery catheter	Hemodynamic monitoring evaluates preload (central venous pressure); afterload (systemic vascular resistance); cardiac output (CO) and cardiac index (CI); and fluid status (pulmonary artery wedge pressure). <b>Goal:</b> Maintain CO >4 L/min, CI >2.1 L/min, pulmonary capillary wedge pressure <18 mm Hg
Three-dimensional echocardiogram	Noninvasive diagnostic study that evaluates structure and function of the heart. Provides estimation of ejection fraction. American Heart Association class I recommendation. <sup>2</sup> <b>Goal:</b> Identification of wall motion abnormalities and left ventricular failure
Cardiac catheterization	Definitive diagnostic procedure to determine exact cause of cardiogenic shock and identify coronary artery stenoses and occlusions. Can also provide hemodynamic measurements, evaluation of left ventricular wall motion, and ejection fraction. Results guide further treatment for revascularization (primary percutaneous coronary intervention versus coronary artery bypass graft surgery). <b>Goal:</b> Quick identification of AMI or cardiogenic shock and transfer patient to cardiac catheterization laboratory
ABGs (arterial blood gases)/arterial line	ABGs monitor acid-base balance and oxygenation. An arterial line provides an access point for ABGs and also provides continuous blood pressure readings. <b>Goal:</b> Maintain mean arterial pressure >60 mm Hg and pH of 7.35-7.45

Source: ECC Committee, American Heart Association. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 8: stabilization in the patient with acute coronary syndrome. *Circulation*. 2005;112(24 suppl I):IV89-IV103.

blood volume and workload of the heart.<sup>11</sup> Mr. B. is a good candidate for IABP therapy because he presented with AMI and is showing signs and symptoms of cardiogenic shock.

Mr. B. was taken directly to the cardiac catheterization laboratory and underwent PCI with drug-eluting stent placement in the left anterior descending coronary artery. An IABP was placed during the procedure to increase coronary artery perfusion and decrease workload of the heart. He was transferred to the cardiac care unit for further management and monitoring and was discharged 5 days later on appropriate medications.

## Prevent complications

Cardiogenic shock is a potential life-threatening complication of AMI. Recognition of early signs and symptoms of cardiogenic shock and rapid interventions enable the nurse and treatment

team to prevent complications and assist the patient to recovery. ♦

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The authors have disclosed that they have no significant relationship with or financial interest in any commercial companies that pertain to this educational activity.

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## Take a rapid treatment approach to cardiogenic shock

**GENERAL PURPOSE:** To provide the registered professional nurse with an overview of cardiogenic shock. **LEARNING OBJECTIVES:** After reading this article and taking this test, the reader will be able to: 1. Describe the signs and symptoms of cardiogenic shock. 2. Discuss the treatment options and nursing considerations for cardiogenic shock.

### 1. The most common cause of cardiogenic shock is

- a. dysrhythmia.
- b. an embolism.
- c. acute myocardial infarction (AMI).
- d. drug toxicity.

### 2. The mortality rate from cardiogenic shock is approximately

- a. 20%.
- b. 30%.
- c. 50%.
- d. 76%.

### 3. Conditions that can lead to cardiogenic shock include all of the following *except*

- a. pleuritis.
- b. acute pulmonary embolism.
- c. tension pneumothorax.
- d. cardiac tamponade.

### 4. In AMI, hemodynamic measurements reveal

- a. persistent hypertension.
- b. high ejection fractions.
- c. low cardiac output.
- d. low filling pressures.

### 5. Signs and symptoms of cardiogenic shock include all of the following *except*

- a. jugular vein distension.
- b. a pathological S3 or ventricular gallop.
- c. pulmonary edema.
- d. hyperthermia.

### 6. Which blood test can detect organ dysfunction at the cellular level early in cardiogenic shock?

- a. potassium
- b. lactic acid
- c. brain natriuretic peptide
- d. C-reactive protein

### 7. Which is *not* an early sign of cardiogenic shock?

- a. altered mental status
- b. oliguria
- c. alkalosis
- d. hypercapnia

### 8. How does the body respond to the failing heart in shock?

- a. The parasympathetic nervous system causes vasodilatation.
- b. The parasympathetic nervous system causes natriuresis.
- c. The sympathetic nervous system causes the stroke volume to increase.
- d. The sympathetic nervous system causes the heart rate and stroke volume to decrease.

### 9. An arterial line is used to measure

- a. continuous blood pressure.
- b. cardiac output.
- c. cardiac index.
- d. ventricular filling pressures.

### 10. Which measurement is consistent with cardiogenic shock?

- a. a central venous pressure/right atrial pressure of 8 mm Hg
- b. a cardiac index of 1.8 L/minute
- c. a pulmonary artery wedge pressure of 11 mm Hg
- d. a mean arterial pressure (MAP) of 70 mm Hg

### 11. Right-sided heart preload is measured by the

- a. cardiac output.
- b. pulmonary artery wedge pressure.
- c. systemic vascular resistance (SVR).
- d. central venous pressure (CVP).

### 12. The resistance against which the ventricles have to pump is called the

- a. preload.
- b. afterload.
- c. cardiac index.
- d. ejection fraction.

### 13. One goal for administration of I.V. epinephrine is to

- a. decrease the SVR.
- b. decrease pulmonary vascular resistance.
- c. increase the CVP but decrease the SVR.
- d. increase the MAP.

### 14. Norepinephrine used during cardiogenic shock

- a. will decrease SVR.
- b. may decrease the MAP.
- c. is mainly limited to combination therapy in severe hypotension.
- d. does not affect cardiac output.

### 15. American Heart Association guidelines recommend that percutaneous transluminal coronary angioplasty be performed within

- a. 30 minutes after arrival to the hospital.
- b. 60 minutes after arrival to the hospital.
- c. 90 minutes after arrival to the hospital.
- d. 2 hours after arrival to the hospital.

### 16. A potential serious complication of percutaneous coronary angioplasty is

- a. renal failure.
- b. aortic dissection.
- c. papillary muscle rupture.
- d. abrupt coronary artery closure.

### 17. Which statement about treating cardiogenic shock is correct?

- a. The intra-aortic balloon pump inflates during diastole to decrease afterload.
- b. Patients in cardiogenic shock should not receive aspirin for acute coronary syndromes.
- c. The purpose of maintaining an adequate MAP is to prevent end-organ damage.
- d. Dilaudid is the treatment of choice for pain not relieved by nitroglycerin.



## ENROLLMENT FORM *Nursing2008 Critical Care, July, Take a rapid treatment approach to cardiogenic shock*

### A. Registration Information:

Last name \_\_\_\_\_ First name \_\_\_\_\_ MI \_\_\_\_\_

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City \_\_\_\_\_ State \_\_\_\_\_ ZIP \_\_\_\_\_

Telephone \_\_\_\_\_ Fax \_\_\_\_\_ E-mail \_\_\_\_\_

**Registration Deadline:** August 31, 2010

Contact hours: 2.5 Pharmacology hours: 0.0 Fee: \$24.95

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Certified by \_\_\_\_\_

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### C. Course Evaluation\*

- Did this CE activity's learning objectives relate to its general purpose? ☐ Yes ☐ No
- Was the journal home study format an effective way to present the material? ☐ Yes ☐ No
- Was the content relevant to your nursing practice? ☐ Yes ☐ No
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