

TCHP

Education
Consortium

Cardiovascular Critical Care Primer

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Introduction/Purpose Statement

Cardiac and vascular diseases are becoming more and more common in American society. Critical care nurses routinely see patients with a wide variety of cardiovascular problems. The purpose of this home study is to increase your understanding of the anatomy, physiology, and pathophysiology of problems such as angina, myocardial infarction, peripheral vascular disease, congestive heart failure, and cardiomyopathy.

Target Audience

This home study was designed for the novice critical care or telemetry nurse. However, other health care professionals are invited to complete this packet.

Content Objectives

1. Identify the normal anatomy and physiology of the cardiovascular system.
2. Describe the pathophysiology of an acute myocardial infarction.
3. Describe the symptoms of heart failure.
4. Describe the pathophysiology of tamponade.
5. Identify the factors that favor the development of a venous thrombosis.
6. Differentiate between dilated, hypertrophic, and restrictive cardiomyopathy.
7. Differentiate between Buerger's Disease and Raynaud's Syndrome.

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Contact Hour Information

For completing this **Home Study and evaluation**, you are eligible to receive:

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Peripheral Vascular Disease

As opposed to cardiac or cerebrovascular disease, peripheral vascular disease refers to problems within the blood vessels in the extremities, thorax, and abdomen. The majority of peripheral vascular disease problems are caused by atherosclerosis, just like cardiac and cerebral vascular disease.

Arterial Vascular Disease

There are three major problems that can arise in the arterial vasculature: atherosclerotic occlusion, aneurysm, and embolization.

Atherosclerosis

Atherosclerosis – or arteriosclerosis in the artery – is the primary cause of all vascular problems in the United States. In this process, there is a gradual build up of plaque on the intimal wall of the artery caused by repeated injury, clotting, and scarring.

Problems in the arterial system arise when the amount of plaque build up has grown to such an extent that blood is no longer able to pass easily through the narrowed arterial diameter. This is called arterial insufficiency. When blood cannot pass through at all, it is called arterial occlusion.



Normal cross-section of an artery



Beginning of atheroma



Partial occlusion with clot formation



Occluded vessel

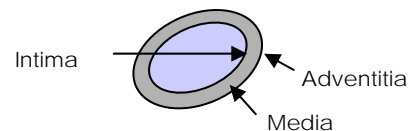
Although the most common sites of arterial insufficiency and occlusion are the carotid, renal, popliteal, aortoiliac and femoral arteries, any junction or branching area can develop problems.

The symptoms of arterial insufficiency or occlusion in the arteries that supply the legs (popliteal, aortoiliac, and femoral) are related to the lack of arterial flow. The distal extremity becomes cool, pale or cyanotic, with poor or absent pulses. Pain often accompanies arterial insufficiency because the distal tissues are starved for oxygen. The symptoms of carotid arterial insufficiency result from brain anoxia – ranging from transient ischemic attacks to completed stroke.

Bruits are common in both iliofemoral and carotid artery insufficiency. A bruit is the sound that is heard when the pressure in the vessel prior to the lumen narrowing is high, and the pressure after the narrowed area is low. The resulting turbulent blood flow causes a low-pitched “whooshing” sound.

Aneurysm

An aneurysm is a weak area in an arterial wall. This weakening can be caused by hypertension, atherosclerosis, smoking, or may be congenital. There are three layers of the artery: the intima, the media, and



the adventitia.

While there is initially just a weakening in the media of the arterial wall, eventually pressure will build up and cause the weakened area of the artery to begin to balloon. There are two main types of aneurysms: Saccular and fusiform.

Fusiform aneurysms appear as a nearly symmetrical bulge around the circumference of the weakened area of the affected vessel. Saccular aneurysms appear as a blister on one side of the vessel. They may be caused by trauma, such as a motor vehicle crash.



Saccular aneurysm

(side view of an artery)

There is an outpouching on one side of the artery



Fusiform aneurysm

(side view of an artery)

There is a large dilation of the artery closest to the heart with a gradual narrowing.

Many aneurysms lie dormant without symptoms for years. Other aneurysms can continue to grow in size until their mass causes symptoms or they rupture.

Although any artery can develop an aneurysm, the most dangerous ones are in the aorta. The aorta is the major artery in the body. It branches from the left ventricle and traverses down through the thorax (thoracic aorta) and the abdomen (abdominal aorta), giving off branches to supply all of the body organs with blood.

Symptoms from an aortic aneurysm may include dyspnea, stridor, hoarseness, hemoptysis, cough, or chest pain. All of these symptoms are related to the mass of the aneurysm impinging on other organs. Pain is the main symptom of descending thoracic aortic aneurysm: pain in the shoulder, lower back, abdomen, shoulders, arms, or neck. Finally, abdominal aneurysms usually have no symptoms until they leak or rupture.

Leaking or rupture of an aortic aneurysm is usually a life-threatening emergency. If the wall is weakened enough, the aneurysm will rupture, resulting in aortic blood being pumped into either the chest or abdominal cavity. The patient may bleed to death (exsanguinate) in a very short time. More commonly, patients who complain of severe, unrelenting pain, shortness of breath, faintness, etc., may be experiencing the leaking of blood from the aneurysm.

Similar to aneurysms, an aortic dissection is more common than an aortic aneurysm rupture. A dissection is said to occur when there is a longitudinal split between the intima and the media of the thoracic aorta. A dissection may occur after trauma, or due to Marfan's syndrome, increasing age, hypertension, or atherosclerosis.

Embolization

The third cause of peripheral vascular insufficiency is embolism. An embolus can begin as either a clot formed in the heart or as a piece of dislodged plaque. An embolus will travel through the arterial system until it

reaches a branch through which it cannot travel. At that point, the embolus will block blood flow distal to the occlusion.

Venous Vascular Disease

The most common form of vascular disease related to the venous system is the development of deep vein thrombosis. Typically found in the lower extremities (particularly the calves), a thrombus occludes venous return to the heart. Pressure backs up from below the thrombus, causing edema to form distal to the occlusion.

The first stage of deep vein thrombosis (DVT) formation is injury. The second stage is intravascular clot formation. If that clot (thrombus) does not become detached and form an embolus, it will adhere to the vein wall within 24-48 hours and eventually be lysed.

The three components needed to cause a DVT are defined in Virchow's triad:

1. **Hypercoagulability of the blood:** blood dyscrasias, trauma, cancer, estrogen therapy, systemic infection, smoking
2. **Venous stasis:** heart disease (CHF), dehydration, immobility, incompetent leg vein valves
3. **Intimal damage:** trauma, infection, venipuncture, IV infusion of irritant solutions.

Buerger's Disease and Raynaud's Syndrome

Buerger's Disease, or Thromboangiitis Obliterans, (TOA) is a rare condition that presents as an inflammation and eventual blockage of the small vessels of the extremities. In rare cases, internal organs are affected. Unlike other vascular diseases, it is neither an embolic nor an atherosclerotic disease. The classic patient is a 20-40 year old smoker, usually male. Non-smoking tobacco users are at risk as well. There is likely a genetic component to it, as it is far more prevalent in certain ethnic groups.

TOA is characterized by reduced blood flow to the extremities, with collateral circulation developing in an ineffective corkscrew pattern (visible on angiogram). Symptoms are related to lack of blood flow: coldness of the extremity, intermittent claudication (pain or cramping that occurs in the legs when walking), and numbness, tingling, and burning sensations. Symptoms start at the tips of the fingers and toes and progress upward. As the disease progresses, there is ulceration of the tips of the

digits, and eventually gangrene. Amputation of the digits can only be avoided by abstaining from all forms of tobacco.

Exposure to cold worsens the symptoms. The extremity is sensitive to loss of blood flow caused by elevation above the level of the heart. Care to avoid cold and constricting medications is important. Treatments such as sympathectomies, (a surgical procedure that destroys nerves in the sympathetic nervous system provide only temporary relief, as do vasodilating drugs).

The cause is unknown, but tobacco is thought to be a trigger for an autoimmune or inflammatory process. TOA is often accompanied by Raynaud's Syndrome.



Figure 1: Buerger's Disease with thrombophlebitis of the great toe. (emedicine.medscape.com)

In Raynaud's Syndrome, the arterioles of the extremities constrict in response to exposure to cold or stress. There is a cyclical response, with the fingers, toes, and the tips of the nose and ears turning pale due to lack of blood flow. As oxygen is consumed, the color turns to bluish. Then, as the arterioles relax and blood refills, the extremity becomes flushed, and then returns to normal color. Attacks may last minutes or hours.

Primary Raynaud's occurs with no associated cause, and is generally a milder form, with little pain. Secondary Raynaud's may be associated with vasoconstricting drugs, frostbite, repetitive motion or vibration injury, smoking, or thoracic outlet syndrome. Secondary Raynaud's is more painful and may also be associated with Buerger's disease.

Treatment is generally aimed at control of symptoms. This involves avoiding offending drugs, including caffeine and tobacco. Protection of the extremities by keeping warm in cold weather and when handling cold and frozen objects can prevent attacks. Patients also need to be aware that significant cooling after exercise can trigger an attack, making it important to cool down slowly.

Some drugs, such as topical nitroglycerin, Viagra, ace inhibitors, and calcium channel blockers may relieve symptoms. Open wounds, blackening of the skin, breaks in the skin, and joint soreness surrounding the affected areas need to be evaluated and treated.



Figure 2 Raynaud's Syndrome (eMedicine.com)

Hypertension & Hypertensive Crisis

Mr. Jerome Atwater enters the Emergency Department with complaints of headache, dizziness, and chest pain. He has a 30 year history of hypertension. His initial vital signs are: HR of 145, sinus tachycardia; BP of 210/138 mm Hg; RR of 24/minute. The initial diagnosis is hypertensive crisis.

What is blood pressure?

The arterial blood pressure is the pressure within the arteries that drives blood into the circulation. The blood pressure is determined by the cardiac output multiplied by the systemic resistance. There are three main elements to blood pressure:

- The *systolic blood pressure (SBP)* is the pressure in the arteries that occurs with ejection from the left ventricle.
- The *diastolic blood pressure (DBP)* is the pressure that has been “stored up” in the arteries during the relaxation of the heart. The sympathetic nervous system (SNS) maintains the muscle tone in the arteries.
- The *mean arterial pressure (MAP)* is the average blood pressure in the systemic circulation.

How is blood pressure controlled?

There are many mechanisms that work to control the blood pressure.

Baroreceptors: receptors in the aortic arch and carotid artery bodies that are sensitive to pressure. Stimulation of these receptors occurs with either a low or high BP. Information sent to the medulla results in a change in the heart rate and vascular tone of the arteries.

Chemoreceptors: situated near the baroreceptors, chemoreceptors are sensitive to changes in the pH, PaO₂, and PaCO₂. The chemoreceptors are stimulated when the PaCO₂ rises and when the pH and PaO₂ falls with hypotension. Information is sent to the medulla.

Autonomic nervous system:

- The parasympathetic nervous system decreases cardiac output (CO) and BP by decreasing the heart rate.
- The sympathetic nervous system increases the CO by increasing the heart rate and cardiac contractility, and by vasoconstricting the blood vessels, thus increasing BP.

Renin-Angiotensin-Aldosterone system: Renin is released by the kidneys in response to a decrease in blood pressure. Renin combines with angiotensinogen to form angiotensin I, which is then converted to angiotensin II. Angiotensin II causes massive vasoconstriction and stimulates aldosterone. Aldosterone causes the kidney tubules to reabsorb water and sodium.

Renal prostaglandins: These hormones weaken the action of the renin-angiotensin-aldosterone system.

Vasopressin: Also known as antidiuretic hormone (ADH), vasopressin is released with a decrease in blood volume or an increase in serum osmolality. It

vasoconstricts the blood vessels and stimulates the kidneys to reabsorb water.

Hypertension is defined as...

Although definitions may vary, the American Heart Association defines hypertension as a consistently elevated blood pressure of > 140 mm Hg systolic and/or > 90 mm Hg diastolic.

What causes hypertension?

Ninety to ninety-five percent of hypertension is called “primary” or “essential” hypertension. There is no known cause for primary hypertension. Risk factors that are suspected in the development of primary hypertension are:

- Family history
- Advancing age
- Race
- High sodium intake
- Obesity
- Excess alcohol consumption
- Low intake of potassium, calcium, magnesium
- Stress
- Use of oral contraceptive drugs

The remaining cases of hypertension are lumped into the category of “secondary hypertension.” Causes for this type of hypertension include renal disease, arteriosclerosis, coarctation of the aorta, pheochromocytoma, elevated levels of adrenocortical hormones, and brain lesions.

Why is hypertension harmful?

The main problem with hypertension is that it places a larger burden on the heart and blood vessels than they are built for. The workload of the heart increases because it has to pump harder against resistance to push blood out. This leads to ventricular muscle hypertrophy, which increases myocardial oxygen demand. Eventually the oxygen demand outstrips the supply, leading to angina, myocardial infarction, and congestive heart failure.

The arteries throughout the body are also affected by hypertension. Hypertension appears to speed the development of atherosclerosis, affecting the coronary arteries and renal vasculature. An elevated blood pressure can also cause an outpouching in a weak part of an arterial wall. This outpouching is called an aneurysm. Aneurysms can occur in any blood vessel in the body; especially the aorta, the retina, and the brain.

What is occurring with Mr. Atwater?

Mr. Atwater is experiencing a hypertensive crisis. Hypertension is a “crisis” when the diastolic BP increases above 120 mmHg, and when there is organ damage. It is generally accompanied by renal disorders, vascular changes and retinopathy. The extremely high pressure causes an intense reflex vasoconstriction in the brain, in the brain’s attempt to preserve itself. This measure is often not successful, and cerebral edema develops, causing papilledema (swelling of the optic nerve at the point of its entrance into the eye), headache, restlessness, confusion, stupor, motor and sensory deficits, and visual disturbances. The continued high BP injures the walls of the arterioles, especially in the kidney and retina. Retinal hemorrhage and kidney failure occur as a result.

Cardiomyopathy

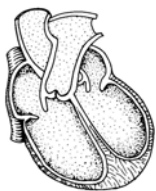
What is cardiomyopathy?

Cardiomyopathy is a term that is used to describe a problem with the functioning of the heart muscle. There are three forms:

1. Dilated (congestive)
2. Hypertrophic
3. Restrictive

Dilated Cardiomyopathy

Dilated cardiomyopathy is the most common form of cardiomyopathy. In this form, the heart muscle fibers are stretched beyond their normal size. This stretching causes the heart chambers to become dilated and the walls of the heart to become thinner. The stretched muscle fibers are not able to contract well, resulting in poor cardiac contractility and inadequate ejection of blood. This eventually leads to increased dilation, and eventually, to congestive heart failure.



See how the ventricles are enlarged and the walls surrounding them are thin?

What are the causes of dilated cardiomyopathy?

The majority of cases of dilated cardiomyopathy are idiopathic – that is, no one knows why they have developed this condition. Alcohol abuse has been linked

to dilated cardiomyopathy, sometimes called alcoholic cardiomyopathy. Other potential causes: pregnancy, viral infections, certain chemotherapy drugs, and a hereditary disposition.

What causes the signs and symptoms of dilated cardiomyopathy?

Some people with dilated cardiomyopathy can live for a long time without symptoms. When symptoms do begin, however, they resemble those of congestive heart failure.

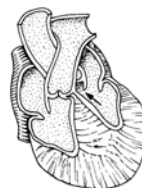
As the left heart fails, the left ventricle is not able to pump out the blood that is filling it. The pressure in the left ventricle rises, eventually causing fluid to back up into the pulmonary system. As the pressure in the pulmonary system rises, fluid is forced out of the capillary bed into the alveoli. Pulmonary edema ensues, with shortness of breath, cough with frothy or blood tinged sputum, and crackles bilaterally.

As the right heart fails, the right ventricle cannot pump out its share of blood, leading to increased pressure in the venous system. Fluid is forced out of the venous system into the peripheral circulation, causing edema of the extremities.

People with dilated cardiomyopathy can also develop cardiac dysrhythmias, particularly atrial fibrillation and ventricular dysrhythmias.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is the second most common form of cardiomyopathy. In this type of cardiomyopathy, there is an abnormal overgrowth of muscle fibers, leading to decreased ventricular chamber size and increased ventricular wall size. As the cardiomyopathy progresses, there is little room for blood in the ventricles, and limited relaxation of the ventricles during diastole, causing a decreased amount of blood to be pumped into the circulation with each beat.



Note how the ventricle chamber size is about the same as the atrial chamber size (much too small) and the walls of the ventricle are very thick.

Another name for hypertrophic cardiomyopathy is hypertrophic obstructive cardiomyopathy (HOCM).

What are the causes of hypertrophic cardiomyopathy?

About 50% of all cases seem to be related to a genetic abnormality transmitted by one or both parents. The other cases do not have an identifiable cause. Hypertrophic cardiomyopathy is **not** caused by exercise; the conditioned heart has more muscle mass, but the ventricular chamber size remains adequate, as does the ability to contract and relax.

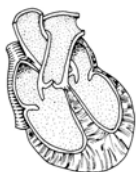
What are the signs and symptoms caused by?

The cause of the symptomology of hypertrophic cardiomyopathy is related to inadequate stroke volume (the amount of blood pumped out of the left ventricle with each contraction). The patient's heart rate may be normal or even fast, but the patient cannot get enough blood to the vital organs. Symptoms arising from inadequate blood supply are: shortness of breath, fainting during activity, palpitations, chest pain, or fatigue. Ventricular fibrillation can be the first indicator of a problem, and may lead to sudden death.

As the condition progresses to the severe stage, congestive heart failure results as the pressure in the heart rises to such an extent that blood "backs up" into the lungs and periphery.

Restrictive Cardiomyopathy

Restrictive cardiomyopathy is rare in the United States. In this case, there is no chamber size or ventricle wall size change; rather, the walls of the ventricle become stiff and noncompliant. Abnormal tissue, caused by amyloidosis, hemochromatosis, or sarcoidosis, invades the walls of the ventricle, causing the contractility to be compromised.



The chamber and wall sizes are normal; however, the wall itself is rigid and noncompliant.

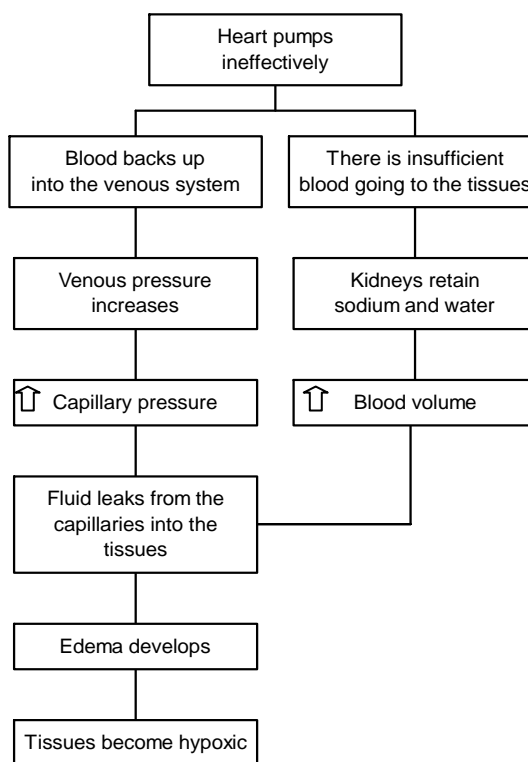
The signs and symptoms of restrictive cardiomyopathy are essentially the same as dilated cardiomyopathy: weakness, fatigue, shortness of breath, edema, nausea and bloating.

Congestive Heart Failure

Mr. Wood enters the hospital after a three-day history of chest pain. The EKG indicates that Mr. Wood has had an anterior wall MI. The day after admission, Mr. Wood begins to show the signs and symptoms of heart failure. He is hypotensive and tachycardic, with a respiratory rate of 26. His respirations are labored; rales are auscultated in the middle and lower lobes of his lungs. He is anxious.

What is heart failure?

The term "heart failure" indicates that the heart has been damaged so that it does not pump blood adequately, causing decreased tissue perfusion and back up into the peripheral circulation. Although heart failure can be classified as left or right in etiology, the failure of one side of the heart usually leads to failure on the opposite side. The pathophysiology for both left and right heart failure is the same, as shown below:



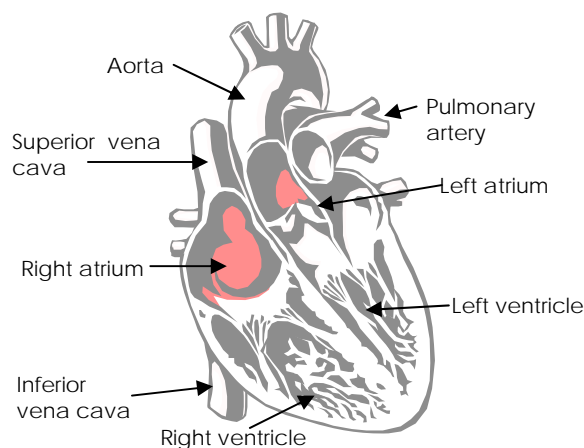
What is the route of blood through the heart?

The right side of the heart is a low pressure, high capacity venous system. Blood enters the **right atrium** from the superior and inferior **vena cava**. Passive filling and active contraction of the atria pushes the blood through

the **tricuspid valve** into the **right ventricle**. As the right ventricle fills, it actively expels the blood through the **pulmonic valve** into the **pulmonary artery** and into the lungs for re-oxygenation.

The left side of the heart is a high pressure arterial system whose size is regulated by different muscle walls. Arteries have the ability to dilate and constrict as stimulated by the sympathetic nervous system in a response to baroreceptor reflexes, oxygenation status, presence of hypercarbia, and CNS stimulation.

When the blood exits the pulmonary circulation through the **pulmonary vein**, it enters the **left atrium**. From the left atrium, the blood is expelled through the **mitral valve** into the **left ventricle**, and is then ejected through the **aortic valve**. The **aorta** is the gateway into the systemic circulation from the aorta's origin on the left ventricle.



What are the pathophysiologic mechanisms for Mr. Wood's symptoms?

Hypotension and Tachycardia

As Mr. Wood's heart fails to pump adequately, the baroreceptors located in the aortic arch and carotid bodies sense a decreased blood pressure. This will stimulate the SNS to release the catecholamines (epinephrine and norepinephrine), which increases the heart rate and the pumping action (contractility) of the heart, and constricts the peripheral blood vessels to attempt to get more blood into the central system.

The renin-angiotensin-aldosterone system is also stimulated, causing further vasoconstriction and the retention of water and sodium.

Rales and Respiratory Rate

The inadequate pumping of Mr. Woods' heart causes a backup into the lungs from the left side of the heart. The pressure inside the pulmonary blood vessels increases to such a point that fluid (plasma) escapes into an area of less pressure - the alveoli and smaller airways. This is known as cardiogenic pulmonary edema. Fluid in the airways will "bubble" with respiration, causing the crackling sound of rales. This fluid also impairs gas exchange, which triggers the medulla to increase respiratory rate and effort.

Anxiety

The decrease in oxygenation from pulmonary edema and the actions of the catecholamines combine to cause anxiety in the heart failure patient. In severe cases, these physiologic mechanisms can cause a feeling of "impending doom."

What signs and symptoms indicate left sided heart failure?

All patients with heart failure will exhibit fatigue and weakness, and an S₃ and S₄ may be auscultated. The patient with **left-sided** heart failure will exhibit signs that relate to the arterial flow of blood.

Symptoms of Left-Sided Heart Failure

- Anxiety
- Orthopnea, dyspnea, tachypnea
- Cough with frothy sputum
- Diaphoresis
- Basilar rales, rhonchi
- Cyanosis, hypoxia, respiratory acidosis
- Elevated PA diastolic, PCWP
- Nocturia
- Mental confusion
- Pulsus alternans

What signs and symptoms indicate right sided heart failure?

Right-sided heart failure manifests in the venous side of blood flow.

Symptoms of Right-Sided Heart Failure

- Hepatomegaly / splenomegaly
- Dependent pitting edema
- Venous distention, hepatojugular reflux
- Bounding pulses
- Oliguria
- Dysrhythmias
- Elevated CVP, RA, and RV pressures
- Kussmaul's sign (JVD does not decrease with inspiration)
- Abdominal pain, anorexia, weight gain

From the Core Curriculum for Critical Care Nurses, J. Alspach (ed.), page 243.

Angina and Myocardial Infarction

Mr. Caleb Ash presents to his family physician with the complaint of chest pain that "comes and goes." His physician suspects that Mr. Ash may have angina. He orders a stress test and echocardiogram.

What is angina?

Angina is a "warning sign" of myocardial damage. Because the heart has no direct pain receptors or messengers, the pain of myocardial ischemia is through nerves sent back to the spinal cord. The spinal cord sends out the urgent pain messages through other spinal nerves, which may manifest as chest pressure; tightness; arm, back or jaw pain; or GI distress may be the patients' symptoms. Whatever manifestation the warning takes, the symptoms are known as "angina."

What causes angina?

Angina is caused by myocardial ischemia. Causes of myocardial ischemia include atherosclerosis, hypertension, anemia, dysrhythmias, shock, congestive heart failure, and coronary artery spasm. Atherosclerosis is the most common cause of myocardial ischemia.

What are the risk factors for Atherosclerosis?

The risk factors that are "positive" or are likely to cause atherosclerosis include:

1. Age: Males \geq 45 years; Females \geq 55 years or premature menopause without estrogen therapy
2. History of premature coronary artery disease (CAD) in a first degree relative (parent, sibling)
3. Current cigarette smoking
4. Hypertension \geq 140/90 or on medication
5. High density lipid (HDL) count $<$ 35

The factor that is "negative" or is likely to subtract another risk factor is:

1. High density lipid (HDL) count \geq 60

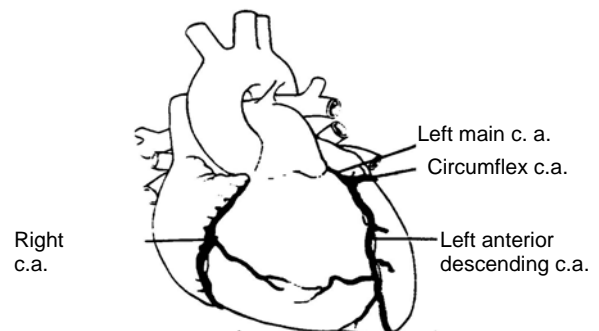
How is angina diagnosed?

Mr. Ash's physician has ordered two of the most common tests for diagnosis of angina. **The stress electrocardiogram** is a test which combines electrical rhythm monitoring with exertion. The patient is placed on a treadmill or other exercise machine and exercises until either angina is experienced or there are EKG changes.

An **echocardiogram** is a diagnostic test that bounces sound waves from the probe off of the structures below it, i.e., the heart. A visual picture forms on the echocardiogram screen. Cardiac wall movement abnormalities, chamber size, valve function, and blood flow can be assessed using this test.

Although the resting echocardiogram was normal, Mr. Ash's stress test was abnormal. His physician ordered a coronary angiogram for the next week.

The most common angiographic procedure done is the coronary angiogram. The term "coronary angiogram" is commonly used to describe a number of diagnostic tests that can be performed in the cardiac catheterization lab. Included in these diagnostic tests are:



- **Coronary arteriogram:** viewing the coronary arteries through the use of dye

- **Right heart cath:** obtaining volumes and pressures in the right heart
- **Left heart cath:** obtaining volumes and pressures in the left heart
- **Aortogram:** obtaining information about the size, function, and pressure of the aorta
- **Ventriculogram:** through the use of dye, obtaining the ejection capability of the heart

During the weekend, Mr. Ash had several episodes of angina. On Sunday evening, the angina was not relieved with three nitroglycerin tablets. As instructed by his physician, his wife called 911. Mr. Ash was brought into the Emergency Room, where he was evaluated for an acute myocardial infarction (AMI).

What is a myocardial infarction?

A myocardial infarction, or MI, is the end result of tissue ischemia. An MI indicates that cells in the myocardium have been anoxic and have died. There are three phases in the evolution of an MI: ischemia, injury, and infarction.

In **ischemia**, the myocardium is deprived of oxygen and/or blood. At this point, if the blood supply or oxygen is returned, the myocardium will return to normal. **Injury** is the next phase of an MI, where the cells have become damaged from the lack of oxygen. If interventions are started in time, the myocardium will return to normal. **Infarction** occurs when the cells are deprived of oxygen long enough to die. Infarction is irreparable.

Immediately to 7 days after an MI, the infarcted area is "mushy" and friable. The cells are dead, and no longer contract or move. After about seven days, collagen scar tissue forms over the area, making the infarcted region more stable. Stretching of the dead tissue before seven days can lead to either a ventricular aneurysm and rupture or to remodeling (thickening of the muscle) of the expanded area, which will lead to congestive heart failure.

What arteries supply the heart?

The heart needs oxygenated blood like any other tissue in the body. All of the coronary arterial blood supply arises from two "holes" in the root of the aorta, called coronary ostia. During contraction of the ventricles, the coronary ostia are compressed. Coronary arteries are supplied with blood only during diastole.

The right coronary artery arises from the right ostia and is located between the right atrium and right ventricle. The right coronary artery is also known as the RCA.

The RCA artery supplies:

- the SA node in 55% of hearts
- the AV node in 90% of hearts
- the RA and RV heart muscle
- the inferior-posterior wall of the LV

For most people, the RCA divides into two branches: the posterior descending artery, which supplies the RV and inferior wall of the LV; and the marginal acute branch, which supplies the inferior surface of the RV.

The left coronary artery comes off of the left coronary ostia as the left main coronary artery (LMCA). It quickly branches into the left anterior descending artery (LAD) and the circumflex artery. The LAD supplies the:

- anterior part of the interventricular septum
- the anterior wall of the LV
- the right bundle branch of the conduction system
- part of the left bundle branch.

The circumflex artery, and its major branch, the obtuse marginal, supplies:

- the AV node in 10% of all hearts
- the SA node in 45% of all hearts
- the lateral posterior surface of the left ventricle.

The coronary arteries supply all of the layers of the heart, which means that they do not lay just on the outside surface of the heart. The large part of the artery lies on the surface of the heart, with smaller branches entering into the myocardial and subendocardial tissues to supply all parts of the heart.

Mr. Ash complains of severe painful pressure in the center of his chest and radiating down his left arm and up his jaw. He is diaphoretic and pale. He complains of some nausea. He is given morphine and started on a nitroglycerin drip, but does not have significant pain relief.

What is causing Mr. Ash's symptoms?

Chest Pressure and Radiation

Because the heart has no direct pain receptors or messengers, the pain of myocardial ischemia is shunted through the spinal nerves of its dermatome. The warning sent by the heart may manifest in many different ways, according to which spinal nerves were stimulated. Chest pressure, tightness; arm, back, or jaw pain; or GI distress may result.

Diaphoresis and Pallor

As the injury has occurred to his heart, Mr. Ash's sympathetic nervous system has kicked into high gear. The catecholamines (epinephrine and norepinephrine) that were released in response to SNS stimulation cause peripheral vasoconstriction and sweating, resulting in cool, clammy, and pale skin.

Nausea

Gastrointestinal distress and heartburn are two of the many symptoms of an MI. Nausea and vomiting, however, are commonly seen with an RCA occlusion. The RCA supplies the AV node, which is also stimulated by the vagus nerve. Irritation of the vagus nerve occurs when the AV node is deprived of oxygen, resulting in nausea.

Mr. Ash is transferred to the CCU after a bolus of a thrombolytic agent is given.

Pericarditis/Pericardial Tamponade

Ms. Louise Comparo is a 36 year old woman who has been undergoing radiation therapy for breast cancer. She enters the ER at approximately 2:30 a.m. complaining of chest pressure, dizziness, and shortness of breath. The assessment shows: distended neck veins, normal breath sounds, distant heart sounds, BP 86/62 mm Hg, HR 156, RR 32. The preliminary diagnosis is cardiac tamponade.

What structures are involved with pericarditis and cardiac tamponade?

There are four layers of the heart: the pericardium, epicardium, myocardium, and endocardium. The **pericardium** is the outermost layer of the heart and makes up a sac that surrounds the heart. This sac is divided into the fibrous and serous pericardium. The **epicardium** is a component of the serous pericardium and

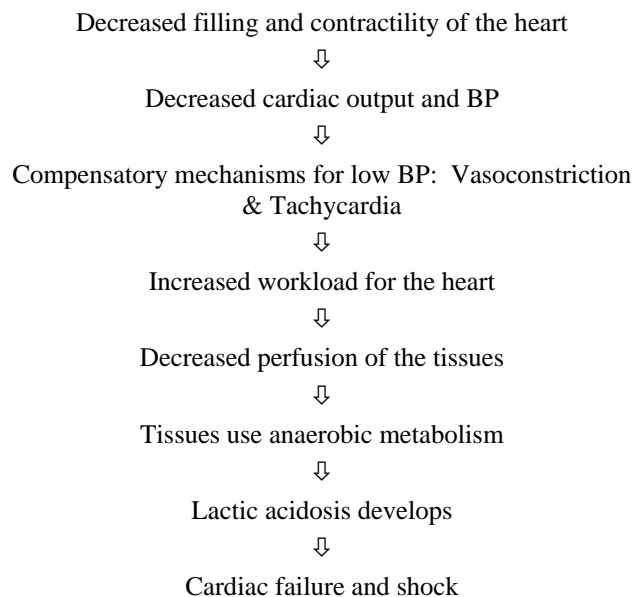
covers the heart and great vessels. The **myocardium** is the muscular portion of the heart which contains conduction fibers, atrial muscle fibers, and ventricular muscle fibers. The **endocardium** is the innermost surface of the heart that covers the chambers of the heart.

What are the possible mechanisms of tamponade in Ms. Comparo's case?

Pericarditis is an inflammation of the pericardial sac, usually as a result of a more generalized disease process, such as an MI or leukemia. Pericarditis will not cause the heart to pump less effectively; however, the complications of pericarditis will cause pericardial effusions and fibrin deposits.

- In a **pericardial effusion**, fluid leaks from the blood vessels supplying the pericardial sac into the space between the fibrous and serous layers. As the effusion grows, the sac becomes "full". When there is no further room for expansion to the outside of the heart, the effusion will either stop, or will continue to grow, causing a decreased pulse pressure and tamponade.
- Fibrin deposition on the serous pericardium from pericarditis causes **constrictive pericarditis**. The constriction does not allow expansion or effective contraction of the heart, rendering it unable to adjust to minute to minute changes in volume and pressure.

Pericardial tamponade is the end result of either pericardial effusion, constrictive pericarditis or pericardial hemorrhage. Cardiac tamponade occurs when the heart is unable to fill or pump adequately, leading to:



What caused Ms. Comparo's symptoms?

Because the heart was being compressed by the fluid in the pericardium, it was not able to pump effectively, causing less and less blood to enter into the system. The net result: **hypotension**. The response of the SNS causes **tachypnea, and tachycardia**. Fluid in the pericardium will uniformly compress the heart, not just one side. This causes an equalization in pressures between the right side and the left side of the heart. This equalization translates into a **narrow pulse pressure**, or a diastolic pressure that is very close to the systolic pressure.

Summary

This program presented information regarding the normal anatomy and physiology of the heart and blood vessels and compared the normal state of affairs to pathological states. Information on angina, myocardial infarction, congestive heart failure, pericarditis, and cardiomyopathy was given to form a foundation of knowledge to apply to clinical practice.

Recommended Reading

1. Brozene S, Russell SS. (1999). *Core Curriculum for Medical-Surgical Nursing*, 2nd ed. Academy of Medical-Surgical Nurses, Janetti NJ.
2. Phipps WJ, Sands JK, Marek JF, eds. (1999). *Medical-Surgical Nursing: Concepts & Clinical Practice*, 6th ed. St. Louis: Mosby, Inc.
3. Seidel HM, Ball JW, Dains JE et al, eds.(2002) *Mosby's Guide to Physical Examination*, 5th ed. St. Louis: Mosby, Inc.
4. Stillwell, S. (2002). *Mosby's Critical Care Nursing Reference*. 3rd ed. St. Louis, Mo: Mosby/Elsevier.
5. Smeltzer SC, Bare BG, eds. (2002) *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 10th ed. Philadelphia: Lippincott William and Wilkins.
6. Wiegand, D.J.L. & Carlson, K.K. (eds.) (2005). *AACN Procedure Manual for Critical Care*. 5th ed. Philadelphia: Elsevier.
7. Functional peripheral arterial disease: Raynaud's Syndrome. (2008). Retrieved March 13, 2010, from <http://www.merck.com/mmhe/sec03/ch034/ch034c.html#sec03-ch034-ch034c-1045>
8. Hanly, E. J. (2009, May 1). Buerger Disease (Thromboangiitis Obliterans) Retrieved March 13, 2010, from, <http://emedicine.medscape.com/article/460027-overview>
9. Occlusive peripheral arterial disease: Thromboangiitis Obliterans. (2008). Retrieved March 13, 2010 from, <http://www.merck.com/mmhe/sec03/ch034/ch034b.html#sec03-ch034-ch034b-1029>

Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. If you are completing this home study as pre-reading for a TCHP class, please bring your post-test and evaluation to class with you for processing. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

HealthEast, HCMC, & MVAMC Employees

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for \$12.00. Please make check payable to **TCHP Education Consortium** and mail to:

**TCHP Education Consortium
Capitol Office Building
525 Park Street, Suite 120
St. Paul, MN 55103**

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Cardiovascular Critical Care Primer Post-Test

Please print all information clearly and sign the verification statement:

Name _____
(please print legal name above)

Birth date (required)

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

For HealthEast, HCMC, or MVAMC, Hospital employees only:

Hospital _____ Unit _____

Personal verification of successful completion of this educational activity (required):

I verify that I have read this home study and have completed the post-test and evaluation.

Signature _____

- Which of the following can be a symptom of angina?
 - chest pressure
 - jaw pain
 - GI distress
 - all of the above
- The right coronary artery supplies:
 - the SA node, the LA and LV
 - the AV node, the LA and LV
 - the AV node, the RA, and RV
 - the bundle branches, the LA and RV
- Baroreceptor stimulation from decreased BP will cause:
 - increased heart rate
 - activation of the parasympathetic nervous system
 - decreased blood pressure

- stimulation of the vagus nerve
- A symptom of left sided heart failure is:
 - pitting edema
 - dysrhythmias
 - basilar rales
 - elevated CVP
 - Arterial insufficiency refers to:
 - total occlusion of the artery
 - blood supply inadequate for tissue need
 - clot formation in the artery
 - arterial wall spasm
 - A bruit is caused by:
 - turbulent blood flow
 - arterial occlusion
 - carotid artery dysfunction
 - myocardial infarction
 - Which of the following is NOT a component of Virchow's triad?
 - hypercoagulability
 - intimal damage
 - arterial insufficiency
 - venous stasis
 - Symptoms of dilated cardiomyopathy are the same as the symptoms of:
 - myocardial infarction
 - subaortic stenosis
 - congestive heart failure
 - hypertension
 - Which of the following disease may result in gangrene and possible amputation of a digit?
 - Buerger's Disease
 - Raynaud's Syndrome
 - both of the above
 - none of the above

Expiration date: The last day that post tests will be accepted for this edition is **December 31, 2020**—your envelope must be postmarked on or before that day.

Evaluation: Cardiovascular Critical Care Primer

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
1. Identify the normal anatomy and physiology of the cardiovascular system.					
2. Describe the pathophysiology of an acute myocardial infarction..					
3. Describe the symptoms of heart failure.					
4. Describe the pathophysiology of tamponade.					
5. Identify the factors that favor the development of a venous thrombosis.					
6. Differentiate between dilated, hypertrophic, and restrictive cardiomyopathy.					
7. Differentiate between Buerger's Disease and Raynaud's Syndrome.					
8. The teaching / learning resources were effective. <i>If not, please comment:</i>					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).

	Yes	No
9. Notice of requirements for successful completion, including purpose and objectives		
10. Conflict of interest		
11. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
12. Sponsorship or commercial support		
13. Non-endorsement of products		
14. Off-label use		
15. Expiration Date for Awarding Contact Hours		
16. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? <i>If yes, please describe the nature of the bias:</i>		

17. How long did it take you to read this home study and complete the post test and evaluation:
_____hours and _____minutes.

18. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?
 ___Yes
 ___No, more contact hours should have been offered
 ___No, fewer contact hours should have been offered

Expiration date: December 31, 2020