Coronary Artery Disease: When Plaques Attack

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Contact hours: 8

Course price: $39

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Course Summary

Traces the gradual development of the atherosclerosis that underlies CAD and includes causes, risk factors, diagnosis, management, and complications. Concludes with the components of a cardiac rehabilitation program.

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Criteria for Successful Completions

80% or higher on the post test, a completed evaluation form, and payment where required. No partial credit will be awarded.

Course Objectives

When you finish this course you will be able to:

1. Define coronary artery disease, including its etiology and major forms.
2. Discuss the prevalence, incidence, mortality, and morbidity of CAD.
3. Name and trace the major coronary arteries.
4. Describe atherosclerotic plaque and explain how it contributes to CAD.
5. List at least 5 symptoms and/or diagnostic procedures that will lead to a diagnosis of CAD.
6. Name 4 of the various medical responses in managing CAD.
7. Discuss 3 major complications of CAD.
8. List at least 6 elements of cardiac rehabilitation.

Recognizing CAD

Finally getting a chance to sit down after another long work day, the electrical contractor, sinks into his easy chair. “This business is killing me,” he says to his wife as she rocks their newborn baby. “My accounts payable can’t collect.”

He rubs his left shoulder and complains “My neck and back are so sore.” Assuming the pain is muscular from working on the construction site, he ignores it for days. The next week is similar but a new pain extends to the front of his chest. “Kyle, you keep rubbing your shoulder. You should get that checked out,” says his wife.

After a week of chest and back discomfort unrelieved by Tylenol, 43-year-old, 6 foot, 260 pound Kyle Hansen goes to a walk-in clinic, where his blood pressure measures 210/98 and his chief complaint is persistent chest and left-shoulder discomfort for 1 week. The workup in the clinic is for acute chest pain but there's much more needed for Mr. Hansen.

As the healthcare professional, what would you do first? Is this a medical emergency? What diagnostic tests should be completed? How could this be a heart problem when he's so young? What were the patient's risk factors? How common is this?

This scenario is all too common for coronary artery disease.

The heart supplies the whole body with life-giving oxygen, but how does the heart receive that oxygen? The blood supply to the heart muscles comes via the coronary arteries located on the outer surface (epicardium) of the heart. Heart muscle requires a constant supply of oxygen and nutrients, and blockage of the coronary arteries can lead to serious and even critical heart problems.

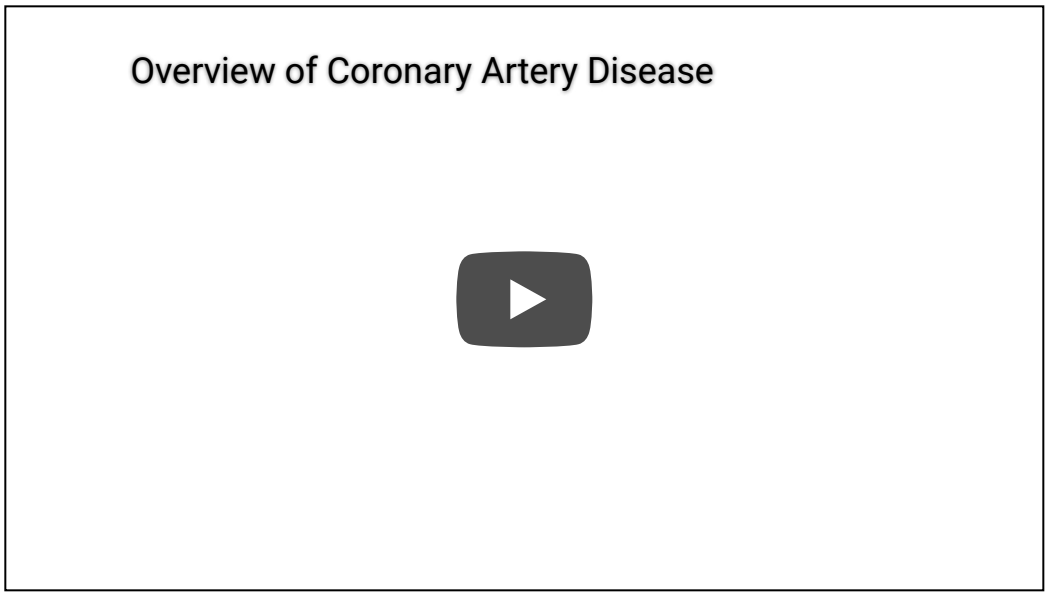
Atherosclerosis, the Underlying Problem

When **atherosclerosis**, a disease that narrows the arteries, begins to limit blood flow through the coronary arteries, the condition is called *coronary artery disease (CAD), coronary heart disease (CHD)* or *atherosclerotic heart disease (AHD)*.

The result of limiting the heart's blood supply is **ischemia**, and CAD is sometimes called *ischemic heart disease* (IHD). Technically, ischemic heart disease is a broader category than coronary artery disease because there are a variety of ways that the blood supply to the heart can be limited, but atherosclerosis is by far the most common cause of ischemic heart disease (Antman et al., 2008).

The term *arteriosclerosis*, is often mistakenly used for atherosclerosis. Arteriosclerosis is the stiffening or hardening of the arterial walls and atherosclerosis is the narrowing of the arterial lumen due to plaque buildup. Atherosclerosis, then, is a specific kind of arteriosclerosis (Nordvist, 2015).

Video (3:01)



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Atherosclerosis leads to the buildup of fatty deposits called **plaque** in the walls of large arteries. All the major arteries are susceptible to atherosclerosis, including the aorta, coronary, carotid, vertebral, cerebral, iliac, and femoral arteries. When atherosclerosis blocks arteries in the limbs, the condition is called *peripheral artery disease (PAD)*; when it blocks the central arteries, the condition is called *cardiovascular disease*.

Atherosclerotic symptoms that appear from blockages in any artery warn the likelihood of atherosclerosis in other arteries; therefore, the appearance of peripheral artery disease makes the presence of CAD more likely, even in patients without symptoms of CAD. A person with CAD has a 1 in 3 chance of having blocked leg arteries (Mitchell & Schoen, 2009).

Once they are present, atherosclerotic plaques in the coronary arteries gradually thicken. The bulging plaques narrow the arterial lumens and reduce the amount of blood that is delivered to the heart muscles. In addition, clots form on the surface of some of the plaques. Occasionally, the clots, along with pieces of plaque, are dislodged and travel downstream, obstructing smaller arteries, which can cause an **embolism** (an obstruction in a blood vessel).

Whether by the gradual thickening of plaque or the sudden formation of a **thromboembolus** (obstruction of a blood vessel by an embolus), atherosclerosis reduces the perfusion of the heart. Heart muscle that does not get sufficient oxygen and nutrients becomes **ischemic**, and ischemic heart muscle contracts weakly or irregularly. Ischemic heart muscle can also stop contracting, and if deprived of oxygen and nutrients for an extended time, heart muscle will die, causing a heart attack or even sudden cardiac death. The irony is that sudden cardiac death isn't sudden, as it has been developing for years.

Heart Ischemia, the Cause of Angina

Ischemic heart disease causes more deaths and disability and incurs greater economic costs than any other illness in the developed world and is likely to become the most common cause of death worldwide by 2020 (Antman et al., 2008).

If the heart becomes ischemic, an uncomfortable feeling called *angina pectoris* is likely. **Angina pectoris** (or, simply, angina) is a deep chest discomfort that is usually described as squeezing, heaviness, aching, or pressure underneath the sternum. This discomfort can sometimes also be felt along the shoulder, arm, neck, jaw, or upper back.

The name *angina pectoris* was coined in 1768 in one of the first written descriptions of the symptoms of CAD. This description was in a medical paper discussing "a disorder of the breast [chest] marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris" [Latin, anguish and choking of the chest] (Silverman & Wooley, 2008). That this condition was specifically a disease of the heart, however, was not agreed upon until the late 1800s, over a century later.

Typical angina is a chronic condition; it occurs with exercise and it is relieved by rest. At first, physicians did not connect angina with clogged coronary arteries because it was thought that blockages of a coronary artery were always fatal. The assumption of lethality came from the observation that autopsies of patients who died of heart attacks frequently found that the patient had blocked coronary arteries, and in the nineteenth century the left anterior descending (LAD) coronary artery was even called "the artery of sudden death" (Silverman & Wooley, 2008).

By 1920, however, it had become clear that obstructions of the coronary arteries could produce a range of effects, not all of which were fatal, and by 1930 "nonfatal myocardial infarction," or heart attack, was becoming a common diagnosis. Today, angina is recognized as a symptom of CAD that can cause ischemic heart problems along a spectrum from reduced coronary blood flow to total coronary artery blockage and from a temporary self-limiting chest ache to sudden cardiac death.

Major Forms of CAD

If a coronary artery has gradually become narrowed, it can usually still deliver sufficient blood to the heart to support normal pumping when the person is at rest. When the heart beats more rapidly and forcefully, such as during stress or exercise, it requires more oxygen. A time will come when the heart needs more oxygen than a narrowed artery can deliver, and at this point the heart muscle becomes ischemic and cardiac tissue may die.

Stable Angina

An individual who has narrowed coronary arteries may be asymptomatic at rest but with exercise can experience angina, or chest pain. When the person stops exercising and returns to a resting state, the angina will subside after a few minutes. This condition of predictable, exercise-dependent angina that is relieved by rest is the form of CAD called **stable angina.**

Acute Coronary Syndromes

More serious forms of CAD are called **acute coronary syndromes**. Acute coronary syndromes occur when a coronary artery suddenly becomes obstructed by clots or pieces of atherosclerotic plaque, causing the heart muscle to receive insufficient oxygen even at rest. Acute coronary syndromes occur unpredictably: the person might be exercising, but could also be relaxing, watching TV, or even sleeping.

Angina from an acute coronary syndrome tends to feel more severe than stable angina, and it usually does not ease with a few minutes of rest or even with sublingual nitroglycerin tablets. Acute coronary syndromes cause ischemia to threaten the viability and function of heart muscle.

When the obstruction causes an acute coronary syndrome but lasts for only a brief time and no significant heart necrosis occurs, it is called an episode of unstable angina. In other cases, the ischemia is substantial and persistent, causing heart muscle death, and the acute coronary syndrome is called a **myocardial infarction (MI).**

Myocardial infarction is one of a number of serious consequences of CAD, which also causes heart failure, arrhythmias, and sudden cardiac death. In the United States, CAD is the leading cause of death for both men and women (CDC, 2016a). Fortunately, early treatment can significantly reduce the likelihood that a patient will develop life-threatening complications.

Overview of Treatment for CAD

Lifestyle changes are the first-line treatment for CAD. People with coronary atherosclerosis should stop smoking, eat reduced-calorie, low-fat/high-fiber diets, and exercise regularly. Diabetes, hypertension, high levels of blood cholesterol, and excess body fat are all CAD risk factors and, if present, should be aggressively controlled. This plan is used both to prevent and to treat CAD.

Medications are usually part of the management of CAD. Medications are also given to reduce the heart's workload and prevent blood clots. Beta-blockers, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors, or antiplatelet drugs such as aspirin may be used. When a patient's coronary arteries have already become significantly narrowed or obstructed, treatment may extend beyond medicines and require surgical reopening of the artery (angioplasty) or bypassing the blockage with arterial or venous grafts (Schoen & Mitchell, 2009; NHLBI, 2016a).

In our earlier scenario, Mr. Hansen was diagnosed with CAD and angina. What symptoms did he present with, and what were his risk factors? His symptoms of hypertension and chest pain are classic for CAD and angina, but because the chest pain wasn't relieved by rest it was unstable. A quick 12-lead ECG demonstrated a non-STEMI. He was given nitroglycerine, oxygen, and morphine for chest pain. He was sent by ambulance to the hospital for a full workup, angiogram, and treatment. His risk factors included BMI >30, hypertension, high-fat diet, male gender, age >40 and stress. So is Mr. Hansen the rare case? What additional tests and procedures will be done at the hospital? What are the statistics for CAD in men and women like Mr. Hansen?

Statistics About CAD

Coronary artery disease is a significant public health problem. One American dies from CAD every minute, and, of every 5 people who experience an acute coronary syndrome, 2 will die from related heart problems within the year. The problem of atherosclerosis begins in youth, and CAD develops gradually over the decades; therefore, health professionals in every field face some aspect of CAD and have a role in helping patients prevent or control the disease and its deadly consequences.

Prevalence

Prevalence indicates how many people currently have the disease, and approximately 1 of every 13 Americans aged 18 years and older has CAD. It is the leading cause of death for people of most racial and ethnic groups in the United States (Mozaffarian, 2015; CDC, 2016a). In the United States, CAD is most common among Hispanic Americans and least common among Asian Americans, Pacific Islanders, American Indians, and Alaska Natives.

Coronary artery disease develops gradually, and the strongest risk factor for CAD is age, so it is typically a disease of older adults. Although the disease may become clinically apparent by age 40 and the trend is increasing, people 65 years of age and older account for approximately 85% of deaths from CAD (Boudi, 2013).

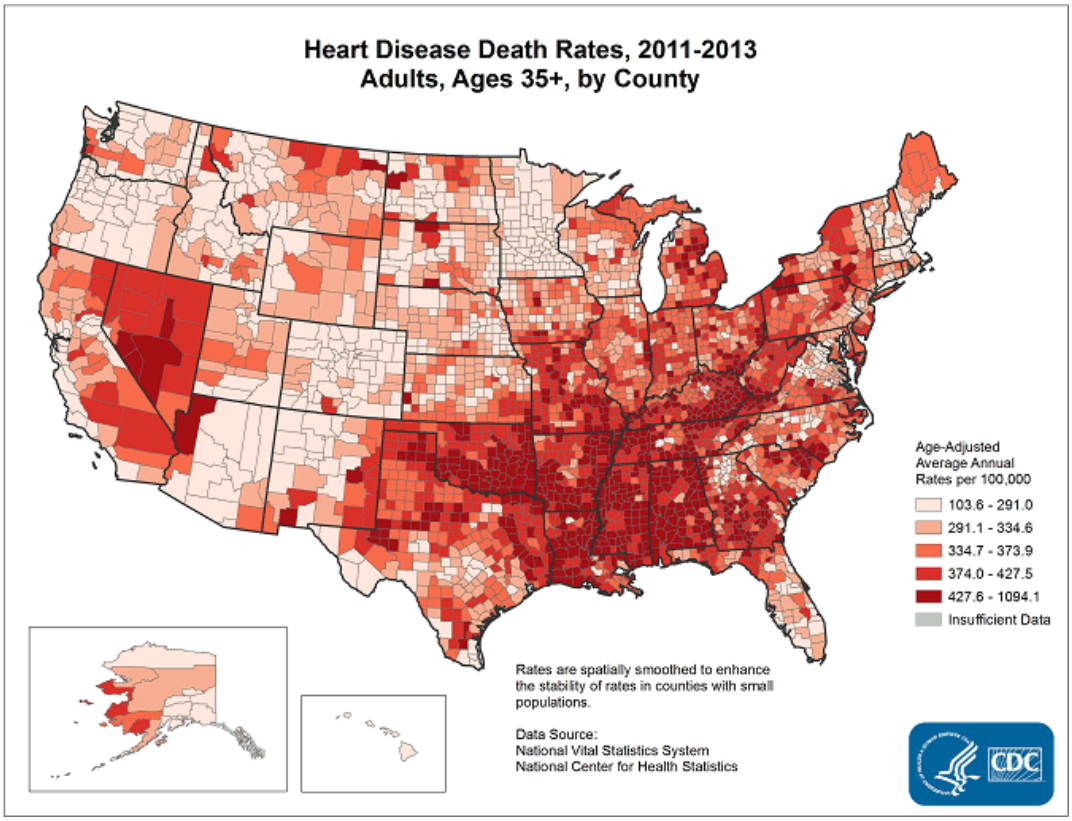
Incidence

Incidence tells how many new cases are diagnosed each year, and about 935,000 Americans have a heart attack. Often CAD is not diagnosed until a cardiac event, such as a heart attack, occurs. Of the almost 1 million MIs annually, 610,000 are a first heart attack and 325,000 are a repeat heart attack (CDC, 2016a).

Mortality

Mortality is the statistic for actual deaths caused by a disease. Heart disease is the number one cause of death for both American men and women, accounting for 1 out of every 4 deaths each year. Decades ago CAD was more common in men, but in the past several decades women have approached the same levels of CAD-related deaths. Every 34 seconds someone has a heart attack and each minute someone dies from a heart disease–related event. CDC reports that 600,000 deaths each year are due to heart disease in the United States (CDC, 2016a). Patterns for age, gender, race and even geographic location have identified those at greater risk to develop CAD.

Although the frequency of CAD is similar in both black and white populations, the death rate from CAD is greater for blacks than for whites. In 2009 African Americans were 30% more likely to die from heart disease than non-Hispanic white men (OMH, 2013).



Death rates vary throughout the United States. The highest death rates (darkest colors) are in the South and around the regions of Mississippi, Alabama, Georgia. Source: CDC, n.d.

There is one hopeful statistic. Despite the fact that CAD is the leading cause of death in the United States, there has been a 30% reduction in the mortality rate since the latter part of the twentieth century. Factors such as increased awareness and patient education, early identification and treatment, availability of coronary artery bypass surgery and stent placement, thrombolytic therapy, and an emphasis on lifestyle modification are some of the reasons for this reduction (Boudi, 2013).

Deaths from CAD often happen to people with no previous heart symptoms. Although some people may feel a rapid heartbeat or dizziness that alerts to a dangerous heart rhythm, over half of the people who have sudden cardiac deaths were previously asymptomatic (WebMD, 2012).

Morbidity

Morbidity is the term for the cost and consequences of a disease. Illness from CAD strains the American health system. Cardiovascular disease, including heart disease and stroke, costs the United States $312.6 billion each year. This total includes the cost of healthcare services, medications, and lost productivity. Cardiovascular diseases are also a leading cause of disability, preventing affected persons from working and enjoying family activities (CDC, 2016a).

Although Mr. Hansen is younger than the expected age, due to his risk factors of obesity, stress, and high-fat diet, his CAD was manifested in chest pain. Because CAD silently progresses, restricting the lumen of arteries, people often are not aware it is developing until they notice symptoms. A common response, however, is **denial**, as people think the chest discomfort, heaviness, neck and back ache are related to stress, poor sleep, and just life.

Test Your Learning

In the United States, coronary artery disease is the number one killer of:

A. Men.
B. Women.
C. Both men and women.
D. Children.

Apply Your Knowledge

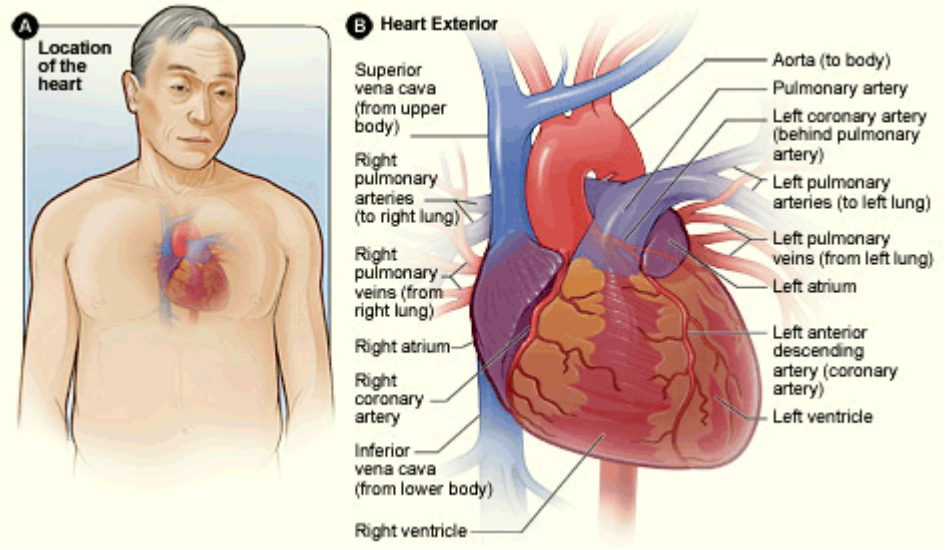
Who would be at great risk for developing CAD in a patient profile? Typically, we think of those older than 60 years of age. We are more likely to identify the symptoms of an MI in an older adult, but what about the variations in symptoms for those in their forties, such as Mr. Hansen, or people with diabetes and cardiac neuropathy? Be aware that CAD is developing in a younger population and that diabetic neuropathy blocks the usual symptom of chest pain that we rely on.

Answer: C

Anatomy of the Coronary Arteries

The coronary arteries are also called the *epicardial arteries* because they run along the outer surface of the heart on the epicardium; the main ones are the left coronary artery and the right coronary artery. The left coronary artery divides into the left anterior descending and the left circumflex arteries. The right coronary artery gives rise to the posterior descending artery along the back surface of the heart.

The Location of Coronary Arteries of the Heart



(A) Location of the heart in the body; (B) front surface of the heart, including the coronary arteries and major blood vessels. Source: NHLBI, 2016b.

Normally, there is little overlap between the areas to which the coronary arteries and their branches provide blood. A secondary circulation does exist, but it is made of small arteries. With enough time, these collateral arteries can widen and offer some circulation to the heart muscle with a partly blocked coronary artery. However, a sudden blockage in a coronary artery will cut off most of the oxygen and nutrients to the corresponding region of heart muscle with no time for secondary circulation to compensate.

Mr. Hansen received an emergency angiogram in the hospital to which he was transferred. It showed a 99% blockage in the LAD (left anterior descending coronary artery), also known as the "widow maker." His wife was very grateful they were able to identify the blockage before she became a young widow! Mr. Hansen received two coronary stents to open the blockage.

Test Your Learning

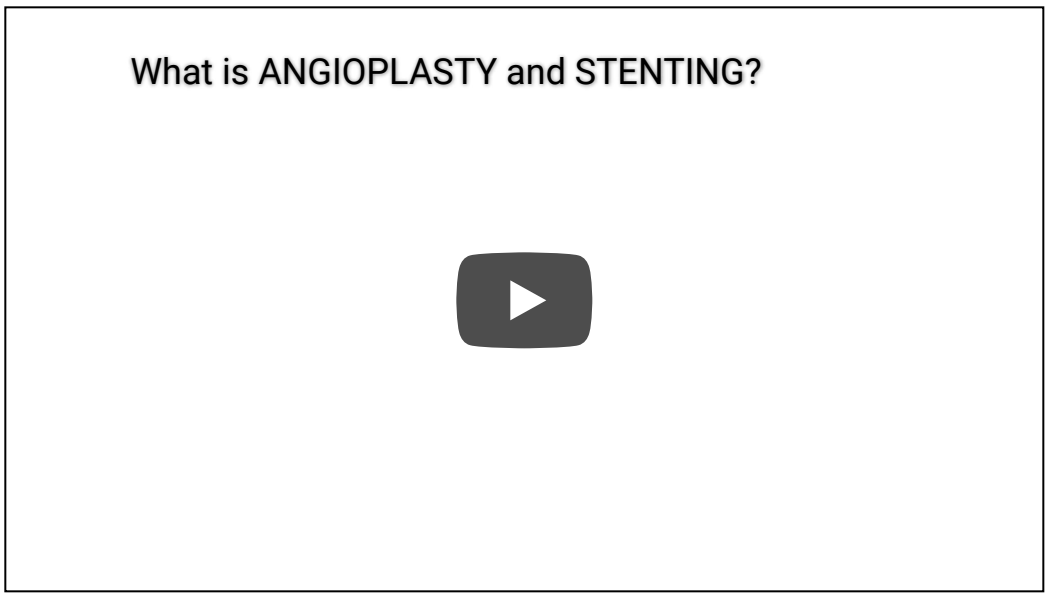
The two main coronary arteries:

A. Run along the inside walls of the heart.
B. Run along the outer surface of the heart.
C. Are called the left anterior descending and the left circumflex coronary artery.
D. Are called the left anterior descending and the right posterior descending coronary artery.

Apply Your Knowledge

How can you help a patient prepare and recover for an angiogram?

Video (1:09)



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Answer: B

Atherosclerosis: Cause of CAD

Atherosclerosis is the disease behind the disease. When atherosclerotic processes take hold in the arteries that supply blood to the heart, the condition becomes **coronary artery disease (CAD).** Atherosclerosis is a degenerative disorder that injures the inner walls of large arteries. In atherosclerosis, thick abnormal patches called **plaques** accumulate at scattered locations along the artery's innermost endothelial layer. The plaques are disorganized masses filled with cholesterol, lipids, and other cells, all covered by a white fibrous coating (Mitchell & Schoen, 2009).

Atherosclerotic plaques narrow an artery and hinder blood flow. The result is that the surface of a bulging plaque sometimes tears, exposing material that stimulates clot formation. Clots and ruptured plaque material can then break away from the wall, be carried by the blood, and clog arteries downstream.

Atherosclerosis damages tissues throughout the body:

- Blockages in the carotid arteries can reduce or block blood flow to the brain, causing a **stroke**.
- Obstructions in the peripheral arteries in the legs can cause claudication (pain when walking), gangrene, and **deep vein thrombosis (DVT).**
- Resultant DVTs can travel to the lungs, causing a **pulmonary embolism** and respiratory arrest.
- Atherosclerotic obstructions in the intestines cause **ischemic bowel**.
- In the coronary arteries, atherosclerotic obstructions cause **heart attack** (NHLBI, 2016c).

Causes of Atherosclerosis

Causes of atherosclerosis include fatty streaks, foam cells, atherosclerotic risk factors, and plaque destabilizers.

Fatty Streaks

Beginning in childhood, a cascade of events slowly and quietly leads to the development of atherosclerosis. Children develop fatty streaks along the walls of their large arteries largely from diets high in fat. These streaks are sites where lipoprotein particles are protected from direct contact with the blood. The lipoproteins become oxidized into destructive molecules and the resulting oxidants injure nearby cells.

Foam Cells: Overwhelmed Macrophages

In the arterial walls, leukocytes (white blood cells) are attracted to the areas of cell injury, and the incoming leukocytes initiate a local inflammatory reaction. Some of the attracted leukocytes are macrophages. Macrophages are clean-up cells, and they begin to engulf the local lipids. When macrophages are overwhelmed by the lipids in their vicinity, they become bloated with fatty debris. The accumulation of fat-filled macrophages, which are called **foam cells,** is a characteristic of atherosclerotic plaque.

In a person with a healthy balance of blood fats such as low blood levels of LDL cholesterol and high blood levels of HDL cholesterol, there is only a modest accumulation of lipids in arterial walls. The available macrophages can swallow and cart off sufficient lipids from the fatty streaks to avoid a lipid buildup, and few foam cells accumulate. When this and the other lipid-removal mechanisms are working smoothly, the amount of sequestered lipid can be controlled and atherosclerosis is minimal. Unfortunately the American diet is high in fat and generally overwhelms the macrophages' ability to clean up damaged arteries.

A total lipid profile for Mr. Hansen revealed total cholesterol >200 mg/dL and low HDLs <25 mg/dL. Atherosclerotic plaques had built up over time and left only a 1% space for blood flow at the junction of the LAD.

Test Your Learning

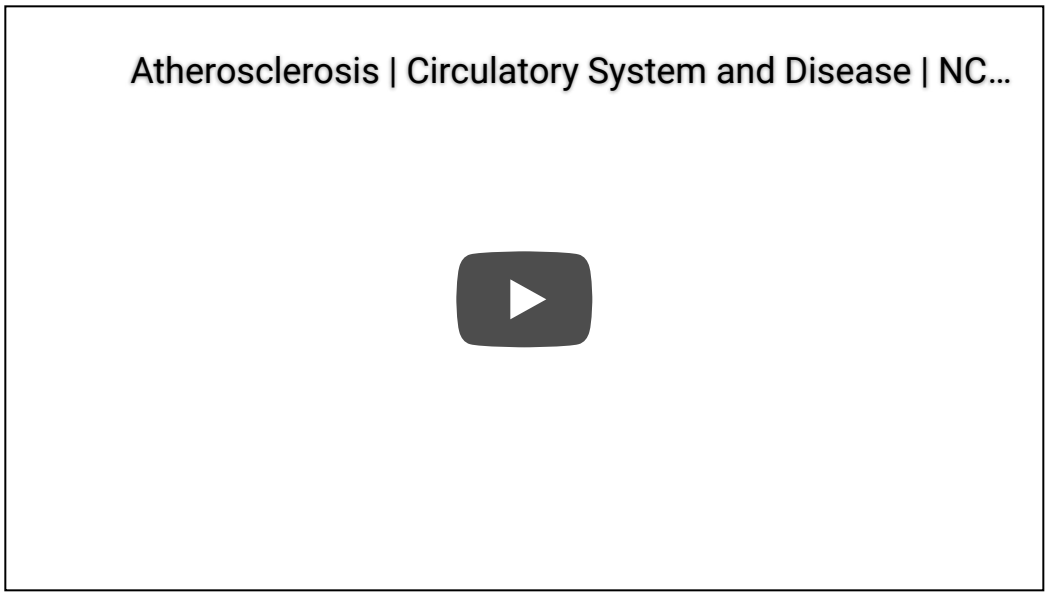
Atherosclerotic plaques are filled mainly with:

A. Blood clots.
B. Lipids and other cells.
C. Embryonic arterial wall tissue.
D. Glycogen.

Foam cells are:

A. Macrophages overfilled with lipids.
B. Lipid droplets.
C. Vacuolated red blood cells.
D. Necrotic, "foamy-looking" smooth muscle cells.

Resource



https://www.youtube.com/watch?v=g3kDdg8r6NY

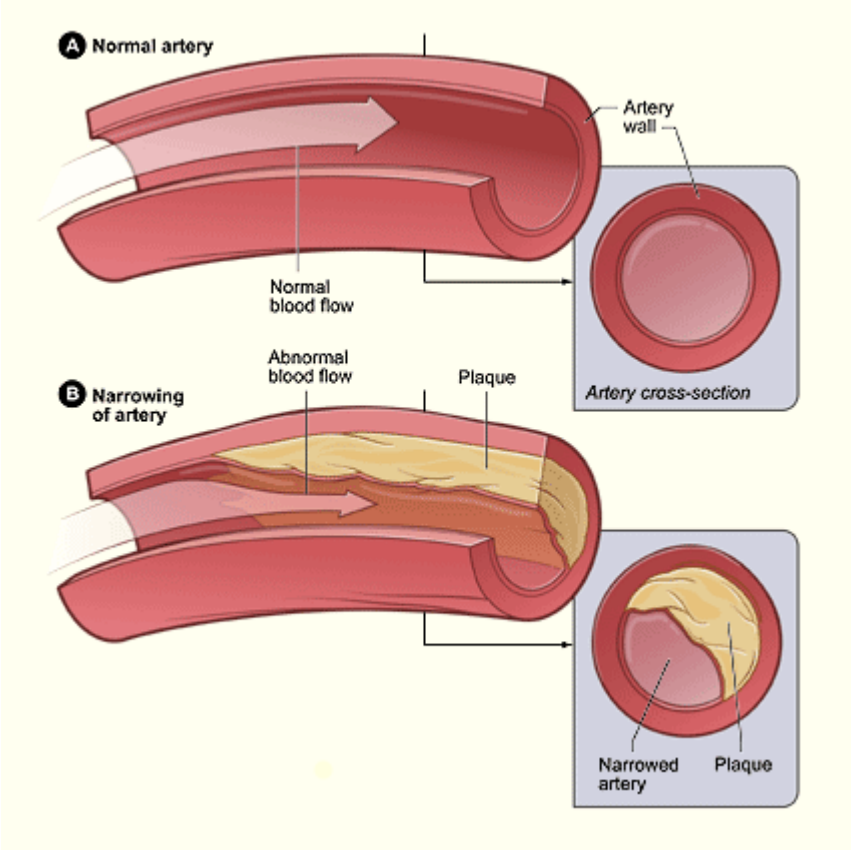
Answers: B, A

Atherosclerotic Risk Factors

A variety of conditions can make the lipid removal systems inefficient; they are known as **atherosclerotic risk factors**. Atherosclerotic risk factors include smoking, diabetes, hypertension, obesity, physical inactivity, diet high in saturated fats, stress, and the accumulated wear of old age. When these conditions interfere with lipid removal, atherosclerosis can slowly clog arteries (Libby, 2008).

In atherosclerosis, the body's lipid removal systems are working poorly. Foam cells die before they can remove lipids, and a core of necrotic cells forms inside the expanding yellow streak. The body attempts to repair the damage through collage, which enter the mass and begins to create a meshwork with other extracellular matrix materials. By this point, the yellow streak has become an atherosclerotic plaque.

Normal and Atherosclerotic Arteries



(A) Normal artery with normal blood flow; (B) artery with plaque buildup and diminished blood flow. Source: NHLBI, 2016b.

Plaque Destabilizers

Gradually, atherosclerotic plaques thicken. Internally, the plaques are disorganized and weak. As part of the local repair efforts, a network of small blood vessels grows into the enlarging plaque. The new vessels tend to be leaky due to the inflammatory process, which dilates blood vessels to more easily allow passage of white blood cells, macrophages, and signaling chemicals. The result is that hematomas and clots form inside the plaque. Some regions in the plaque end up with little oxygen and few nutrients. In these poorly oxygenated areas, the local cells—mainly leukocytes and smooth muscle cells—die and create additional necrotic pockets. As in many tissues with chronic inflammation, calcium salts are slowly deposited, making parts of the plaque brittle, causing arteriosclerosis, or hardening of the arteries.

Along with hematomas, these necrotic pockets make the entire plaque susceptible to erosion, crumbling, and rupture. Tears in the fibrous coat of a plaque allow the extracellular matrix inside to come in contact with blood. This stimulates blood-clotting mechanisms and clots form along the edges of the eroded plaque.

Many people go through their entire lives unaware that they have atherosclerosis; however, even asymptomatic atherosclerosis is a constant threat because plaques continue to enlarge. One consequence of this continued growth is that the pressure of an enlarging plaque weakens the muscular layer of the arterial wall and makes the region susceptible to ballooning into an **aneurysm** (rupture of the wall).

Another consequence is that atherosclerotic plaques become less stable. At some point, the pressure of local blood flow can push the clots and plaque debris downstream to become artery-clogging emboli. The obstructed arteries leave their surrounding tissues ischemic, and the ischemic tissue eventually dies (Libby, 2008).

Test Your Learning

As they age and thicken, some atherosclerotic plaques accumulate deposits of:

A. Copper.
B. Potassium.
C. Sodium.
D. Calcium.

Answer: D

Atherosclerotic Plaque

Atherosclerotic plaques are white or yellowish bulges filled with cells, cholesterol, and cellular debris. When the surface of a plaque becomes abraded or torn, clots form, and these areas appear red-brown. Individual atherosclerotic plaques are 0.3 to 1.5 cm in diameter, but adjacent plaques can merge into larger lesions.

Atherosclerotic plaques develop in patches, and they are more likely to develop in places where epithelial cells have been injured. Regions of blood turbulence, such as arterial branch points, have chronically injured epithelial cells, and, in atherosclerosis, plaque tends to develop near branch points inside large arteries.

Certain arteries are more likely to have atherosclerotic plaques than others. Here, in order, are the arteries most likely to have significant atherosclerosis:

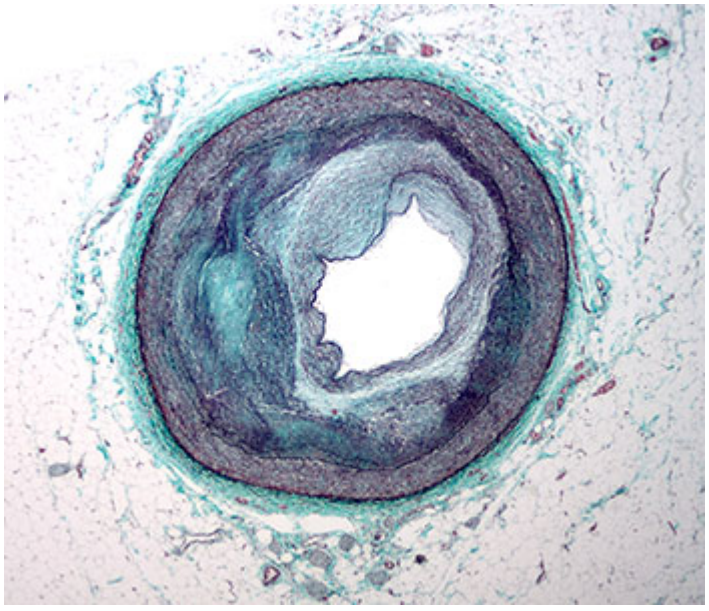
- Abdominal aorta
- Coronary arteries
- Popliteal arteries (behind the knees)
- Internal carotid arteries
- Arteries in the circle of Willis, which supply blood to the cerebral hemispheres of the brain (Mitchell & Schoen, 2009)

Atherosclerosis of the Coronary Arteries

The two main areas most commonly thickened by atherosclerosis are the lower abdominal aorta and the coronary arteries. Within the coronary arteries, the branch points, shaped like forks in the road, tend to accumulate the thickest buildup of atherosclerotic plaque.

Atherosclerotic plaque forms as a mass inside the arterial wall. Initially, this mass causes a bulge on the outside of a coronary artery, increasing the artery's overall diameter. Later, as the disease progresses, the plaque begins to bulge into the lumen (inner passageway) of the artery, and at this stage, the plaque reduces the effective inner diameter of the artery and the stenosis can be detected by angiography.

Atherosclerotic Artery with Narrowed Lumen



Micrograph of the distal right coronary artery with complex atherosclerosis and luminal narrowing. Source: Wikimedia Commons.

Coronary arteries that have lost 50% to 75% of their inner diameter can no longer deliver sufficient blood flow to meet the increased needs of heart muscle during exercise. That level of stenosis will lead to ischemic symptoms, typically angina, when the patient exercises. If atherosclerotic plaque thickens sufficiently to fill 80% to 90% of the diameter of a coronary artery, ischemic heart symptoms appear even when the person is resting (Antman et al., 2008).

Over time, the section of blood vessel that lies beyond a narrowed segment will dilate, any small collateral arteries will widen, and new collaterals will grow in efforts to deliver life-giving oxygen. In these ways, the arteries of the heart can partly compensate for a narrowed coronary artery when the stenosis has developed gradually.

As brilliant as the body is in adapting for survival, there is no natural protection against sudden arterial narrowing, such as the obstruction produced by clots or debris from a ruptured plaque. Sudden narrowing can lead to ischemia and heart muscle death. This is the most common cause of the death of heart muscle: greater than 95% of all MIs are caused by blood clots and debris from atherosclerotic plaque.

Test Your Learning

Atherosclerotic plaques are likely to accumulate:

A. At branch points and forks inside large arteries.
B. In capillaries and small arterioles.
C. On the surface of mucous membranes.
D. In the large veins, such as the vena cava.

The arteries that supply blood to the heart can partly compensate for a narrowed artery when the stenosis:

A. Is caused by a sudden blood clot.
B. Is caused by a sudden disruption of atherosclerotic plaque.
C. Has been caused by a sudden vasospasm.
D. Has developed gradually.

Heart muscle cells that become ischemic will:

A. Die within 1 to 2 minutes unless they are immediately reperfused.
B. Stop working but can "hibernate" for weeks and then revive when reperfused.
C. Die in 20 minutes to 4 hours if not reperfused.
D. Keep working for 2 to 5 days before they slowly die.

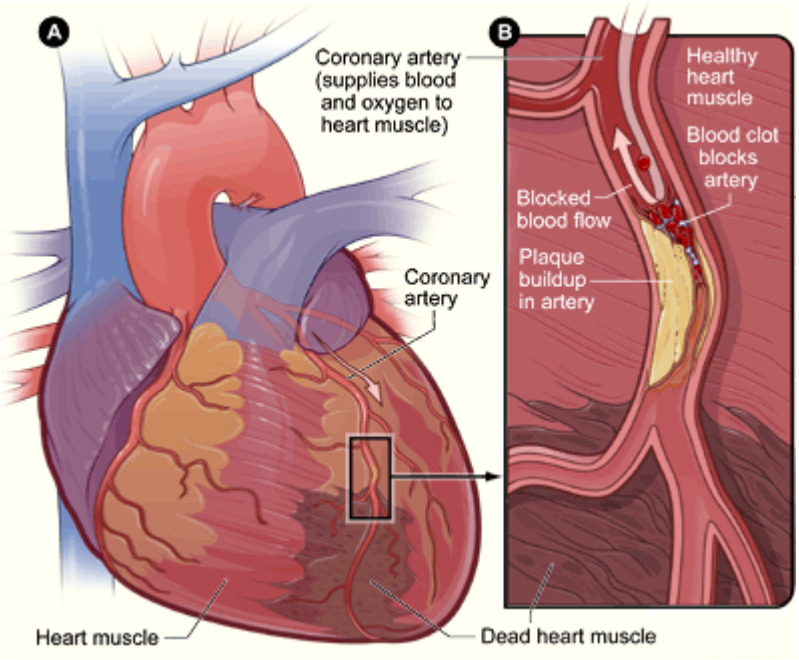
Answers: A, D, C

Effects of Atherosclerosis on the Heart

Normally, when heart muscle works harder, the blood vessels inside the heart—those arteries downstream from the main coronary arteries—dilate to increase blood flow to the muscle cells. However, if atherosclerosis has narrowed the coronary arteries, dilation of the downstream arteries is not enough to prevent ischemia (Antman et al., 2008).

The region of heart muscle that becomes ischemic depends on which artery has been blocked. Approximately half of all MIs are caused by blockages of the left anterior descending artery, 30% to 45% are caused by blockages of the right coronary artery, and 15% to 20% are caused by blockages of the left circumflex artery (Burke & Virman, 2008). After a sudden blockage, the heart muscle cells fed by these arteries become ischemic and, unless they are quickly reperfused, ischemic heart muscle cells will die in 20 minutes to 4 hours, depending on the existing collateral circulation.

Myocardial Infarct in the Wall of the Left Ventricle



(A) Blood flowing down the left anterior descending (LAD) branch of the left coronary artery has been obstructed. Heart muscle beyond the blockage has died from prolonged ischemia (brown patch). (B) A cutaway diagram of the LAD shows plaque (yellow) that had gradually narrowed the arterial lumen. Later, a blood clot, broken loose from upstream plaque, has become wedged into the narrowed section of the LAD, completely obstructing blood flow Source: NIH, 2016.

Symptoms of CAD

Coronary artery disease can become a chronic disease called *stable angina*. It can also give rise to sudden cardiac emergencies called *acute coronary syndromes*. Acute coronary syndromes range from temporary episodes of significant ischemia (unstable angina) to permanent heart muscle damage (myocardial infarction) to sudden cardiac death.

Stable Angina

In **stable angina,** coronary arteries are partly occluded. At rest, dilation of the downstream arteries allows sufficient blood flow to meet the demands of the heart muscle cells. However, during exercise, the increased oxygen and nutrients needed by the heart exceed the capacity of the already-dilated arteries. Therefore, when the person exercises, the heart muscles become ischemic, and typically the person feels angina. In most cases, the ischemia of stable angina does not cause significant muscle cell death.

Symptoms in stable angina occur when the demands on the heart exceed the blood flow through pre-existing stenosis. These symptoms occur predictably, whenever the patient's heart accelerates to a certain level of activity. In stable angina, the event that initiates symptoms is generally external such as exercise, stress, or cold weather. Internally stressors such as psychological stress can also create hypertension and resultant demands on ischemic heart vessels.

Acute Coronary Syndromes

In **acute coronary syndromes**, the event that initiates symptoms is an internal change in the atherosclerotic plaque in the patient's coronary arteries. When a patient suffers an acute coronary syndrome:

- Plaque has expanded, eroded, or ruptured, or
- A thrombus has moved or broken loose, or
- A coronary artery has suddenly tightened in a vasospasm.

When arterial obstruction persists, causing sufficient ischemia to kill muscle cells, this is called a **myocardial infarction (MI, or heart attack).** After an MI, intracellular proteins leak from the damaged cells and circulate in the bloodstream. An MI can be diagnosed by finding cardiac-specific intracellular proteins (cardiac biomarkers) in the blood of a person who has the signs and symptoms of an acute coronary syndrome.

Myocardial infarctions can be definitively diagnosed in symptomatic patients from blood tests. When heart muscle cells die, heart-specific intracellular molecules (cardiac biomarkers such as troponin) leak into the bloodstream.

Sudden Cardiac Death

The abrupt release of atherosclerotic thrombi that causes myocardial ischemia can also trigger fatal ventricular arrhythmias. This appears to be the critical event behind most cases of **sudden cardiac death,** a condition in which patients die unexpectedly and within minutes of the onset of symptoms (Schoen & Mitchell, 2009). This fatal symptom is the end stage of CAD and can only be altered with cardiopulmonary resuscitation.

Mr. Hansen had developed stable angina over years of sedentary lifestyle, high-fat diet, and work stress. Unstable angina had developed quickly as new stress worsened at work and a new baby was born to the busy family. Even happy events such as marriage or a baby can result in stress on the body.

Test Your Knowledge

The chest discomfort of stable angina is often described as:

A. Sharp pain.
B. Fleeting pain.
C. Squeezing or tightness.
D. Made worse by the movements of coughing or breathing.

Cardiac biomarkers are intracellular heart muscle molecules that:

A. Are released into the blood when heart muscle cells die.
B. Are released into the blood all the time.
C. Become dense and visible in CT scans when heart muscle cells first becomes ischemic.
D. Become dense and visible in CT scans when heart muscle cells die.

Answers: C, A

Diagnosis and Evaluation of CAD

Chest discomfort is the most telling symptom of CAD. It can take the form of angina or manifest as acute angina in an MI.

Chest Discomfort

Frequently the chief complaint of a person with CAD is chest discomfort. When a patient presents with a nonacute history of chest discomfort, the workup for possible CAD should include blood work for cholesterol, baseline ECG, stress treadmill, statin or aspirin therapy, and lifestyle modification education.

When a patient comes to a medical facility with very recent or ongoing chest discomfort, a rapid triage and assessment should occur. In the emergency department, clinic, or office, the clinician must quickly distinguish those patients who have potentially life-threatening conditions—MIs, aortic dissection, pulmonary embolus, tension pneumothorax—from nonlife-threatening causes of chest pain or discomfort (Lee, 2008). Conditions such as heartburn, swallowing disorders, pancreatic and gallbladder problems, muscle and bone problems, and lung disorders can all cause symptoms similar to those caused by the heart (Mayo Clinic, 2016). For more details, see Medical Management of CAD below.

Classic Angina

The classic symptom of CAD is a form of chest discomfort called angina. The following symptoms are some of the ways that patients describe angina (Schoen & Mitchell, 2009).

Quality: Squeezing Pressure

The nouns commonly used to describe the feeling of angina include *constriction, heaviness, pressure*, and *tightness*. The adjectives commonly used include *aching, choking, crushing*, *smothering*, and *squeezing*. Patients typically hold a clenched fist over their chest when describing the feeling of angina.

Location: Substernal

Most commonly, patients say angina is located substernally, ie, inside the center or lower center of the chest. Patients may also locate the feeling in the epigastric region. Some patients describe angina as a deep ache in their teeth, jaw, neck, shoulder, or arm, on either side of the body.

Duration: A Few Minutes

The angina of stable angina is temporary, lasting 2 to 5 minutes and coming in a wave that worsens, reaching a peak and then subsiding. Unstable angina lasts longer, typically 10 to 20 minutes. The angina of MI has a variable duration, often lasting longer than 30 minutes.

Triggers: Exercise

Angina can be triggered by exercise, sexual activity, exposure to cold weather, emotional stress (anger, fright, frustration), or a large meal. Any physical or emotional stress that causes tachycardia can induce angina.

The angina of acute coronary syndromes (unstable angina or an MI) can be precipitated by exercise, but it can also occur at rest or it can wake a person from sleep. (Occasionally, the angina of *stable angina* will also occur at night, especially if the patient has sleep apnea.)

Occurrence: Predictable or Unpredictable

The angina of stable angina comes predictably when the patient engages in a certain level of exercise. The angina of unstable angina arises unpredictably, sometimes at increasing or even decreasing levels of exercise. The angina of an MI happens unpredictably.

Relievers: Rest or Nitroglycerin Tablets

The angina of stable angina resolves in about 5 minutes with rest and sublingual nitroglycerin tablets. The angina of unstable angina is usually not relieved by a brief rest, and if it is lessened by nitroglycerin, the pain typically recurs. The angina of an MI usually does not respond to rest or to nitroglycerin.

Accompanying Symptoms: Variable

The angina of an acute coronary syndrome and angina can be accompanied by a variety of other symptoms, including shortness of breath, lightheadedness, nausea, or sweating (Lee, 2008).

Descriptors Not Often Used: Sharp and Brief

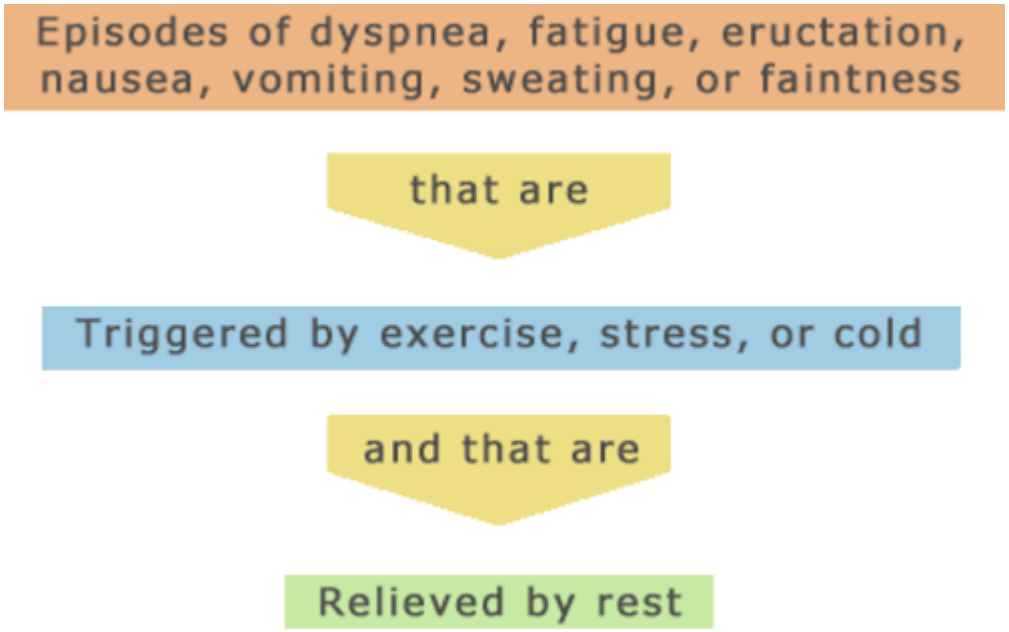
Chest discomfort can be caused by many thoracic disorders so it essential to rule out cardiac issues first as they potentially can be the most lethal. For example, chest pain can manifest with gastrointestinal nausea and gastric ulcers can present as a burning sensation in the chest. Here are some comments **not** typically offered by patients when describing classic angina:

- Sharp
- Brief
- Worsened by chest movements of breathing
- Changes with the patient's position
- Reproduced by an examiner tapping or pressing on the chest
- Below the umbilicus or above the jaw
- Prolonged ache deep to the left breast

Anginal Equivalents

Angina is the complaint that usually triggers a workup for CAD. However, not all people with CAD get angina. Even during an MI, some people do not experience significant chest discomfort, which is why some people may be surprised to hear their ECG shows that a previous MI had occurred.

A few complaints other than chest discomfort can also be caused by heart ischemia. Clinicians suspect heart ischemia when patients complain of the following.



When they are caused by heart ischemia, these symptoms behave like angina and are called **anginal equivalents**. Among patients with CAD, angina is more often reported by men than by women: instead of classic angina, women often report anginal equivalents. Anginal equivalents are also more common for CAD in older adults and in people with diabetes. One of the arts of clinical practice is distinguishing angina—chest discomfort caused by heart ischemia—from other types of chest discomfort.

Test Your Learning

The main symptom of CAD is:

A. Arrhythmias (usually, atrial fibrillation).
B. Chest discomfort called angina.
C. Gradually increasing shortness of breath, fatigue, and lower limb edema.
D. Sudden inability to get enough air.

An anginal equivalent is:

A. A symptom, such as shortness of breath other than chest pain that is caused by heart ischemia, brought on by stress, and relieved by rest or nitroglycerin.
B. The dose of nitroglycerin that can relieve typical angina in a given patient.
C. Sharp chest pain worsened by coughing.
D. Angina brought on by emotion not by exercise.

Answers: B, A

A number of medical conditions other than heart ischemia present with acute chest discomfort. These conditions include aortic dissection, aortic stenosis, esophageal reflux, esophageal spasm, gallbladder disease, herpes zoster, musculoskeletal disease or injury, peptic ulcer, pericarditis, pleuritis, pneumonia, psychological/psychiatric problems (eg, panic disorder), spontaneous pneumothorax, pulmonary embolus, and pulmonary hypertension.

Laboratory tests, ECG recordings, and imaging studies may be needed to distinguish these health problems definitively from heart ischemia. However, the patient's description of chest discomfort can point the clinician toward or away from a diagnosis of heart ischemia. A differential diagnosis for chest discomfort can be determined by evaluating the variations of the following characteristics of pain:

Quality

- Aortic dissection is a tear in the inner wall of the aorta. The pain is severe and sudden.
- Pneumonia, pleuritis, and spontaneous pneumothorax pains are often described as "sharp."

Location

- Aortic dissection pain typically radiates to the back between the shoulder blades.
- Herpes zoster (shingles) pain follows the pattern of the chest dermatomes.
- Gallbladder and peptic ulcer disease usually produce abdominal as well as epigastric pain.
- Pericarditis pain can radiate to the trapezius muscles.

Duration

- Aortic dissection and spontaneous pneumothorax have a sudden onset of pain that does not stop.
- Gallbladder and peptic ulcer pains can continue for hours.
- Pericarditis pain lasts for hours and sometimes days.

Triggers

- Aortic dissection occurs in a weakened area of the aorta. Chronic hypertension, some inherited conditions such as Marfan syndrome, and trauma to the chest wall are risk factors (Johns Hopkins Medicine, n.d.).
- Musculoskeletal pains are usually caused by injury or trauma.

- Esophageal reflux pain can be brought on by alcohol, aspirin, or lying down after meals.
- Gallbladder and peptic ulcer pains typically appear 1 to 2 hours after a meal.
- Musculoskeletal pain is worsened by movement and can be reproduced by local pressure by the examiner.
- Pericarditis pain is usually aggravated by coughing or by certain postural changes.
- Pneumonia, pleuritis, and spontaneous pneumothorax pains are worsened by the chest movements of breathing and coughing.

Relievers

- Esophageal reflux and peptic ulcer pains can be relieved by antacids.
- Pericarditis pain is sometimes relieved by sitting up.

Accompanying Symptoms

- Aortic dissection can cause loss of peripheral pulses.
- Herpes zoster pain can be accompanied by a vesicular rash in the region.
- Pericarditis pain can be accompanied by a pericardial friction rub.
- Pneumonia, spontaneous pneumothorax, pulmonary embolus, and pulmonary hypertension are typically accompanied by significant dyspnea.
- Pneumonia usually causes cough and fever.
- Pulmonary hypertension can cause edema and jugular venous distension.
- Spontaneous pneumothorax causes decreased breath sounds and respiratory distress. (Lee, 2008)

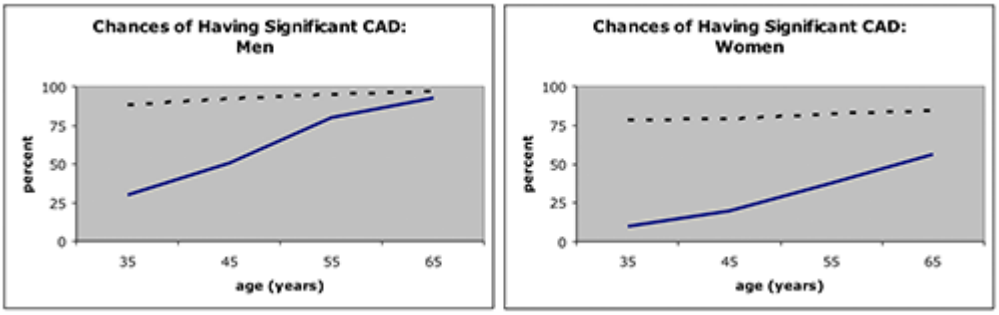
Medical History

Although the pace and the focus of a workup differ for acute and nonacute angina, much of the collected cardiovascular information is the same. In all cases, the history, physical examination, lab work, imaging, and stress tests will look for evidence of coronary atherosclerosis and of heart ischemia.

Age of Patient

The typical CAD patient is middle-aged or older. Men tend to have their first symptoms when they are older than 50 years. Women tend to have their first symptoms when they are older than 60 years. Symptomatic CAD is much less common in premenopausal women, unless they are smokers or have metabolic syndrome.

Differences in CAD Between Men and Women



Source: Douglas et al., 2008.

The solid and dashed lines in both of the above graphs represent the percent of people with stable angina who actually have significant CAD. The solid line is for people who have angina but who do not have diabetes, hyperlipidemia, or a history of smoking. The dashed line is for people who have angina and diabetes, hyperlipidemia, and a history of smoking, showing the strong correlation between those conditions and CAD.

Family Medical History

Coronary artery disease is most likely to be found in people with first-degree relatives who have had ischemic heart problems.

Patient's Lifestyle

Atherosclerosis is associated with smoking, a sedentary lifestyle, and diets that are high in fats and calories.

Physical Findings

Atherosclerosis is a whole-body disease because it affects the circulatory system throughout the body. People with atherosclerosis of the coronary arteries usually have similar problems in other arteries, so they may have such complaints as intermittent claudication, foot pain, cold feet, transient ischemic attacks (TIAs), or stroke. Additionally, they may have other health problems that foster atherosclerosis, such as hypertension, dyslipidemia, diabetes, or obesity.

A patient with asymptomatic CAD or with stable angina may have an unremarkable physical examination. Sometimes, however, the patient will have signs of atherosclerosis, such as carotid, femoral, or renal artery bruits, diminished pulses in the legs, ankles, or feet, or visible venous occlusions in the retina. A patient with CAD may also have hypertension and funduscopic signs of hypertensive retinopathy (Antman et al., 2008).

The heart examination, too, may be normal. On the other hand, CAD can produce unrecognized (silent) myocardial infarctions, and the heart examination of a patient with CAD will sometimes uncover signs of previous ischemic damage. For example, there can be murmurs (from weakened papillary muscles), widened split sounds (from damage to the heart's conduction system), or a diminished first sound (from weakened ventricular contractions).

Long-standing CAD can cause heart failure. A patient with heart failure may have an enlarged heart, lower-extremity edema (swollen legs, ankles, or feet), and distension of the jugular veins. Heart failure can also lead to cyanosis, pulmonary edema, heart murmurs, or heart gallops.

Laboratory Tests: Cardiac Biomarkers

There are no laboratory tests specifically for CAD. The laboratory workup for CAD, instead, is an assessment of risk factors, using blood tests for diabetes, kidney disease, dyslipidemia, and chronic inflammation (C-reactive protein levels and homocysteine) (Antman et al., 2008).

When heart muscle cells die, intracellular proteins leak into the circulation, and some of these proteins can be identified using blood tests. In investigating a potential myocardial infarction, blood is tested for these intracellular heart molecules—**cardiac biomarkers**—which would be released only by dying heart muscle cells. The appearance of cardiac biomarkers in the bloodstream is a reliable sign of heart muscle death (myocardial necrosis). The tests are repeated 6 to 9 hours later, because the damage caused by heart ischemia can continue for a number of hours. After an MI, blood levels of cardiac biomarkers can remain high for 1 to 2 weeks.

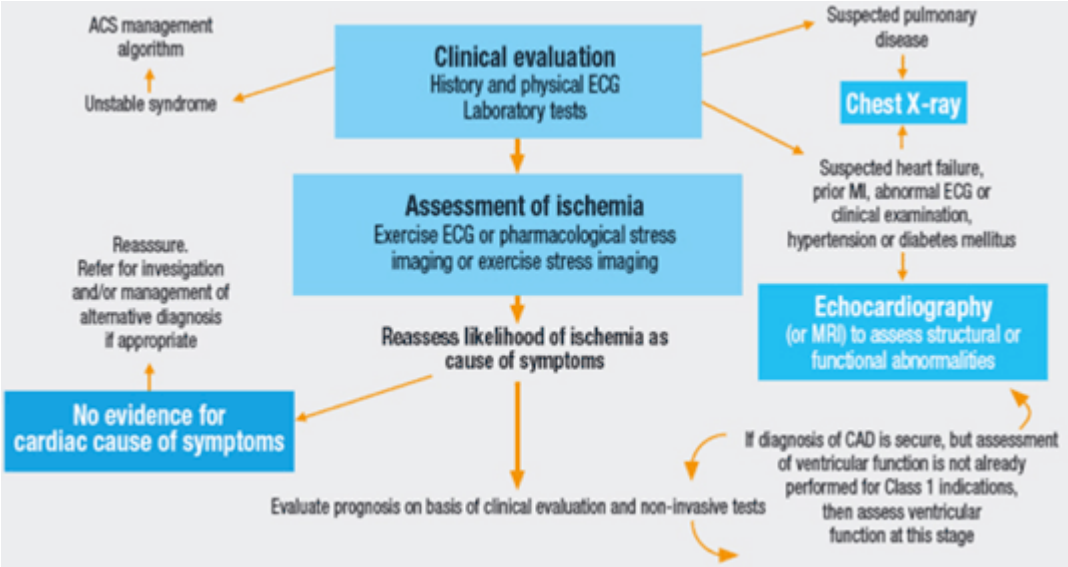
Currently, **cardiac troponin T** and **cardiac troponin I** are considered more sensitive indicators than other commonly used biomarkers such as creatinine kinase-MB (CK-MB). Normally, blood levels of cardiac troponins are practically undetectable; therefore, detectable blood levels indicate heart muscle damage. Higher levels of cardiac troponins indicate damage that is more extensive.

While an elevated level of cardiac biomarkers is a trustworthy measure of heart muscle injury, detectable biomarkers do not reveal the cause of the injury. Heart muscle damage is often caused by ischemia, but it can also be caused by infection, inflammation, heart failure, or metabolic disorders. Elevated cardiac biomarkers will accompany trauma (including surgery), myocarditis, aortic dissection, pulmonary embolism, congestive heart failure, arrhythmias, renal failure, poisoning, and even extreme exertion.

In diagnosing myocardial infarctions, elevated cardiac biomarkers are best used as confirmation of heart muscle damage when there are other indications that heart ischemia may have occurred.

Imaging Studies

A full range of imaging techniques is employed in diagnosis of cardiac disease.



Algorithm for the initial evaluation of patients with clinical symptoms of angina. Source: The CLARIFY Registry: Management of Stable Coronary Artery Disease in Clinical Practice.

Chest X-Ray

In diagnosing CAD, a chest x-ray, which shows heart size, is a priority when considering the possibility of accompanying heart failure.

Echocardiography

Echocardiography, or "echo," is a painless test that uses sound waves to create pictures of the heart in motion. The pictures show the size and shape of the heart and how well the heart's chambers and valves are working. Echo also can pinpoint areas of heart muscle that aren't contracting well because of poor blood flow or injury from a previous heart attack.

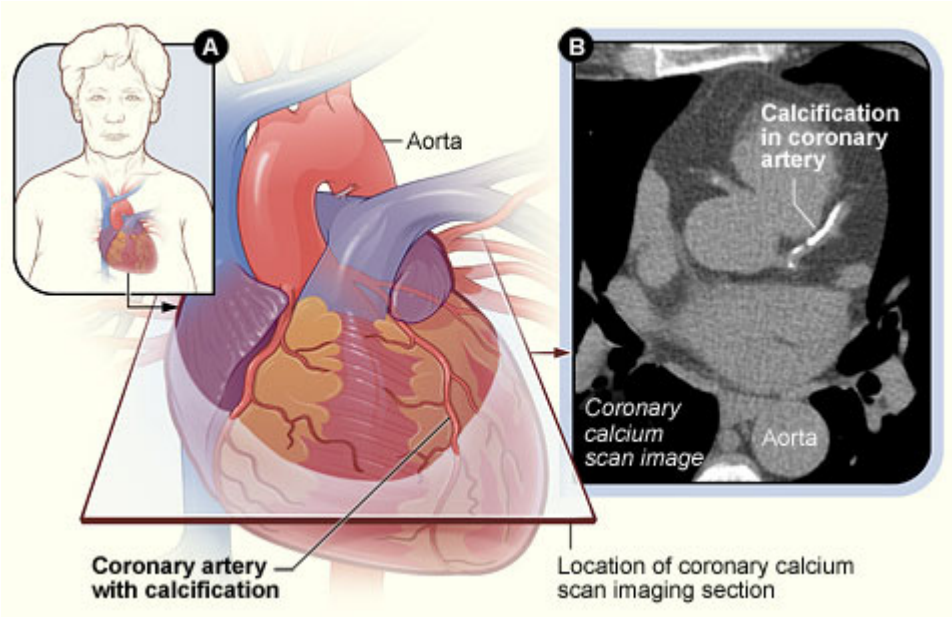
A type of echo called **Doppler ultrasound** shows how well blood flows through the heart's chambers and valves. Echo can detect blood clots inside the heart, fluid buildup in the pericardium (the sac around the heart), and problems with the aorta (NHLBI, 2016d).

Computed Tomography (CT)

Cardiac **computed tomography (CT)** uses x-rays to produce a series of images of each part of the heart, then assembles them to make a three-dimensional picture. Sometimes an iodine-based dye is injected into an IV during the scan to further highlight the coronary arteries. This is called **coronary CT angiography**.

Coronary calcium scans are used to scan the coronary arteries for calcium deposits, which are usually a sign of longstanding atherosclerotic plaque (Lloyd-Jones et al., 2009). Two imaging techniques can show calcium in the coronary arteries: electron-beam computed tomography (EBCT) and multi-detector computed tomography (MDCT) (NHLBI, 2012b).

Coronary Calcium Scan Image



(A) The position of the heart in the body and the location and angle of the coronary calcium scan image; (B) scan image showing calcification in a coronary artery. (In the CT scan, the patient's back is at the bottom of the image, and the patient's sternum is at the top.) Source: NHLBI, 2016e.

Multi-slice CT is a high-resolution technique that provides good visualization of the structure of the coronary arteries. In many cases, multi-slice CT can allow an accurate diagnosis of CAD, although this is at the expense of subjecting patients to higher than usual doses of radiation (Lee, 2008).

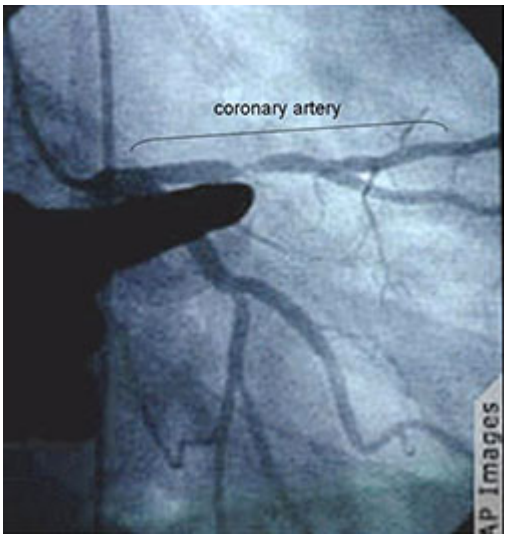
Magnetic Resonance Imaging (MRI)

Cardiac **magnetic resonance imaging (MRI)** gives high-resolution images of the coronary arteries without subjecting patients to radiation. When contrast agents are used, MRI images can also show the relative perfusion of various regions of the heart (Antman et al., 2008). Cardiac MRI creates both still and moving pictures of the heart and major blood vessels.

Cardiac Catheterization Angiography

Cardiac catheterization angiography (cardiac arteriography) reveals the outlines of the flow space inside the coronary arteries. During this procedure, a catheter is inserted into an artery in the groin or arm and dye is injected into a coronary artery to search for stenotic segments. Guided by x-rays, the catheter can be moved up into the heart; there, in ideal conditions, narrowed (stenotic) arterial segments or blockages can be seen clearly. Angiography cannot detect early atherosclerotic plaque, which builds up inside the arterial wall but does not yet protrude into the arterial lumen. Cardiac angiography is also used to assess the performance of the cardiac valves and the left ventricle.

Angiograph of a Stenotic Segment in a Coronary Artery



The finger is pointing to the narrowed region of a dye-filled coronary artery. Source: Gebel, 2008.

Cardiac catheterization is an invasive procedure and it brings risks, so it is not done on all patients with diagnosed or suspected CAD. Individuals who qualify for coronary angiography include patients who have:

- STEMI or non-ST elevation MI (NSTEMI)
- Chronic stable angina and who are being considered for reperfusion surgery, a percutaneous coronary intervention (PCI), or a coronary artery bypass graft (CABG)
- Anginal equivalent
- Patients with a moderate to large area of intact heart and no signs or symptoms or mild symptoms of ischemia
- Patients with moderate to severe ischemia (Antman et al., 2008; Stouffer, 2012)

Test Your Learning

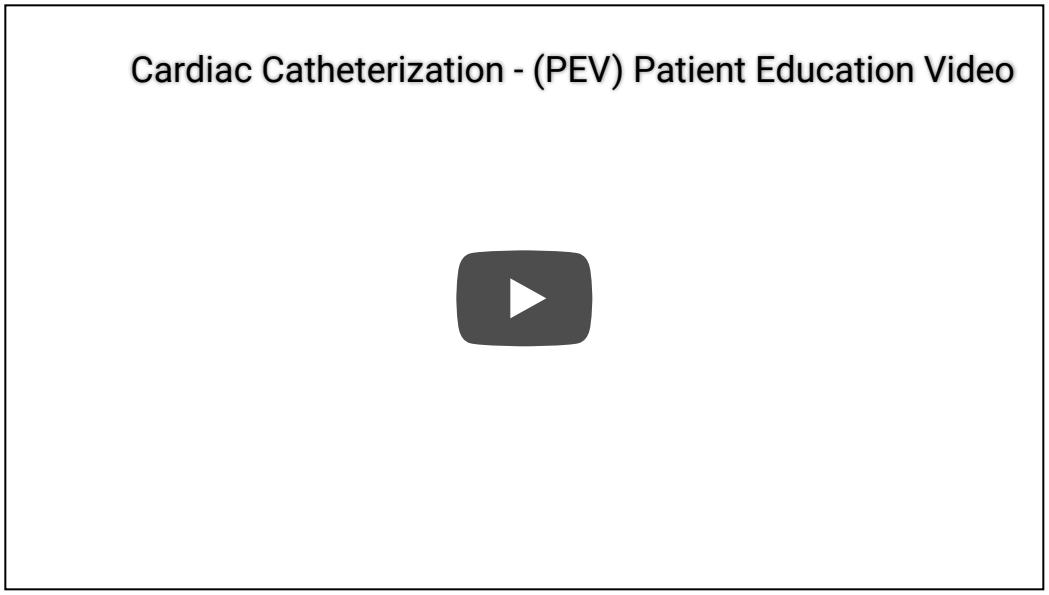
When examining patients with suspected CAD, chest x-rays:

A. Can give a definitive diagnosis.
B. Are not useful.
C. Will highlight the ischemic regions of the heart.
D. Are used to recognize possible co-existing heart failure.

Cardiac catheter angiography:

A. Uses dye injected into a coronary artery to search for stenotic segments.
B. Is the definitive diagnostic technique for an MI.
C. Has risks and is only used in preparation for coronary artery bypass graft surgery.
D. Has risks and cannot be used if a person has ever had an MI.

Online Resource (7:20)



https://www.youtube.com/watch?v=cUIbLRO2pnU

Answer: C, A

Electrocardiography

All patients with possible heart problems should have a 12-lead electrocardiogram (ECG). In a patient with stable angina, the heart muscles may not be ischemic at rest and a resting ECG can be normal. However, if areas of heart muscle are ischemic, or if past episodes of ischemia have left damaged muscle, there will be characteristic signs on the ECG.

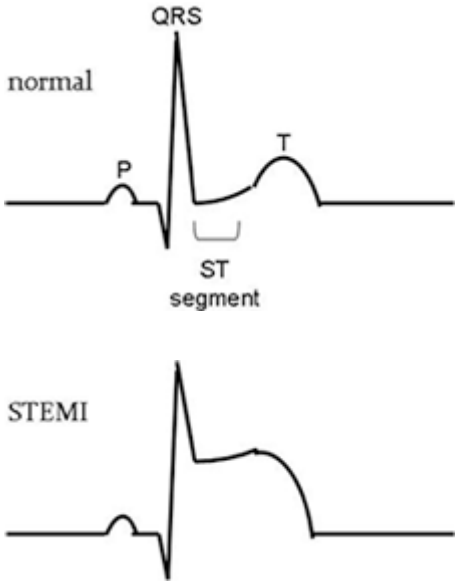
On ECG recordings, ischemia is typically diagnosed from changes in the shape and position of the ST segment of the waveform:

- The vector of the most significant ST changes indicates the region of heart muscle that is suffering ischemia.
- The elevation or depression of the ST segment indicates the portion of the heart wall (subendocardium, epicardium, or the entire thickness, ie, transmural) that is suffering ischemia.

A myocardial infarction with elevated ST segments (a STEMI) suggests epicardial or transmural damage. This type of damage is the most likely to be rescued or reduced by immediate reperfusion therapy (Goldberger, 2008). The most common location of cardiac tissue damage is in the anterior lower leads. The formal definition of a STEMI is at least 2 contiguous leads of >2 mm elevation. Regardless of the type of STEMI, all will be treated the same—by quick coronary revascularization.

During an acute coronary syndrome, the patient's ECG can change repeatedly; therefore, physicians will take successive ECG recordings to monitor evolving events during the first 48 hours.

ECG Waveforms



Top: Normal ECG waveform. Bottom: ECG waveform in which the ST segment is raised significantly, indicating an ST-segment elevation myocardial infarction (ie, a STEMI).

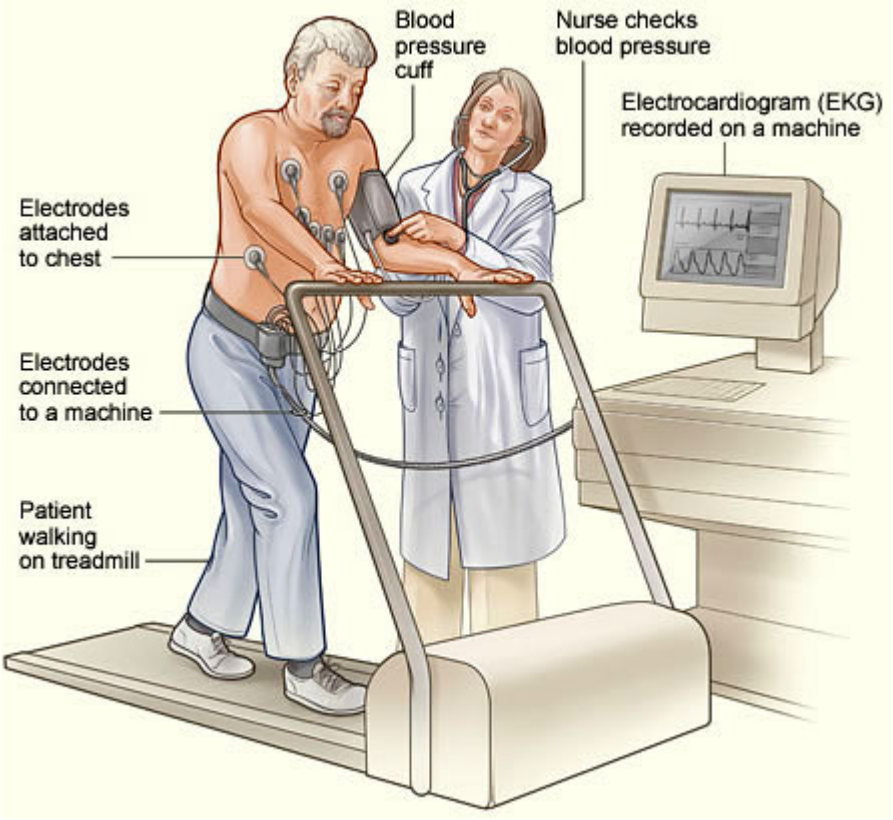
Stress Testing

Stress tests increase the oxygen demands of the heart in a controlled setting to search for evidence of activity-dependent ischemia.

ECGs and Echocardiograms with Increased Activity

Sometimes patients who report episodes of chest discomfort during exercise have no evidence of heart ischemia on ECGs taken in the office while the patient is resting. To check whether the patient's exercise-dependent chest discomfort is angina, ECG recordings can be taken while the patient exercises (usually on a treadmill) in a medically monitored environment.

Patient Undergoing Stress Test



An exercise stress test with the patient closely monitored. Source: NHLBI, 2016f.

Echocardiograms, which show heart movements and ventricular wall thicknesses, can also be used in stress tests. Stress echocardiography is a more sensitive test for heart ischemia than is stress ECG testing.

If a patient cannot exercise sufficiently to complete the test, the heart can instead be stressed pharmacologically using drugs such as adenosine, dipyridamole, or dobutamine.

Stress testing should not be done during a potential acute coronary syndrome.

Test Your Learning

To document episodes of heart ischemia, stress tests have:

A. Actors berate the patient.
B. AThe patient exercise, generally on a treadmill.
C. The patient attempt to do two tasks at once.
D. The patient eats a large meal followed by coffee and dessert.

Answer: B

Confirming Diagnoses/Identifying Patients at Risk

Stress tests are most helpful when they confirm an uncertain diagnosis of CAD. Negative stress tests are not as helpful, because about 1 out of 4 patients with CAD will show no diagnostic changes during a stress test.

Certain findings in stress tests can identify patients who have a high risk of developing an acute coronary syndrome. Worrisome findings include signs or symptoms of heart ischemia:

- At low levels of exercise
- But no increase in blood pressure with continuing increases in exercise
- That persist >5 minutes after the end of the exercise (Douglas et al., 2008)

Prognosis

From a patient's medical profile and test results, clinicians can estimate the patient's risk of suffering an acute coronary syndrome. Tables are available for quantifying this risk, but the first step is to distinguish low and high-risk patients.

Low-Risk Patients

In patients with CAD, the chances of developing an acute coronary syndrome are lowest when:

- A stress test finds no problems, and
- The left ventricle appears to be functioning normally, and
- The coronary arteries look normal in angiograms

High-Risk Patients

Patients with CAD have higher chances of developing an acute coronary syndrome in any of the following situations:

- The patient has significant risk factors for atherosclerotic CAD, including:
  - Age >75 years
  - Diabetes
  - Extreme obesity
  - Peripheral or cerebrovascular artery disease
  - Dyslipidemia
  - History of smoking

    - Hypertension
    - Elevated C-reactive protein
- The patient has a history of heart problems, including:
    - Episodes of unstable angina
    - Past myocardial infarctions
    - Heart failure or heart failure signs, such as pulmonary edema, a heart gallop, an enlarged heart, mitral regurgitation, or a reduced ejection fraction (<40%)
- The patient has worrisome stress test results, including:
    - Inability to exercise >6 minutes
    - Ischemic signs or symptoms at low stress levels
    - Severe ischemia during testing
- The patient has worrisome cardiac catheterization results, including:
    - Increased left ventricular end-diastolic pressure or volume
    - Reduced ejection fraction
    - Stenosis >50% in the left main or the left anterior descending coronary artery
- The patient has extensive coronary artery calcification (Antman et al., 2008)

Management of CAD

The foundation for treating all forms of CAD is the basic treatment plan for stable angina. This plan begins with therapeutic lifestyle changes, adds medications when necessary, and considers using reperfusion therapy for more serious levels of disease.

Initial Management of Acute Angina

Patients with acute chest discomfort that does not respond to rest or to nitroglycerin or that lasts more than 20 minutes should immediately be taken to an emergency department, preferably by ambulance (Antman et al., 2007).

In choosing among these treatment paths, clinicians rely on the history, physical examination, ECG, and blood tests for cardiac biomarkers. If the discomfort appears to be acute angina, physicians will stabilize the patient and send them along 1 of 4 treatment paths. These paths lead to:

- Immediate treatment: those with ST elevation on initial ECG (percutaneous coronary intervention or IV thrombolytic drugs)

- Admission to a coronary intensive care unit, a telemetry unit, or an observation unit: those without ST elevation but at high risk
- Collection of further data for those who have symptoms that warrant evaluation (stress testing or radionuclide imaging) in the ED
- Discharge home with followup by primary care physician for those who have an obvious non-cardiac cause for their symptoms (Kontos et al., 2010)

Patients most likely to benefit from immediate treatment are those with a major acute myocardial infarction, which can usually be identified by biochemical evidence of heart muscle necrosis (increased blood levels of cardiac biomarkers) and elevations of the ST segments of the ECG waveform.

Patients who should be admitted to coronary units are those most likely to continue developing acute problems over the next 72 hours. These are patients with any of these conditions:

- Recently changing or accelerating angina
- History of myocardial infarction
- Evidence of heart failure
- ECG signs of ischemic changes
- Elevated cardiac biomarkers
- Over 70 years of age

In addition, patients are admitted if there are serious contributing conditions, such as uncontrolled hypertension, hypotension, new cardiodynamic problems, mitral regurgitation, or lung disease (Lee, 2008).

Patients being worked up for acute coronary syndrome (ACS) must first be stabilized. Antithrombotic therapy should then be started and ischemic pain eliminated. Aspirin at 162 to 325 mg should be given (300–600 mg loading dose of clopidogrel for those allergic to aspirin), and chest pain should be treated with sublingual nitroglycerin. For continued chest pain, morphine or fentanyl can be used if blood pressure tolerates nitrates. Intravenous nitroglycerin can be used when other medications have failed to alleviate chest pain (Coven, 2013).

Medical personnel have traditionally been taught the mnemonic MONA (morphine, oxygen, nitroglycerin, aspirin) to remind them of the initial treatment of any suspected ACS patient. However, according to current American Heart Association guidelines, “there is insufficient evidence to support the routine use of oxygen in uncomplicated ACS.” If the patient is short of breath, hypoxemic, or has obvious signs of heart failure, oxygen should be titrated to saturation levels ≥94% (AHA, 2015).

The concern is oxygen toxicity and vasoconstriction. Oxygen causes constriction of the coronary, cerebral, renal, and other key vasculatures. If perfusion decreases with blood hyperoxygenation, the administration of oxygen may place tissues at increased risk of hypoxia. Hyperoxia reduces coronary blood flow by 8% to 29% in normal individuals and in patients with coronary artery disease or chronic heart failure. The reduction in coronary artery flow is associated with a reduction in myocardial-tissue oxygen delivery and oxygen consumption (Iscoe et al., 2011).

Test Your Learning

When people call to ask advice about the sudden occurrence of chest discomfort:

A. Advise them to be taken immediately to the hospital, preferably by ambulance.
B. Check to see whether they suffer from panic attacks, gastroesophageal reflux disease (GERD), or peptic ulcer disease (PUD) before advising them.
C. Tell them to call their physician immediately.
D. Do a quick telephone triage of symptoms to determine the likelihood of an MI.

For patients who are having a suspected myocardial infarction, chewing 2 to 4 tablets of 81 mg aspirin is:

A. Not recommended if the patient is a candidate for IV fibrinolytic therapy.
B. Not recommended if the patient is already taking daily aspirin.
C. No longer recommended for most patients.
D. Recommended for all patients unless the patient is allergic to aspirin.

The use of supplemental oxygen:

A. Is never recommended for patients with suspected ACS.
B. Is only used during cardiac arrest.
C. Is recommended only when a patient is short of breath, hypoxemic, or has signs of heart failure.
D. Should be titrated to keep oxygen saturation at 100%.

Patients with stable angina who get chest discomfort with exercise:

A. Must resign themselves to living with lower levels of exercise or risk having an MI.
B. Can safely ignore chest discomfort because their disease is stable.
C. Can increase their exercise tolerance with a medically supervised cardiac rehabilitation program.
D. Should be put on bed rest to avoid sudden cardiac death.

Answers: A, D, C, C

Therapeutic Lifestyle Changes

With commitment and perseverance, a person can significantly reduce the threats posed by CAD.

Following a peak incidence around 1968, death from CAD declined significantly in the United States. It is estimated that 47% of the decrease in mortality from coronary heart disease in the United States between 1980 and 2000 was attributed to advances in medical therapies, including treatment of acute coronary syndromes and heart failure. Approximately 44% of the reduction was secondary to a decline in cardiovascular risk factors, including hypercholesterolemia, hypertension, smoking, and physical inactivity (Chiha et al., 2012).

Unfortunately, this reduction in the death rate from CAD was partly offset by increases in diabetes and body mass index (BMI). Unfortunately, the cardiovascular disease epidemic continues to evolve rapidly on a global level and is currently responsible for twice as many deaths in developing countries as in developed countries. In low- and middle-income countries, cardiovascular risk factors, especially smoking and obesity, continue to increase in incidence and affect a larger proportion of younger patients. Cardiovascular mortality has been reported 1.5 to 2 times higher among the working population in India, South Africa, and Brazil compared to the United States (Chiha et al., 2012).

Therapeutic lifestyle changes have been scientifically demonstrated to be good treatments for heart disease—namely, stop smoking, eat a diet low in saturated fat and higher in fruits and vegetables, exercise daily, and lose weight. It is the job of the healthcare team to work with the patient to personalize these familiar recommendations. Healthcare providers must offer practical advice that the patient can reasonably follow and that the patient believes is worth following.

In as many as three-quarters of all CAD patients, supervised lifestyle change programs can reduce the amount and severity of angina within three months. During that time, patients can increase their exercise capacity and improve their quality of life. Comprehensive lifestyle change programs can also reduce the chance that patients will require a coronary reperfusion procedure (Frattaroli et al., 2008).

Therapeutic lifestyle changes can prevent coronary artery disease and reduce its severity. Young people, who are increasingly at risk for developing CAD, should be encouraged to follow the same principles of no smoking, low-fat/high-fiber meals, and plenty of physical activity (Libby, 2008).

Smoking Cessation

Therapeutic lifestyle changes begin with smoking cessation. Carbon monoxide and other poisons in cigarette smoke damage many types of cells in the body. Carbon monoxide also reduces blood oxygenation, stressing the oxygen-hungry heart. This stress is compounded by the nicotine in cigarette smoke. Nicotine constricts blood vessels and causes the heart to work harder, raising heart rate and blood pressure, two effects that increase the heart's workload (Mitchell & Schoen, 2009).

Cigarette smoking accelerates coronary atherosclerosis in both sexes and at all ages and increases the risk of thrombosis, plaque instability, MI, and death. In addition, by increasing myocardial oxygen needs and reducing oxygen supply, it aggravates angina (Antman et al., 2008).

People who stop smoking reduce their risk of death from CAD; however, many people find it difficult to stop smoking. Clinicians can begin by telling patients that continued smoking increases their risk of serious heart problems and death, while quitting reduces this risk. They should then ask patients who smoke if they have thought about quitting. Whatever the answer, clinicians should follow with the offer "When you would like to stop smoking, I'll be happy to help set up an effective program for you." For more information, see Cardiac Rehabilitation below.

Low-Fat/High-Fiber Diet

The American Dietetic Association has collected evidence demonstrating that a low-fat diet with 12 to 33 g per day of fiber from whole foods or up to 42.5 g per day from supplements can help to reduce blood pressure, correct dyslipidemia, reduce indicators of chronic inflammation, and reduce weight (Am. Diet. Assoc., 2008). A low-fat/high-fiber diet has also been shown to reduce the risk of developing CAD.

In one large study of older adults, eating whole-grain fiber in the equivalent of an extra 2 slices of whole-grain bread per day reduced the number of:

- Deaths from CAD by 13%
- Nonfatal myocardial infarctions by 6%
- Ischemic strokes by 24%

Compared with medical or surgical interventions, nutritional changes are relatively low-risk, low-cost, and widely available. Therefore, the practical importance of even a small change in risk may be significant on a population or public health level (Mozaffarian et al., 2003).

Recommended Therapeutic Lifestyle Changes

- Less than 7 percent of daily calories should come from saturated fat. This kind of fat is found in some meats, dairy products, chocolate, baked goods, and deep-fried and processed foods.
- No more than 25 to 35 percent of daily calories should come from all fats, including saturated, trans, monounsaturated, and polyunsaturated fats.
- Cholesterol intake should be less than 200 mg a day.
- Foods high in soluble fiber help prevent the digestive tract from absorbing cholesterol. These foods include:
  - Whole-grain cereals such as oatmeal and oat bran
  - Fruits such as apples, bananas, oranges, pears, and prunes
  - Legumes such as kidney beans, lentils, chick peas, black-eyed peas, and lima beans
- Choose a diet rich in fruits and vegetables to decrease cholesterol. These compounds, called plant stanols or sterols, work like soluble fiber.
- Fish such as salmon, tuna (canned or fresh), and mackerel are a good source of omega-3 fatty acids and should be eaten twice a week.
- Limit sodium intake. Choose low-salt and "no added salt" foods and seasonings.
- Limit alcohol intake. Too much alcohol raises blood pressure, triglyceride level and adds extra calories.
  - Men should have no more than two drinks containing alcohol a day.
  - Women should have no more than one drink containing alcohol a day.

Source: NHLBI, 2006.

Increased Physical Activity

Regular exercise helps to correct dyslipidemia. It also reduces insulin resistance, decreases platelet aggregation, aids weight loss, improves sleep, and gives people a sense of well-being. For low-risk patients who get no cardiac symptoms with exercise, the minimum amount of recommended activity is 30 minutes of moderate-intensity activity such as brisk walking, on at least three different days each week.

A recommended goal is for everyone to include 30 to 60 minutes of moderate-intensity aerobic activity in their schedule every day in addition to their daily activities such as gardening or housework (Fraker et al., 2007).

To have a significant effect, regular activity must become a continuing part of a patient's life. Patients stick to exercise goals more consistently when the activities are undertaken in a structured, supervised program—for example, when the patients attend regular classes or when they keep a record and report regularly to someone.

Exercise programs may have to be introduced gradually. At first, patients with stable angina may be limited by the occurrence of angina and will probably need to adapt even their normal activities. The appearance of angina or anginal equivalents indicates that an activity is too strenuous, so patients should revise their normal activities to avoid the angina.

In many cases, patients can reduce the heart's workload and prevent angina simply by doing stressful activities at a slower pace. Patients should be warned that their exercise tolerance should be customized to their comfort and daily activities. Angina occurs more frequently in the morning, after meals, and in cold weather.

After finding an angina-free daily routine, most patients should then aim to increase their exercise tolerance. Regular exercise, beyond their daily activities, is beneficial for almost all patients with CAD. For symptomatic and high-risk patients, a medically supervised cardiac rehabilitation program is recommended. The goal for most patients is to progress to a minimum of 30 minutes of moderate-intensity aerobic activity on at least five different days each week (Antman et al., 2007). See Cardiac Rehabilitation below for more details.

Weight Loss

During the past twenty years there has been a dramatic increase in obesity in the United States, and rates remain high. Thirty-five percent of U.S. adults and approximately seventeen percent (12.5 million) of children and adolescents aged 2 to 19 years are obese. Obesity-related conditions include heart disease, stroke, type 2 diabetes, and certain types of cancer—some of the leading causes of preventable death (CDC, 2012c).

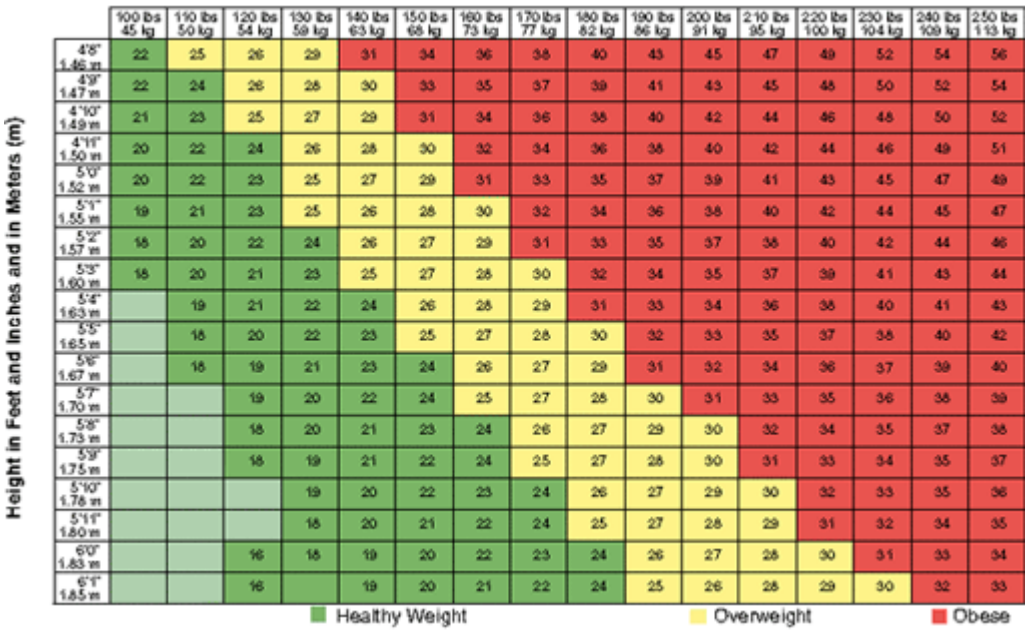
Excess body weight makes the heart work harder, and excess fat fosters atherosclerosis. A person is considered overweight if their body mass index (BMI, see chart below) is 25 to 29.9 kg/m^2, while obesity is defined to be BMI >30 kg/m^2.

BMI Chart

Source: Courtesy of webmd.com.

Calculating BMI is one of the best methods for population assessment of overweight and obesity. Because calculation requires only height and weight, it is inexpensive and easy to use for clinicians and for the general public. The use of BMI allows people to compare their own weight status to that of the general population. The standard weight status categories associated with BMI ranges for adults are shown in the following table (CDC, 2011).

Body Mass Index (BMI): Weight in Pounds (lbs) and Kilograms (kg)



BMI values for selected heights between 4'10" and 6'3" and for selected weights between 100 lbs and 248 lbs. BMI values are kilograms of body weight per square meter of body surface area (kg/m^2) (NHLBI, n.d.).

Excess body weight is a CAD risk factor. A person need not be obese to suffer a higher risk of CAD: overweight people with a BMI >26.5 have an increased likelihood of developing atherosclerosis, and overweight people with a BMI >27.5 have an increased rate of death from CAD (Lewis et al., 2009).

When a person's excess fat is visceral (ie, inside the abdomen as opposed to directly under the skin), the effect on atherosclerosis is worse. A quick and effective measure of intra-abdominal fat is a person's waist circumference. Waist circumferences >102 cm (40 in) in men and >89 cm (35 in) in women represent increased coronary artery risks.

Weight management is a key part of the therapeutic lifestyle changes recommended for people with CAD. Patients should be encouraged to maintain a BMI <25 kg/m^2. Men should aim for a waist circumference <102 cm (40 in), while women should aim for a waist circumference <89 cm (35 in) (Antman et al., 2007; Fraker et al., 2007). To measure waist size correctly, one should stand and place a tape measure around the middle, just above the hipbones and measure the waist just after breathing out.

Both low-carbohydrate diets (<130 g carbohydrates/day) and low-fat diets seem to be equally effective. The most effective way to lose weight and to maintain the lower weight is participation in a comprehensive weight-loss program that combines low-calorie diets, behavior modification, and regular exercise. Healthy weight loss must include a lifestyle of long-term changes in daily eating and exercise habits.

Evidence shows that people who lose weight gradually and steadily (about 1 to 2 pounds per week) are more successful at keeping weight off. In order to lose weight, a person must reduce the daily caloric intake and use up more calories than are taken in. Since 1 pound equals 3,500 calories, caloric intake must be reduced by 500 to 1000 calories per day to lose about 1 to 2 pounds per week (CDC, 2016b).

Even a modest weight loss, say, 5% to 10% of total body weight, is likely to produce health benefits, such as improvements in blood pressure, blood cholesterol, and blood sugars. Long-term success is achieved through healthy eating, and physical activity most days of the week (about 60–90 minutes, moderate intensity) (CDC, 2016b).

Stress Management

It is generally believed that psychological stress worsens CAD. In one small study, an intensive stress management program was shown to actually reduce the amount of stenosis in coronary arteries (Pischke et al., 2008). Anger, frustration, fear, anxiety, depression, and insomnia are thought to foster an internal biochemistry conducive to atherosclerosis and perhaps to acute coronary syndromes. Physicians therefore advise CAD patients to lessen as possible the stressors in their lives and to learn and use simple relaxation techniques (Davis, 2008). A CAD patient struggling with any form of psychological stress should be offered a referral to a mental health professional.

Medications

Medications are used to manage all forms of CAD:

- **Stable angina**. Drugs that dilate arteries, slow heart rate, and lower blood pressure can temper the cardiovascular effects of stressful activities. Drugs in this category include nitrates, beta-blockers, and calcium channel blockers.
- **Acute coronary syndromes**. Antiplatelet drugs can be taken daily to reduce the size and prevalence of thrombotic clots. Drugs in this category include aspirin and clopidogrel.

Nitrates

Nitrates are drugs that release nitric oxide (NO) when metabolized. Nitric oxide relaxes arterial walls, causing the blood vessels to dilate, and the dilation of coronary arteries will ease ischemic heart pain.

Sublingual nitroglycerin tablets are the most effective of the nitrates. These tablets will relieve angina within 5 minutes. Nitroglycerin tablets can also be taken 5 minutes before exercise or other stresses to prevent angina. The most common side effects of nitroglycerin are headache, dizziness, and a pulsing feeling in the head. Longer-acting nitrates can also be effective; however, responses vary, and the appropriate drug and dosage must be discovered empirically for each patient.

Patients with stable angina often get relief from an episode of angina by resting. If a few minutes of rest are not sufficient, sublingual nitroglycerin tablets will usually relieve the angina. As many as three tablets can be taken at 3- to 5-minute intervals.

When rest and nitroglycerin do not work within a few minutes, the individual may be having an acute coronary syndrome. In these circumstances, the person should be taken immediately by ambulance to an emergency department (Antman et al., 2008).

Patients should keep a diary, recording episodes of angina and the responses to rest and to nitroglycerin. This diary can help physicians to recognize when stable angina is becoming unstable.

Nitroglycerin tablets deteriorate. They last for 3 to 5 months when tightly capped and stored in a refrigerator. When kept in a pill container and carried by a patient, the tablets deteriorate more rapidly.

Nitrates Prescribed for Angina

Short-term (lasting 2 to 10 minutes)

- nitroglycerin (sublingual tablets, sublingual spray)
- isosorbide dinitrate (sublingual spray)

Intermediate-term (lasting ~1 hour)

- isosorbide dinitrate (sublingual tablets)

Long-term (lasting >2 hours)

- nitroglycerin (ointment, transdermal patch, oral sustained release, IV)
- isosorbide dinitrate (oral pills, chewable tablets, oral slow release, IV)
- isosorbide mononitrate (oral pills)

Beta-Adrenergic Blockers

Exercise and other stressors raise both heart rate and blood pressure via the sympathetic nervous system. The sympathetic neurotransmitters are adrenergic chemicals such as epinephrine and norepinephrine. Adrenergic blocking drugs will reduce the ability of the sympathetic nervous system to stimulate end organs, such as the heart and the smooth muscles in arteries.

Sympathetic stimulation has different effects on different tissues, and it has been possible to find drugs that selectively block specific sympathetic effects. Beta-adrenergic blockers (**beta blockers**) are especially effective at limiting the cardiovascular effects of sympathetic stimulation. Beta blockers inhibit the increases in heart rate and blood pressure that are normally caused by exercise and stress, while having little effect on the heart at rest (Antman et al., 2007).

To reduce angina and to lower the risk of an MI, many CAD patients take long-acting or sustained-release beta blockers once daily. Beta blockers are also prescribed as protection after an acute coronary syndrome (Alaeddini, 2012).

Side effects can require a beta blocker to be taken at a lower dose or to be discontinued entirely. When being discontinued, beta blockers should be tapered and not stopped suddenly. The possible side effects of beta blockers include fatigue, reduced exercise tolerance, nightmares, impotence, cold hands and feet, intermittent claudication, bradycardia, impaired atrioventricular conduction, left ventricular failure, bronchial asthma, and, in diabetics, an intensification of the hypoglycemia produced by oral hypoglycemic agents or insulin. Beta blockers are usually not given to patients with asthma or other types of reversible airway obstruction, severe bradycardia, atrioventricular conduction disturbances, Raynaud's phenomenon, or a history of clinical depression (Antman et al., 2008).

Beta Blockers Prescribed for Angina

Selective beta 1 blockers

- Acebutolol
- Atenolol
- Betaxolol
- Bisoprolol
- Esmolol (an IV drug)
- Metoprolol

Nonselective beta blockers

- Carteolol
- Carvedilol
- Labetalol
- Nadolol
- Penbutolol
- Pindolol
- Propranolol
- Sotalol
- Timolol

Calcium Channel Blockers

Calcium channel blockers dilate coronary arteries, lower blood pressure, and reduce the heart's oxygen requirements. However, calcium channel blockers can produce hypotension, edema, and bradycardia and can worsen heart failure.

Overall, the effects of calcium channel blockers are similar to the effects of beta blockers, and calcium channel blockers are often prescribed when beta blockers cannot be used, such as with asthma or chronic obstructive pulmonary disease, or when the side effects of beta blockers such as depression, sexual disturbances, or fatigue pose too much of a problem.

Diltiazem and the dihydropyridine calcium channel blockers can be combined with beta blockers and nitrates, although the specific doses must be customized for each patient.

In 2006 the FDA approved the use of a metabolic modulator, **ranolazine** (Ranexa), a new anti-anginal medicine, for treatment of refractory angina. Ranolazine is a sodium current inhibitor that, unlike other anti-anginal drugs, has no effect on heart rate or blood pressure. It is given in twice-daily oral doses of 500 to 1000 mg, and is usually prescribed in combination with another anti-anginal drug such as a nitrate, a beta blocker, or amlodipine (Antman et al., 2008). Ranolazine is used to relieve or prevent chronic angina in patients with moderately severe symptoms (O'Rourke et al., 2008).

Calcium Channel Blockers Prescribed for Angina

Dihydropyridines

- Amlodipine
- Felodipine
- Isradipine
- Nifedipine, slow release

Other calcium channel blockers

- Diltiazem, slow release
- Verapamil, slow release

Ranolazine has recently been shown "to reduce angina frequency and sublingual nitroglycerin use in patients with type 2 diabetes, coronary artery disease, and chronic angina," according to results of a study presented at the American College of Cardiology's 62nd Annual Scientific Session (Hughs, 2013).

Antiplatelet Drugs

Blood clots and an activated clotting pathway are major causes of acute coronary syndromes. Aspirin and clopidogrel (Plavix), two drugs that inhibit platelet aggregation, have been shown to reduce a person's risk of having an acute coronary syndrome.

Aspirin

Aspirin is the most widely used and tested antiplatelet drug in CAD, and it is the cornerstone of antiplatelet therapy in treatment and prevention of CAD. In acute coronary syndrome and thrombotic stroke, acute use of aspirin can decrease mortality and recurrence of cardiovascular events. As secondary prevention, aspirin is believed to be effective in acute coronary syndrome, stable angina, revascularization, stroke, and atrial fibrillation (Dai & Ge, 2012).

For ACS patients, the current American Heart Association/American College of Cardiology (AHA/ACC) guidelines recommend that aspirin should be administered as soon as possible, with an initial loading dose of 162 to 325 mg and continued indefinitely with a dose of 75 to 162 mg daily. The use of aspirin (162 mg chewed, to ensure rapid therapeutic blood levels) was associated with a 23% reduction of vascular mortality rate in MI patients and close to a 50% reduction of nonfatal re-infarction or stroke, with benefits seen in both men and women. In unstable angina and NSTEMI patients, aspirin has been shown to reduce the risk of fatal or nonfatal MI by 50% to 70% during the acute phase and by 50% to 60% at 3 months to 3 years (Dai & Ge, 2012).

The highest benefit of aspirin was seen in those undergoing coronary angioplasty, with a 53% reduction in MI, stroke, or vascular deaths. In percutaneous coronary intervention (PCI), the use of aspirin significantly reduces abrupt closure after balloon angioplasty and significantly reduces stent thrombosis rates (Dai & Ge, 2012).

Long-term aspirin therapy reduces the yearly risk of serious vascular events (nonfatal myocardial infarction, nonfatal stroke, or vascular death), which corresponds to an absolute reduction of nonfatal events and to a smaller reduction in vascular death. Although the long-term use of aspirin has been associated with gastrointestinal and other bleeds, the benefits outweigh the risks. For secondary prevention, aspirin is recommended in conjunction with lifestyle changes and smoking cessation to reduce an individual's overall risk of further cardiovascular events (Dai & Ge, 2012).

Clopidogrel (Plavix)

Clopidogrel, also known as Plavix, is an antiplatelet drug used for peripheral vascular disease, ACS, recent MI, or stroke. Clopidogrel is given as an initial 300-mg loading dose followed by 75 mg daily (Mechcatie, 2012). Aspirin and clopidogrel can be combined, however caution should be taken regarding an increase risk of gastrointestinal bleeding.

Influenza Vaccination

Patients who have CAD should get a seasonal flu shot every year. Those over age 65 should also consider a pneumonia vaccine.

Treating Metabolic Syndrome

Metabolic syndrome is a cluster of metabolic risk factors defined by the International Diabetes Federation as central obesity (measured by waist circumference) plus any two of the following four risk factors (Edwardson et al., 2012):

- Elevated blood pressure (systolic 130 or above or diastolic 85 or above)
- Triglycerides above 150 mg/dl

- Reduced high-density lipoprotein (HDL)
- Elevated fasting blood sugar (greater than 100 mg/dl)

Research has shown that individuals with metabolic syndrome are at an increased risk of diabetes, cardiovascular events, and mortality from CAD. Approximately one-quarter of European, American, and Canadian adults have metabolic syndrome. The high prevalence of the syndrome and the associated health consequences demonstrate the importance of understanding the causes of metabolic syndrome in order to implement prevention strategies (Edwardson et al., 2012).

Having just one of the metabolic risk factors does not meet the criteria for metabolic syndrome. However, any of these conditions increases the risk of serious disease. If more than one of these conditions occur in combination, the risk of serious disease is even greater (Mayo Clinic, 2016). Each of these cardiometabolic disorders promotes the development of the others. For example:

- Insulin resistance can lead to dyslipidemia and hypertension.
- Hypertension increases the likelihood of developing diabetes.
- Central obesity can lead to insulin resistance.

People tend to have more than one of these disorders at a time (Buse et al., 2008). Aggressive lifestyle changes can delay or even prevent the development of serious health problems. Treatment of metabolic syndrome involves the separate treatment of each of its components.

In addition to metabolic syndrome, certain other medical conditions are especially problematic for people with CAD. These conditions include:

- Aortic valve disease
- Hypertrophic cardiomyopathy
- Hyperthyroidism
- Pulmonary disease
- Anemia

Controlling these problems can sometimes reduce or eliminate the angina of concurrent CAD (Antman et al., 2008).

Diabetes

Diabetes and CAD are a deadly combination. Diabetes accelerates coronary atherosclerosis and increases the risk of angina, myocardial infarction, and sudden coronary death. Yet, according to Libby (2008), "most patients with diabetes mellitus die of atherosclerosis and its complications."

Diabetics are at least 4 to 6 times as likely as nondiabetics to have heart disease and tend to develop heart disease at an earlier age than nondiabetics. Women who have not gone through menopause usually have less risk of heart disease than men of the same age. But women of all ages who have diabetes have an increased risk of heart disease because diabetes cancels out the protective effects of being a woman in her childbearing years (NDIC, 2012).

People with diabetes who have already had one heart attack run an even greater risk of having a second one. In addition, heart attacks in diabetics are more serious and more likely to result in death (NDIC, 2012).

It has been shown that maintaining strict control of blood glucose levels reduces the coronary artery risk posed by diabetes and taking metformin, controlling blood lipid levels (with statins), reducing hypertension, and instituting therapeutic lifestyle changes all lessen the serious complications of CAD in diabetics (Amer. Diet. Assoc., 2016; Fraker et al., 2007; Brunzell, 2008; Libby, 2008).

Dyslipidemia

Lipid disorders underlie atherosclerosis, and treating lipid abnormalities is critical to slowing the progression of CAD. The therapeutic lifestyle changes described above such as stop smoking, eat a low-fat, high-fiber diet, exercise more, and lose weight are the first steps in avoiding or reversing lipid abnormalities. When target blood lipid levels cannot be achieved via lifestyle changes, the next step is the addition of medications, namely statins (Libby, 2008). Low-fat diet means saturated fats are <7% of daily calories, minimal trans fats, and dietary cholesterol <200 mg/day.

Target Blood Lipid Levels for Patients with CAD

- LDL cholesterol <100 mg/dl or <70 for those with metabolic syndrome
- HDL cholesterol >40 mg/dl for men, >50 mg/dl for women
- Triglycerides <150 mg/dl

Source: NDIC, 2016.

To manage CAD, the primary lipid goal is a reduction of LDL cholesterol levels. In the general adult population, the target LDL level is <100 mg/dl. However, for people with CAD, the target LDL level is <70 mg/dl. The recommended type of medication for lowering LDL cholesterol is a statin. Statins can reduce LDL cholesterol by 25% to 50%; they can also raise HDL cholesterol by 5% to 9% and lower triglycerides by 5% to 30% (Brunzell et al., 2008).

Hypertension

High blood pressure (hypertension) worsens atherosclerosis and increases the risks of acute coronary syndrome. High blood pressure was a primary or contributing cause of death for 348,000 Americans in 2008, or nearly 1,000 deaths per day. Of the 68 million American adults who have high blood pressure, 36 million do not have it under control (CDC, 2016c).

About 30% of American adults have prehypertension—blood pressure measurements that are higher than normal, but not yet in the high blood pressure range. Better hypertension management leads to improved health outcomes. A large systematic review of 147 trial reports on the management of hypertension has shown that a reduction of 10 mm Hg in systolic blood pressure and 5 mm Hg in diastolic was associated with a 20% reduction of coronary heart disease and 32% reduction in stroke in one year (Al-Ansary et al., 2013).

Normal, At-Risk, and High Blood Pressure Levels

Normal

- Systolic: less than 120 mmHg
- Diastolic: less than 80 mmHg

At Risk (Prehypertension)

- Systolic: 120–139 mmHg
- Diastolic: 80–89 mmHg

High

- Systolic: 140 mmHg or higher
- Diastolic: 90 mmHg or higher

Source: CDC, 2016c.

As with all health problems related to CAD, treatment of hypertension begins with therapeutic lifestyle changes. If lifestyle changes are not sufficient, medications should be added (Rosendorff et al., 2007).

Drugs prescribed for hypertension in patients with CAD include:

- **Beta blockers**. Beta blockers are the first-line antihypertensive drugs for patients with CAD. If necessary, certain calcium channel blockers can be added to or substituted for beta blockers.
- **ACE inhibitors**. Angiotensin-converting enzyme (ACE) inhibitors are antihypertensive drugs and are part of the standard drug therapy for CAD patients who have diabetes or left ventricular dysfunction, even when the patients do not have hypertension. Angiotensin receptor blockers can often be substituted for ACE inhibitors.
- **Thiazide diuretics**. Thiazide diuretics can be effectively added to other antihypertensive medicines when needed but caution must be taken to maintain serum potassium levels within the normal range.

Reperfusion Therapies

After therapeutic lifestyle interventions are in place, drugs are added to the treatment regimen to control both angina and hypertension. When these medicines still do not reduce the anginal episodes, the next level of treatment is reperfusion therapy (also called *revascularization* or *recanalization therapy*).

There are two types of reperfusion procedures: percutaneous interventions to open blocked coronary arteries via a catheter, and surgical artery or vein grafts to bypass obstructed segments of coronary arteries.

In some situations, physicians recommend reperfusion therapy even before learning how well the prescribed drugs will succeed in controlling a patient's angina. For example, in addition to lifestyle changes and medications, reperfusion is often recommended for patients with any of these conditions:

- >50% stenosis of the left main coronary artery
- Significant stenosis of three major coronary artery branches
- Stenosis of the left anterior descending coronary artery and one additional major coronary artery branch

Percutaneous Interventions

Percutaneous interventions (PCIs) are accomplished by threading a balloon-tipped catheter into the stenotic segment of an artery. The balloon is inflated until it flattens the offending plaque against the arterial wall and reopens the arterial lumen. In the larger branches of the coronary arteries, the cleared lumen is held open by a metal stent that is left in place permanently.

Obstructed arteries that have been dilated by percutaneous intervention are susceptible to restenosis, so patients are given antiplatelet therapy after the procedure. Currently, by using stents coated with antithrombotic chemicals (drug-eluting stents), the restenosis rates have been reduced to about 10% over six months. When the reopened arteries eventually become blocked again, percutaneous intervention is repeated.

Coronary Artery Bypass Graft Surgery

Coronary artery bypass graft surgery (CABG) uses a grafted blood vessel to deliver blood around the obstructed segment of a coronary artery. Often, the new blood conduit is a length of saphenous vein that has been removed from the patient's leg. Arteries make better conduits than veins, and an arterial graft can be made by detaching the distal end of the internal thoracic artery (also called the *internal mammary artery*) and re-attaching it to the stenotic coronary artery beyond the obstructed region; this reroutes blood from the subclavian artery into the coronary circulation.

Bypass grafts are preferred over percutaneous interventions for the simultaneous revascularization of three or more obstructed coronary arteries. Bypass grafts are also preferred when other heart repairs, such as a valve replacement, are needed by the patient.

Test Your Learning

Coronary artery bypass grafts (CABG):

A. Are always the therapy of choice in a hospital with capacity to perform them.
B. Typically use nonhuman materials, such as Teflon tubes or porcine arteries.
C. Are the therapy of choice when multiple vessels are blocked and additional heart repairs need to be made.
D. Are no longer recommended because studies show that other interventions lead to better survival rates.

Answer: C

The long-term survival advantage after CABG, cited above, was consistent across multiple subgroups based on gender, age, race, diabetes, body mass index, prior heart attack history, number of blocked coronary vessels, and other characteristics. For example, the insulin-dependent diabetes subgroup that received CABG had a 28% increased chance of survival after four years compared with the PCI group (NIH, 2016a).

According to an international study supported by the NHLBI, adults with diabetes and multi-vessel coronary heart disease who underwent cardiac bypass surgery had better overall heart-related outcomes than those who underwent PCI. The study compared the effectiveness of CABG and PCI that included insertion of drug-eluting stents. After five years, the CABG group had fewer adverse events and better survival rates than the PCI group. The survival advantage of CABG over PCI was consistent regardless of race, gender, number of blocked vessels, or disease severity (NHLBI, 2016g).

Test Your Learning

New studies that compare CABG and PCI in diabetics with multi-vessel CAD:

A. Found fewer adverse events and better survival rates after 5 years for those who underwent CABG.
B. Found fewer adverse events for those who underwent PCI.
C. Found no difference in survival rates and adverse events after 5 years.
D. Found neither procedure to be effective for diabetic patients.

Answer: A

Laser Transmyocardial Revascularization

Another technique is available for reducing the exercise-limiting angina suffered by some patients with severe chronic stable angina. **Laser transmyocardial revascularization** uses a laser to cut thin channels through the heart's walls. It is thought that the laser treatment stimulates the formation of new blood vessels that can increase local blood flow to heart muscle. It is also thought that the laser may destroy some of the nerves that are causing the angina (Texas Heart Institute, 2012).

Silent Ischemia

Patients often know when they are having an episode of heart ischemia because it causes angina or an anginal equivalent. However, heart ischemia can also be silent. When coronary artery patients are continually monitored by ECG during their daily lives, ischemic episodes can be detected electrically at times when the patients do not feel any symptoms. This is true both for patients who occasionally experience angina in normal life and for CAD patients who have never experienced angina. People can also have silent heart attacks, especially diabetics who have developed autonomic neuropathy. When patients are discovered to have had a silent MI, it is treated in the same way as a symptomatic one.

Test Your Learning

When patients with stable CAD have an episode of heart ischemia, they:

A. Almost always have angina or an anginal equivalent at the same time.
B. May have no symptoms.
C. Must always be taken immediately to an emergency department.
D. Should expand their coronary arteries by performing warm-up exercises for 5 to 10 minutes.

Answer: A

Major Complications of CAD

The ischemia of CAD can lead to serious heart damage. **Acute ischemia** can cause heart muscle to die, thereby weakening the heart and reducing its efficiency. Acute ischemia can also initiate a fatal arrhythmia and sudden cardiac death. **Chronic ischemia**, with accumulating damage from even small ischemic episodes, can lead to heart failure.

Acute Coronary Syndromes

Acute coronary syndromes are ischemic episodes caused by a sudden worsening of the atherosclerosis in coronary arteries. All acute coronary syndromes should be evaluated in an emergency department.

The acute coronary syndromes fill a spectrum from self-limiting ischemic episodes that cause little muscle death to myocardial infarctions, which cause areas of heart muscle necrosis.

Some myocardial infarctions threaten so much heart damage that the patient may need immediate reperfusion therapy. The need for immediate reperfusion is best diagnosed in a prepared emergency department that is attached to a cardiac reperfusion facility.

Always send patients with acute chest discomfort to an ED!

Health practitioners frequently receive telephone calls from patients or family members, friends, or caregivers who are concerned that their symptoms could be due to a heart attack.

Rather than dismiss such symptoms over the telephone, healthcare providers, EMS dispatchers, and staff positioned to receive these calls should advise patients to come in for an evaluation and testing. Only through a full physical examination, ECG, and appropriate blood tests to measure cardiac biomarkers can the correct diagnosis be made and appropriate interventions be given (Andersen et al., 2007).

During the last 25 years, the management of patients with acute MI has undergone many transformations. Until 1984, treatment was limited to providing symptomatic relief plus management of complications such as arrhythmias, acute heart failure, or post infarction angina. In the 1980s, the introduction of antithrombotic treatment with aspirin and intravenous (or intracoronary) fibrinolysis resulted in significant mortality reductions in patients with STEMI. In the 1990s, clinicians in some settings introduced immediate (pre-hospital) initiation of thrombolytic treatment following pre-hospital triage of patients with an acute MI and an indication for reperfusion therapy (Nauta et al., 2011).

Although more effective thrombolytic agents became available, reperfusion of the infarcted vessel often failed and bleeding complications were a limiting factor. Gradually, mechanical percutaneous techniques improved, and in the last two decades PCI became the treatment of choice in patients presenting with a STEMI (Nauta et al., 2011).

In the same time period, patients with NSTEMI benefitted from improved antithrombotic and anticoagulant therapy, better risk stratification, and tailored treatment with selective coronary revascularization in high-risk patients. In addition, effective secondary prevention was introduced with aspirin, beta blockers, statins, and ACE inhibitors in subjects with left ventricular dysfunction and, subsequently, in high-risk MI survivors. In combination, all these developments reshaped the treatment map of the patient with an MI (Nauta et al., 2011).

While they are being evaluated, patients who may have unstable angina or NSTEMI must have their ECGs monitored continuously. They also need to be near emergency resuscitation equipment including a defibrillator, because sudden cardiac death is possible (Andersen et al., 2007). These patients are usually given antiplatelet/anticoagulation therapy (aspirin, clopidogrel or prasugrel with or without heparin), oxygen when needed, nitroglycerin, additional pain relief (eg, morphine), beta blockers, and bed rest (Becker et al., 2008; Hirsh et al., 2008; Bashore et al., 2009; Smith, 2012). Coronary angiography within 24 hours is recommended for some unstable angina or NSTEMI patients.

The 2012 NSTEMI guidelines from the American Heart Association and the American College of Cardiology Foundation recommend the antiplatelet drug ticagrelor as an alternative to prasugrel and clopidogrel. Ticagrelor "significantly reduced the rate of death from vascular causes, myocardial infarction, or stroke compared with clopidogrel, although ticagrelor was associated with a higher risk of bleeding" (Smith, 2012).

STEMIs

When an acute coronary syndrome is accompanied by elevated cardiac biomarkers and elevations in the ST-segment of the ECG waveform, the event is called an **ST-elevation myocardial infarction (STEMI).** STEMIs are myocardial infarctions that affect large areas of heart muscle and that cause **transmural ischemia** resulting in decreased oxygenation through the entire thickness of the heart wall. Patients with STEMIs should be identified early in the chest pain triage, because they are candidates for immediate reperfusion therapy.

The two types of acute reperfusion therapy are percutaneous coronary intervention and IV fibrinolysis. In general, percutaneous interventions are more effective than fibrinolytic therapy. However, if there is no catheter laboratory capable of performing percutaneous coronary intervention, "the appropriate and timely use of some form of reperfusion therapy is likely more important than the choice of therapy" (Levine, 2012).

Speed is important. For percutaneous coronary interventions, the time goal is to have the obstructed artery recanalized in less than 90 minutes after the patient has come into the ED. For thrombolysis, the time goal is to have injected fibrinolytic agents no later than 30 minutes after the patient has come into the ED.

The most commonly used fibrinolytic agents are reteplase, alteplase, and tenecteplase. Giving these drugs quickly is critical because they are most effective when used on new clots. Intravenous injections of a fibrinolytic agent achieve the best results when administered within 1 to 2 hours of the onset of symptoms. The benefit of this therapy decreases markedly 6 hours after symptom onset and there is hardly any benefit from thrombolytics after 12 hours (AHRQ, 2012).

Fibrinolytic therapy has the risk of inducing bleeding. Intracranial bleeding is a serious complication that occurs in about 1% to 2% of cases (AHRQ, 2012).

STEMI Guidelines

Percutaneous coronary intervention, often with stenting, is performed emergently in patients having a STEMI. Thrombolytics are also used for STEMIs when PCI is not available.

According to the 2013 American Heart Association STEMI guidelines, PCI is the best treatment for patients having a STEMI if it can be performed in a timely manner and by experienced clinicians. Previous guidelines recommended "door-to-balloon time" or "door-to-needle time" of 90 minutes or less. This means that a patient who qualifies for PCI should have a cardiac catheterization within 90 minutes of arrival to the ED (Husten, 2012).

The 2013 guidelines now recommend "first medical contact (FMC) to device time" of 120 minutes or less (Levine, 2012). Many EMS providers have the ability to pre-notify a hospital with a cardiac catheter laboratory by sending the ECG electronically to the ED. If the ECG is identified as a STEMI or new left bundle branch block (LBBB) by the ED diagnostician, the catheter laboratory is then notified to be ready for a procedure. Upon arrival, the patient is quickly prepped in the ED and sent for PCI. The goal is to get the patient to angiography as soon as possible because, with every moment without oxygen, more heart muscle dies.

Research suggests that most patients with LBBB do not have an occluded artery at cardiac catheterization and are therefore needlessly exposed to the risks of fibrinolytic therapy (NIH, 2016a).

Therapeutic Hypothermia

The 2013 American Heart Association STEMI guidelines stress the importance of therapeutic hypothermia for unconscious cardiac-arrest survivors. During cardiac arrest, the brain is deprived of oxygen with resulting irreversible brain damage. If blood flow is restored, cerebral edema caused by tissue injury or blood–brain barrier disruption can cause even more brain damage. It has been found that these patients are likely to have a better neurologic outcome if mild hypothermia is induced.

For out-of-hospital cardiac arrest caused by ventricular fibrillation, the American Heart Association class I recommendation is induction of hypothermia to temperatures of 32°C to 34°C for a period of 12 to 24 hours in an unconscious patient who has had **return of spontaneous circulation (ROSC).** Hypothermia induction is a class IIB recommendation for other cases of cardiac arrest, with ROSC in an unconscious patient occurring both in and out of the hospital (Erb & Hravnak, 2012).

Coronary Artery Bypass Graft (CABG)

In a recent study sponsored by the National Heart Lung and Blood Institute, PCI outcomes were compared to cardiac bypass in diabetic patients. There was a substantial advantage of coronary artery bypass grafting (CABG) over PCI in diabetics with coronary artery disease (JournalWatch, 2012).

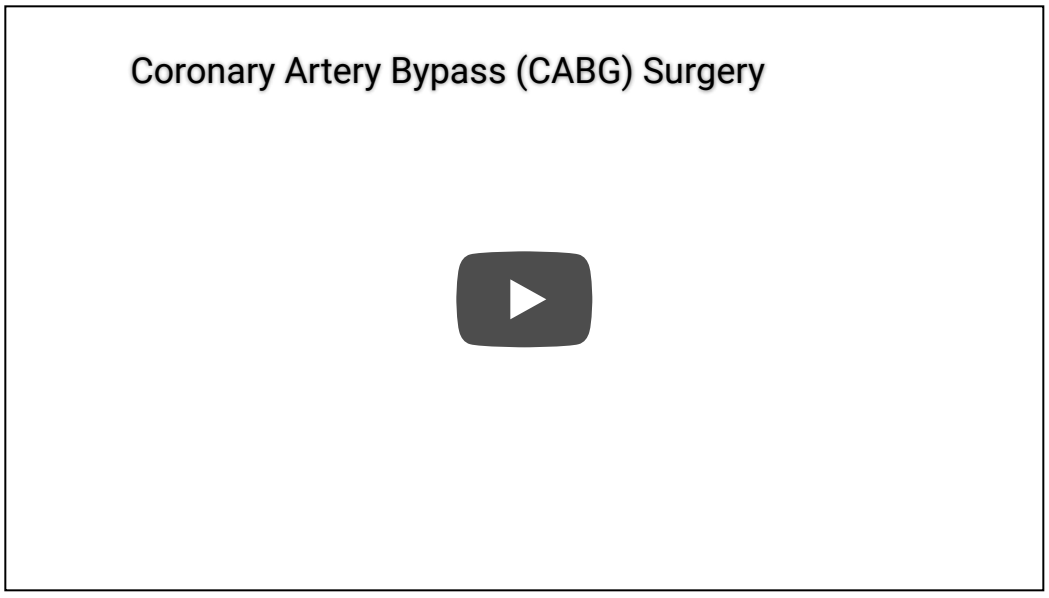
A new study supported by the National Institutes of Health (NIH) found older adults with stable coronary heart disease who underwent bypass surgery had better long-term survival rates than those who underwent PCI to improve blood flow to the heart muscle. While there were no survival differences between the two groups after one year, after four years the CABG group had a 21% lower mortality rate (NHLBI, 2016g).

Test Your Learning

Fibrinolytic therapy, used to reopen arteries after certain myocardial infarctions:

A. Consists of three intramuscular injections of tissue plasminogen activator.
B. Is most effective when given >3 hours after the MI to avoid excess bleeding.
C. Carries a 1% to 2% risk of inducing serious bleeding, such as intracranial hemorrhages.
D. Uses a percutaneous, balloon-tipped catheter to redilate clogged coronary arteries.

Online Resource (9:25)



https://www.youtube.com/watch?v=WM_tcf5Ogy0

Answer: C

Sudden Cardiac Arrest

Each year as many as 400,000 Americans die unexpectedly from an acute cardiac event. Typically, these sudden cardiac deaths (SCD) are due to a lethal arrhythmia, such as ventricular fibrillation. The risk of sudden cardiac arrest (SCA) increases with age or a history of underlying heart disease. Men are 2 to 3 times more likely to have SCA than women (NHLBI, 2016c).

Scar tissue replaces dead heart muscle cells after a heart attack. The scar tissue can disrupt and damage the heart's electrical system and cause the electrical signals to spread abnormally throughout the heart. These changes to the heart increase the risk of dangerous arrhythmias and SCA (NHLBI, 2016c).

An acute coronary syndrome seems to cause most cases of SCA in adults. Many of these adults, however, have no signs or symptoms of CAD before having SCA. The highest risk for SCA is during the first 6 months after a heart attack (NHLBI, 2016c).

Approximately 65% of deaths caused by MI occur in the first hour. Sudden cardiac death can strike a person without previous heart symptoms, but 80% to 90% of the victims of sudden cardiac death are found to have significant stenosis of at least one major branch of the coronary arteries (Schoen & Mitchell, 2009; Zafari, 2013).

Ischemic Heart Failure

A heart is in failure when it cannot pump sufficient blood to oxygenate the body. Coronary artery disease causes heart failure through myocardial infarctions. A patient can have a single infarction that injures a large region of heart muscle, or there can be a series of small infarctions that cumulatively injure a significant amount of heart muscle.

The ischemic heart failure produced by CAD is characterized by a dilated, hypertrophic left ventricle and by scars and fibrosis in the heart muscle. Ischemic heart failure is the most common form of congestive heart failure, and it accounts for about half of all the hearts that are replaced by transplants (Schoen & Mitchell, 2009).

Heart failure increases the oxygen requirements of the heart. Medicines are prescribed based on how severe the heart failure is, the type of heart failure, and the patient response to medications (NHLBI, 2016h). Patients with both CAD and heart failure are usually given an angiotensin-converting enzyme (ACE) inhibitor, a diuretic, and digoxin. Together, these drugs reduce the size, the wall tension, and the oxygen demands of the heart. If the ACE inhibitor does not reduce ongoing hypertension, a diuretic is added (Rosendorff et al., 2007). Occasionally, beta blockers can be given, but calcium channel blockers are contraindicated.

The following medications are commonly used to treat heart failure:

- Diuretics (water or fluid pills) help reduce fluid buildup in the lungs and swelling in feet and ankles.
- ACE inhibitors lower blood pressure and reduce strain on the heart. They may also reduce the risk of a future heart attack.
- Aldosterone antagonists trigger the body to get rid of salt and water through urine. This lowers the volume of blood that the heart must pump, decreasing the work load of the heart.
- Angiotensin-receptor blockers relax blood vessels and lower blood pressure to decrease the heart's workload.
- Beta blockers slow the heart rate and lower blood pressure to decrease the heart's workload.
- Isosorbide dinitrate/hydralazine hydrochloride helps relax blood vessels so the heart does not work as hard to pump blood.
- Digoxin makes the heart beat stronger and pump more blood. (NHLBI, 2016h)

Patients with both CAD and heart failure have a relatively poor prognosis. Even when the CAD takes the form of stable angina, reperfusion therapies are considered in an attempt to improve the oxygenation of a failing heart (Antman et al., 2008).

Cardiac Rehabilitation (Secondary Prevention)

For CAD the techniques of prevention, treatment, and rehabilitation overlap considerably. Cardiac rehabilitation, which is the long-term treatment of patients who have suffered an episode of severe heart ischemia, is a variant of the same medical care that patients receive in all phases of their CAD. In some medical centers the cardiac rehabilitation program that is prescribed for patients recovering from MIs is also offered as preventive therapy for patients at high risk for acute coronary syndromes.

Whereas primary prevention strategies are interventions during an acute phase of CAD, cardiac rehabilitation is referred to as secondary prevention. For CAD a key part of both rehabilitation and prevention programs is the reduction of the patient's atherosclerotic risk factors. Both programs strongly urge smoking cessation, supervised weight loss, physical exercise, and help in planning nutritionally balanced, low-fat/high-fiber/low-calorie meals. In addition, both programs stress aggressive management of hypertension, diabetes, and dyslipidemia.

Team Care

As with the management of all chronic illnesses, the most effective cardiac rehabilitation programs are organized utilizing a team of physicians, nurses, physical therapists, dieticians, social workers, occupational therapists, and clinical psychologists. Within their own area of expertise, healthcare professionals focus on helping to restore a cardiac patient's comfort, sense of well-being, and normal daily activities. At the same time, each worker aims to reduce specific risk factors to prevent future episodes of ischemia (Graham et al., 2008).

Individual Medical and Social Profiles

Cardiac rehabilitation programs are individualized. When a rehabilitation team takes over the care of a cardiac patient, they begin by compiling a medical and social history. Besides the standard health history, the team needs to know the details of the patient's normal daily home, work, and recreational activities. The physical examination information in the profile should include ECG recordings at rest and during exercise. Stress test results are added to the profile and can be used to set exercise guidelines.

Supportive and Protective Medications

After a serious episode of heart ischemia, patients will be taking a number of medications. A typical drug regimen includes a statin (with the goal of lowering LDL cholesterol blood levels to 70 mg/dl), a beta blocker (to ease the work of the left ventricle), and aspirin and clopidogrel (to reduce the risk of clots).

If patients have a poorly functioning left ventricle, they may also be taking an ACE inhibitor or an angiotensin-receptor blocker. When a patient continues to have hypertension on the existing drug and lifestyle regimens, a thiazide diuretic is added (Bashore et al., 2009).

Smoking Cessation

Smoking is dangerous for patients who have had an MI. Health professionals need to tell patients who smoke that stopping will cut in half their risk of dying from another ischemic episode.

Clinicians are encouraged to take five steps—the **Five A's**—with their patients who smoke:

- **Ask**. Ask the patient if they smoke.
- **Advise**. Strongly advise quitting.
- **Assess**. Ask the patient whether they are ready to quit.
- **Assist**. Help to formulate a workable smoking cessation plan, including medications and regular interactions with a counselor.

- **Arrange**. Take steps to put the plan into action: organize the necessary medications, counseling, and follow-up visits.

Healthy Diet

Eating that fulfills psychological or emotional needs is not always heart-healthy eating. Many cardiac patients eat out of habit and in response to psychological or emotional stress. These patients need guidance and support when trying to improve their diet. A cardiac diet should be prescribed, which includes low fat, high fiber and fresh fruits and vegetables.

Dieticians

Ideally, a dietician will be part of the cardiac rehabilitation team. Dieticians can help plan meals that:

- Meet the rehabilitation goals
- Patients can afford
- Patients will prepare and eat
- Is customized to their culture and needs

Rehabilitation Goals

A major rehabilitation goal is to keep blood levels of LDL cholesterol and triglycerides low and levels of HDL cholesterol high. Diets that help to meet these goals are low in saturated fats (<7% of daily calories), have minimal trans fats, and have about 25 g of dietary fiber daily for women and 38 g for men (Am. Diet. Assoc., 2008).

Other dietary advice includes:

- Drinking excess alcohol can raise blood pressure, an atherosclerotic risk factor.
- Salt and certain salt substitutes can contribute to hypertension, especially in those patients with a sensitivity to excess sodium.
- Excess body weight is an atherosclerotic risk factor, so diets should be calorie-controlled.
- Saturated fats should be replaced by monounsaturated fats, polyunsaturated fats, and omega-3 fatty acids (found in oily fish).
- Fruits, vegetables, and grains are recommended.

Dietary Counseling Programs

A dietary counseling program begins with the dietician seeing each patient individually. The dietician takes a dietary history and measures the patient's height, weight, and waist circumference. Patients are then given diaries in which to record all their food and drink intake for five days.

Patients mail or email their diaries to the dietician, and at the next visit the dietician suggests specific ways that the patient can improve what and how they eat. Regular followup visits continue. At each visit, the patient's height, weight, and waist circumference are measured, the patient's progress is charted, and specific dietary recommendations are suggested. This dietary rehabilitation program should continue until the patient has found a stable, healthy eating routine.

Weight Management

Excess weight is a heart stressor and an atherosclerotic risk factor, so weight reduction is an important component of cardiac rehabilitation for overweight patients. Heart-healthy weight goals are:

- BMI <25 kg/m^2 and
- Waist circumference <102 cm (40 in) in men and <90 cm (35 in) in women

To lose weight, a person must eat fewer calories. People should maintain a nutritional balance while they reduce their caloric intake, and crash diets should be discouraged. By itself, exercise rarely leads to a significant weight loss, but exercise can be an important weight loss aid.

For weight reduction and weight maintenance, regular one-on-one sessions with a dietician are usually the most successful tools. Experienced dieticians can help patients to devise a healthy reduced-calorie eating plan that the patient can stick with for the long term.

Exercise

Well-planned exercise training can restore many patients to a normal or near-normal lifestyle and weight. Modifications should be made for those with retinopathy or neuropathy, and for those on beta blockers.

Ineligible Patients

Some cardiac patients will not yet be sufficiently stable, mobile, or resilient to begin the exercise component of cardiac rehabilitation. Ineligible patients include those with:

- Worrisome ischemic symptoms (eg, unstable angina, significant resting ST-segment displacements, BP drop >20 mm Hg during episodes of angina)

- Heart rhythm or conduction problems (eg, uncontrolled tachycardia, atrial arrhythmias, ventricular arrhythmias, third-degree heart block)
- Severe aortic stenosis
- Uncompensated heart failure
- Recent thrombi (eg, from thrombophlebitis)
- Uncontrolled high blood pressure (systolic >200 mm Hg or diastolic >110 mm Hg)
- Uncontrolled diabetes
- Recent heart infections or inflammation (endocarditis, myocarditis, or pericarditis)
- Acute systemic illness or fever
- Acute metabolic problems
- Physical problems that prohibit exercise

Medically supervised exercise can be started for most other cardiac patients, including those who are stable following an MI, a coronary reperfusion procedure, or a heart transplant.

Structured Exercise Rehabilitation

To many people, cardiac rehabilitation is synonymous with post heart attack exercise programs. The exercise component of cardiac rehabilitation reconditions a patient's musculoskeletal and cardiovascular systems as well as strengthening the heart.

Reconditioning begins in the hospital. Supervised programs then continue for many months. The final goal is for the patient to develop an independent exercise plan that will last for years and become a part of his lifestyle.

Phase I: In the Hospital

Exercise rehabilitation can begin as soon as patients are medically stable. Breathing exercises and leg exercises get patients to use their muscles, and these exercises help to re-establish the patient's confidence that it will be safe to become active again. As the patient heals, assisted walking and light physical therapy can be added. By day four, patients are usually able to walk for 5 to 10 minutes in the corridors 3 to 4 times/day.

Phase II: After Discharge

Following discharge from the coronary care unit, a reconditioning program is begun. This usually includes having the patient walk indoors on a level floor at a speed that does not raise the patient's pulse >20 beats/minute above its resting rate. Patients should be reassured that they may fatigue easily, which is normal after a cardiac event.

Over 4 to 6 weeks, patients who are recovering well should be encouraged to increase gradually the total distance they walk until they are walking a total of 1 to 3 miles/day. Patients are asked to keep a diary of their daily exercise and the occurrences of any problems, and the physical therapist checks the diary regularly.

Phase III: Supervised Exercise Program

When they have been medically cleared by their physician, patients can begin a 6- to 12-month program of regular exercise. Often, the physician will perform a symptom-limited stress test to establish the patient's initial exercise capacity for the exercise program.

One commonly followed rehabilitation plan offers supervised exercise programs in 8-week sessions of 2 to 5 classes per week. The intensity of exercise is increased slowly over the sessions.

The main cardiac rehabilitation exercises are aerobic (walking on treadmills, stationary bicycling) and the intensity of exercise is limited by the patient's heart rate or feeling of fatigue (perceived exertion). Exercising to 60% to 75% of their maximum heart rate is a typical goal for cardiac patients. A rough calculation of maximum heart rate is 220 minus the patient's age. Those on beta blockers may not be able to reach the maximum heart rate.

Each session begins with 5 to 15 minutes of gentle exercise to decrease peripheral vascular resistance. Patients then undertake 5 to 30 minutes of aerobic exercise. On their physician's advice, some patients should have ECG monitoring during the exercises. Classes end with a 10-minute cool down period.

Phase IV: Continued Independent Exercising

The effects of exercise programs will fade unless patients continue to exercise regularly. Some patients have the self-discipline to stick to a lifelong independent exercise regimen. Many patients are more successful when they enroll in structured exercise programs, which can be found at community or health centers in most cities. After the formal cardiac rehabilitation program ends and the patient is exercising under self-direction, physicians should check on the patient's lifestyle at each medical visit and encourage remaining active and continuing a healthy diet plan.

Resuming Normal Daily Activities

The goal of cardiac rehabilitation is to enable patients to resume a normal life. Patients can be fearful after a heart attack, and they will need specific advice as to how much daily exercise they should attempt and when to return to sexual activities, although they may hesitate to ask. Physicians should introduce the subject and offer a simple guide: one common rule is that people can resume sexual activities when they can walk up and down two flights of stairs without any cardiac symptoms.

Patients should wait to resume driving a car for 4 weeks after an MI and 6 weeks after heart surgery. As they ease into driving independence, patients should begin by driving with a companion and avoid long trips or heavy traffic (Davis, 2008).

Psychological and Emotional Help

Patients who worry about suffering another serious cardiac ischemic event can feel overwhelmed, and they often need help making realistic decisions about their lifestyles. Many patients become overly timid, anxious, or depressed. Some patients deny the seriousness of their heart disease. Other patients become angry.

Few patients are able to manage these emotions without help. Physicians should be proactive by probing for clues that their patient might be suffering emotional or psychological distress. Because unapparent emotional and psychological problems can become disabling, all patients should be offered the opportunity to talk with a mental health professional (Antman et al., 2008).

So what happened to Mr. Hansen?

After stabilization from his angina and double stent placement, he took cardiac rehabilitation seriously to prevent an acute coronary episode and full myocardial infarction. The angiogram showed he had no permanent myocardial tissue damage and was very lucky.

He began to lose weight with the help of his wife, who wanted to lose her pregnancy weight. He joined a gym and lost 40 pounds. His blood pressure lowered to normal levels, his cholesterol levels decreased, and his HDL was 60 mg/dL. He ultimately ran a sprint triathlon!

Mr. Hansen continued to follow up with his cardiologist annually for lab tests to measure liver function and general health. He was fully adherent to medication management of statins, antihypertensives and antiplatelets. He developed a chronic cough from an ACE inhibitor and his medications were readjusted to reduce the side effect. He learned meditation and stress management strategies.

Every several years Mr. Hansen repeat a treadmill stress test and he has an angiogram every five years, which has resulted in no manifestations of progressive CAD. He and his wife went on to have another baby and both learned that the price for good health must be paid in healthy living choices day by day.

Test Your Learning

After an MI, patients can usually resume sexual activity:

A. When a coronary arteriogram has documented they now have an unobstructed arterial tree.
B. After a few sessions of counseling for patients and their partners.
C. When they can walk up and down two flights of stairs without cardiac symptoms.
D. In 4 to 8 days for men and 6 to 10 days for women.

Online Resource (3:59)

https://www.youtube.com/watch?v=fXRdrF0DKsQ

Answer: C

Summary

Causes and Symptoms of Angina

When a person’s epicardial heart arteries contain significant atherosclerosis, the condition is called coronary artery disease. Coronary artery disease is the number one killer of Americans, accounting for 1 of every 4 deaths.

Patients with CAD can be asymptomatic. As the disease progresses, however, it often produces angina pectoris. Angina pectoris, or simply angina, is the chest discomfort caused by ischemic heart muscle. The feeling of angina is typically described as a substernal pressure, tightness, heaviness, or suffocation rather than as pain; sometimes, the discomfort of angina is felt in an arm, a shoulder, the neck, or the jaw. Instead of angina, patients may experience anginal equivalents, such as shortness of breath or fatigue that follow a pattern typical of angina.

Angina that appears at a predictable level of exercise and that is relieved by rest or nitroglycerin is a characteristic of the form of CAD called stable angina. When the angina becomes unpredictable, the disease has moved to a form called unstable angina. Brief (<10 minutes) angina that is relieved by rest usually indicates an ischemic episode without significant heart damage. Extended angina (>20 minutes) usually indicates an MI, which means that some heart muscle has died (myocardial necrosis).

Acute Complications of CAD

Unstable angina can present as an acute coronary syndrome. An acute coronary syndrome is an episode of angina that is unusually severe, that is not relieved by rest or nitroglycerin, or that lasts >20 minutes. An acute coronary syndrome signals heart ischemia that may be severe enough to cause an MI, and a patient with an acute coronary syndrome should immediately be taken to an emergency department.

The damage done by major heart ischemia can be reduced significantly if the blood supply to the heart is restored by cardiac reperfusion therapy within about an hour. A more modest recovery of ischemic muscle can be achieved by reperfusion therapies done between 2 and 12 hours after the beginning of an MI. The two forms of immediate reperfusion therapy are percutaneous coronary interventions (angioplasty with or without stenting) and IV injections of fibrinolytic agents.

The acute coronary syndrome patients who are most likely to benefit from an aggressive reopening of the obstructed coronary arteries are those in whom the ischemia affects a large region of muscle or in whom an entire cross section of heart wall has become ischemic (transmural ischemia).

Blood tests and ECGs are used to identify candidates for reperfusion. The appearance of cardiac biomarkers (eg, cardiac troponins) in the bloodstream is a reliable sign of an MI. The appearance of elevations in the ST-segment of the patient's ECG waveform suggests that the area of the infarction is large or transmural. Together, these results indicate that immediate reperfusion therapy would be appropriate.

A systematic checklist for managing an acute coronary syndrome includes:

- Patients with worrisome chest discomfort should be taken to an emergency department immediately.
- After being stabilized, patients should chew 162 to 325 mg aspirin, blood tests for cardiac biomarkers should be analyzed, and ECGs evaluated. Patients with ECG changes indicating an acute ST-elevation myocardial infarction (STEMI) are candidates for immediate reperfusion therapy.
- All patients with a possible acute coronary syndrome should have pain relief (usually, nitroglycerine, with or without morphine). Patients with acute coronary syndromes are given a beta blocker, and patients with heart failure may receive an ACE inhibitor.
- A combination of antiplatelet/anticoagulant therapy and cardiac monitoring is the next step for those acute coronary syndrome patients who will not have reperfusion therapy.
- Patients with no elevation of their cardiac biomarkers and with a normal or nearly normal ECG should be treated with sublingual nitroglycerin. Depending on the circumstances, morphine and oral beta blockers might also be administered. If the heart symptoms resolve and the patient improves, continued treatment with antiplatelet medications is considered.
- Begin (or continue) patients on a cardiac rehabilitation program. (Brady et al., 2009)

Management of CAD

Coronary artery disease will always benefit from therapeutic lifestyle changes that can slow, stop, or occasionally reverse atherosclerosis. These lifestyle changes include a low-fat/high-fiber diet, weight loss, increased physical activity, and smoking cessation.

Management of CAD also includes controlling problematic co-existing disorders. Blood lipids should be kept in safe ranges:

- LDL cholesterol <100 mg/dl (<70 mg/dl in patients with significant artery disease)
- Triglycerides <150 mg/dl, HDL cholesterol >40 mg/dl in men and >50 mg/dl in women

- Blood pressure lower than systolic 140 mm Hg and diastolic 90 mm Hg

Lower pressures are better, and a reduction to a systolic pressure <130 mm Hg and a diastolic pressure <80 mm Hg is often advised. Angina indicates heart ischemia. Typical angina can be relieved by rest and by sublingual nitroglycerin tablets. Patients with stable angina can often avoid heart symptoms by slowing the rate at which they do their normal daily activities. When certain unavoidable activities cause angina, the angina can be reduced by taking a sublingual nitroglycerin tablet 5 minutes before the stressful activity.

A systematic checklist for managing CAD includes:

- Institute a prophylactic drug regimen of nitroglycerin, beta blockers, aspirin and, when needed, ACE inhibitors.
- Ask the patient to keep a record of symptom triggers, symptom details, and the success or failure of symptom relievers.
- Consider coronary angiography if symptoms interfere with the patient's daily activities.

Cardiac Rehabilitation

Long-term treatment for patients who have had an acute coronary syndrome is called secondary prevention, or cardiac rehabilitation. The goal of cardiac rehabilitation is to reduce the patient's chances of another acute coronary syndrome and to strengthen the cardiovascular system so the patient can resume normal daily activities.

Cardiac rehabilitation attempts to reduce the same atherosclerotic risks that are the subjects of primary treatment. Similar lifestyle changes and medications are recommended for rehabilitation and for primary treatment, although cardiac rehabilitation usually includes a more aggressive antithrombotic drug regimen.

The restrengthening component of cardiac rehabilitation consists of a gradually increasing exercise program that is initially supervised. By 6 months to 1 year, the supervised program should evolve into a lifetime of patient-directed regular exercise, along with weight watching and healthy eating.

Resources and References

Resources

American Heart Association website
Clear, up-to-date information presented for patients, caregivers, and healthcare professionals
http://www.heart.org/HEARTORG/

National Heart Lung and Blood Institute (NIH) website
Detailed, up-to-date information in two categories: for the public and for health professionals (in general, not as well written as the American Heart Association website)
http://www.nhlbi.nih.gov/

Women's Heart Foundation

Heart information specifically for and about women, presented both at the level of the patient and at the level of the healthcare professional
http://www.womensheart.org/

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Post Test

Use the answer sheet following the test to record your answers.

1. In the United States, coronary artery disease is the number one killer of:

    a. Men.
    b. Women.
    c. Both men and women.
    d. Children.

2. The two main coronary arteries:

    a. Run along the inside walls of the heart.
    b. Run along the outer surface of the heart.
    c. Are called the left anterior descending and the left circumflex coronary artery.
    d. Are called the left anterior descending and the right posterior descending coronary artery.

3. Atherosclerotic plaques are filled mainly with:

    a. Blood clots.
    b. Lipids and other cells.
    c. Embryonic arterial wall tissue.
    d. Glycogen.

4. Foam cells are:

    a. Macrophages overfilled with lipids.
    b. Lipid droplets.
    c. Vacuolated red blood cells.
    d. Necrotic, "foamy-looking" smooth muscle cells.

5. As they age and thicken, some atherosclerotic plaques accumulate deposits of:

    a. Copper.
    b. Potassium.
    c. Sodium.
    d. Calcium.

6. Atherosclerotic plaques are likely to accumulate:

a. At branch points and forks inside large arteries.

b. In capillaries and small arterioles.

c. On the surface of mucous membranes.

d. In the large veins, such as the vena cava.

7. The arteries that supply blood to the heart can partly compensate for a narrowed artery when the stenosis:

a. Is caused by a sudden blood clot.

b. Is caused by a sudden disruption of atherosclerotic plaque.

c. Has been caused by a sudden vasospasm.

d. Has developed gradually.

8. Heart muscle cells that become ischemic will:

a. Die within 1 to 2 minutes unless they are immediately reperfused.

b. Stop working but can "hibernate" for weeks and then revive when reperfused.

c. Die in 20 minutes to 4 hours if not reperfused.

d. Keep working for 2 to 5 days before they slowly die.

9. The chest discomfort of stable angina is often described as:

a. Sharp pain.

b. Fleeting pain.

c. Squeezing or tightness.

d. Made worse by the movements of coughing or breathing.

10. Cardiac biomarkers are intracellular heart muscle molecules that:

a. Are released into the blood when heart muscle cells die.

b. Are released into the blood all the time.

c. Become dense and visible in CT scans when heart muscle cells first becomes ischemic.

d. Become dense and visible in CT scans when heart muscle cells die.

11. The main symptom of CAD is:

a. Arrhythmias (usually, atrial fibrillation).

b. Chest discomfort called angina.

c. Gradually increasing shortness of breath, fatigue, and lower limb edema.

d. Sudden inability to get enough air.

12. An anginal equivalent is:

a. A symptom, such as shortness of breath other than chest pain that is caused by heart ischemia, brought on by stress, and relieved by rest or nitroglycerin.

b. The dose of nitroglycerin that can relieve typical angina in a given patient.

c. Sharp chest pain worsened by coughing.

d. Angina brought on by emotion not by exercise.

13. When examining patients with suspected CAD, chest x-rays:

a. Can give a definitive diagnosis.

b. Are not useful.

c. Will highlight the ischemic regions of the heart.

d. Are used to recognize possible co-existing heart failure.

14. Cardiac catheter angiography:

a. Uses dye injected into a coronary artery to search for stenotic segments.

b. Is the definitive diagnostic technique for an MI.

c. Has risks and is only used in preparation for coronary artery bypass graft surgery.

d. Has risks and cannot be used if a person has ever had an MI.

15. Regardless of the location of the STEMI what therapy is essential to open up the coronary vessels?:

a. Immediate reperfusion therapy.

b. Oxygen and steroid therapy.

c. Cardiac rehabilitation.

d. Radiation and nuclear medicine.

16. During an acute coronary syndrome, an important indicator that immediate coronary reperfusion therapy could be helpful is:

a. A normal ECG.

b. Depressed ST segments on ECG recordings.

c. Elevated ST segments on ECG recordings.

d. No P waves and irregular heart rate on ECG recordings.

17. To document episodes of heart ischemia, stress tests have:

a. Actors berate the patient.

b. The patient exercise, generally on a treadmill.

c. The patient attempt to do two tasks at once.

d. The patient eats a large meal followed by coffee and dessert.

18. When people call to ask advice about the sudden occurrence of chest discomfort:

a. Advise them to be taken immediately to the hospital, preferably by ambulance.

b. Check to see whether they suffer from panic attacks, gastroesophageal reflux disease (GERD), or peptic ulcer disease (PUD) before advising them.

c. Tell them to call their physician immediately.

d. Do a quick telephone triage of symptoms to determine the likelihood of an MI.

19. For patients who are having a suspected myocardial infarction, chewing 2 to 4 tablets of 81 mg aspirin is:

a. Not recommended if the patient is a candidate for IV fibrinolytic therapy.

b. Not recommended if the patient is already taking daily aspirin.

c. No longer recommended for most patients.

d. Recommended for all patients unless the patient is allergic to aspirin.

20. The use of supplemental oxygen:

a. Is never recommended for patients with suspected ACS.

b. Is only used during cardiac arrest.

c. Is recommended only when a patient is short of breath, hypoxemic, or has signs of heart failure.

d. Should be titrated to keep oxygen saturation at 100%.

21. Patients with stable angina who get chest discomfort with exercise:

a. Must resign themselves to living with lower levels of exercise or risk having an MI.

b. Can safely ignore chest discomfort because their disease is stable.

c. Can increase their exercise tolerance with a medically supervised cardiac rehabilitation program.

d. Should be put on bed rest to avoid sudden cardiac death.

22. For angina, sublingual nitroglycerin tablets:

a. Release nitric oxide, which dilates arteries.

b. Are used because they have no side effects.

c. Take about 30 minutes to produce relief.

d. Should continue to be taken every 3–5 min for 1–2 hr.

23. When managing a CAD patient, the primary lipid goal is:

a. Reducing triglycerides, preferably to <200 mg/dl.

b. Reducing HDL cholesterol levels, preferably to <40 mg/dl for men and <50 mg/dl for women.

c. Reducing the A1C level to <11.

d. Reducing LDL cholesterol levels, preferably to <70 mg/dl.

24. Coronary artery bypass grafts (CABG):

a. Are always the therapy of choice in a hospital with capacity to perform them.

b. Typically use nonhuman materials, such as Teflon tubes or porcine arteries.

c. Are the therapy of choice when multiple vessels are blocked and additional heart repairs need to be made.

d. Are no longer recommended because studies show that other interventions lead to better survival rates.

25. New studies that compare CABG and PCI in diabetics with multi-vessel CAD:

a. Found fewer adverse events and better survival rates after 5 years for those who underwent CABG.

b. Found fewer adverse events for those who underwent PCI.

c. Found no difference in survival rates and adverse events after 5 years.

d. Found neither procedure to be effective for diabetic patients.

26. When patients with stable CAD have an episode of heart ischemia, they:

a. Almost always have angina or an anginal equivalent at the same time.

b. May have no symptoms.

c. Must always be taken immediately to an emergency department.

d. Should expand their coronary arteries by performing warm-up exercises for 5 to 10 minutes.

27. Fibrinolytic therapy, used to reopen arteries after certain myocardial infarctions:

a. Consists of three intramuscular injections of tissue plasminogen activator.

b. Is most effective when given >3 hours after the MI to avoid excess bleeding.

c. Carries a 1% to 2% risk of inducing serious bleeding, such as intracranial hemorrhages.

d. Uses a percutaneous, balloon-tipped catheter to redilate clogged coronary arteries.

28. Sudden cardiac death:

a. Is a misnomer, because sudden "cardiac" deaths are usually due to strokes.

b. Is usually triggered by an acute coronary syndrome.

c. Occurs most often in women.

d. Is thought to be due to paroxysmal atrial fibrillation.

29. After an MI, patients can usually resume sexual activity:

a. When a coronary arteriogram has documented they now have an unobstructed arterial tree.

b. After a few sessions of counseling for patients and their partners.

c. When they can walk up and down two flights of stairs without cardiac symptoms.

d. In 4 to 8 days for men and 6 to 10 days for women.

Answer Sheet

Coronary Artery Disease: When Plaques Attack

Name (Please print your name): _____

Date: _____

Passing score is 80%

1. _____
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Course Evaluation

Please use this scale for your course evaluation. Items with asterisks * are required.

- 5 = Strongly agree
- 4 = Agree
- 3 = Neutral
- 2 = Disagree
- 1 = Strongly disagree

* Upon completion of the course, I was able to:

a. Define coronary artery disease, including its etiology and major forms.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

b. Discuss the prevalence, incidence, mortality, and morbidity of CAD.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

c. Name and trace the major coronary arteries.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

d. Describe atherosclerotic plaque and explain how it contributes to CAD.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

e. List at least 5 symptoms and/or diagnostic procedures that will lead to a diagnosis of CAD.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

f. Name 4 of the various medical responses in managing CAD.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

g. Discuss 3 major complications of CAD.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

h. List at least 6 elements of cardiac rehabilitation.

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

* The author(s) are knowledgeable about the subject matter.

5 4 3 2 1

* The author(s) cited evidence that supported the material presented.

5 4 3 2 1

* This course contained no discriminatory or prejudicial language.

Yes No

* The course was free of commercial bias and product promotion.

Yes No

* As a result of what you have learned, do you intend to make any changes in your practice?

Yes No

If you answered Yes above, what changes do you intend to make? If you answered No, please explain why.

* Do you intend to return to ATrain for your ongoing CE needs?

- Yes, within the next 30 days.
- Yes, during my next renewal cycle.
- Maybe, not sure.
- No, I only needed this one course.

* Would you recommend ATrain Education to a friend, co-worker, or colleague?

- Yes, definitely.
- Possibly.
- No, not at this time.

* What is your overall satsfaction with this learning activity?

○ 5 ○ 4 ○ 3 ○ 2 ○ 1

* Navigating the ATrain Education website was:

- ○ Easy.
- ○ Somewhat easy.
- ○ Not at all easy.

* How long did it take you to complete this course, posttest, and course evaluation?

- ○ 60 minutes (or more) per contact hour
- ○ 50-59 minutes per contact hour
- ○ 40-49 minutes per contact hour
- ○ 30-39 minutes per contact hour
- ○ Less than 30 minutes per contact hour

I heard about ATrain Education from:

- ○ Government or Department of Health website.
- ○ State board or professional association.
- ○ Searching the Internet.
- ○ A friend.
- ○ An advertisement.
- ○ I am a returning customer.
- ○ My employer.
- ○ Other
- ○ Social Media (FB, Twitter, LinkedIn, etc)

Please let us know your age group to help us meet your professional needs.

- ○ 18 to 30
- ○ 31 to 45
- ○ 46+

I completed this course on:

- ○ My own or a friend's computer.
- ○ A computer at work.
- ○ A library computer.
- ○ A tablet.
- ○ A cellphone.
- ○ A paper copy of the course.

Please enter your comments or suggestions here: _____

_____

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Registration Form

Please print and answer all of the following questions (* required).

* Name: _____

* Email: _____

* Address: _____

* City: _____ * State: _____ * Zip: _____

* Country: _____

* Phone: _____

* Professional Credentials/Designations:

_____

Your name and credentials/designations will appear on your certificate.

* License Number and State: _____

* Please email my certificate:

○ Yes ○ No

(If you request an email certificate we will not send a copy of the certificate by US Mail.)

Payment Options

You may pay by credit card or by check.
Fill out this section only if you are **paying by credit card.**
8 contact hours: $39

Credit card information

* Name: _____

Address (if different from above): _____

* City: _____ * State: _____ * Zip: _____

* Card type:

○ Visa ○ Master Card ○ American Express ○ Discover

* Card number: _____

* CVS#: _____

* Expiration date: _____