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Contact Hours: **7**

Coronary Artery Disease (CAD)

BY Michael Jay Katz, MD, PhD; Sheryl M. Ness, MA, BSN, ADN, RN, OCN

COURSE OBJECTIVE: The purpose of this course is to enable healthcare practitioners to plan, deliver, and evaluate evidence-based preventative and therapeutic care for patients with or at risk for coronary artery disease (CAD).

LEARNING OBJECTIVES
Upon completion of the course, you will be able to:

- Discuss the incidence and impact of coronary artery disease.
- Describe the anatomy and normal blood circulation of the heart.
- Review the pathophysiology of CAD.
- Differentiate between the major clinical presentations of CAD.
- Discuss nonpreventable and preventable risk factors.
- Describe evidence-based prevention measures.
- Identify the signs, symptoms, and clinical test outcome criteria used to screen and diagnose coronary artery disease.
- State the principles underlying the acute management of the disease.
- Explain the components of a comprehensive plan of care and monitoring for patients.
- Discuss the complications, comorbidities, and other diseases associated with CAD.

INTRODUCTION

Coronary artery disease (CAD) is caused by atherosclerosis of the coronary arteries that leads to a restriction of blood flow to the heart. Atherosclerosis is a process that develops slowly over time. Typically, atherosclerosis begins in a person's teenage years or earlier, and the disease worsens quietly for decades. As people age, their atherosclerosis becomes more likely to involve the arteries of the heart and to become coronary artery disease.

Atherosclerosis is a chronic condition that narrows arteries by building fat-filled bulges in the arterial walls. These bulges are called atherosclerotic plaques, or simply plaques. In some people, the plaques eventually break open and the contents cause blood clots. If these clots are swept into the bloodstream, they can lodge in the smaller arteries downstream and completely block blood flow beyond that point.

The heart muscle is constantly active, and it requires a continuous blood supply. When a heart artery is blocked suddenly, the heart muscle it supplies stops working within a few minutes. If the blood supply remains blocked for a half hour or more, the heart's muscle cells will begin to die.

Angina

Complete, sudden blockage of an artery is not the only problem. Even a reduced blood supply will reduce the oxygen supply to heart muscle, and an oxygen-starved heart muscle responds with a characteristic feeling of pain or discomfort called **angina**.

When its arteries are narrowed by atherosclerosis, a heart may still get enough oxygen to pump blood at rest. On the other hand, exercise increases the work of the heart, and narrowed arteries cannot always deliver the excess oxygen required by an exercising heart. Under these circumstances, a person with narrowed coronary arteries will develop angina when exercising. One of the first symptoms of coronary artery disease is the appearance of angina when a person is working strenuously.

Acute Coronary Syndrome and Myocardial Infarction

As atherosclerosis progresses, some of the plaques rupture and clots are formed. If a clot temporarily shuts down an artery, the patient will get sudden angina that lasts until the clot is broken or swept away. If the clot remains for an extended period of time, some heart muscle begins to die: this is a "**heart attack**," or **myocardial infarction**.

In general, the sudden blockage of a coronary artery or one of its main branches is called an acute coronary syndrome, and myocardial infarctions are one form of acute coronary syndrome. Acute coronary syndrome is a medical emergency and must be treated immediately in an emergency room.

Preventative Measures

The progression of atherosclerosis can be slowed or even stopped by a few preventive measures. These include stopping smoking, staying thin or losing weight, exercising regularly, and eating a low-fat, balanced diet. To control atherosclerosis, it is also important to keep blood pressure low, to reduce high blood cholesterol levels, and to treat diabetes.

People who develop symptomatic coronary artery disease should begin or continue these anti-atherosclerotic programs. In addition, they should take aspirin daily, and they should probably take other medicines (typically, beta blockers) to reduce the workload of the heart. Nitroglycerin tablets can be used to alleviate occasional anginal pain, and surgical procedures are available to widen narrowed arteries.

Incidence and Impact

Coronary artery disease is the most common form of heart disease. It is estimated that nearly one half of all middle-aged men and one third of middle-aged women in the United States will develop some form of the disease (Wilson & Douglas, 2015). CAD is the number one killer in the developed world, with over 7.4 million deaths attributed to CAD in 2012 (WHO, 2015). In the United States, it is estimated that one in seven deaths is due to heart disease. In addition, heart disease is the primary cause of death in women, taking more lives than all cancers combined (AHA, 2015).

The proportion of deaths in the United States that are due to coronary artery disease has been decreasing slowly but continuously over the past half century. Nonetheless, coronary artery disease remains the single most common cause of death in the United States.

Coronary artery disease is not just an American problem. Throughout the developed world, coronary artery disease causes more deaths and disabilities and is responsible for more economic costs than any other single illness (WHO, 2015).

TERMS RELATED TO CAD

Coronary artery disease is the result of atherosclerosis of the coronary arteries of the heart.

Coronary artery disease is also called:

- Coronary heart disease (CHD)
- Ischemic heart disease (IHD)
- Atherosclerotic heart disease
- Coronary atherosclerotic disease

The main forms of coronary artery disease are:

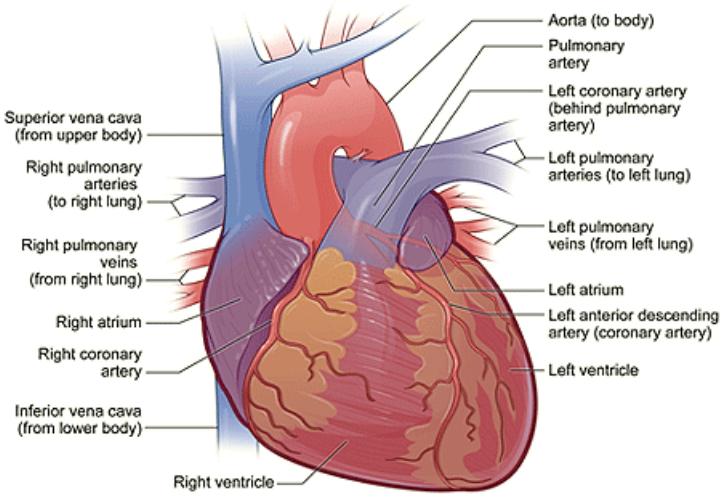
- Chronic stable angina
- Acute coronary syndromes

The three main acute coronary syndromes are:

- Unstable angina
- Myocardial infarction (heart attack)
- Sudden cardiac death

CIRCULATION OF THE HEART

The heart is made up almost entirely of muscle. Cardiac muscle, which differs from the skeletal and smooth muscle of the rest of the body, is dependent on aerobic metabolism. This means that the heart cannot function without a constant supply of oxygen.



A drawing of the heart, seen from the front. The coronary arteries and their main branches are large, and they run along the outer surface of the heart. The smaller arteries, which directly feed the heart muscle, dive deep into the walls of the heart. (Source: NHLBI, 2011a.)

Coronary Arteries

Just beyond the aortic valve—the outflow valve of the left ventricle of the heart—the right and left **coronary arteries** are the first branches of the aorta. The two coronary arteries and their main branches run in grooves along the outside of the heart; these grooves separate the left and right ventricles and they also separate the atria from the ventricles. The coronary arteries and their main branches are called epicardial arteries because they run on the outer surface of the heart.

Each major coronary artery is 2 mm to 4 mm wide, about half the diameter of a pencil. From the coronary arteries and their major branches, many small arteries run into the muscular walls of the heart, and these small arteries give rise to rich capillary networks that bathe the cardiac muscle cells with blood. All arteries inside the heart walls are fed by branches of either the right or left coronary arteries.

In most people, the left coronary artery supplies most of the blood used by the left ventricle and the interventricular septum, while the right coronary artery supplies most of the blood used by the walls of the right ventricle. However, people vary in the way the blood supply to the heart is divided between the right and left coronary arteries.

There is not much overlap between the territories of the major branches of the coronary arteries. Therefore, if one of the major branches suddenly becomes blocked, there is no other blood supply to the territory served by that branch, and muscle in that territory will be deprived of oxygen (Warnica, 2013).

A common finding in coronary artery disease is collateral circulation, the development of additional arteries that form a natural bypass from one side of a blocked artery to the other. Research suggests that coronary collateral circulation may help to reduce ischemia, preserve ventricular function, and improve prognosis in patients with coronary artery disease (Seiler et al., 2013).

LEFT CORONARY ARTERY

The left coronary artery splits into two main branches, the left anterior descending (LAD) coronary artery and the left circumflex coronary artery. The LAD coronary artery runs down the front of the heart along the groove between the left and right ventricles. In most people, the LAD supplies blood to the front wall of the left ventricle and to the interventricular septum. Forty to fifty percent of heart attacks are caused by an obstruction of the left anterior descending coronary artery.

The left circumflex coronary artery runs to the left (at a right angle to the LAD) along the groove between the left atrium and the left ventricle. The left circumflex coronary artery supplies blood to the side or lateral wall of the left ventricle. Fifteen to twenty percent of heart attacks are caused by an obstruction of the left circumflex coronary artery.

RIGHT CORONARY ARTERY

The right coronary artery runs to the right, along the groove between the right atrium and the right ventricle. The right coronary artery branches behind the heart and gives rise to the posterior descending coronary artery, which parallels the LAD in front. The right coronary artery supplies the bottom and backside of the heart, and in most people, it supplies blood to the right ventricle and to the sinus and AV nodes of the heart's electrical conduction system. Thirty to forty percent of heart attacks are caused by an obstruction of the right coronary artery.

Normal Blood Flow to the Heart

The blood flow through the heart usually keeps up with the body's demand. The demand is increased by exercise and strong emotions, both of which make the heart pump more quickly and more forcefully, causing the heart to use more oxygen. As a rough rule, when the heart beats twice as fast, it needs twice as much oxygen (Depre et al., 2011). Normally, the extra oxygen needed during exercise is supplied by a faster and a more voluminous blood flow through the coronary arteries.

HEART RATE

Faster blood flow is a direct result of a faster heart rate. Blood flow to the heart automatically speeds up as the heart beats more quickly because the coronary arteries are fed directly by the outflow of the heart.

LOCAL MUSCLE ACTIVITY

Throughout the body, the volume of blood flow is regulated by the size of the arteries. Arteries have an innate tension in their walls. This tension keeps arterial volume at a particular level, and the tension also creates a resistance to blood flow. When the arterial wall tension is reduced, the artery stretches more easily and can carry a larger volume of blood.

The natural state of coronary arteries and their main branches is relatively wide open, and in general, these arteries do not limit the volume of blood getting to the muscle cells inside the heart. Instead, it is the small arteries inside the walls of the heart that widen and narrow and in this way control the volume of blood flow to the muscle cells.

The control of the arterial wall tension—the force that widens and narrows the arteries—is local. As muscle cells work harder, they change the concentration of molecules (e.g., oxygen) surrounding them. Most molecular changes resulting from hard work relax the arteries in the vicinity. In addition, during exercise or stress, sympathetic nerves reduce the tension in the walls of arteries. Together, these factors relax the walls of the arteries and increase the local blood flow.

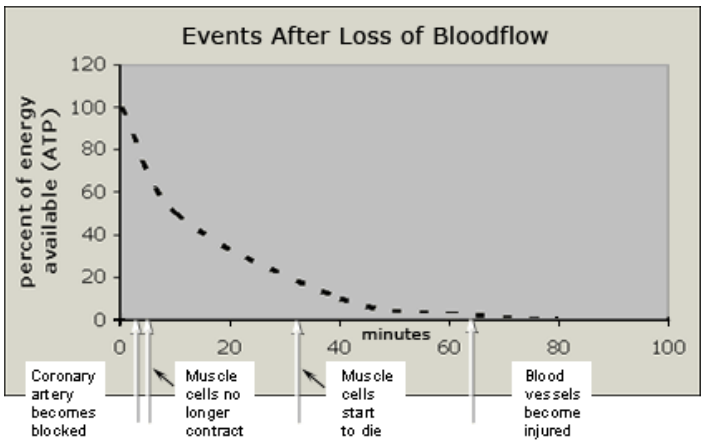
Myocardial Ischemia

Myocardial ischemia occurs when blood flow and blood volume are insufficient to supply all the oxygen needed by the heart muscle.

BLOOD LOSS TO MUSCLE CELLS

What happens to heart muscle cells when they become ischemic? As soon as the blood flow to an area of heart muscle is stopped, the cells begin to lose their energy stores, and within a few minutes the muscle cells are no longer able to contract. Any region of the heart that loses all its blood flow will stop working almost immediately.

Although muscle cells stop working, they do not begin to die until 20 to 40 minutes after losing their blood supply. If blood flow is restored within a half hour, most muscle cells will eventually recover; however, the recovery can take from 10 minutes to several days. During that time, the heart acts “stunned” and may not contract well unless stimulated by ionotropic drugs (Schoen, 2010).



When the blood flow is entirely stopped to a region of the heart, the muscle cells stop working almost immediately. The muscle can recover, however, if blood flow is restored in less than a half hour. (Source: Adapted from Schoen, 2010.)

Another effect of sudden ischemia of the heart is electrical irregularity. Before muscle cells begin to die, they become electrically unstable. After the blockage of a major coronary artery, the electrical instability of some people's hearts leads to ventricular fibrillation, and this can cause sudden death.

SYMPTOMS OF ISCHEMIA

Chest Pain

Heart ischemia usually produces symptoms, and the classic symptom of reduced oxygen supply to the heart is a particular type of chest pain called angina pectoris, or simply angina. Angina arises many seconds or even minutes after a sudden arterial blockage.

Typically, angina pain feels like crushing or squeezing, although sometimes it is described as burning. The sensation is usually felt inside the chest behind the sternum, and the feeling can radiate to the lower part of the neck, jaw, shoulder, back, or down the ulnar side (inside) of the left arm; in some cases the feeling can radiate to either or both arms. In some people, the discomfort of angina is mild, but other people get diffuse unbearable pain.

Although women tend to visit their physicians more often than men and therefore report more symptoms, including chest pain, their angina symptoms usually present in the form of upper abdominal discomfort, neck or jaw pain, or shortness of breath as opposed to crushing or squeezing chest pain. Women are also more likely than men to associate their angina with emotional or mental stress (Charney, 2011).

Angina is a classic symptom of myocardial ischemia. However, angina is not a perfect indicator of heart problems. Myocardial ischemia can occur without angina; moreover, some people get angina although they have no detectable ischemia.

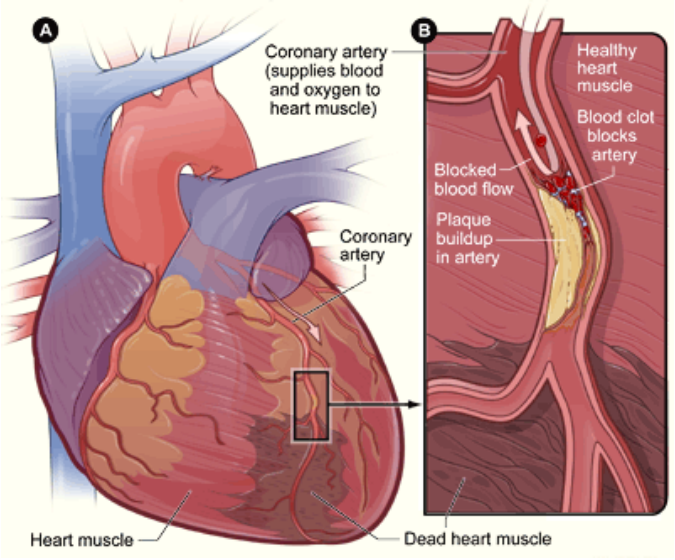
- >20% of heart attacks (myocardial infarctions) occur with no symptoms.
- Only 18% of heart attacks are preceded by long-term angina.
  (Lloyd-Jones, 2010)

Arrhythmias

Another significant result of sudden ischemia is a change in the heart's rhythm. Such changes can be serious. The arrhythmias (notably, ventricular fibrillation) that sometimes result from heart ischemia are the cause of most sudden deaths after an acute myocardial infarction.

PATHOPHYSIOLOGY

Coronary artery disease is the umbrella term for various syndromes of heart ischemia that are caused by atherosclerotic obstruction of the coronary arteries. The atherosclerotic damage ranges from gradual narrowing of the coronary arteries (due to bulging patches of plaque) to the sudden obstruction of a coronary artery by a blood clot that has been dislodged from the surface of a ruptured plaque.

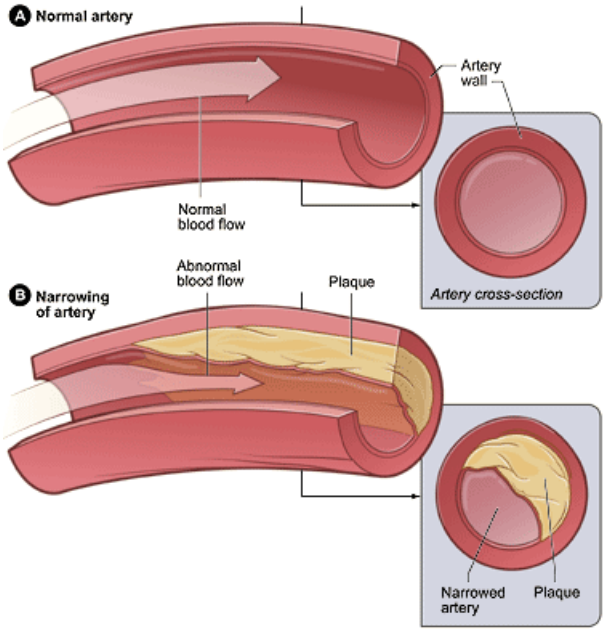


The heart damage in coronary artery disease ranges from narrowing of a coronary artery to complete blockage of a coronary artery. (Source: NHLBI, 2013.)

Atherosclerosis

Atherosclerosis is the disorder that underlies coronary artery disease. Atherosclerosis thickens the walls of medium and large arteries. The atherosclerotic thickenings occur as bulges, called plaques, in the arterial walls. Plaques contain lipids, white cells, smooth muscle cells, and

connective tissue in a poorly organized mass that lies just under the endothelial lining of the artery wall.



In atherosclerosis, fat and cells collect in bulges just below the surface of the lining of arteries. These bulges are called plaques. Over time, the plaques thicken and reduce the inner diameter of the arteries, allowing less blood to get to tissues beyond the plaques. (Source: NHLBI, 2014a.)

In the United States, atherosclerosis usually begins in childhood or adolescence and then gradually worsens over many decades. Any medium or large artery in the body can be affected. Most atherosclerosis causes no clinical problems. Many people have atherosclerosis throughout their bodies but develop no serious medical symptoms, and the disease is only discovered at autopsy (Lam, 2012).

When atherosclerosis causes the coronary arteries to become very narrow or when plaques rupture and send clots into the arteries of the heart, a person is said to have coronary artery disease.

When the effects of atherosclerosis reduce the circulation in nonheart arteries, a person can develop **peripheral artery disease (PAD).**

- In the carotid arteries, PAD can cause strokes.
- In the aorta, it can lead to aneurysms.
- In the legs, it can cause episodic pain when walking (intermittent claudication) and sometimes gangrene of the feet.
- In the gastrointestinal arteries, it can cause mesenteric ischemia.
- In the renal arteries, it can cause stenosis leading to hypertension.

ATHEROSCLEROTIC PLAQUE FORMATION

The hallmarks of atherosclerosis are atherosclerotic plaques, which develop slowly and in three stages.

Stage One: Fatty Streaks Appear

As atherosclerosis begins, the first detectable changes are the appearance of fatty streaks along artery walls. These streaks are places where excess fat is accumulating.

Most of the fat (lipids) in the blood is carried by proteins in molecular complexes called **lipoproteins**. The surface of a lipoprotein is made of the more water-soluble lipids—cholesterol and phospholipids. The least soluble lipids—cholesteryl esters and triglycerides—are carried in the centers of the lipoproteins.

Lipoproteins come in five sizes. From the largest to the smallest, these are: chylomicrons, VLDL, IDL, LDL, and HDL. Each size of lipoprotein has its own characteristic balance of lipids. The largest lipoproteins—chylomicrons and VLDL—are especially rich in triglycerides, while 70% of all blood cholesterol is contained in the LDL lipoproteins.

When there is an excess of lipoproteins in the blood, as happens in hypercholesterolemia, more lipoproteins than normal get through the endothelial cells and into the artery walls. These excess lipoproteins stick to extracellular molecules, and eventually enough excess fat becomes stuck just below the endothelial cells to form visible yellowish streaks—fatty streaks—along the arterial walls.

Stage Two: Foam Cells Develop

White blood cells are attracted to these unhealthy accumulations of lipids. Some of the white blood cells are phagocytes, scavenger cells that begin to "swallow" the lipids. These cells swell as they become filled with fat droplets; the cells look puffed up, and they are called foam cells.

Stage Three: Smooth Muscle Cells Move In

Fatty streaks containing foam cells continue to thicken. Soon smooth muscle cells from deeper in the arterial walls move into the expanding plaques. Muscle cells secrete extracellular molecules such as collagen, and the whole fatty lesion bulges into the bloodstream and narrows the space inside the artery. As they continue to evolve, some plaques also accumulate calcium, which can sometimes be seen in X-rays.

Stage Four: Blood Clots Begin to Form

In time, the endothelial cells covering the bulge begin to rip, letting blood come in contact with the underlying collagen and other extracellular molecules. Extracellular

molecules are stimulants of blood clotting. Therefore, small blood clots and clumps of platelets form along the rips in the endothelial lining of the artery. Disrupted plaques create blood clots, and if the clots break loose, they are carried into the smaller arteries downstream. The result can be a blocked artery.

INFLAMMATION

In response to infection, the body produces inflammation—redness, tenderness, warmth, and swelling. The inflammatory response can be triggered by things other than foreign substances, and inflammation plays a key role in worsening atherosclerosis.

Inflammation is the activation of white blood cells. Just as white blood cells are activated by foreign antigens, they can also be activated by the contents of atherosclerotic plaques. As a plaque develops, white blood cells (notably, lymphocytes and macrophages) collect underneath the endothelial cells, and their reaction to the material they encounter is to begin releasing inflammatory molecules (e.g., cytokines, proteolytic enzymes). The macrophages also ingest the excess lipoproteins in the vicinity, and these macrophages become bloated foam cells.

Many of the processes in the inflammatory response weaken the structure of an atherosclerotic plaque. The foam cells do not hold together strongly. The secreted inflammatory molecules destabilize the plaque by breaking bonds between extracellular matrix molecules. In these and other ways, inflammation makes a plaque prone to rupture, leading to the formation of blood clots (Falk & Fuster, 2011).

CLOTS AND VASOSPASMS

Atherosclerotic plaques bulge into arteries and narrow the space available to carry blood. Under some conditions, these plaques also generate blood clots and vasospasms.

The rupture of a plaque can also cause the walls of the artery to constrict in that region. The resulting vasospasm narrows the artery suddenly and causes ischemia downstream. Alone and together, clots and vasospasms can cause emergency medical conditions, including heart attacks and sudden death.

The rupture of an atherosclerotic plaque can happen quickly. It can be set off by a sudden spurt of output from the sympathetic nervous system. Such spurts can occur when people are waking in the morning or when people are subjected to strong emotional stress. External stresses, however, do not disrupt stable plaques. External stresses only rupture those plaques that have already become weakened and destabilized by inflammation or other internal changes (Falk & Fuster, 2011).

ATHEROSCLEROSIS OF THE CORONARY ARTERIES

Rather than uniformly thickening arterial walls, atherosclerosis is patchy and unevenly distributed. The specific coronary arteries affected by atherosclerosis vary from person to person,

but there is a common feature: within a coronary artery, plaques are found most often at branch points, places where the blood flow naturally becomes turbulent.

The narrowing of coronary arteries usually occurs slowly, and in response, new small collateral arteries have time to grow into the fields of the atherosclerotic arteries to help bolster the local oxygen supply. These collateral arteries will sometimes provide enough extra blood flow to keep the heart muscle working comfortably at a resting rate. The collateral arteries are small, however, and they do not have the capacity to keep up with the oxygen demands of heart muscle during exercise.

Even with the growth of small collateral arteries, the continual narrowing of the coronary arteries by atherosclerosis can eventually produce ischemia and anginal pain. Initially, these symptoms occur only when the patient is exercising; later, the symptoms begin to occur even when the patient is at rest.

Besides slowly narrowing the coronary arteries, atherosclerosis can cause a sudden medical crisis. The degeneration of a plaque can seed clots into the bloodstream and can also trigger local vasospasm. These lead to a marked reduction of blood flow, and the resulting damage can range from temporary to permanent and from mild to fatal.

CAUSES AND CONTRIBUTORS

Some people have a genetic propensity for developing atherosclerosis, but it appears that the disease can occur in almost everyone. A critical causative agent is dietary fat, especially the cholesterol that is carried in the blood in **low-density lipoproteins (LDL).** High blood levels of LDL cholesterol can cause and worsen atherosclerosis.

Other contributors to atherosclerosis are cigarette smoking, high blood pressure, type 2 diabetes, age, gender, physical inactivity, and obesity (Boudi, 2014a).

CLINICAL FORMS OF CAD

Many people live their entire lives symptom-free even when they have atherosclerosis of the coronary arteries. Other people, however, develop symptoms and heart damage from their atherosclerosis. The ischemic heart problems of atherosclerotic coronary artery disease fall into two general classes: chronic coronary syndromes and acute coronary syndromes (Schoen, 2010).

CLINICAL FORMS OF CORONARY ARTERY DISEASE

Chronic Coronary Syndromes

- Stable angina
- Stable ischemic heart disease

Acute Coronary Syndromes

- Sudden cardiac death
- Myocardial infarction (MI)
- Unstable angina

Chronic Coronary Syndromes

Coronary artery disease is a chronic disease that is punctuated by sudden medical emergencies—the acute coronary syndromes. The long, chronic phases of the disease have two forms: stable angina and stable ischemic heart disease (Schoen, 2010).

STABLE ANGINA

When one or more coronary arteries have become narrowed and cannot meet the demands of a hard-working heart, the patient has **chronic stable angina**. This syndrome is characterized by ischemic heart pain that shows up when patients exercise and that goes away in a few minutes after they rest. Blood flow to the heart must be reduced by two thirds to three fourths before a patient develops the symptoms of chronic stable angina.

The chest pain of chronic stable angina is short-lived and occurs predictably. Particular amounts of exercise, trauma, weather changes, or strong emotions may trigger angina. In chronic stable angina, resting or nitroglycerin tablets will relieve the chest pain in a few minutes.

The occurrence of angina is influenced by the general tone of the sympathetic nervous system (which tends to be, for example, higher in the mornings) and by the demands of blood flow by the gastrointestinal tract after a meal. Therefore, although the symptoms of chronic stable angina are fairly predictable, the amount of exercise or stress that will produce these symptoms varies during the course of a day.

The chest pain of chronic stable angina can also be brought on by any medical condition that increases the work of the heart, such as hypertension, aortic stenosis, systemic infections, or thyrotoxicosis. Likewise, conditions that reduce the oxygenation of the blood, such as COPD, anemia, or high altitudes, can also cause angina.

STABLE ISCHEMIC HEART DISEASE

A second chronic syndrome is **stable ischemic heart disease**, or ischemic cardiomyopathy, in which years of damage from ischemia have weakened the heart sufficiently that it gradually fails. Stable ischemic heart disease is a major cause of congestive heart failure in older adults.

Most patients with this condition have had acute myocardial infarctions in the past, although not all infarctions may have been symptomatic. In people who have had "silent" myocardial infarctions, heart failure from stable ischemic heart disease can be the first evidence of their coronary artery disease.

PROGNOSIS

A patient with any form of coronary artery disease has a higher chance of dying when the left ventricle of the heart has been weakened. Signs of a failing left ventricle include an enlarged heart, pulmonary edema, leg and ankle edema, jugular venous distension, or a third heart sound (S_3). Previous myocardial infarctions weaken the heart, so a history of past heart attacks also worsens a patient's prognosis (Warnica, 2011).

Patients who have chronic stable angina with no history of myocardial infarction, a normal resting ECG, and normal blood pressure have a mortality rate of approximately 1.4% per year. Systolic hypertension raises the mortality rate to 7.5%, an abnormal ECG raises the rate to 8.4%, and the two together raise the rate to 12%. Diabetes doubles all these rates.

Acute Coronary Syndromes

Sudden unpredictable episodes of severe heart ischemia are called acute coronary syndromes. Acute coronary syndromes result from a disruption of plaque that then causes ischemia severe enough to injure or kill muscle cells in the heart.

An acute coronary syndrome needs immediate treatment in a prepared emergency room (Kim et al., 2011). People with the highest risk of developing an acute coronary syndrome are those who already have serious cardiovascular disease or diabetes.

Similarly to other types of heart disease, **risk factors** for acute coronary syndromes include:

- Older age (above age 45 for men and above age 55 for women)
- High blood pressure
- High blood cholesterol
- Cigarette smoking
- Physical inactivity
- Type 2 diabetes
- Family history of chest pain, heart disease, or stroke
  (Mayo Clinic, 2013a)

SUDDEN CARDIAC DEATH

The most catastrophic of the acute coronary syndromes is **sudden cardiac death**, an unexpected death from cardiac causes that happens quickly, usually within an hour of the first symptoms. In adults, sudden cardiac death is usually (in 80% to 90% of patients) associated with coronary artery disease.

The direct cause of these deaths is fatal arrhythmias, such as ventricular fibrillation. The arrhythmias develop in heart cells that have been made overly excitable because of sudden ischemia from a blood clot or a vasospasm.

MYOCARDIAL INFARCTION

Heart attacks (myocardial infarctions) are a type of acute coronary syndrome. Most myocardial infarctions are caused by clots dislodged from atherosclerotic plaque with or without vasospasms (i.e., sudden, temporary contractions of the muscles inside the walls of a heart artery).

Myocardial infarctions occur when a chunk of plaque, a blood clot, a vasospasm, or some combination of these completely obstructs a coronary artery or one of its major branches. If the obstruction persists for more than about 30 minutes, some of the cell injury will be permanent. The area of the heart damaged by a myocardial infarction is called a myocardial infarct.

A myocardial infarction produces distinctive ECG changes. In addition, the cell necrosis in the infarct allows intracellular muscle molecules to leak into the bloodstream, and these heart molecules can be detected in blood tests (Mayo Clinic, 2013a).

UNSTABLE ANGINA

A third common acute coronary syndrome is **unstable angina**. An episode of unstable angina includes symptoms of heart ischemia that do not go away after 5 to 10 minutes of rest. These episodes are caused by sudden disruptions of plaques, although the resulting arterial blockages resolve spontaneously. In unstable angina, the level of heart damage is much less than occurs in a myocardial infarction, but unstable angina often foreshadows a subsequent myocardial infarction (Tan, 2014).

PROGNOSIS

Heart attacks are the cause of most deaths from coronary artery disease. Thirty percent of myocardial infarctions are fatal, with half of those fatalities occurring before the patient reaches the hospital. Of the patients with a myocardial infarction who get to a hospital alive, about 10% die in the hospital.

Patients who survive a heart attack have a 5% to 10% chance of dying within a year, and most of these fatalities occur in the first 4 months after their hospitalization. Larger areas of heart injury

lead to higher mortality rates. Approximately 50% of all patients with a myocardial infarction are rehospitalized within one year (Maziar Zafari, 2014).

Unstable angina is sometimes called preinfarction angina. About 30% of patients who develop unstable angina have a myocardial infarction within 3 months. Unstable angina has a mortality rate of about 4% per year.

RISK FACTORS AND PREVENTION MEASURES

Coronary artery disease creeps up quietly. In most patients, atherosclerosis builds over decades. Although it probably begins before most people are out of their teenage years, the coronary effects of atherosclerosis usually do not show up until middle age.

The most common symptom of coronary artery disease is chest pain, which can be accompanied by shortness of breath and tiredness, and these are the symptoms that often bring the patient with CAD to the doctor. However, CAD can be symptomless and "silent" for years. Even those patients who have been diagnosed with coronary artery disease because of occasional temporary chest discomfort can at the same time be suffering acute myocardial infarctions without apparent symptoms.

More than half of the patients who die suddenly from CAD have had no previous symptoms. Frequently, those patients who suffer from silent myocardial infarctions also have type 2 diabetes.

In spite of the variation in the overt signs and symptoms of coronary artery disease, there are some characteristics and risk factors present in most patients with the disease (Boudi, 2014b).

Nonpreventable Risk Factors

AGE

Age is the strongest risk factor for coronary artery disease; most cases occur in patients aged 40 years or older, although mortality and morbidity are higher in the elderly. More than 80% of people who die of CAD are aged 65 years or older (Boudi, 2014b). Elderly women who have heart attacks are more likely than men are to die from them within a few weeks (AHA, 2014a).

GENDER

Overall, CAD is slightly more common in men than in women; in the United States, 9.1% of men and 7.0% of women have the disease. Women tend to develop symptomatic coronary artery disease about 10 years later than men. In the United States, men over 40 years of age have a 49% chance of developing the disease in their lifetime, while the chance for women over the age of 40 years is 32% (Boudi, 2014b). It is thought that the higher estrogen levels in premenopausal

women protect them from some of the heart damage done by atherosclerosis, but this protection disappears after menopause.

RACE/HEREDITY

African Americans have a higher prevalence of, and a higher death rate from, CAD than European Americans. In part, the difference results from the higher incidence of hypertension, obesity, and metabolic syndrome among African Americans. This racial disparity is also thought to result from the fact that African Americans, on average, tend to seek treatment later than European Americans and are less likely to receive invasive treatment (Boudi, 2014b).

Americans of Asian Indian origin are 2 to 3 times as likely as European Americans to develop coronary artery disease (Boudi, 2014b). Heart disease risk is also higher among Mexican Americans, American Indians, native Hawaiians, and some Asian Americans. This may be due in part to higher rates of obesity and diabetes in these populations (AHA, 2014a).

Children of parents with heart disease are more likely to develop it themselves. African Americans tend to have more severe high blood pressure than Caucasians and a higher risk of heart disease. Most people with a strong family history of heart disease have one or more other contributing risk factors (AHA, 2014a).

Individuals with familial hypercholesterolemia, an inherited metabolic disorder affecting the LDL receptors, carry a genetic mutation that makes it difficult for their cells to remove LDL from their blood (McLaughlin, 2014).

Recent studies have shown that genetic predisposition is responsible for around 50% of the risks associated with CAD. Several genes may contribute to the predisposition, with each having only a mild to moderate influence on whether a person will get CAD purely from a genetic perspective. Over 50 genetic variants related to coronary artery disease have been identified and are currently being studied. Ongoing clinical trials are continuing to investigate how genes influence a person's risk for CAD (Roberts, 2014).

Preventable Risk Factors and Evidenced-Based Prevention Measures

SMOKING

People who smoke have a risk of developing CAD that is 2 to 4 times higher than that of nonsmokers. Nicotine causes the sympathetic nervous system to constrict arteries and raises blood pressure, causing arterial wall damage. The damage encourages the formation of atherosclerotic plaque.

Cigarette smoking is also an important independent risk factor for sudden cardiac death in patients with CAD. Cigarette smoking adds a cumulative effect when other risk factors are present to greatly increase the risk for CAD. People who smoke cigars or pipes seem to have a

higher risk of death from CAD as well. Exposure to second-hand smoke also increases the risk of heart disease for nonsmokers (AHA, 2014a).

Patients who smoke should be strongly encouraged to quit smoking. An important factor is to educate patients on the risks of smoking and offer assistance in developing an action plan to help the patient stop smoking. The best smoking cessation programs include a combination of the following components:

- Behavioral modification therapies
- Medications such as antidepressants
- Nicotine replacement strategies, such as patches or gum
  (McLaughlin, 2014)

HIGH CHOLESTEROL

As blood cholesterol rises, so does the risk of CAD. When other risk factors (e.g., hypertension and smoking) are present, this risk increases even more.

Low HDL cholesterol is a risk factor for heart disease. Likewise, a high triglyceride level combined with low HDL cholesterol or high LDL cholesterol is associated with atherosclerosis, which increases a person's risk for CAD.

Cholesterol level is affected by age, gender, heredity, and diet. Genetic factors, type 2 diabetes, and certain drugs, such as beta blockers and anabolic steroids, also lower HDL cholesterol levels. Smoking, being overweight, and being sedentary can all result in lower HDL cholesterol.

HYPERTENSION

Hypertension causes inflammation, which can damage the lining of arteries and increases fatty deposits contributing to the development of atherosclerosis and CAD. For people at increased risk for CAD, blood pressure control is an important factor. A diagnosis of hypertension is confirmed when two or more elevated blood pressure readings are obtained on separate visits.

CLASSIFYING BLOOD PRESSURE

Category	Systolic (mm/Hg)	Diastolic (mm/Hg)
Normal	<120	<80
Prehypertension	120–139	80–89
Stage I hypertension	140–159	90–99
Stage II hypertension	≥160	≥100

Source: Adapted from McLaughlin, 2014.

Treating hypertension is an important factor in preventing CAD and includes the following strategies:

- Lifestyle modifications, such as smoking cessation and dietary changes
- Medications to control blood pressure, such as beta blockers, calcium channel blockers, angiotensin receptor blockers, and thiazide diuretics

PHYSICAL INACTIVITY

A sedentary lifestyle is a risk factor for CAD. Patients with a sedentary lifestyle are also more likely to also be overweight or obese, which contributes to the risk developing CAD.

Patient goals for physical activity should begin with 10 to 15 minutes a day and gradually work up to a goal of 30 minutes a day of moderate to vigorous exercise. The more vigorous the activity, the greater the benefits. The level of activity should be based on the patient's baseline condition and other comorbid diseases. Patients should always work with their healthcare provider prior to starting an exercise program.

The cardiovascular benefits of exercise include a positive impact on:

- Lipid metabolism
- Blood pressure
- Insulin sensitivity
- Calories burned

(AHA, 2014a; McLaughlin, 2014)

Although a program of regular exercise does not typically reduce LDL cholesterol levels to a significant degree, it will reduce insulin resistance and blood levels of triglycerides, and it will increase blood levels of HDL cholesterol.

For patients who are just starting an exercise program, it is important to start slowly and consult a professional, such as an exercise physiologist, for assistance in developing a plan that will work for them. For high-risk patients with comorbidities who are deconditioned or have had recent cardiac events, careful supervision of physical rehabilitation is recommended. Referral to a physical therapist to evaluate, plan, and monitor the patient's progress with his or her exercise program is an important consideration.

OBESITY

Obesity increases the risk for heart disease by causing the heart to work harder, which leads to hypertension. With obesity, high blood cholesterol and triglyceride levels also increase, while HDL levels decrease. Obesity is defined as a body mass index (BMI) of 30 or greater. Patients who have a larger waist measurement than hip measurement are at increased risk for CAD

(McLaughlin, 2014). Obese patients are also at increased risk for developing metabolic syndrome and diabetes (AHA, 2014a).

Exercise alone rarely leads to significant weight loss. A reduced-calorie diet is usually necessary as well. Overweight people with CAD may need to reduce the total number of calories that they eat each day. Referral to a dietitian may be indicated to assist patients with meal planning and monitoring.

Even a modest weight loss makes a difference. Patients who are overweight should be encouraged to follow a comprehensive weight loss plan. A goal of achieving a 10% weight loss will lower a person's risk for CAD. A small but consistent weight loss of 1/2 to 2 pounds per week is the safest way to accomplish this (AHA, 2014a).

DIABETES

Diabetes is a strong risk factor for developing CAD. Even when glucose levels are under good control, diabetes increases the risk of heart disease and stroke. The risks are even greater if blood sugar is not well controlled. Around 65% of people with diabetes die of some form of heart or blood vessel disease (AHA, 2014a).

Patients with type 2 diabetes may have an increased risk of CAD because of disturbances in protein and fat metabolism, which may lead to weight problems. As a result, most patients with type 2 diabetes are overweight or obese. Maintaining a normal weight with diet and exercise as well as taking prescribed medications is important to maintain adequate blood sugar control (McLaughlin, 2014).

OTHER RISK FACTORS

Stress may be a contributing factor for developing CAD. For example, stress may cause people to overeat, start smoking, or smoke more than they otherwise would.

Alcohol is also a risk factor. Drinking too much alcohol can raise blood pressure and contribute to high triglycerides. However, the risk of heart disease in people who drink moderate amounts of alcohol is lower than in nondrinkers (AHA, 2014a).

Nutrition is also an important factor. Eating habits can affect other controllable risk factors such as cholesterol, blood pressure, diabetes, and weight. Evidence has shown that including a diet rich in vegetables, fruits, whole-grain and high-fiber foods, fish, lean protein, and fat-free or low-fat dairy products may lower a person's risk for developing CAD. The revised AHA guidelines place emphasis on foods and an overall eating pattern rather than on percentages of food components such as fat (AHA, 2014a).

MEDITERRANEAN DIET

The Mediterranean diet has been studied and shown to have a positive effect on heart health. The diet is characterized by:

- High intake of monounsaturated fatty acids, primarily from olives and olive oil
- Daily fruits, vegetables, whole grain cereals, and low-fat dairy products
- Weekly intake of fish, poultry, tree nuts, and legumes
- Lower intake of red meat, approximately twice a month
- Moderate daily consumption of alcohol, normally with meals

Large studies have reported positive results comparing the Mediterranean diet to low-fat diets in people at high risk for cardiovascular disease. Reduction of incidence of myocardial infarction, stroke, or CV death was shown in people following the Mediterranean diet (Ward, 2015). Adherence to the diet is associated with reduced HDL cholesterol and triglyceride levels. The Mediterranean diet can be adopted by all population groups and cultures and is cost-effective (Mohamad, 2014).

DAILY ASPIRIN THERAPY

Aspirin can be taken to prevent heart disease and stroke in some individuals. The U.S. Preventive Services Task Force (USPSTF) recommends that men with no history of heart disease or stroke aged 45 to 79 years use aspirin to prevent myocardial infarctions and that women with no history of heart disease or stroke aged 55 to 79 use aspirin to prevent stroke.

These recommendations apply only when the benefit of aspirin use outweighs the potential harm of gastrointestinal hemorrhage or other serious bleeding. Patients should always discuss aspirin use and dosage with their healthcare provider.

USPSTF, 2013.

ASSESSMENT, SCREENING, AND DIAGNOSIS

Chief Complaint: Chest Pain

Chest discomfort is a key identifying symptom of coronary artery disease, particularly in men. When a man with coronary artery disease comes to the office, clinic, or hospital with heart symptoms, the typical chief complaint is chest discomfort. Most often, the patient does not describe this discomfort as pain but instead as heaviness, pressure, squeezing, smothering, or a choking sensation.

By contrast, a woman with coronary artery disease is more likely to complain of nausea or abdominal discomfort; neck, throat, or jaw pain; shortness of breath; or weakness or fatigue rather than the more classic symptom of chest pain. Coronary ischemia should therefore be considered in women who appear to be acutely ill even if they do not complain of chest pain (Charney, 2011; McLaughlin, 2014).

ANGINAL PAIN

Chest discomfort or chest pain can originate from many places other than the heart, but the characteristic pain of angina almost always points to ischemia of heart muscles (McLaughlin, 2014).

Classic symptoms associated with angina include:

- Chest pain or discomfort
- Pain in arms, neck, jaw, shoulder, or back accompanying chest pain
- Nausea
- Fatigue
- Shortness of breath
- Sweating
- Dizziness

(Mayo Clinic, 2015)

Specific questions will help a healthcare provider assess whether a patient with chest pain has angina. These include the following:

- Onset: "When did you first notice the pain?"
- Provocation: "What activities bring on the pain or make the pain worse?"
- Quality: "Describe what the pain is like?"
- Location/Radiation: "Where is the pain located?" "Does the pain radiate anywhere?"

(Humphreys, 2011)

Onset and Provocation

Anginal pain is caused when heart muscle does not get enough oxygen. Most activities have fairly predictable oxygen requirements, and with stable angina, the patient gets chest discomfort at predictable levels of activity. In contrast, in unstable angina, people get chest discomfort at rest and at unpredictable times.

Any situation that increases heart rate can trigger angina in people with coronary artery disease. Exercise is a classic cause of anginal pain: hurrying, walking up an incline, walking against a strong cold wind, working with the arms extended above the shoulders, and sexual activity are all exercises that can produce ischemic heart pain. In addition,

strong emotions or nightmares activate the heart through the sympathetic nervous system, and these too can cause angina.

In the case of stable angina, although the amount of exertion needed to produce chest pain is fairly predictable, the threshold for angina will vary during the day and with the weather and temperature. After a heavy meal, for example, blood flow is diverted to the gastrointestinal organs and, under these conditions, less exertion than usual can cause angina. Lying down changes the dynamics of blood flow, and some people get angina when they get in bed at night. Women with chronic stable angina are more likely than men to get chest discomfort when they are resting or sleeping or when they are in stressful situations.

Other medical conditions can precipitate angina in a person with coronary artery disease. For example, anemia, systemic infections, pneumonia, or atrial fibrillation change the balance between the heart's need for oxygen and the available supply; therefore, these can bring on ischemic heart pain.

Predictable triggers are characteristic of coronary artery disease with narrowed arteries (stable angina). Acute coronary syndromes are different. When coronary artery disease produces a sudden significant obstruction of blood flow, the chest pain can occur without any apparent trigger. Unstable angina, for example, is characterized by worsening and unpredictable episodes of chest pain.

Time Course

The chest discomfort of stable angina typically lasts from 2 to 5 minutes; it rarely persists for as long as 10 minutes. The angina begins dully and progressively worsens for a minute or two, and then it fades away as the patient stops and rests. (Lying down does not always relieve the pain of angina as quickly.) Nitroglycerin tablets or sprays will usually end or lessen angina in a few minutes.

In contrast, the angina of acute coronary syndromes lasts for more than 10 minutes, and with myocardial infarctions, the pain can last for hours. When rest does not relieve classic anginal pain, then it is more likely that the patient has an acute coronary syndrome.

Quick (10 to 15 seconds), sharp chest pains are rarely ischemic heart pains. Also, a long, steady, dull ache in the lower left chest is rarely ischemic heart pain.

Quality

The quality or sensation of angina has a special character. The word *angina* means "choking, narrowing, or tightening," and this is typically how it is described. Rather than saying "pain," patients most often use words such as squeezing, tightening, constricting, pressing, or strangling to describe the feeling of heart ischemia. They may say that they feel like there is "a band across my chest," "a heavy weight in the center of my chest," or "a vise that is tightening my chest."

As patients describe the uncomfortable feeling of angina, they often clench their fist and hold it in front of their sternum. This gesture is so common that it has been given a name, **Levine's sign.**

Sometimes, a patient with chronic stable angina will describe angina in more painful terms, such as "burning," but when patients give descriptions of true pain, it is often because the underlying ischemia is caused by a complete blockage of an artery, as is found in a myocardial infarction. Even then, anginal pain is described as diffuse; it is rarely described as sharp and localized.

Location

When asked, "Where do you get this uncomfortable feeling?" patients with angina usually put a hand or fist over their sternum in the middle of their chests and say "Inside here!" meaning retrosternally. When asked, "Does this discomfort extend anywhere else?" angina patients will often say that the feeling extends to the left shoulder, to the inside (the ulnar) half of either or both arms, to the neck and jaw, or sometimes to the middle of the upper back. Additionally, women with angina may complain of pain or discomfort in the abdominal area.

The pain or discomfort of angina is broad, and patients do not point to it with a finger, saying "It's right here." Also, patients rarely feel angina above the jaw, below the umbilicus, in the lower right chest, or localized below the left nipple. Moreover, the examiner usually cannot reproduce the pain by pushing gently on the skin or the chest wall.

History

In addition to a description of individual occurrences of angina, the overall history of these episodes is telling. Chronic stable angina gives fairly predictable episodes of chest discomfort over many months, although the exact pattern of the episodes differs from patient to patient. In some patients, episodes of chest pain may occur several times a day; in others, there may be symptom-free intervals of weeks, months, or years. Occasionally, anginal attacks gradually decrease or disappear if adequate collateral coronary circulation (i.e., growth of new blood vessels) develops; this does not mean, however, that the disease has gone away.

In contrast, acute coronary syndromes give unpredictable or steadily worsening episodes of ischemic symptoms. As acute coronary syndromes are developing, the symptoms may change from being occasional to happening constantly. A myocardial infarction may give prolonged severe chest discomfort and continuous fatigue.

The chest discomfort of chronic stable angina is predictable for a given patient. Therefore, any changes in the pattern or the intensity of angina should be considered serious (Mayo Clinic, 2015).

ANGINAL PAIN RATING SYSTEM

People vary in how they report angina. To compare the symptoms of different patients with coronary artery disease, the New York Heart Association has developed a rating system for classifying anginal pain. In this system, patients with known coronary artery disease and angina are put in one of four categories or classes according to their physical limitations. In brief, the classes are:

- **Class I.** Patient can do almost any daily physical activity without getting cardiac symptoms. Angina only occurs with strenuous or prolonged exertion.
- **Class II.** Patient has slight limitation of physical activity, as certain normal active exercise brings on symptoms. For example, walking or climbing one flight of stairs may be symptom-free, but running or climbing many flights of stairs will bring on cardiac symptoms.
- **Class III.** Patient has no symptoms at rest and can perform many activities of daily living without symptoms, but mild activity can bring on symptoms. For example, dressing, showering, and walking slowly for a short distance may be symptom-free, but carrying shopping bags, climbing stairs, and walking for 1 to 2 blocks will bring on cardiac symptoms.
- **Class IV.** Patient sometimes has cardiac symptoms at rest, and basic activities of daily living will bring on cardiac symptoms.

Source: Adapted from McLaughlin, 2014.

OTHER CAUSES OF CHEST PAIN

Chest discomfort is a classic symptom of myocardial ischemia. It is also a key symptom of other medical problems, the most common of which are gastroesophageal diseases (gastroesophageal reflux, other esophageal problems, ulcer disease, gallbladder disease) (McLaughlin, 2014).

CAUSES OF CHEST PAIN

Origin	Causes
Cardiovascular	• Aortic aneurysm • Aortic dissection • Myocardial ischemia • Pericarditis
Pulmonary	• Pneumonia • Pneumothorax • Pulmonary embolism

Musculoskeletal	• Chest wall injury • Costochondritis • Herniated intervertebral disc • Spinal arthritis
Gastrointestinal	• Esophageal spasm • Esophageal tear (Mallory-Weiss) • Esophagitis • Gallbladder disease • Pancreatitis • Ulcer disease
Infectious	• Herpes zoster
Neurologic	• Panic attack

Other Symptoms of CAD

In addition to chest pain, other symptoms are frequently caused by myocardial ischemia. These symptoms are sometimes called "anginal equivalents," and they include:

- Shortness of breath, especially when it feels localized to the middle of the chest
- Weakness and tiredness
- Faintness or dizziness

These three symptoms are especially common in older (age >75 years) patients and in diabetic patients when they have episodes of heart ischemia.

Other general anginal equivalents are:

- No chest pain, but discomfort in the shoulders, inside (ulnar side) of the left arm, neck, or lower jaw
- Indigestion or nausea

When accompanying angina, certain additional symptoms signal potential emergencies. For example, chest pain with sweating and nausea or vomiting suggests myocardial infarction.

WOMEN AND MYOCARDIAL ISCHEMIA SYMPTOMS

Healthcare professionals should be alert to the fact that women are more likely than men to present with the following as the primary symptoms of an acute coronary syndrome:

- Dyspnea
- Gastrointestinal complaints (nausea and vomiting)
- Back pain or pressure

- Jaw pain
- Shortness of breath
- Fatigue

(AHA, 2014b; McLaughlin, 2014)

Women also are more likely to attribute cardiac symptoms to other causes (such as the flu, stress, and normal aging) and may delay reporting symptoms (AHA, 2014b).

SILENT HEART ATTACKS

Not all patients with myocardial ischemia have symptoms. Angina is a very common indicator of myocardial ischemia, and the characteristics described above are frequent and typical. Nonetheless, patients with all forms of CAD can have atypical feelings of chest discomfort or anginal equivalents. Moreover, ischemia severe enough to cause myocardial infarctions can occur without any chest pain, giving what are called silent heart attacks (asymptomatic myocardial ischemia).

CASE

Claire Brown is a 62-year-old female with a family history of CAD and a previous history of smoking (20 years in total). Claire has arrived to the emergency department with complaints of anxiety, dizziness, weakness, and ongoing fatigue.

The nurse on duty is Robert, who questions Mrs. Brown about her current medications, which include Zocor, Atenolol, and Xanax. As Robert continues to triage Mrs. Brown, he is initially concerned that she may be having an anxiety attack since she also seems short of breath and perspiring.

As Robert considers this case, he is concerned about Mrs. Brown's symptoms. In order to uncover more, he asks probing questions about any pain that she is noticing. Mrs. Brown reports that she has been having some pain in her jaw and thought it was a tooth problem and that she has had more heartburn lately and felt nauseous with cold sweats.

She tells Robert that she called into her local doctor with her concerns a week ago and was instructed to continue to take her Xanax and start taking an over-the-counter antacid to help with the heartburn symptoms. Mrs. Brown states that "she originally thought she might have the flu, but now she knows that there is something wrong and that she has had the symptoms for a couple of weeks."

Even though this patient has a few classic symptoms of an anxiety attack, Robert recognizes that Mrs. Brown may be having cardiac symptoms, as women do not always present with obvious complaints of chest pain. Robert discusses the case with the ED physician. Together, they continue to question Mrs. Brown about her symptoms. When asked if she had tried

anything to relieve her symptoms, the patient stated that she has been taking Tylenol for the jaw pain and antacids for the heartburn symptoms.

The ED team proceeds with a cardiac evaluation along with other testing in order to rule out myocardial ischemia. Mrs. Brown's evaluation shows that she is experiencing angina pain. She is admitted for continuing monitoring and medical management.

Patient History

MEDICAL HISTORY

The medical history of patients with coronary artery disease may suggest that they have or are at high risk for atherosclerosis. The primary elements in a person's medical history that should alert the healthcare provider to the possibility of an increased risk for atherosclerotic CAD include:

- High blood cholesterol
- High blood pressure
- Diabetes
- Metabolic syndrome
- Family history of CAD
- Smoking
- Fatty diet

Ischemia

When taking a medical history, the healthcare provider may find that atherosclerosis of the coronary arteries has already revealed itself. A patient with CAD may already have had episodes of heart ischemia, such as myocardial infarctions.

Peripheral Artery Disease

Atherosclerosis is a whole-body disease. Patients with coronary artery disease will often have indications of atherosclerosis in arteries outside the heart. For example, they may have a history of intermittent claudication (a result of atherosclerosis in the leg arteries), strokes or transient ischemic attacks (results of atherosclerosis in the carotid arteries), or abdominal aortic aneurysms (results of atherosclerosis in the aorta).

Lipid Abnormalities

High levels of blood lipids predispose a patient to atherosclerosis. Specifically, high levels of LDL cholesterol can cause atherosclerosis, and a patient with CAD may already have a diagnosis of high cholesterol.

Hypertension

High blood pressure is another major risk factor for developing atherosclerosis. For example, middle-aged men with blood pressures >169/95 are 5 times more likely to have atherosclerotic heart disease than middle-aged men without high blood pressures (<140/90). A patient with CAD may already be taking antihypertensive medicines.

Diabetes

Diabetes puts a patient at high risk of developing coronary artery disease. Diabetes—especially type 2 diabetes—tends to increase the level of blood cholesterol and to worsen atherosclerosis. Nearly 70% of people with diabetes die from some form of cardiovascular disease, and people with diabetes are 2 to 4 times more likely to have heart disease or a stroke than people without diabetes. Patients who have undiagnosed or poorly controlled diabetes are at highest risk (AHA, 2014a).

Metabolic Syndrome

Metabolic syndrome is the name for a cluster of health conditions that are frequently found together. The core problems are obesity and insulin resistance, and the additional three problems are high blood pressure, high levels of triglycerides, and low levels of HDL cholesterol. Having metabolic syndrome increases a patient's risk of developing type 2 diabetes and also puts a person at high risk of developing serious atherosclerotic vascular disease with coronary artery blockage (McLaughlin, 2014).

FAMILY HISTORY

Patients are much more likely to develop coronary artery disease if they inherit a genetic propensity for the disease. When assessing a patient for CAD, a good indicator of this propensity is the existence of close (first-degree) relatives who have had an acute coronary syndrome, such as a heart attack, at an early age. For men, this would be when they were younger than 45 years, and for women, it would be when they were younger than 55 years.

SOCIAL HISTORY

Two features of a patient's lifestyle may put them at high risk for developing CAD: smoking and a high-fat diet. Assessment should include taking a careful history of current or previous smoking as well as asking about dietary habits.

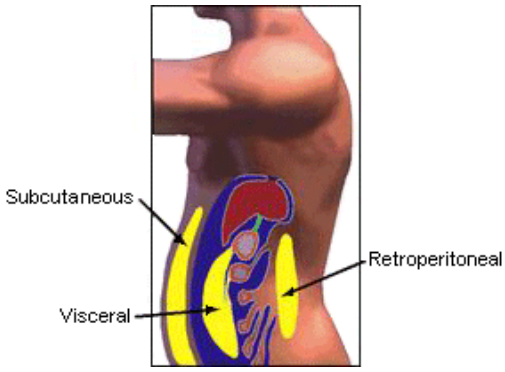
Smoking one or more packs of cigarettes a day for several years doubles a person's chance of dying from CAD; however, a person who stops smoking can reduce this extra risk. Likewise, a diet high in cholesterol, saturated fats, and trans fats increases a patient's chances of developing artery problems from atherosclerosis, while low-fat diets may reduce the risk.

Physical Exam Components

A patient with CAD who presents to the emergency department with serious cardiac symptoms can show many abnormalities on physical examination. On the other hand, a patient with CAD who comes to the clinic or office for a check-up may have only a few signs of the underlying disease. During a routine physical examination, the following findings would fit with a diagnosis of CAD (Boudi, 2014b).

WEIGHT

Patients with excess intra-abdominal or visceral fat (an "apple-shaped" build) are more likely to have atherosclerotic cardiovascular disease. Waist circumference is a good measure of intra-abdominal fat content: a waist circumference >102 cm (>40 inches) in men or >88 cm (>35 inches) in women is in the high-risk range (Mayo Clinic, 2014a).



Subcutaneous fat lies just under the skin. Visceral fat is inside the abdomen, surrounding the abdominal organs. Visceral fat is more likely to contribute to coronary artery disease than is subcutaneous fat. (Source: NHLBI.)

VITAL SIGNS

During a routine office visit, the pulse may have a normal rate and rhythm in a patient with coronary artery disease. Tachycardia is common, however, when a patient is suffering from an episode of myocardial ischemia. Bradycardia during an acute coronary syndrome can be an ominous sign.

Patients with CAD often have hypertension (BP ≥140/90), and the higher the blood pressure, the greater the risk of heart disease. Hypotension during a myocardial infarction is an ominous sign.

The respiration rate is usually normal in a routine office visit, however patients will breathe more rapidly under the stress of heart ischemia.

SKIN

No unusual sweating is expected on a routine office visit, but acute coronary syndromes, especially myocardial infarctions, are often accompanied by profuse sweating (diaphoresis).

Smoking is a strong risk factor for atherosclerosis, and patients with CAD may have nicotine stains on their fingers or teeth.

HEAD AND NECK

The blood vessels of the retina may show the effects of hypertension or atherosclerosis (i.e., widened light reflections from the arteries, copper- or silver-colored arteries, white sheaths along the arteries, venous tapering or "nicking" at arterial-venous crossings, hemorrhages, or papilledema). Diabetes, which worsens coronary artery disease, produces a characteristic retinopathy.

Atherosclerotic plaque can produce local blood turbulence, which will sometimes give a murmur or bruit that can be heard when listening to the carotid arteries.

THORAX

The pain of heart ischemia is usually diffuse and "somewhere inside." If a patient's chest pain can be reproduced by the examiner pressing on some point along the chest wall, the pain is unlikely to be angina. (In some patients with myocardial infarctions, however, broad regions of the chest become tender.)

On a routine exam, the lungs of a patient with coronary artery disease can be clear and unremarkable. With myocardial infarction, on the other hand, the patient may be breathing rapidly and may complain of shortness of breath. When ischemia has brought on some degree of heart failure, valve dysfunction, or arrhythmia, patients can have fluid in their lungs and rales can be heard.

A routine physical exam of a patient with CAD may find no overt heart problems. If the patient has a history of ischemic episodes, however, there may be a number of findings. Previous heart surgeries will have left chest scars. Hypertension or heart failure may have enlarged the heart. Murmurs suggest valve or papillary muscle damage, and gallops suggest heart wall damage. In addition, an ischemic heart is more susceptible to arrhythmias.

ABDOMEN

An abdominal aortic aneurysm usually indicates atherosclerosis. Likewise, bruits from other major abdominal arteries, such as the renal arteries, can be due to atherosclerosis.

EXTREMETIES

Leg edema may be from heart failure due to chronic ischemic heart disease. Atherosclerosis can give weakened peripheral pulses. Diabetes can produce neuropathies, which show up as a decrease in the patient's ability to sense stimuli in the feet.

Laboratory Studies

A patient being evaluated for CAD should be given a number of laboratory tests. Certain tests are especially helpful in assessing a patient's risk of serious heart damage from atherosclerosis. These include blood tests of lipid levels, complete blood count, fasting glucose levels, A1C, creatinine levels, and the possible presence of cardiac markers, which are indicators of recent heart cell damage (Humphreys, 2011; Boudi, 2012).

BLOOD LIPIDS

High serum cholesterol levels markedly increase a patient's risk for developing atherosclerosis-induced heart injury. The LDL fraction of cholesterol is the specific culprit. The box below shows both healthy and unhealthy fasting blood lipid levels. Patients with CAD often have one or more lipid levels in the unhealthy range (McLaughlin, 2014).

FASTING BLOOD LIPID LEVELS

Lipids	Optimal Levels	Unhealthy Levels
Total cholesterol	<200 mg/dl	>240 mg/dl
HDL cholesterol	≥60 mg/dl	<40 mg/dl for men <50 mg/dl for women
LDL cholesterol	<100 mg/dl	>160 mg/dl
Triglycerides	<150 mg/dl	>200 mg/dl

FASTING PLASMA GLUCOSE

Patients with diabetes have a higher than normal chance of developing CAD. Diabetes will manifest as a fasting plasma glucose level of ≥126 mg/dl when measured on at least two different days.

SERUM CREATININE

Renal disease worsens atherosclerosis. The level of creatinine in a patient's blood can be used to screen for a number of kidney problems.

CARDIAC MARKERS

When heart muscle is damaged, intracellular molecules leak into the bloodstream. After a myocardial infarction, specific heart proteins (cardiac markers) can be detected in a patient's blood within hours and then for many days afterward. The standard cardiac markers are the cardiac troponin molecules. Other commonly measured proteins are the creatine kinase molecules. Cardiac markers are used for diagnosing and following emergency cardiac events and are not measured at routine checkups for coronary artery disease.

CARDIAC MARKERS		
Marker	**Normal Levels**	**Duration of Elevation after MI**
Creatinine kinase (CK)	• 38–190 units/L (men) • 10–150 units/L (women)	Peaks at 12 to 18 hours; remains elevated up to 72 hours.
Myoglobin	0.0–0.09 mcg/ml	Peaks within 6 to 7 hours; returns to baseline within 24 hours.
Troponin I	<0.1 mcg/ml	Peaks within 14 to 20 hours; returns to baseline in 10 to 15 days.
Source: Adapted from McLaughlin, 2014.		

Electrocardiogram (ECG)

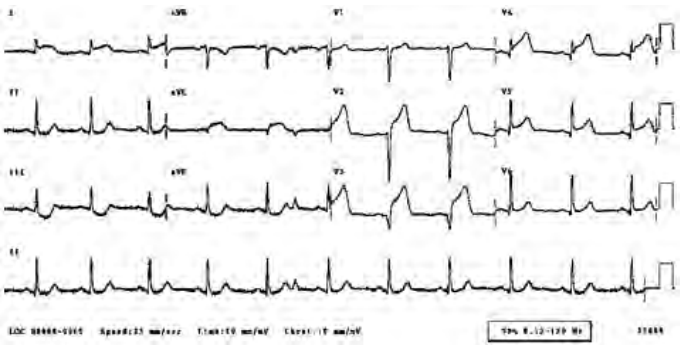
Twelve-lead **electrocardiography** (ECG or EKG) is the standard method for identifying arrhythmias and conduction problems. In terms of coronary artery disease, the ECG is a quick, accurate, and noninvasive way to detect myocardial injury, ischemia, and the presence of prior myocardial infarction (Levine, 2013).

An acute coronary syndrome changes the electrical properties of a region of heart muscle, and these changes can be seen in the ECG. The location of the ischemic heart region can often be identified by the particular segments of the wave pattern that have changed. The segments of the electrical wave pattern produced during a heartbeat have been named, and changes in the ST segment and the T wave are the clearest indicators of a myocardial infarction.

About one quarter of patients with stable angina will have a normal ECG wave pattern when they are resting. To determine the degree of heart ischemia that a patient with chronic stable angina suffers when the heart is stressed, an ECG can be taken while the patient exercises—typically, walking on a treadmill or pedaling a bicycle. However, not all patients with CAD show ECG changes during stress testing.

CASE

Joanne Lang is a 63-year-old woman with a history of stable angina and heart valve disease. She is admitted to the emergency department complaining of new chest pain, shortness of breath, sweating, and chest tightness. The ED nurse takes her vital signs, which yield a blood pressure reading of 140/90 mm Hg and a heart rate of 98 beats/min. Electrocardiography is administered; Joanne's electrocardiogram appears at the right. The ED nurse suspects acute anterior myocardial infarction and refers Joanne for further evaluation by the attending cardiologist.



(Source: ECG Library, 2014.)

Stress Testing

Stress testing directly assesses the ability of a patient's heart to cope with exercise. A stress test is a controlled way to increase the workload of the heart, and stress tests are used to find the threshold beyond which coronary arteries cannot supply sufficient blood to meet the heart's oxygen needs. The lower the threshold (i.e., the smaller the stress) at which symptoms appear, the worse the patient's coronary artery disease.

Stress tests can confirm that a patient's complaint of chest discomfort is actually anginal pain. The tests can also establish the level of activity that brings on chest discomfort. Subsequent stress tests can objectively monitor both the progression of the CAD and the efficacy of treatments (Akinpelu, 2014).

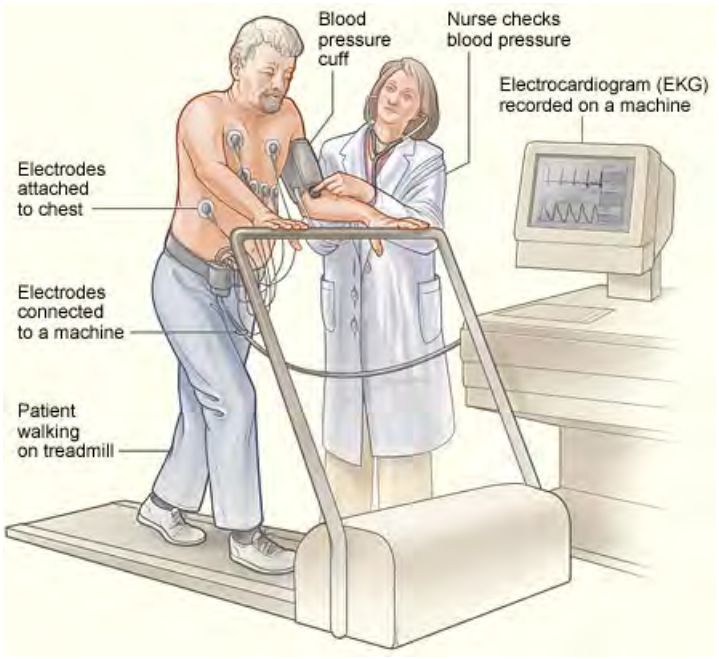
EXERCISE STRESS TESTING

The preferred heart stressor is graded exercise, either walking on a treadmill or pedaling a stationary bicycle.

As part of exercise stress testing, patients should be instructed as follows:

- To withhold certain medications prior to the test (e.g., beta blockers may limit the patient's ability to increase heart rate during the test)
- To wear comfortable clothing and shoes for the test
- About procedures that will take place before, during, and after the test
- That they may need to have a thallium injection during the test to evaluate coronary blood flow
- To immediately report any chest pain, leg pain, shortness of breath, or fatigue during the testing

Monitoring of the patient's ECG and blood pressure are necessary during and after the test until the patient is back to a baseline state (McLaughlin, 2014).



Stress testing uses graded exercise in a supervised session to assess the heart's response to increases in its workload. (Source: NHLBI, 2011b.)

During a stress test, symptoms of heart problems—angina, shortness of breath, severe fatigue, lightheadedness, or fainting—usually appear when patients go beyond their exercise threshold. At the same threshold, signs of heart problems—gallops, arrhythmias, hypotension, inappropriate increases or decreases in heart rate, pulmonary rales, or cyanosis—will also appear.

In addition to watching for these symptoms and signs of cardiac problems, the stress test supervisor will use more objective monitoring. The typical objective monitor is an ECG, which shows the rate and rhythm of the heart's electrical wave pattern, and echocardiography can be used to follow changes in the heart's anatomy during exercise.

ECG stress testing is most useful in the following clinical scenarios:

- Trying to make a diagnosis of coronary artery disease in an unclear case
- Measuring the exercise limitations imposed by a patient's coronary artery disease

Approximately one fifth of ECG stress tests give false positives, so the test is not recommended for routine examinations of people who are not likely to have coronary artery disease. At the other end of the spectrum, a similar percent of ECG stress tests give false negatives, and an ECG stress test that appears normal cannot be used to discard an otherwise convincing diagnosis of CAD.

DRUG-INDUCED STRESS TESTING

When patients cannot tolerate exercise, their heart can be stressed with a vasodilator drug such as dipyridamole or adenosine in a monitored and controlled setting. A physician needs to be present at all stress tests, and the tests need to be tailored to the individual patient's health.

Dipyridamole (Persantine) or dobutamine is administered intravenously. Depending on the patient's history, thallium or sestamibi (a radioactive tracer) may also be administered with the stress test. The drugs will stimulate the heart to react as if the patient is exercising.

The tracer drugs travel through the bloodstream to the heart, where they are picked up by the muscle cells. The areas of the heart that lack adequate blood supply pick up the tracer very slowly or not at all. Baseline images are compared with images taken 3 to 4 hours later. A cardiologist will determine if areas of the heart have suffered permanent damage from a previous ischemia (McLaughlin, 2014).

Imaging Tests

Pictures of the heart and the coronary arteries can be obtained in a variety of ways. The least invasive techniques are chest X-rays and echocardiograms. Another technique, coronary arteriography, produces excellent views of the coronary arteries, but it is an invasive procedure using arterial catheters.

CHEST X-RAYS

A chest X-ray shows the size and shape of the heart and the condition of the lungs. Patients with coronary artery disease can have normal chest X-rays, and usually chest X-rays do not help to diagnose CAD. Sometimes, however, chest films will show consequences of the disease, such as heart enlargement, aortic aneurysms, aortic dissections, or pulmonary signs of heart failure.

ECHOCARDIOGRAPHY

An echocardiogram uses ultrasound technology to show the size and thickness of the atria and ventricles of the heart, and it also shows the heart valves in action. Used during stress testing for coronary artery disease, echocardiography can show which heart walls or valves are most affected by ischemic episodes. Echocardiographic stress tests are not recommended as screening tools, but many doctors use these tests to confirm a clinical diagnosis of CAD in unclear cases.

CORONARY ARTERIOGRAPHY

Coronary arteriography (also called coronary angiography or cardiac catheterization) uses X-rays to follow dye injected into the heart or the coronary arteries. Coronary arteriography gives as definitive a diagnosis of arterial narrowing and blockage as is possible without major surgery.

Nonetheless, its high cost, mortality rate (about 0.1%), and morbidity rate (1%–5%) limit its use as a routine diagnostic tool. Currently, coronary arteriography is most often used in CAD patients when preparing them for possible bypass grafts or other heart operations. Coronary arteriography is also used when other tests cannot determine the cause of debilitating cardiac symptoms of ischemia.

NUCLEAR IMAGING STUDIES

Nuclear imaging involves the use of small amounts of radioactive material known as radiopharmaceuticals or radiotracers to diagnose and assess the severity of various diseases. The radiopharmaceutical is either injected into the body, swallowed, or inhaled as a gas and eventually accumulates in the part of the body being examined. A special camera or imaging device is used to detect radioactive emissions from the radiopharmaceutical, yielding pictures and detailed molecular information (RadiologyInfo.org, 2014a).

In coronary artery disease, nuclear imaging studies can be useful in assessing patients for significant **stenoses** (narrowing of coronary arteries). In this setting, stress and rest nuclear scintigraphic studies using the radiopharmaceuticals thallium or sestamibi (Cardiolite) are occasionally helpful. A nuclear imaging examination can be administered as a treadmill nuclear stress test, a dobutamine nuclear stress test, or a dipyridamole (Persantine) or adenosine nuclear stress test (Boudi, 2014b).

Radionuclide stress myocardial perfusion imaging using thallium-201 (^{201}Tl) or sestamibi can be used to quantify coronary flow reserve, which is usually assessed during exercise or with pharmacologic coronary vasodilators (Boudi, 2014b).

OTHER STUDIES

Computed tomography, also known as CT scanning, is another common imaging tool in coronary artery disease. **Coronary calcium scoring** involves administration of a cardiac CT scan to collect information about the presence, location, and extent of calcified plaque in the coronary arteries. Because calcium is a marker for CAD, the coronary calcium score—a number reflecting the degree and extent of calcium deposits in the walls of the coronary arteries—can be a useful prognostic tool in coronary artery disease (RadiologyInfo.org, 2014b).

Magnetic resonance angiography (MRA) uses MRI technology combined with injected contrast dye to check for areas of narrowing or blockages in the coronary arteries. This technology is not as precise as coronary arteriography.

MANAGEMENT OF ACUTE CAD

Patients with CAD may have mild symptoms (stable angina) that can be monitored and treated over time. Other patients may present with chest pain, dyspnea, profuse sweating, extreme fatigue, or other acute symptoms that may need to be seen in an emergency department and evaluated immediately.

Emergency Treatment

Emergency treatment for patients with CAD can be guided by the American Heart Association's "chain of survival," a series of actions that, when put into motion, can reduce the patient's chance of dying from cardiac arrest. The links in the **chain of survival** are:

1. Immediate recognition of cardiac arrest and activation of the emergency response system (call 911)
2. Early CPR with an emphasis on chest compressions
3. Rapid defibrillation
4. Effective advanced life support
5. Integrated post–cardiac arrest care
   (AHA, 2014c)

Unless patients have already been diagnosed with stable angina and recognize that they are having a typical short-lived anginal attack, they should **call 911 and be transported quickly by emergency responders** to an emergency department whenever experiencing an episode of chest pain.

BEFORE THE HOSPITAL

Because quick treatment of a heart attack is so beneficial, bystanders should start cardiopulmonary resuscitation (CPR) as soon as they see someone collapse, call 911, and use an automated external defibrillator (AED) if one is available (John & Ewy, 2011).

Emergency response professionals (EMT and/or nurses) who encounter patients experiencing chest pain or a sudden onset of dyspnea should treat the symptoms as myocardial ischemia and begin active interventions.

- The patient should be assessed and stabilized.
- If available, an intravenous (IV) access line and a pulse oximetry monitor should be placed.
- A conscious patient should chew and swallow 325 mg of aspirin, unless they have an allergy to aspirin or have active signs or recent history of gastrointestinal bleeding.

- If chest pain continues, the patient can be given sublingual nitroglycerin (patients with existing angina may have nitroglycerin on hand).
- Supplemental oxygen can be provided.
  (McLaughlin, 2014)

IN THE EMERGENCY DEPARTMENT

Triage

When a patient experiences ischemic heart symptoms, it is a potential life-threatening emergency. Triage of patients by the medical team with acute chest pain in the ED includes the following assessment steps coordinated by the triage physician:

1. The triage physician will assess for and reverse circulatory system failure and respiratory insufficiency. Then, when the patient has been clinically stabilized, go to step 2.
2. Next, the patient will be assessed for immediate life-threatening conditions (medical crises associated with chest pain):
   - Cardiovascular
     - Acute, massive myocardial infarction
     - Pulmonary embolism
     - Aortic dissection
     - Cardiac tamponade
   - Pulmonary
     - Pulmonary embolus
     - Tension pneumothorax
3. Working together with the triage physician, nurses' responsibilities may include assessing the patient's complaints, symptoms, and vital signs. Patients presenting with the complaint of chest pain should be evaluated with a focused history and physical examination conducted by a physician. Chest pain typically requires additional diagnostic testing including ECG and blood tests for cardiac markers of ischemic heart injury.

   Meanwhile, the ED nurse monitors the patient's basic vital signs at regular intervals to watch for the development of any medical crises.

   Older patients (age >75 years), patients with diabetes, and female patients are more likely to present with the sudden onset of dyspnea and fatigue as the primary symptom of an acute coronary syndrome, and new dyspnea can be the equivalent of chest pain in these individuals (AHA, 2014b).

Evaluation of Stabilized Patients

After stabilizing patients, a triage protocol for chest pain/sudden dyspnea should be implemented.

It is important for the team of providers in the ED to remember that one third of people with acute myocardial infarction do not mention chest pain as their chief complaint. Many patients are more likely to describe other symptoms as their primary complaint, even when they are suffering a heart attack.

Atypical presentations tend to come from patients with diabetes, older adults, women, patients of nonwhite ethnicities, and patients with dementia. Besides dyspnea, atypical symptoms include nausea; profuse sweating; fainting; and pain in the neck, shoulder, arms, or upper abdomen (McLaughlin, 2014).

To begin the medical evaluation of a patient with stable chest pain, the triage physician working with a stabilized adult patient may order an immediate 12-lead ECG to look for STEMI (see box below). It is thought that patients with STEMIs usually have a completely blocked artery, whereas patients whose infarctions do not produce ST-elevations have an incompletely blocked artery (AHA, 2013).

ST ELEVATION MYOCARDIAL INFARCTIONS (STEMIs)

Fast treatment gives the best outcome for all myocardial infarctions. In addition, certain types of myocardial infarction will benefit dramatically from quick reperfusion therapies, i.e., drugs and other techniques that open the blocked arteries and that restore blood flow. These myocardial infarctions can usually be identified on an ECG by the abnormal elevation of the ST segments of the heartbeat wave forms, and they are called ST elevation myocardial infarctions (STEMIs).

The chances of a patient dying from a STEMI heart attack can be decreased by about half if the blocked arteries are reopened in the first 1-1/2 hours after the symptoms begin (AHA, 2013). Quick reperfusion therapy will also reduce the amount of permanent muscle damage resulting from a STEMI. (Heart damage does not happen all at once after the blockage of a coronary artery; a myocardial infarct continues to enlarge over 5 to 6 hours if the blockage is not reduced or removed.)

For these reasons, the American Heart Association criteria recommend that emergency rooms aim for reperfusion within 90 minutes of the first STEMI symptoms, with an emphasis of treating STEMI patients within 90 minutes of presentation at the hospital (AHA, 2013).

When STEMI is identified, a reperfusion plan should be formulated for the patient by the triage team. The two major choices for reopening a blocked artery are pharmacologic and mechanical. The pharmacologic option is administration of a fibrinolytic drug

(streptokinase, alteplase, or reteplase), unless contraindications exist, to weaken and disrupt the damaging clot (McLaughlin, 2014).

The mechanical option consists of a percutaneous coronary intervention (PCI, also known as PTCA), meaning balloon angioplasty, with or without the placement of a stent, to break up or remove the clot.

Distinguishing STEMI from non-STEMI infarctions is important. Either type of reperfusion technique will benefit STEMI patients when done quickly. Patients without the characteristic ECG changes of STEMI may have either non-STEMI infarction or unstable angina.

Additional diagnostic tests coordinated by the triage physician for all chest pain/sudden dyspnea patients include a chest X-ray. As the patient is evaluated, the triage nurse continuously makes assessments for changes in symptoms, vital signs, and blood oxygen levels, since these are important indicators of a worsening medical condition. Electrocardiograms may also be repeated.

Stabilized patients who are unlikely to have an acute coronary syndrome still need to be evaluated for the cause of their chest pain. The triage physician conducts a thorough patient history to uncover underlying causes for chest pain. Among the causes that should be considered include pneumonia, pulmonary embolism, pneumothorax, pericarditis, rib fracture, costochondral separation, esophageal spasm, aortic dissection, renal calculus, splenic infarction, abdominal disorders, or chest injuries.

CASE

Nelson Martinez is a slightly overweight, 56-year-old Hispanic male with a history of hypertension, CAD, and chronic stable angina. At a family gathering in a local park, he joined in on a soccer game but started to feel nauseated and short of breath after running around for a few minutes. Because he has a history of heart disease and angina, his wife called 911, and an ambulance brought him to the closest emergency department.

Nelson was admitted to the ED 30 minutes after his angina symptoms emerged. He described his initial symptoms as shortness of breath, nausea, and arm pain to the nurse in the ED. The nurse recognized these as potential myocardial ischemia and immediately initiated the chest pain protocol along with the medical team. This included a chest X-ray, a 12-lead ECG, and initiating oxygen therapy. The nurse communicated this to Mrs. Martinez and explained the process of assessment of the chest pain in order to stabilize her husband's angina. She was reassured that she did the right thing by calling 911, since quick treatment can improve outcomes.

The ECG revealed a ST elevation myocardial infarction (STEMI). The physician ordered the administration of a fibrinolytic drug to achieve reperfusion. The nurse working with Mr. Martinez explained the indication for the therapy to the patient and his wife by educating them about ST elevations and the need to treat symptoms quickly in order to provide blood supply to the heart muscle.

Because of the quick action of Mrs. Martinez and the identification of the STEMI, Mr. Martinez was treated within 60 minutes after his first symptoms appeared. His condition was stabilized, and he was admitted to the cardiology service for further monitoring of his symptoms.

Goals of ED Care for Patients with Acute Coronary Syndromes

For all patients with acute coronary syndromes, the primary goals of care include:

- Revascularize the coronary artery
- Stabilize heart rhythm
- Preserve myocardial tissue and function
- Reduce cardiac workload
- Provide pain relief
  (McLaughlin, 2014)

While the type of acute coronary syndrome is being identified, the following medical treatments may be ordered for the patient:

- Supplemental oxygen ensures that the existing blood supply is maximally oxygenated.
- Antiplatelet drugs are a key treatment. Aspirin reduces the mortality from an acute myocardial infarction, unless contraindications exist, and all conscious patients with a possible acute coronary syndrome should have chewed and swallowed 160 mg to 325 mg of nonenteric-coated aspirin. Aspirin can also be given as a suppository.
- Fibrinolytic drugs (streptokinase, alteplase, or reteplase) are also utilized, unless contraindications exist, to weaken and disrupt the damaging clot in the coronary artery. Thrombolytic therapy can be used within three hours of the onset of symptoms (McLaughlin, 2014).
- Vasodilators can increase blood flow to heart muscle and can reduce the force need to pump blood through the arterial system. The standard vasodilator for heart arteries is nitroglycerin, which can ease ischemic pain and can also reduce mortality rates. In the ED, nitroglycerin is administered either sublingually, by spray, or via IV. (Certain patients, such as those with hypotension, require graded doses of nitroglycerin and careful monitoring.)
- Beta blockers, such as atenolol, esmolol, metoprolol, or propranolol, are used to lessen the oxygen requirements of the heart by slowing the heart rate and lowering the arterial tension against which the heart is working. Beta blockers also reduce the risk of developing heart arrhythmias, which can accompany heart ischemia. The use of beta blockers has been shown to minimize the size of infarcts and to reduce mortality rates.

- Angiotension-converting enzyme (ACE) inhibitors are administered to patients with evolving MI with ST-segment elevation or left bundle-branch block.
- Antiarrhythmic drugs, such as lidocaine, amiodarone, and epinephrine, may also be indicated to stabilize heart rhythm if the patient has arrhythmias.
- Transcutaneous pacing patches or external defibrillation may also be needed if the arrhythmia continues.
- Glycoprotein IIb/IIIa inhibitors (such as abciximab) may be administered if a patient continues to have unstable angina or acute chest pain to reduce platelet aggregation.
- Anticoagulants can keep new blood clots from forming. Heparin and the low-molecular-weight heparins are often used to lower the risk that unstable angina will progress to myocardial infarction. Heparin administration requires careful monitoring for bleeding, and when the drug is stopped, the patient must be watched for "rebound" ischemic episodes that sometimes occur during the subsequent 24 hours.
- Analgesics (pain relievers), such as morphine sulfate, reduce chest pain and also reduce the sympathetic nervous system's demands on the heart muscles (McLaughlin, 2014).
- Laser angioplasty, arthrectomy, or stent placement may also be initiated during this time (see also the section below on "Percutaneous Coronary Intervention").
- Emergency cardiac surgery may also be performed for patients who are unable to undergo percutaneous interventions (see also the section below on "Coronary Artery Bypass Graft").

Treating Stable CAD in the ED

Some patients who come to the ED with chest discomfort will have stable angina instead of an acute coronary syndrome. As with all patients with possible heart ischemia, these patients should follow a similar treatment protocol, including aspirin, nitroglycerin, a beta blocker, supplemental oxygen, and a blood draw to search for cardiac marker molecules.

In patients with stable angina, the symptoms that brought them to the ED should resolve and not return over the 2 to 3 hours that they are being monitored. Their ECG and vital signs will remain unremarkable for the next few hours, and repeated blood tests will find no cardiac marker molecules.

If the evaluation of noncardiac causes of their chest discomfort identifies no serious problems, these patients do not need further medical treatment in the ED. Instead, they can be monitored and followed as an outpatient by a CAD treatment team.

NURSING CARE OF THE PATIENT WITH STABLE AND UNSTABLE ANGINA

Care of the patient in the ED includes collaborative care with a team approach. Team members may include emergency medical personnel, nurses, a cardiologist, a cardiothoracic surgeon, and a rehabilitation specialist.

Nursing assessment and care of the cardiac patient in the ED is as follows:

- Assess and monitor vital signs (blood pressure, heart rate, temperature, heart and breath sounds), communicating any changes to the team.
- Monitor the patient during episodes of angina and before and after administering medication (especially nitroglycerin).
- Assess and monitor pain symptoms. Include severity, location and duration of pain, medications administered, and any related symptoms.
- Obtain a 12-lead ECG to assess heart rate and arrhythmias (monitor at baseline and during acute episodes of angina).
- Assess and monitor urine output (hourly).
- Monitor oxygen saturation status and make changes as needed.
- Provide patient education regarding medications administered and any procedures anticipated.
- Communicate changes and status updates to family as needed.
- Assess the patient and family for any ongoing psychosocial needs and refer to appropriate supportive services as needed (e.g., social work, community resources, psychologist, counselor, etc.).

Source: McLaughlin, 2014.

Interventional Cardiac Procedures and Surgery

Treatment for patients with stable coronary artery disease is medical therapy and lifestyle modification. In some cases, however, surgery to increase blood flow to ischemic areas can be added to the treatment program to improve a patient's heart function. The general term for these procedures is **coronary revascularization**, which is commonly performed throughout the United States.

In general, coronary revascularization should be considered for patients who still have debilitating angina after optimal medical therapy. The two types of coronary revascularization procedures are percutaneous coronary interventions (PCI) and coronary artery bypass grafts (CABG).

- **PCI** is usually indicated for patients with significant narrowing of one, two, or—at most—three major coronary arteries when the left ventricle is functioning normally.

- **CABG** is indicated for patients with more than two arterial constrictions, with weakened left ventricles, or with diabetes.

There are other therapies for patients whose medical treatment does not improve the symptoms of their coronary artery disease but who are not good candidates for either PCI or CABG. The alternatives include laser transmyocardial revascularization (using a carbon dioxide laser), enhanced external counterpulsation to reduce the frequency of angina, and spinal cord stimulation to relieve the pain of angina (Bhimji, 2013; McLaughlin, 2014).

PERCUTANEOUS CORONARY INTERVENTION (PCI)

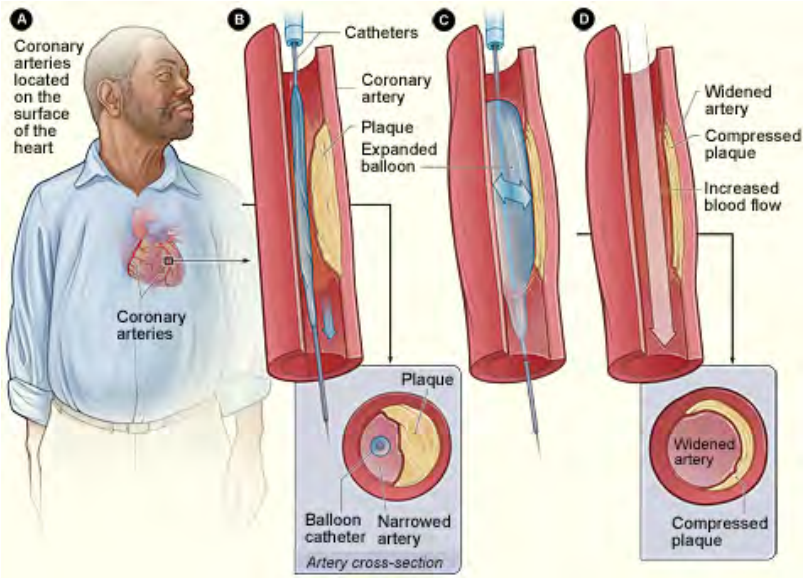
PCI, also commonly known as **coronary angioplasty** or simply angioplasty, is used to unclog blocked coronary arteries. If PCI is recommended, the patient may be transferred to an interventional radiology suite, where the procedure takes place. The procedure involves threading a catheter into the constricted region of an artery and expanding a balloon to flatten the plaque back against the walls of the artery.

Usually, a wire mesh support called a **stent** is left in the region to hold the artery open. Some stents are coated with medications that are slowly and continuously released into the artery. These are called drug-eluting stents. The drugs help prevent the artery from becoming blocked with scar tissue that can form in the artery.

Typically, the PCI catheter is inserted under local anesthesia using X-ray fluoroscopy. The PCI catheter is threaded through the femoral artery into the heart to the area where the coronary artery is narrowed. The procedure can take between 30 minutes and 2 hours.

PCI gives a sufficient increase in blood flow to initially reduce angina in >95% of cases. Approximately one fifth of treated arteries narrow again within 6 months, and angina returns within 6 months in about 1 of 10 patients (NHLBI, 2014b).

In-stent restenosis (narrowing) is a continued concern with coronary angioplasty. Recent studies have shown that using drug-eluting balloon angioplasty to reopen a blocked stent is a promising treatment option in this situation (Indermuehle et al., 2013).



In PCI, a catheter is threaded into the region of the artery that is narrowed by plaque. A balloon near the tip of the catheter is inflated, flattening the plaque against the arterial wall and widening the space inside the artery. Often a wire support (a stent) is left in place to hold the artery open. (Source: NHLBI, 2014b.)

NURSING CARE OF THE PATIENT DURING AND AFTER PCI

Care of a patient during and after PCI includes primary nursing assessments and measures as follows:

- Prepare and educate the patient on what to expect during and after the procedure.
- Ensure adequate patient protection from radiation exposure.
- Monitor and assess vital signs throughout the procedure (blood pressure, respiration, heart rate).
- Apply manual compression (15 to 20 minutes) to achieve homeostasis (mechanical compression may also be used).
- Apply and assess pressure dressing to femoral puncture site.
- Assess and monitor puncture site for any bleeding.
- Instruct patient on duration of bed rest and plan for progression to ambulation.
- Review home instructions, restrictions, new medications (including anticoagulation), and follow-up appointments.

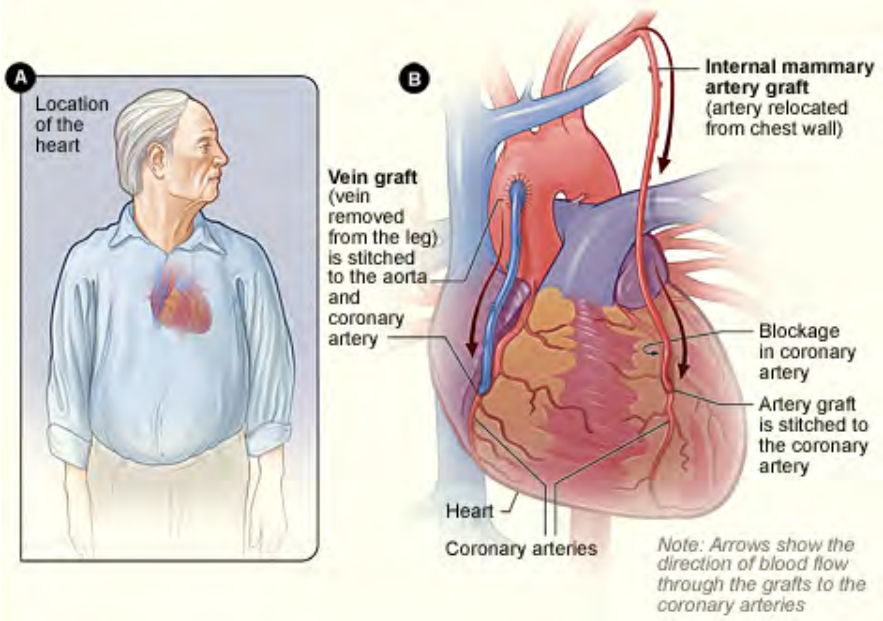
Source: Merriweather & Sulzbach-Hoke, 2012.

CORONARY ARTERY BYPASS GRAFT (CABG)

Coronary artery bypass surgery is the most common open-heart operation performed in the United States, with over 500,000 procedures performed each year. CABG may be contraindicated in elderly patients and in patients with end-stage kidney disease, lung disease, and peripheral vascular disease, as these patients are at higher risk for complications (University of Michigan, 2014).

The procedure involves attaching an unclogged blood vessel to a blocked coronary artery beyond the obstruction. One or both internal thoracic (also called internal mammary) arteries can be rerouted or a piece of the saphenous vein or the radial artery can be made into a conduit.

The surgery is done under general anesthesia and takes between 3 to 6 hours. Usually, the procedure is done by temporarily stopping the heart and oxygenating the blood with a cardiopulmonary bypass machine. When patients have no other serious disease, there is <1% mortality from a first-time CABG surgery.



In coronary artery bypass graft (CABG) revascularization surgery, blood is routed past blockages in coronary arteries. Figure B shows how vein and artery bypass grafts are attached to the heart. (Source: NHLBI, 2012.)

There are two main types of bypass surgery: conventional (arrested heart) and "beating heart" CABG.

Conventional CABG

Conventional (or "on pump") CABG is performed on an arrested (stopped) heart through an incision down the middle of the patient's chest. The patient's heart is stopped with medications, and blood is routed to a heart-lung bypass machine, which removes

CO_2 and supplies oxygen, thus bypassing the processes carried out by the heart and lungs. The reoxygenated blood is returned to the body to nourish it.

The patient may need blood transfusions (donor blood, blood harvested during the procedure and returned to the patient, or self-donations made in advance of surgery) to replenish blood volume, red blood cells, or platelets. To reduce oxygen demand, the patient is placed in therapeutic hypothermia.

"Beating Heart" CABG

In **"beating heart" (or "off pump") bypass surgery**, the heart is not stopped, the heart-lung bypass machine is not used, and the patient remains at normal or only slightly lowered temperature. Indications for this type of surgery include patients who have diabetes, lung disease, kidney disease, or a previous history of stroke.

Beating heart surgery often allows patients to be discharged from the hospital more quickly than with conventional CABG, and the avoidance of the heart-lung machine has been shown to reduce the need for transfusions. Patients with this procedure may also have a lower risk for infection, stroke, and kidney complications (University of Michigan, 2014).

MINIMALLY INVASIVE CABG SURGERY

Robotic-assisted coronary artery bypass grafting is a minimally invasive procedure. In this procedure, the surgeon makes several small incisions between the ribs and then inserts a small camera and small robotic arms through the incisions. During the procedure, the surgeon sits at a console and controls the robotic instruments to perform the CABG.

This procedure may not be indicated for everyone and requires specialized training for the surgeon. The robotic approach has been shown to improve postoperative outcomes for patients, including less pain and a faster recovery period.

In addition, the mini-thoracotomy and totally endoscopic port-only approaches to perform CABG reduce surgical trauma while providing cosmetic benefits and preserve chest wall structure and function. Many studies show a positive reduction of the recovery period and a rapid return to full activity for the patient.

Source: Bonatti et al., 2011.

Postoperative Care and Management

Care of the patient in the postoperative setting includes collaboration and a team approach. Team members may include respiratory therapists, nurses, a cardiologist, a cardiothoracic surgeon, and rehabilitation specialists.

Postoperative care after a CABG is as follows:

- Monitor vital signs, watching for signs of hemodynamic changes such as severe hypotension, decreased cardiac output, and shock.
- Initiate warming procedures according to hospital protocol.
- Assess and record vital signs every 5 to 15 minutes until the patient's condition is stable.
- Administer medications as ordered and titrate according to patient response.
- Monitor ECG for any heart rate changes or arrhythmias.
- Evaluate and assess the patient's peripheral pulses, capillary refill time, and skin temperature.
- Auscultate heart sounds, noting and reporting any changes.
- Monitor chest tube drainage and negative pressure.
- Assess breathing and breath sounds, monitor ventilator settings, and check arterial blood gas (ABG) results every two hours.
- Monitor mean arterial pressure (MAP), pulmonary artery pressure (PAP), central venous pressure (CVP), left arterial pressure, and cardiac output as ordered.
- Measure intake and output (I & O) and assess for any electrolyte imbalances.
- Assess the patient's pain and provide pain medications as needed.
- Monitor the patient for signs and symptoms of stroke, pulmonary embolism, pneumonia, and impaired renal function.
- Encourage incentive spirometry, coughing, and deep breathing (while splinting the incision) after the patient is weaned from the ventilator.
- Assist with range of motion (ROM) exercises to enhance peripheral circulation and prevent formation of thrombus.

  (McLaughlin, 2014)

COMPLICATIONS IN THE ACUTE POSTOPERATIVE PERIOD

Adverse events can occur in the postoperative period. It is important for nurses to assess the patient for postoperative complications, which may include atrial fibrillation, stroke, cognitive decline (including delirium), surgical site infections, depression, and acute renal failure (Diodato & Chedrawy, 2014).

Atrial Fibrillation

Postoperative atrial fibrillation (AF) is the most common post-CABG complication, occurring in 20% to 50% of patients. Factors that may increase a patient's risk include

peripheral artery disease, COPD, valvular heart disease, previous cardiac surgery, male gender, and advanced age. First-line treatment includes beta blockers and amiodarne. Post-CABG AF typically has an onset within 2 to 5 days postoperatively and usually resolves within 6 weeks of surgery (Diodato & Chedrawy, 2014).

Stroke

Postoperative stroke occurs in 1% to 4% of patients. Risk factors include age, previous stroke, diabetes, hypertension, and female gender. Along with vital signs, nursing assessment includes postoperative neuro status checks in addition to any functional or cognitive changes that may be due to sudden stroke (Diodato & Chedrawy, 2014).

Cognitive Decline

Postoperative delirium and cognitive decline occurs in <10% of patients. Nursing assessment includes monitoring for any cognitive changes, especially in patients at high risk. Risk factors for cognitive decline include preexisting cerebral vascular disease, central nervous system disorders, and cognitive impairment (Diodato & Chedrawy, 2014).

Surgical Site Infection

Surgical site infections occur in 10% to 20% of patients. Risk of deep sternal wound infections is increased if a patient has a history of diabetes, smoking, obesity, and COPD. Infection rates and a risk for sepsis also increases with the use of blood transfusions, prolonged intubation, and surgical reexploration (Diodato & Chedrawy, 2014). Careful nursing assessment for any signs or symptoms of infection includes monitoring patient temperature, pain, swelling, and incision site redness/discharge.

Depression

Postoperative depression is common and can occur weeks after discharge (Humphreys, 2011). The risk of depression following a heart attack is high, with reports that up to 65% of patients suffer from depressive symptoms following acute myocardial infarction (Diodato & Chedrawy, 2014).

Depression is strongly linked to patients with low physical activity and limited mobility. This fact stresses the importance of initiating physical activity and rehabilitation as soon as possible following a cardiac event. Nurses should place an emphasis on educating patients and their families about the development of depressive symptoms with resources and strategies to address depression.

SYMPTOMS OF DEPRESSION

- Sleep disturbance and fatigue
- Changes in physical activity, ranging from slowing down to agitation or hyperactivity
- Loss of interest in favorite activities
- Hopelessness about the future
- Changes in appetite and/or weight
- Inability to concentrate or make decisions
- Complaints about poor memory and concentration
- Thoughts of suicide

Source: NIMH, 2014; Tabloski, 2014.

Acute Renal Failure

Incidence of acute renal failure after CABG is 2% to 3%. A small percentage of patients will go on to need dialysis. Careful nursing monitoring includes kidney function (urinary output, creatinine clearance, and other kidney function tests), especially for those patients with high risk factors including preexisting renal dysfunction, decreased cardiac output, insulin-dependent diabetes, peripheral artery disease, advanced age, African race, and female gender (Diodato & Chedrawy, 2014).

POSTOPERATIVE PHYSICAL AND OCCUPATIONAL THERAPY GOALS

Frequently, a patient with physical limitations following a cardiac event will be referred to a rehabilitation specialist. Based on the assessment and evaluation, the physical and/or occupational therapist creates an individualized treatment plan that includes the patient's goals for treatment and addresses the individual physical limitations.

Early and frequent physical therapy (PT), starting as soon as one day after surgery, can help restore a normal pattern of daily functioning in a patient with CAD. A customized PT program may include exercises for range of motion, muscle strengthening, and coordination. Exercises will vary depending on the patient's baseline condition.

Initial physical therapy may include range of motion (ROM) exercises to move the patient's arms, legs, hands, and feet while in the initial recovery period. Gradually, the patient will increase activity levels to learning how to transfer to the chair, participate in activities of daily living (ADLs), and eventually walk. Whole-body movement may progress from walking in brief bursts to walking the length of the corridor to climbing stairs (Moroz, 2013a).

INITIAL PHYSICAL THERAPY EVALUATION

Patients who are stable after a cardiac event will have an initial evaluation while recovering in the hospital. Elements of a physical therapy evaluation include the following:

- Medical history
- Heart rate
- Blood pressure
- Oxygen saturation
- Upper body strength and range of motion
- Lower body strength and range of motion
- Level of functional mobility and ability to perform self-care
- ECG measurements at rest and during activity
- Other measurements of baseline functional status (e.g., Timed Up-and-Go [TUG] test or 6-Minute Walk Test)

Source: Sears, 2015a.

Exercise is a key component of physical rehabilitation and involves specific exercises and muscle strengthening exercises focused on maintaining and improving strength, endurance, and balance (Brown & Flood, 2013). The first phases of cardiac rehabilitation may involve walking, using a treadmill, or riding a stationary bicycle, all supervised by a physical therapist trained in rehabilitation. If patients tolerate these exercises well, they may progress to stair climbing (see also "Cardiac Rehabilitation Phases" later in this course).

In patients who experience shortness of breath, lightheadedness, or chest pain during exercise, the exercise should be stopped immediately and the patient's cardiac status reassessed. ECG monitoring is often used to monitor the patient's cardiac status as well (Moroz, 2013a).

Occupational therapists in particular have specialized knowledge and skills that address the limitations that patients may experience with the performance of ADLs and instrumental ADLs. The **occupational therapy process** begins with a thorough evaluation that identifies the baseline status of the patient and any secondary disabilities, the need for any assistive devices, home safety, and any areas of occupation or skills that are currently difficult for the patient to perform (Moroz, 2013a).

An occupational therapist can assist the patient with **ADLs (activities of daily life)**, such as learning how to transfer safely, progressing to achieve more independent self-cares, such as showering, cooking, housework, and driving. Patients who are planning to return to a work environment may need to adjust their work schedules and limits on physical activities (such as lifting) depending on the type of work they are engaged in (Moroz, 2013a).

STERNAL PRECAUTIONS

Sternal precautions will help the patient recover from a sternal incision and prevent separation of the breastbone as it heals. These precautions should be followed by the patient for 4 to 6 weeks until the sternal incision is well healed.

Patients should be instructed on the following precautions:

- No pushing or pulling with the arms
- Limit lifting to 5 to 8 pounds or less
- No reaching of arms directly over the head
- No extension of both arms out to the side
- No reaching with the arms to the back

Physical and occupational therapy strategies to assist the patient in protecting the sternum include instructions on the following:

- Scooting in when rising from a chair
- Walking up stairs with pulling on the rail
- Rolling in bed and gradually sitting up
- Using assistive devices if recommended (e.g., walker or quad cane)
- Strategies to assist the patient in performing ADLs, such as bathing, dressing, and brushing hair

Source: Sears, 2015b.

POSTOPERATIVE CARDIAC REHABILITATION GOALS

There are four phases of cardiac rehabilitation. In the immediate postoperative period, phase I of cardiac rehabilitation is initiated. Cardiac rehabilitation programs are supervised and monitored by trained rehabilitation professionals. The goals of cardiac rehabilitation are to maximize strength, prevent regression of coronary artery disease, and reduce the likelihood of future cardiac problems (McLaughlin, 2014).

Comprehensive rehabilitation programs that include exercise, education, counseling, and help with lifestyle changes can:

- Increase exercise tolerance
- Decrease symptoms (such as angina and shortness of breath)
- Improve blood lipid levels
- Reduce stress
- Make it easier to stop smoking
- Improve mood

Many patients with CAD, and especially those who have had a heart attack or heart surgery, may be fearful of exercise. The first step in reassuring patients is to educate them about the disease in general and their condition in particular. General advice should include a review of the symptoms of ischemia, rules on managing an episode of angina or dyspnea, and an explanation of what symptoms require a quick trip to an ED. The patient's family should be included when educating the patient.

Patients with signs and symptoms of depression are less likely to complete their cardiac rehabilitation programs, and it is important to identify these patients and to get the appropriate help for them early in the program.

Rehabilitation specialists are involved in advising patients on resuming their normal activities after discharge from the hospital. Recommendations may include:

- Daily walking can be encouraged immediately.
- Sternal precautions may be recommended (see box above).
- Patients can often resume their previous level of sexual activity in 2 to 4 weeks, depending on their tolerance for exercise. (If patients have no symptoms of angina, dyspnea, or palpitations with moderate exertion physical activity, this is a good indication that they will not have symptoms during sexual intercourse.)
- Routine driving can usually be resumed within a week (in those states that allow it).
- Patients can return to work with recommended modifications to their schedule or duties, as needed.
- Long-distance travel should be postponed until the patient is stable. Patients should take caution when planning travel to high altitudes or extreme temperatures.
  (Humphreys, 2011)

(See also "Cardiac Rehabilitation" below in this course for more detailed information.)

CASE

John Townsend, age 65, is recovering in the hospital from CABG surgery to reopen a blocked coronary artery. The day after his surgery he is visited by a nurse and a physical therapist, who each brief him on the cardiac rehabilitation regimen he is about to undergo. John expresses anxiety about having to undergo cardiac rehabilitation so soon, but the nurse and physical therapist reassure him that the regimen will be helpful and manageable and that it will start slowly.

John starts his early-stage (phase I) cardiac rehabilitation the next day. On performing an initial evaluation of John's functional mobility status and activity tolerance, the physical therapist helps teach him how to transfer from the bed to the chair and later helps him get out of bed and walk a short distance to the door of his room and back again. That exercise is repeated twice more the same day.

The nurse helps John to begin taking care of his ADLs, such as personal care, bathing, eating, and understanding new medications. The physical therapist addresses planning for his transition to the home environment by asking John about safety issues, anticipated barriers in the home, and assistive devices needed or used in the past. Together, they plan for strategies to address showering at home and his plan for home ambulation.

The next day, after his physical therapist has determined the level assistance needed in order for John to ambulate safely, the nurse helps him venture out to the corridor, and he is able to walk slowly to the nurses' station, which is 60 feet from his room. John takes two more corridor walks that day and four such walks each of the next two days.

By the time he is discharged on day five postsurgery, John is able to walk in the corridor for 10 minutes at a time. The nurse reinforces the safety recommendation instructions from the physical therapist and reviews the detailed instructions with John on continuing with his exercise plan while at home.

John has support at home from his wife, however the nurse schedules a home health visit twice a week for one month to follow-up on his progress. John is scheduled to see his doctor and the physical therapist for a follow-up visit in two weeks to start his long-term rehabilitation program.

DISCHARGE PLANNING AND EDUCATION

Discharge planning following a cardiac event or procedure may include the following patient education and instructions:

- Monitor for signs of infection (redness, swelling, discharge, drainage, fever, or sore throat).
- Understand the warning signs for arterial reocclusion (angina, dizziness, dyspnea, rapid or irregular pulse, and shortness of breath).
- Monitor body weight and notify the primary care provider if the patient gains more than 3 lbs. (1.4 kg) in one week.
- Follow any special diet instructions (especially any sodium and cholesterol restrictions).
- Review any restrictions on lifting (limit to <10 lbs. for 4 to 6 weeks).
- Maintain a good sleep routine, with at least 8 hours of sleep each night and short rest periods throughout the day.
- Participate in an exercise program and cardiac rehabilitation recommendations.
- Follow any lifestyle modifications recommended (smoking cessation, nutrition, and exercise programs).
- Understand the dose, indication, frequency, and side effects of all prescribed medications.

- Understand the follow-up plan of care, including visits with cardiology, the surgeon, and the primary care provider.

  (McLaughlin, 2014)

COMPREHENSIVE MANAGEMENT OF CHRONIC CAD

A patient who has chronic CAD should be enrolled in a long-term treatment plan. These patients include people with chronic stable angina and people with stable coronary artery disease after having been treated for acute coronary syndromes.

Outpatient Monitoring and Guidance

Each patient is different and each will need an individualized treatment program. Such programs include education, medications, therapeutic lifestyle changes, possible revascularization (reperfusion) surgery, and treatment of associated disorders.

The **primary goals of care for patients with CAD** include strategies that focus on stabilizing any progression of disease while improving physical function, quality of life, and psychosocial well-being.

LONG-TERM GOALS FOR TREATMENT

- Support the patient in living a comfortable life without pain and with the fewest possible restrictions
- Prevent the development of an acute coronary syndrome
- Slow or reverse the degree of atherosclerosis
- Reduce the cardiovascular risk factors in the patient's life

Medications

Drug therapy is a key part of the treatment of coronary artery disease. To reduce the likelihood of developing obstructive clots, patients who have CAD or are at high risk of developing CAD should take antiplatelet drugs daily. To lessen the work of the heart, most patients with CAD also take beta blockers. For relief of angina, nitrates are prescribed.

The standard **medication therapies for CAD** include:

- Aspirin
- Beta blocker
- ACE inhibitor
- LDL-lowering drug, when needed
- Nitroglycerin for relief of angina

Medications are essential to the care of heart patients. The approximately 10% of elderly patients with CAD who do not take their prescribed medications regularly are twice as likely to develop acute coronary syndromes. By asking patients at each visit whether they are taking their medicines all the time, it is possible to intervene and to lower the risk of serious complications (Marcum et al., 2013).

ASPIRIN

Long-term antiplatelet therapy makes acute ischemic episodes less likely in all forms of coronary artery disease. **Aspirin** is the first-line antiplatelet drug, unless the patient has aspirin allergy or a history of or risk for gastrointestinal bleeding.

The starting dose is typically between 75 mg and 162 mg/day and should be continued indefinitely unless contraindicated. Clopidogrel (Plavix) can be added for up to 12 months to increase the inhibition of clot formation, and it can be given to patients when aspirin is contraindicated (Humphreys, 2011).

Patients may need to discontinue antiplatelet or anticoagulant therapy before undergoing elective surgery, as not doing so can lead to cancellation or postponement of the operation or, worse, cardiac events or other potentially catastrophic developments during or following surgery (Reich et al., 2011). Patients considering elective surgery should therefore coordinate their antiplatelet/anticoagulant regimens with their primary care provider, cardiologist, and surgeon.

NITROGLYCERIN

Nitrates, such as **nitroglycerin**, dilate blood vessels throughout the body. By lowering the arterial resistance to blood flow, nitrates ease the work of the heart, and by dilating heart arteries, they increase the blood flow to the heart muscles.

Nitroglycerin relieves the pain of angina, and if taken approximately 5 minutes before exercise or stress, it can prevent angina. The nitroglycerin in sublingual tablets is absorbed quickly and completely, and it generally works within 2 to 3 minutes and lasts for 1/2 hour. All patients with angina should be given sublingual nitroglycerin with specific instructions about its use. Nitroglycerin is also available as an oral spray and as long-lasting tablets and patches.

USE OF SUBLINGUAL NITROGLYCERIN	
Purpose	• To relieve chest pain from heart disease • To prevent chest pain in stressful or active situations
When to use	• As soon as chest pain or tightness begins • 5 to 10 minutes before an event expected to cause chest pain or tightness (e.g., climbing stairs, going outdoors in cold weather, having sex)
How to use	• Sit down to prevent falling if feeling faint after taking nitroglycerin. • Place one tablet under the tongue. • Let the tablet dissolve naturally. Don't swallow it whole; if swallowed by mistake, put another tablet under the tongue. • While the tablet is dissolving, don't eat, drink, smoke, or chew tobacco.
What to expect if it works	Chest discomfort should decrease in 1 to 5 minutes.
What to do if it doesn't work	If discomfort does not decrease after taking 1 tablet, call 911 immediately and report chest pain. Alternately, for those used to taking nitro, take up to 3 tablets before calling 911.
Typical side effects	• Burning or tingling under the tongue • Dizziness, lightheadedness, or fainting • Flushing of the face or neck • Headache • Burning/tingling under the tongue
Side effects to report immediately to primary care provider	• Blurred vision • Dry mouth • Skin rash, itching, or swelling • Sweating • Feeling of extreme pressure in the head • Unusual tiredness or weakness • Pale skin • Fast heartbeat • Difficulty breathing
Drugs that can be taken before or after nitroglycerin	• Aspirin • Other pain relievers approved by the primary care provider
Drugs **not** to be taken with nitroglycerin	Erectile dysfunction medicines (Viagra, Cialis, Levitra)
Storage	Keep the tablets tightly sealed in their original container and away from heat, light, and moisture.
Source: Adapted from MedicineNet, 2014.	

BETA BLOCKERS

Beta-adrenergic blocking agents are antihypertensive drugs that also reduce heart rate and heart muscle tension, and in these ways, they reduce the heart's demand for oxygen. Beta blockers will lower the incidence of episodes of angina, and they will also reduce the likelihood of myocardial infarctions and death in CAD patients.

Special care must be taken when prescribing beta blockers to patients with asthma, other obstructive airway conditions (COPD), intermittent claudication, insulin-requiring diabetes, certain heart conduction problems, and clinical depression. When the side effects of beta blockers become a problem, calcium channel blockers, such as diltiazem or verapamil, or Ranolazine can be substituted (Humphreys, 2011; Mayo Clinic, 2013b).

ACE INHIBITORS

Angiotensin-converting enzyme (ACE) inhibitors, such as ramipril (Altace), are antihypertensive drugs that can reduce the likelihood of acute ischemic episodes, strokes, and death in patients with CAD (Humphreys, 2011; Mayo Clinic, 2013b).

STATINS

Lipid-lowering drugs are frequently prescribed for people with coronary artery disease. High levels of LDL cholesterol initiate and worsen atherosclerosis. In patients with high blood levels of cholesterol, the first medical intervention is lifestyle changes, especially a low-fat diet and increased exercise (see below). When this does not lower a patient's cholesterol to safe levels, lipid-lowering drugs are prescribed.

Statins, such as atorvastatin (Lipitor) and simvastatin (Zocor), are the preferred lipid-lowering drugs for coronary artery disease, but some lipid abnormalities should be treated with nicotinic acid or fibric acid (Humphreys, 2011).

In the Heart Protection Study, a large trial of more than 20,000 high-risk patients, longer-term statin therapy was associated with greater reductions in vascular events; even after patients in the study stopped taking statins, the benefits persisted for at least 5 years without evidence of emerging hazards (Bulbulia et al., 2011). Patients with liver disease should not take statins.

OTHER MEDICATIONS

Patients who have moderate to severe depression may be prescribed medications as part of their management program. Selective serotonin reuptake inhibitors (SSRIs), including sertraline and citalopram, are one of the only forms of antidepressant therapy safe to use with patients who have CAD (Humphreys, 2011).

Cardiac Rehabilitation

A cardiac rehabilitation program is designed to support and assist a patient recovering from a myocardial infarction, other forms of heart disease, or surgery to treat heart disease. Cardiac rehabilitation improves the long-term survival of patients with heart disease. The American Heart Association and the American College of Cardiology recommend cardiac rehabilitation programs (Mayo Clinic, 2014c).

Hospitalization for a cardiac event or surgery is often the time when the first phase of cardiac rehabilitation begins. Once the patient is discharged, referral to an outpatient rehabilitation program is initiated. Patients can begin formal outpatient cardiac rehabilitation programs as early as 10 days postoperatively depending on their condition. Cardiac rehabilitation may last 3 to 6 months or longer.

Cardiac rehabilitation may begin in an acute care hospital. Rehabilitation hospitals or units may provide the most extensive and comprehensive care and should be a consideration for patients who have good potential for recovery and can participate in and tolerate aggressive therapy.

Rehabilitation can also be offered in nursing homes or in the home environment with a less intensive approach that lasts longer and is better suited to patients less able to tolerate therapy (e.g., frail or elderly patients).

Ideally, the patient's care is coordinated by a multidisciplinary team who sees the patient regularly. For patients recovering from myocardial infarctions or surgical cardiac procedures, the team should include cardiac rehabilitation specialists. Cardiac rehabilitation specialists may include a cardiologist, nurse educator, dietitian, exercise physiologist, occupational therapist, physical therapist, psychologist, and psychiatrist who are trained in cardiac rehabilitation programs. Also, family members may need help learning how to adjust to the patient's disability and how to help the patient (Moroz, 2013b).

COMPONENTS OF CARDIAC REHABILITATION

Elements of a cardiac rehabilitation program are comprehensive and include the following primary components:

- Individualized exercise program
- Diet, nutrition, and weight management
- Stress management
- Risk factor reduction
- Lipid and cholesterol control

Source: McLaughlin, 2014.

Cardiac rehabilitation models are continuing to evolve to meet a variety of age groups and needs. Advanced age is associated with a higher prevalence of CAD as well as increased morbidity and mortality. Cardiac rehabilitation programs designed to meet the needs of older patients (>65 years of age) should include strength, balance, coordination, and flexibility. Evidence-based programs show that elderly patients can realize positive benefits from an exercise-based cardiac rehabilitation program to increase functional capacity, glucose control, quality of life, enhanced ability to perform ADLs, and reduced incidence of hospitalization (Menezes et al., 2014).

INNOVATION IN CARDIAC REHABILITATION

A unique telephone-based coaching program was implemented for participants who were not able to attend an in-person rehabilitation program. The program was designed to coach participants with lifestyle modification tools to assist in meeting outcomes. Tools included a pedometer to monitor physical activity, nutrition guidelines and weight guidance, and regular check-in calls (six telephone coaching sessions in total over a period of eight weeks) from trained behavioral coaches.

The program results showed modest improvements in weight and increased physical activity. The low-contact, telephone-based intervention may be a feasible model for delivering cardiac rehabilitation to patients who are in rural and underserved communities.

Source: Sangster et al., 2014.

CARDIAC REHABILITATION PHASES

Cardiac rehabilitation may be divided into four phases (Humphreys, 2011; Morris et al., 2011):

Phase I (Inpatient)

The first phase of cardiac rehab takes place before the patient is discharged from the hospital.

This phase generally consists of evaluation and assessment of the patient's condition, motivation, and risk factors, accompanied by education and discharge planning.

The patient is gradually introduced to exercise on day 2 of cardiac rehab, with an intensity of exercise up to four metabolic equivalents (METS, i.e., four times the resting metabolic rate, or four times the amount of oxygen consumed at rest). Ideally, by day 4, the patient will be walking in the corridor for 5 to 10 minutes 3 to 4 times a day.

Phase II (After Discharge)

The patient is given clear instructions on his or her individualized exercise plan. The rehabilitation team may include the following professionals who work closely with the patient: exercise physiologists, occupational therapists, and physical therapists.

If a patient is considered home bound, a home physical therapist evaluation is completed and a program of home exercises outlined for the patient.

The initial mode of exercise is usually walking on level ground, with an intensity goal of between 2 and 4 METS or a score of 11 to 12 on the Rating of Perceived Exertion Scale (i.e., moderate intensity) (CDC, 2011b). Patients are generally advised to stay indoors for the first day or two because they may expect to feel fatigued and/or anxious, though patients with uncomplicated coronary artery disease may be advised to increase their walking distance progressively to 3 to 5 kilometers a day after 4 to 6 weeks.

During Phase II rehabilitation, exercises may include:

- Treadmill walking
- Stationary bike
- Using an upper body ergometer (UBE)
- Rowing
- Upper and lower body strengthening using free weights
- Stretching

As the patient gains strength, these same exercises may progress in intensity and duration as the patient transitions from Phase II to Phase II (Sears, 2015c).

Phase III (Outpatient Exercise Program)

The goal of this phase is to enable the patient to exercise safely in a structured environment and to understand the benefits of exercise. Before starting an exercise program, it is common for a patient to undergo an exercise stress test until symptoms become apparent. The exercise test can be used as either a diagnostic or prognostic tool or as a test of functional capacity.

Cardiac patients should exercise in the low to moderate range of exercise intensity, corresponding to 60% to 75% of maximum heart rate or 60% to 70% of maximum heart rate reserve, which is equivalent to a score of 12 to 14 on the Rating of Perceived Exertion Scale. (The maximum heart rate is usually calculated by subtracting the patient's age from 220. The heart rate reserve is calculated by subtracting the resting heart rate from the maximum heart rate.) The outpatient exercise program may last from 8 to 12 weeks, and patients generally attend 2 to 3 times per week.

Exercises during this phase promote total physical conditioning and include:

- Treadmills
- Cycle and arm ergometers
- Stair climbers
- Rowing machines

The exercise session should be preceded by a warm-up period lasting approximately 15 minutes, and the session itself lasts for 30 to 35 minutes, followed by a 10-minute cool-down period.

While the above exercises are largely aerobic in nature, resistance training can also be used in patients at low to moderate risk. However, patients are advised to spend some time on aerobic-type exercises before they initiate resistance exercise.

Phase IV

The patient exercises independently and maintains the recommended lifestyle modifications. Increased physical activity and enhanced physical fitness can promote cardiovascular health, provided that the patient keeps up with the exercise program. Indeed, the change in exercise behavior that the patient achieves during phase III must be lifelong in order to have any lasting benefit.

Physical therapists' evaluation of a patient undergoing cardiac rehabilitation may include:

- Sternal precautions and scar mobility
- Exercise endurance level
- Assessment of range of motion and strength
- Assessment of gait, balance, and mobility
- Functional mobility tests (6-Minute Walk Test, Timed Up-and-Go Test)
  (Sears, 2015d)

Occupational therapists' role in evaluating and treating patients during cardiac rehabilitation may include:

- Evaluating self-care skills and other activities of daily living
- Home safety evaluation
- Self-care skills training
- Recommendations for home management tasks and instrumental activities of daily living
- Teaching, strategies, and tools for health management (e.g., medication reminders and appointment schedules)
  (AOTA, 2015)

EXERCISE PROGRAMS

Physical conditioning from a regular exercise program generally:

- Improves the body's metabolism as well as conditioning the heart muscles
- Increases the amount of activity a patient can do before developing chest discomfort

- Helps with losing weight and in maintaining weight loss
- Makes smoking cessation easier
- Improves lipid levels
- Lowers blood pressure
- Increases the feeling of well-being
- Increases the chances of surviving a myocardial infarction

Formal cardiac exercise programs are supervised and tailored to the abilities of the patient, and these programs increase exercise levels appropriately but gradually.

The patient is evaluated for **risk of cardiovascular complications** before starting an exercise program. Patients are stratified by risks according the following:

- Class A: Patients who have a healthy baseline with no clinical evidence of increased cardiovascular risk with exercise
- Class B: Patients with established CAD that is stable (patients at low risk for cardiovascular complications with vigorous exercise)
- Class C: Patients who are at moderate to high risk for cardiac complications during exercise because of previous history of myocardial infarction or cardiac arrest
- Class D: Patients with unstable disease who require activity restriction with contraindications for exercise
  (Braun et al., 2014)

LIFESTYLE MODIFICATIONS

For patients with existing CAD, lifestyle changes will improve their quality of life and their sense of well-being as well as slow or even reverse their illness. Patients may have modifiable risk factors that will put them at increased risk for continued medical problems related to CAD (see also "Preventable Risk Factors" earlier in this course).

Smoking cessation, reducing dietary calories and fats (especially saturated fats), and increasing exercise can significantly reduce a patient's risk of further developing atherosclerotic cardiovascular disease. Therapeutic lifestyle changes are also the cornerstones of the treatment of diabetes, obesity, hypertension, insulin resistance, and most dyslipidemias (Boudi, 2014b).

Smoking Cessation

Smoking injures cells throughout the body. Smoking contributes to the development of atherosclerotic cardiovascular disease, insulin resistance, type 2 diabetes, dyslipidemia, a variety of cancers, many lung diseases, gastrointestinal diseases, reproductive problems, osteoporosis, cataracts, age-related macular degeneration, and hypothyroidism.

Patients should be educated on the medical consequences of smoking and strongly advised to stop smoking. It may be difficult for smokers to quit on their own. Counselors working with patients should encourage them to set a goal for a specific date they will begin to wean themselves from cigarettes. Patients may be referred to programs that include support, counseling, and the availability of antismoking medications.

Weight Management

The ideal goal for a patient's body mass index (BMI) should be between 18.5 and 24.9 kg/m^2, and the waist circumference should be <102 cm (40 inches) for men and <88 cm (35 inches) for women. Excess weight strains the heart, and excess fat leads to continuous high levels of blood lipids. Weight loss improves blood lipid profiles and helps lower blood pressure in overweight and obese people. For coronary artery disease patients who are overweight, weight loss can reduce the severity of their angina (McLaughlin, 2014).

Exercise alone rarely leads to significant weight loss; a reduced calorie diet is necessary. Reducing patients' overall calorie intake will also improve their lipid profile. Besides eating fewer calories, scheduled meals and preplanned menus make weight loss easier. Weight loss programs include these and other techniques, and formal programs with regular advice, counseling, and supervision usually have the most success. From any starting weight, a loss of 10% should be considered a success if the patient manages to maintain the lower weight (McLaughlin, 2014).

Nutrition

Eating nutritiously will slow the development of atherosclerosis. Simply reducing the overall calories in a patient's diet will improve the lipid profile, and reducing the amount of fat will improve lipid levels even further.

For a heart-healthy diet, it is especially important to remove or limit foods that are high in saturated fats and trans fats. Instead, diets should focus on fresh fruits, vegetables, and whole grains. In addition, daily plant sterols and 10 to 25 g/day of soluble fiber (oat bran, beans, soy products, psyllium) are recommended. Moderate alcohol intake (20 g/day or less) in men is associated with a reduced incidence of coronary artery disease events, although the mechanism behind this benefit is not well understood (Boudi, 2014b).

NUTRIENT COMPOSITION OF A HEART-HEALTHY DIET

Nutrient	Recommended Intake
Saturated fat	<7% of total calories
Polyunsaturated fat	≤10% of total calories
Monounsaturated fat	≤20% of total calories
Total fat	25%–35% of total calories

Carbohydrate	50%–60% of total calories
Fiber	20–30 grams/day
Protein	Approximately 15% of total calories
Cholesterol	<200 mg/day
Total calories (energy)	Balance energy intake and expenditure to maintain desirable body weight and prevent weight gain
Source: Cleveland Clinic, 2012b.	

Oral health problems can indirectly increase the risk of developing cardiovascular disease. Patients with mouth problems—such as cavities, sore gums, periodontal disease, and missing teeth—prefer to eat soft foods, which are low in fiber. Dental care and tooth replacement are an often-forgotten part of improving a patient's diet.

Nutritional evaluation, counseling, and monitoring are essential to helping patients improve their diet. However, it is unrealistic to expect that a single nutritional educational session or program will result in long-term adherence to a sensible diet. Moreover, patients may find it difficult to absorb a large amount of information in a short period of time. Some patients—particularly those with comorbidities such as diabetes, obesity, or heart failure, as well as those from culturally and linguistically diverse backgrounds—may require more nutritional information and counseling than they can obtain in the context of a group program (Graham et al., 2011).

These factors make it especially important for patients and their family members to consult with a dietitian on a regular basis. Many hospitals offer preventive and therapeutic nutrition classes with an emphasis on cardiovascular health.

Emotional Support

For many patients, adjusting to the lifestyle changes needed to manage CAD can take time. Some patients may feel anxious or depressed and lose touch with their support system. Patients may also need to be away from their work for several weeks during treatment and recovery.

Counseling may be helpful for patients with depressive symptoms. Antidepressants may also be helpful for patients who have more severe or chronic symptoms. Occupational therapists can help in teaching new skills if a patient needs to modify activity levels because of their work or vocation.

Patients should be encouraged to learn stress reduction strategies that work for them. These may include mind-body techniques such as tai chi, yoga, journaling, guided imagery or other creative outlets.

OVERALL PATIENT EDUCATION GOALS

Patients should be taught the **basics of the disease**. They should learn that their sensitivity to ischemia will vary during the day (for example, angina is more likely in the early morning and just after meals) and according to the weather (cold weather is more stressful).

Patients can control their angina by the way they live their daily lives. Heart ischemia is brought on when the heart muscle is asked to work hard. Many tasks that cause chest pain can be done without discomfort simply by doing them more slowly or in smaller chunks.

Instruction and education from both physical and occupational therapists can assist patients if modifications are needed in **activities of daily living (ADLs)** in order to prevent ischemic symptoms. Walking, climbing stairs, vacuuming, raking, and lifting can all be done in a more leisurely way. Washing, carrying, and lifting should be done with fewer items. In their jobs, cardiac patients may have to learn to allot more time to each task.

For some people, anger, frustration, and other strong emotions can cause ischemic episodes. These patients need help in calming their emotions, and they should be referred to therapy programs that emphasize behavioral modification and that provide practical **coping techniques** for stressful situations. In addition, **relaxation techniques**, mental focusing strategies, guided imagery, and yoga have all proven useful in reducing stress for patients with coronary artery disease.

Along with cardiac rehabilitation, patient and family education regarding the management of CAD may include the following:

- **Understanding the warning signs of angina:**
    - Chest pain (may be described as heaviness, tightness, pressure, aching, burning, numbness, fullness, or squeezing)
    - Pain or discomfort in other areas of the upper body, including the arms, left shoulder, back, neck, jaw, or stomach
    - Difficulty breathing or shortness of breath
    - Sweating or “cold sweat”
    - Fullness, indigestion, or a choking feeling (may feel like heartburn)
    - Nausea or vomiting
    - Lightheadedness, dizziness, extreme weakness, or anxiety
    - Rapid or irregular heart beats
- To **call 911 for severe chest pain that does not go away after five minutes**
- **Decreasing risk factors**, including:
    - Smoking or the use of tobacco products
    - High blood cholesterol

  - High blood pressure
  - Uncontrolled diabetes
  - Sedentary lifestyle
  - Being obese or overweight
  - Stress
  - High fat diet
- **Taking medications as directed**
- Understanding that cardiac procedures may be needed (now or in the future) to **treat unstable disease**.
- The importance of **regular visits to the cardiologist**

(Cleveland Clinic, 2012a)

CASE

Linda Ortiz, a 60-year-old Hispanic American woman with a history of type 2 diabetes and hypertension, was recently diagnosed with coronary artery disease. She has come to the doctor's office for a follow-up appointment two weeks after an episode of angina that brought her to the emergency room.

Linda tells the nurse that she has been compliant with her newly prescribed drug regimen of daily aspirin, an ACE inhibitor, and a statin. However, when the nurse asks her about her lifestyle, Linda admits that she has been having a hard time adhering to the recommended lifestyle changes.

The nurse counsels Linda about the importance of smoking cessation, regular exercise, and a sensible diet and gives her referrals to a dietitian and a physical therapist. As the nurse talks with Linda, she also provides her with an educational brochure about the benefits of implementing lifestyle changes, including an example of healthy heart nutrition choices and a diary for Linda to record and track her daily activities, food intake, and medication doses.

The nurse also discusses a goal with Linda to agree to stop smoking within two months and recommends that the doctor prescribe a medication to help her quit. Additionally, the nurse helps Linda establish some exercise goals, starting with moderate walking 15 to 20 minutes every day and gradually progressing to more intense exercise.

Linda has some hesitation with this exercise goal, stating, "I just don't feel like I have the strength to start exercising." After discussing this, the nurse refers Linda to a physical therapist for evaluation of her mobility status and exercise tolerance and to assist Linda in establishing and implementing a realistic long-term physical fitness regimen.

Finally, the nurse has Linda schedule another follow-up appointment in four weeks so they can track and assess her progress on these lifestyle changes.

COMPLICATIONS AND COMORBIDITIES

Diabetes

People with diabetes have a higher incidence of atherosclerotic heart and artery disease than people without diabetes, and 80% of the people with type 2 diabetes die from some form of cardiovascular disease. All coronary artery disease patients with diabetes should be enrolled in a comprehensive diabetes management program. A reasonable goal for patients with diabetes is to reduce their glycosylated hemoglobin (A1C) level to below 7%.

Metabolic Syndrome

CAD is the most striking risk posed by metabolic syndrome. By themselves, the dyslipidemias of metabolic syndrome (i.e., high triglycerides and low HDL cholesterol levels) encourage plaque to form along the walls of arteries. When combined with the other components of metabolic syndrome, these atherogenic dyslipidemias (i.e., those that tend to cause atherosclerotic plaque) put a person at high risk for developing serious atherosclerotic vascular disease with coronary artery blockage.

People who have metabolic syndrome often also have low-level inflammation throughout the body and blood-clotting defects that increase the risk of developing blood clots in the arteries. These conditions contribute to increased risk for cardiovascular disease (NDIC, 2014).

Metabolic syndrome also worsens heart failure, and even when no heart disease is apparent, metabolic syndrome makes a person more likely to develop certain arrhythmias (notably, paroxysmal atrial fibrillation or flutter).

Hyperlipidemia

Even a mildly elevated blood level (fasting level >100 mg/dl) of LDL cholesterol gives patients with CAD a higher risk for myocardial infarctions and sudden cardiac death. Therefore, any degree of hyperlipidemia should be treated in patients with CAD.

Besides lifestyle changes, treatment of hyperlipidemia frequently requires medication. Statins are the recommended drugs for treating high levels of LDL cholesterol, except in people with liver disease. When needed, niacin is added, and further reductions can be achieved by adding fibrates. Patients with CAD should aim for LDL levels of <100 mg/dl (McLaughlin, 2014).

Hypertension

High blood pressure contributes to and worsens atherosclerosis. In a person with coronary artery disease, the goal is to reduce blood pressure to below 130/80 mm Hg (Mayo Clinic, 2014b). The

lifestyle changes recommended for CAD—smoking cessation, regular physical exercise, weight management, improved diet, and stress reduction—will all lower blood pressure.

If these do not reduce a patient's blood pressure sufficiently, then medications are added. Beta blockers are basic drugs for all patients with coronary artery disease, and these agents can also be used to treat hypertension. ACE inhibitors are also used to treat CAD, and these too can be used to treat hypertension.

Depression

Patients with clinical depression or with symptoms of depression have a higher incidence of myocardial infarction, poorer recovery, and a higher rate of mortality (Cowles, 2011). Psychotherapy, behavioral therapy, and serotonin reuptake inhibitors (SSRIs), which are safe to administer after an acute coronary syndrome, have been shown to reduce mortality and improve recovery from myocardial infarctions (Humphreys, 2011).

Immunizations

Patients with CAD are advised to receive a pneumonia vaccine as well as annual vaccination for influenza.

CASE

Mrs. Crawford is a patient on the cardiac unit who is recovering from a myocardial infarction, which was managed medically. She is 72 years old, with a daughter and son who live in the local area. Her family has been to visit throughout her hospitalization. Mrs. Crawford's husband died two years ago from lung cancer.

Claire is her nurse today and is preparing Mrs. Crawford for discharge in 1 to 2 days. Claire is going over the discharge plans and reviewing all of Mrs. Crawford's instructions and medications (including a beta blocker, aspirin, and nitroglycerin as needed for angina). Claire also reviews Mrs. Crawford's exercise program and verifies her appointments to see a physical therapist to continue her cardiac rehabilitation program.

As Claire discusses the transition to the home environment, Mrs. Crawford states, "It's pretty lonesome around the house with my husband gone. My daughter stops by when she can, but she is busy with her work, so she does not have much time." Mrs. Crawford has not progressed as well as expected with her activity level in the hospital. Claire is concerned that Mrs. Crawford may not do well with recovery if she does not progress with her exercise program. She is also concerned that Mrs. Crawford may be depressed.

In order to understand more about how Mrs. Crawford is coping, Claire asks a couple of questions to assess depressive symptoms. Claire asks Mrs. Crawford about her sleep habits, social contacts, and hobbies. When asked directly, "Do you feel like you are blue or depressed

after this heart attack?" Mrs. Brown starts to cry. She says "I just am not sure if I want to go home alone. I am afraid, and I have no one to be with me. I miss my husband."

With this feedback, Claire discusses her assessment with the cardiologist in charge of her care in the hospital. Mrs. Crawford is started on Zoloft therapy (an SSRI, which is safe to take with her cardiac meds). Claire reviews with Mrs. Crawford and her family members which depressive symptoms they should be aware of. Claire also reiterates how important it will be to continue her rehabilitation program because exercise and physical activity may potentially lift her spirits. Claire also encourages Mrs. Crawford to be in close contact with friends, which will give her the opportunity to get out of the house and be with others.

Mrs. Crawford seems to understand the importance of following her rehabilitation plan. Claire confirms her follow-up appointment in one week with the team. Mrs. Crawford and her family verbalize understanding of the discharge plans.

CONCLUSION

Coronary artery disease continues to be one of the most common health problems in the United States and around the globe. Great strides are being made in understanding how to prevent and treat CAD. Treatment outcomes are also improving, which means that more patients are now living with CAD. Therefore, it is vital for all healthcare professionals to understand the key components of managing CAD.

Prevention of CAD begins with understanding individual risk factors and implementing therapeutic lifestyle changes. Weight loss, improved diet, medications, and regular physical exercise are the elements of the initial treatment program. Drugs and interventional procedures are used to treat those components of CAD that do not improve sufficiently with therapeutic lifestyle changes alone.

Patients with CAD are managed by a multidisciplinary team to assure that they have success in treating, managing, and living with the best quality of life and outcomes possible.

QUESTIONS PATIENTS MAY ASK ABOUT CAD

Healthcare professionals who advise patients should know straightforward answers to basic questions. Here are a few important questions and answers about coronary artery disease.

Q: I think I'm having a heart attack, but I'm not sure. Should I call my doctor? Should I drive to the hospital?

A. Don't waste time calling your doctor, and don't take any chances. Don't drive yourself to the hospital. Hang up and call 911 now. Emergency medical technicians can start to treat you on

the way to the hospital. While you wait for the ambulance, if you can take aspirin, chew 1/2 of a regular 325 mg aspirin or two baby aspirins (81 mg each), then sit down and try to relax.

Q: What does a heart attack feel like?

A: Most people get a very uncomfortable pressure, squeezing, or pain in the center of their chest. This chest pain lasts for more than a few minutes; sometimes it goes away briefly, but it soon comes back. Some people feel the pain in their arms, shoulders, back, jaw, or stomach. There can also be a feeling of breathlessness, lightheadedness, cold sweat, or nausea.

Q: How can I tell whether I am a person likely to have a heart attack?

A: One good way is to ask your primary care provider. You can also get an idea by counting how many of the following characteristics apply to you:

1. You have a father, mother, brother, or sister who had heart disease in middle age or earlier.
2. You are older than 45 years if you are a man or older than 55 years if you are a woman.
3. You have high blood cholesterol.
4. You have high blood pressure.
5. You have already had a heart attack, heart pain, heart surgery, stroke, or blocked arteries.
6. You are overweight.
7. You get little or no physical exercise.
8. You smoke cigarettes.
9. You have diabetes.

These are nine things that increase your risk of having a heart attack. The more that apply to you, the greater your chances of heart trouble. Most items on the list can be fixed or controlled. Each thing that you fix will reduce your risk of a heart attack.

Q: Will I die if I have a heart attack?

A: Most people who have heart attacks survive, and the survival rates have been improving as new medicines and new medical procedures are developed. If you have a heart attack, your chances of doing well go way up if you get to an emergency department quickly. If you think you are having a heart attack, don't take chances. Call 911 if your symptoms don't go away in a few minutes.

Q: But I would be embarrassed having an ambulance zooming up to my house with lights flashing and sirens blaring. It would be even worse if I weren't really having a heart attack.

A: Of course, those are normal feelings. The paramedics in the ambulance and the healthcare professionals in the emergency department know that it isn't easy for a person to figure out if they are really having a heart attack. They also know that when people wait too long to get help, they are more likely to die. No one will give you a hard time if you are not actually having a medical crisis.

If there is even a small chance that you could have a heart attack, your primary care provider should have already warned you. Your life is worth more than a little embarrassment, so call 911 if there is any possibility that you might be having a heart problem.

Q: What is a stress test?

A: In a stress test, you exercise in a safe place to see how well your heart handles increased activity. Usually, you walk on a treadmill or pedal a bicycle while a physician watches your pulse rate, your blood pressure, and your ECG.

You will probably be asked to come to the hospital in comfortable clothes and soft shoes. When you arrive in the exercise room, electrode pads will be stuck to the skin of your chest, and the wires will be attached to an ECG machine, which records the electrical activity of your heart.

Then you will exercise—slowly at first, and gradually harder. Your heart rate will get faster, your blood pressure will go up, and you will breathe harder. Meanwhile, the physician will keep an eye on the electrical activity of the heart. If you feel any heart symptoms, the test will be stopped. The goal is to measure exactly how much work (stress) your heart can cope with and, if your heart has difficulty, what specific heart problem is occurring.

Q: I've heard that women get different heart disease than men. Is this true?

A: As far as we know, women and men get the same disease, called coronary artery disease or coronary heart disease. This disease is caused by the same atherosclerosis in both men and women, and it affects the arteries of the heart the same way in everyone.

Just as with men, coronary artery disease is the number one killer of women in the United States. For both men and women, the likelihood of getting heart disease increases as a person gets older. The same factors also increase the chances of getting the disease for both men and women: cigarette smoking, a fat-filled diet, being overweight, having high cholesterol, doing little or no physical exercise, having diabetes, having high blood pressure, and coming from a family that tends to have heart disease.

Nonetheless, there are some differences in how the disease affects men and women. Before menopause, women are less likely to get heart disease than men of the same age. After menopause, a woman's risk increases to levels similar to a man's, but this risk can be reduced earlier in a woman's premenopausal years by improving her lifestyle (stopping smoking, maintaining a moderate weight, eating nutritiously, exercising regularly, keeping her blood pressure low, and treating diabetes).

Another difference between the sexes is that, while many women get the same kind of chest tightening (called angina) as men, women with heart attacks get other symptoms more often than men. When having a heart attack, women are more likely than men to feel sharp chest pains, excess tiredness, dizziness, difficulty breathing, nausea, or indigestion.

Q: I am taking birth control pills. Are they bad for my heart?

A: The older variety of birth control pills had a higher dose of hormones, and this increased a woman's risk of getting a heart attack or a stroke. Today's birth control pills and birth control patches contain lower doses of hormones and do not make women more likely to have a heart attack. The evidence is not entirely clear for strokes, but if there is any increase in the risk of strokes, the increased risk is small.

The safety of modern, low-dose birth control pills is clear for women who don't have any other problems that might put them at risk for heart attacks or strokes. A woman who already has a higher chance than usual of getting heart or artery disease needs to talk with her primary care provider about the best birth control choices for her particular situation.

Q: My doctor says my medicine is a beta blocker. What's that, and what is it blocking?

A: A beta blocker is a drug that slows your heart rate and lowers your blood pressure. This kind of drug blocks the stress caused by the particular nerves that make you tense when you are frightened.

Q: I've heard that trans fats are bad for your heart. What are trans fats?

A: Trans fats are the worst kind of fats for your heart and arteries. Trans fats are also called hydrogenated fats. Most trans fats are manmade and added to processed foods to make the food last longer. On ingredient labels, trans fats are usually called "partially hydrogenated" oils or fats, and on nutrition labels, they are listed as trans fats (usually a subclass of saturated fats). Trans fats are often found in vegetable shortenings, margarines, cakes, crackers, cookies, snack foods (potato chips, corn chips, popcorn), and foods like fried potatoes that have been cooked in partially hydrogenated oils.

When choosing foods, look at the nutrition label. The U.S. Food and Drug Administration offers these practical suggestions about managing the fats in your diet:

- Choose foods with the lowest saturated fat, trans fat, and cholesterol.
- Avoid palm kernel oil.
- Avoid solid shortenings, hard margarines, and animal fat (including butter).
- Monounsaturated and polyunsaturated fats are safe in moderate amounts (examples

of monounsaturated fats are olive oil and canola oil; examples of polyunsaturated fats are soybean oil, corn oil, sunflower oil, and the oils in nuts).

- Fish usually have healthy oils.
- Poultry without the skin has less fat than most cuts of beef.
- Limit your eating of liver, kidney, and egg yolks because they are high in cholesterol.
- Whole-grain foods, fruits, and vegetables are healthy foods.
  (FDA, 2014)

Q: I'm afraid of taking too many medicines. What natural remedies can I use for my coronary artery disease?

A: Your fears are understandable. All medicines have side effects, and all medicines can be dangerous in higher-than-recommended doses. If you are having side effects that make your life difficult or if you are worried about something, then talk directly to your primary care provider. Don't be shy about telling him or her what is bothering you.

Natural remedies, such as herbs and plant or animal extracts, are chemicals just like the medicines that you are taking. "Natural" often means that the chemical is not as pure or as precisely measured as a prescription drug. When the natural remedy is not purified, you are taking all the impurities as well as the chemical—in fact, you don't know exactly what things you are taking, which can be dangerous. When the natural remedy is not as well measured as a prescription drug, you don't know exactly how much you are taking, which can also be dangerous.

There are a couple of herbs and other natural products that are especially dangerous for patients with coronary artery disease:

- Don't take anything with ephedra in it because it puts too much strain on the heart. Ephedra is sometimes found in weight loss products.
- Don't take concentrated licorice or licorice root. Licorice is sometimes used as an herbal remedy for breathing or stomach problems, but it can cause high blood pressure and salt imbalances in your body.
- Don't take danshen, dong quai, evening primrose oil, garlic, ginkgo, ginseng, or St. John's wort, as these may interfere with medications that you are taking for your CAD.
  (Mayo Clinic, 2014d)

Often, there are safe alternatives to the standard therapy for a disease. Talk with your primary care provider and ask for an alternative that is safe.

Q: Is vitamin E good for my heart?

A: At one time, vitamin E supplements were recommended to protect people's hearts. More recently, however, it was found that vitamin E supplements should not be taken to prevent or to treat coronary artery disease.

RESOURCES

About Heart Disease in Women (AHA—Go Red for Women)
https://www.goredforwomen.org/home/about-heart-disease-in-women/

Cleveland Clinic Heart and Vascular Institute
http://my.clevelandclinic.org/heart/

Coronary Artery Disease (AHA)
http://www.heart.org/HEARTORG/Conditions/More/MyHeartandStrokeNews/Coronary-Artery-Disease---Coronary-Heart-Disease_UCM_436416_Article.jsp

Risk Assessment Tool for Estimating Your 10-year Risk of Having a Heart Attack (National Heart, Lung, and Blood Institute)
http://cvdrisk.nhlbi.nih.gov/calculator.asp

What Is Coronary Heart Disease? (National Heart, Lung, and Blood Institute)
http://www.nhlbi.nih.gov/health/health-topics/topics/cad/

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TEST

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1. Atherosclerosis can lead to coronary artery disease by causing:

a. Heart valve failure.
b. Reduced blood flow to the brain.
c. Congestive heart failure.
d. Reduced blood flow to the heart.

2. The path of the two main coronary arteries:

a. Runs along the outside of the heart.
b. Runs along the outside of the pulmonary trunk.
c. Dives directly into the heart muscle.
d. Dives directly into the muscular wall of the pulmonary trunk.

3. Forty to fifty percent of heart attacks are caused by a blockage in the main branch of which coronary artery?

a. The right pulmonary artery
b. The left circumflex coronary artery
c. The right carotid artery
d. The left anterior descending coronary artery

4. Myocardial ischemia results from:

a. Insufficient glucose stored inside the heart muscle cells.
b. High glucose concentration inside the heart muscle cells.
c. Blood flow failing to meet the oxygen demands of the heart.
d. High blood flow to certain regions of the heart.

5. Classic chest pain associated with myocardial ischemia is called:

a. Infarction.
b. Shock.
c. Angina.
d. Stroke.

6. Atherosclerosis thickens the walls of:

a. Capillaries.
b. Arteries.
c. Veins.
d. Lymph vessels.

7. Which is a stage in atherosclerotic plaque formation?

a. Extracellular molecules stimulate blood thinning and bleeding.
b. Deposits of fat in the blood dilate arteries.
c. Extracellular fluid collects in the walls of arteries.
d. Foam cells develop from fat and white blood cells.

8. The rupture or disruption of an atherosclerotic plaque:

a. Reduces the potential for heart ischemia.
b. Releases steroids into the coronary arteries.
c. Allows the blood to safely wash away the unwanted fatty tissue.
d. Causes the formation of blood clots and/or vasospasms.

9. A major contributor to the development of atherosclerosis is high blood levels of:

a. LDL cholesterol.
b. Sodium.
c. Acetylcholine.
d. Lactic acid.

10. Stable ischemic heart disease is a major cause of:

a. Diabetes.
b. Atherosclerosis.
c. Congestive heart failure.
d. High LDL cholesterol levels.

11. The underlying cause of sudden cardiac death is often:

a. From low blood sugar associated with diabetes.
b. The result of external trauma to the heart.
c. The result of a fatal arrhythmia.
d. From the management of stable angina.

12. A common clinical sign of myocardial infarction is:

a. Facial paralysis.

b. Electrocardiogram (ECG) changes.

c. Aphasia.

d. High LDL cholesterol levels.

13. A wife brings her 60-year-old husband to the emergency department (ED) for chest pain that persists despite administering repeat doses of his "heart medicine" and resting for over 10 minutes at home, as directed by his provider. The nurse in the ED documents that the patient's serial electrocardiograms, laboratory blood tests, and vital signs are normal. The patient also tells the nurse that his chest pain is now much improved. The most likely cause of the patient's chest pain is:

a. Myocardial infarction.

b. Stable ischemic heart disease.

c. Unstable angina.

d. Left ventricular hypertrophy.

14. Which finding in the personal health history of a 50-year-old patient places him or her at a higher risk for CAD?

a. Eating a diet high in protein

b. Smoking half a pack of cigarettes daily

c. Drinking a glass of wine daily

d. Regular recreational marijuana use

15. Which is a recommended daily preventive measure for CAD?

a. Aspirin therapy

b. Vitamin E supplements

c. Vitamin C supplements

d. Menopausal hormone therapy

16. Which is a **true** statement about the onset of stable angina?

a. Exercising rarely brings about the pain associated with stable angina.

b. Drinking a glass of wine with dinner commonly triggers stable angina.

c. The pain associated with stable angina has a predictable pattern.

d. The threshold for stable angina is unaffected by the weather, temperature, or time of day.

17. The time course of chest pain caused by acute coronary syndromes is best characterized as:

a. Brief periods of chest pain (2 to 3 minutes).
b. Chest pain that lasts 10 minutes or more.
c. Sharp chest pain lasting a few seconds.
d. Long, steady, and dull ache.

18. When patients describe the feeling of angina, they commonly:

a. Cough.
b. Sigh.
c. Clench their fists.
d. Roll their eyes.

19. The pain or discomfort of angina is **rarely** located:

a. Below the belly button.
b. In the center of the chest.
c. In the shoulder or neck.
d. Down the arm.

20. Women with angina are more likely than men to present with symptoms of:

a. Difficulty breathing.
b. Sharp chest pain.
c. Dull chest pain.
d. Chest heaviness.

21. A patient is being evaluated for symptoms of heart ischemia in the clinic. Which finding indicates a high risk for having coronary artery disease?

a. High HDL cholesterol
b. Metabolic syndrome
c. Hypotension
d. Low LDL cholesterol

22. Which lipid profile result may indicate CAD?

a. A HDL cholesterol level of 70 mg/dl
b. A LDL cholesterol level of 180 mg/dl
c. A total cholesterol level of 165 mg/dl
d. A triglyceride level of 120 mg/dl

23. After a myocardial infarction, cardiac markers indicate damage to the heart muscle by detecting:

a. Light spots on X-rays.
b. Proteins that leak into the blood.
c. Changes in the retina.
d. Lipids that accumulate in the blood.

24. When an emergency response team encounters an adult with acute chest pain outside the hospital setting, the best course of action is to:

a. Confirm a definitive patient diagnosis before beginning treatment.
b. Treat the patient immediately for myocardial ischemia.
c. Transport the patient to the emergency department and then start treatment.
d. Establish IV access only and transport the patient to the emergency department.

25. When a patient is diagnosed with an ST elevation myocardial infarction (STEMI), the next step in managing the patient is to:

a. Wait and watch.
b. Maintain bed rest.
c. Begin the protocol of administering a fibrinolytic drug when chest pain stops.
d. Achieve the goal of reperfusion within 90 minutes of first symptoms.

26. A patient with chest pain is transported to the emergency department. Routine care for the patient includes X-ray films to:

a. Monitor the brain for evidence of stroke.
b. Prevent the need for more invasive procedures.
c. Detect physical injury and any lung or heart changes.
d. Diagnose ischemia and monitor the ischemic damage to the heart muscle.

27. If a patient is having symptoms of acute coronary syndrome, aspirin is:

a. Not administered because it promotes gastrointestinal bleeding.
b. Not administered because it does not improve survival.
c. Administered orally in a low, one-time dose (160–325 mg).
d. Administered by high-dose continuous IV infusion (1,000 mg/hr).

28. The administration of morphine for patients experiencing acute coronary syndrome:

a. Is avoided for nonsurgical pain due to its addictive qualities.

b. Can dangerously increase the heart rate and stress the heart muscle.

c. Reduces chest pain and eases the work of the heart muscle.

d. Is only appropriate when other pain medications fail to work.

29. Which is the most common postoperative complication of cardiac bypass surgery?

a. Pulmonary hypertension

b. Pneumonia

c. Atrial fibrillation

d. Ventricular tachycardia

30. Upon discharge from the hospital, a patient who underwent coronary angioplasty is instructed by the nurse to:

a. Limit walking to less than 10 minutes per day.

b. Monitor for signs of angina, dyspnea, and dizziness.

c. Schedule a later visit with the primary care provider to understand cardiac drug therapy.

d. Resume a regular diet once swallowing returns to normal.

31. The primary long-term treatment goals for patients with CAD include:

a. Limiting physical function to reduce angina pain.

b. Stabilizing or reversing progression of atherosclerosis.

c. Gradually decreasing cardiac medications.

d. Providing medications to relieve angina pain.

32. Aspirin therapy is initiated for patients with a history of CAD to:

a. Relieve chest pain.

b. Reduce blood pressure.

c. Inhibit blood clot formation.

d. Prevent chest pain.

33. Nitroglycerin increases blood flow to the heart by:

a. Constricting coronary arteries.

b. Dilating coronary arteries.

c. Decreasing sodium levels in the body.

d. Decreasing potassium levels in the body.

34. Which medication is contraindicated when taking sublingual nitroglycerin tablets?

a. Viagra
b. Plavix
c. Beta blockers
d. Aspirin

35. Beta blockers are classified as:

a. Antihypertensive drugs.
b. Lipid-lowering drugs.
c. Oral hypoglycemic drugs.
d. Fibrinolytic drugs.

36. Older adult patients who participate in exercise-based cardiac rehabilitation programs benefit through improvement in functional capacity, glucose control, quality of life, and the ability to perform:

a. Activities of daily living.
b. Strenuous physical activities.
c. Repetitive muscle contractions.
d. Quick movements in a short period of time.

37. What is a lifestyle change patients with CAD can implement to improve their health?

a. Avoiding exercise training
b. Participating in risk factor reduction counseling
c. Scheduling revascularization surgery
d. Taking drugs to increase oxygen delivery to the heart

38. Weight loss in an overweight patient with CAD can lead to an improved blood lipid profile, lower blood pressure, and:

a. Greater absorption of nutrients.
b. Increased caloric expenditure during exercise.
c. Reduced severity of angina.
d. Lower likelihood of smoking cessation.

39. Patients with severe angina lasting more than five minutes should be instructed to:

a. Meditate to calm the mind and body.
b. Give up all activities that cause chest pain.
c. Stay home and rest as much as possible.
d. Call 911 and report it as an emergency.

40. The psychiatric disorder most associated with poor recovery from a myocardial infarction is:

a. Schizophrenia.
b. Mania.
c. Depression.
d. Neurosis.