Heart Disease: Coronary Artery Disease (CAD)

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

- Identify the statistics and impact of coronary artery disease.
- Understand the anatomy, normal blood circulation and electrical system of the heart.
- Understand the pathophysiology of CAD.
- Identify the major clinical signs and symptoms of acute cardiac syndrome.
- Identify non-preventable and preventable risk factors.
- List methods that can reduce preventable risk factors.
- Identify the signs, symptoms, and clinical test used to screen and diagnose coronary artery disease.
- Understand the clinical indications for PCI and CBAG.
- Understand the components of a comprehensive plan of care and monitoring for patients.
- List the complications, comorbidities, and other diseases associated with CAD.

Heart Disease:

- Heart disease is the leading cause of death for both men and women worldwide.
- Heart disease kills more people annually then all forms of cancer combined.
- Over 600,000 people die of heart disease in the United States every year—that’s 1 in every 4 deaths.
- Coronary heart disease (CHD) also called coronary arterial disease (CAD) is the most common type of heart disease, killing more than 370,000 people each year.
- CAD can lead to heart attacks, over 700,000 Americans have heart attacks and approximately 210,000 of those individuals are people who have already had a heart attack.
- In the United States, someone has a heart attack every 43 seconds. Each minute, someone in the United States dies from a heart disease-related event.
- Heart disease is the leading cause of death for people of most racial/ethnic groups in the United States, including African Americans, Hispanics, and whites. For Asian Americans or Pacific Islanders and American Indians or Alaska Natives, heart disease is second only to cancer.
- Coronary heart disease alone costs the United States $108.9 billion each year. This total includes the cost of health care services, medications, and lost productivity.

Anatomy of the Heart

The heart is the hollow, muscular organ in the thoracic cavity (chest) that maintains the circulation of blood throughout the body. It is surrounded by a membrane called the pericardium. The pericardium consists of a layer of fibrous connective tissue and a layer of thin, serous (i.e., produces a secretion) tissue and is attached to the vena cava, the aorta, the diaphragm, and the sternum. The pericardial cavity—the potential space between the pericardium and the heart—contains the watery pericardial fluid. This fluid prevents friction between the pericardium and the heart. The heart wall consists of the epicardium (inner layer), the myocardium (middle layer comprised of cardiac muscle tissue), and the endocardium (lining of the myocardium that covers the heart valves). The heart has a right side and a left side. Each side has a relatively thin-walled chamber that receives blood returning to the heart (atrium) and a muscular chamber that pumps blood out of the heart (ventricle).
Blood Flow

The flow of blood through the heart is controlled by the opening and closing of valves and the contraction and relaxation of the myocardium. Heart valves are controlled by pressure changes within each chamber and contraction and relaxation are controlled by the heart's conduction system. Blood that has traveled through the body returns to the heart and enters the right atrium. This blood flows through the tricuspid valve into the right ventricle. The right ventricle pumps the blood to the lungs, where it absorbs oxygen. Oxygen-rich blood returns from the lungs and enters the heart through the left atrium. Blood passes from the left atrium through the mitral valve and into the left ventricle. The left ventricle, the largest and most muscular of the four chambers, is the main pumping chamber of the heart. As the left ventricle contracts, blood is pumped through the aortic valve into the main artery of the body (aorta). The aorta supplies blood to smaller arteries that travel to the head, arms, abdomen, and legs. These arteries supply oxygen-rich blood to the organs and tissues of the body, which require oxygen to function. The coronary arteries supply oxygen-rich blood to the tissues of the heart. Oxygen-poor blood travels from organs and tissues to the heart through veins. The vena cava is the major vein that returns blood to the right atrium of the heart. The vena cava superior returns blood from the head, neck, upper extremities, and chest. The vena cava inferior returns blood from the lower extremities, the pelvis, and the abdomen. The coronary sinus drains blood from the coronary arteries into the right atrium.

Conduction System

An electrical impulse travels through the heart and initiates contractions of the chambers. The heart's "spark plug" is an area of specialized heart tissue called the sinoatrial node (SA node), which is located in the right atrium. Each time the SA node "fires," an electrical impulse is generated that travels through the right and left atria, signaling these chambers to contract and pump blood into the ventricles. The impulse then travels into another area of
specialized heart tissue called the atrioventricular node (AV node), which is located between the atria and the ventricles. The electrical impulse is conducted through the AV node and wire-like pathways (Purkinje fibers) to the ventricles, signaling the ventricles to contract and pump blood into the lungs and throughout the body. The normal sequence of electrical activation of the chambers of the heart is called sinus rhythm. It occurs each time the heart beats, usually about 60 to 80 times every minute. In a normal heartbeat, the atria contract simultaneously while the ventricles relax. Then, the ventricles relax and the atria contract. The term systole refers to contraction and the term diastole refers to relaxation. A heartbeat consists of the systole and diastole of the atria and the systole and diastole of the ventricles.

Heart Muscle

Cardiac muscle (heart muscle) is involuntary striated muscle that is found in the walls and histological foundation of the heart, specifically the myocardium. The cells that constitute cardiac muscle, called cardiomyocytes or myocardiocytes, contain only three nuclei. The myocardium is the muscle tissue of the heart, and forms a thick middle layer between the outer epicardium layer and the inner endocardium layer.

Coordinated contractions of cardiac muscle cells in the heart propel blood out of the atria and ventricles to the blood vessels of the left/body/systemic and right/lungs/pulmonary circulatory systems. This complex mechanism illustrates systole of the heart.

Cardiac muscle cells, unlike most other tissues in the body, rely on an available blood and electrical supply to deliver oxygen and nutrients and remove waste products such as carbon dioxide. The coronary arteries help fulfill this function.

Blood Supply to the Heart

The myocardium (heart) is a very specific type 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. Occlusion (blockage) of the coronary arteries by atherosclerosis and/or thrombosis can lead to myocardial infarction (heart attack), where part of the myocardium is injured due to ischemia (not receiving enough oxygen). This occurs because coronary arteries are functional end arteries - i.e. there is almost no overlap in the areas supplied by different arteries (anastomoses) so that if one fails, others cannot adequately perfuse the region, unlike in other tissues.
Coronary circulation is the circulation of blood in the blood vessels of the myocardium. The vessels that deliver oxygen-rich blood to the myocardium are known as coronary arteries. The vessels that remove the deoxygenated blood from the heart muscle are known as cardiac veins. These include the great cardiac vein, the middle cardiac vein, the small cardiac vein and the anterior cardiac veins.

As the left and right coronary arteries run on the surface of the heart, they can be called epicardial coronary arteries. These arteries, when healthy, are capable of autoregulation to maintain coronary blood flow at levels appropriate to the needs of the heart muscle. These relatively narrow vessels are commonly affected by atherosclerosis and can become blocked; causing angina or a heart attack. The coronary arteries that run deep within the myocardium are referred to as subendocardial.

The coronary arteries are classified as "end circulation", since they represent the only source of blood supply to the myocardium; there is very little redundant blood supply, which is why blockage of these vessels can be so critical.

Left coronary artery

The left coronary artery is an artery that arises from the aorta above the left cusp of the aortic valve and feeds blood to the left side of the heart. It is also known as the left main coronary artery and the left main stem coronary artery.

It typically runs for 10 to 25 mm and then bifurcates into the anterior interventricular artery (also called the left anterior descending and the "Widow maker") and the left circumflex artery. Sometimes an additional artery arises at the bifurcation of the left main artery, forming a trifurcation; this extra artery is called the ramus or intermediate artery.

The part that is between the aorta and the bifurcation only is known as the left main artery, while the term 'LCA' might refer to just the left main, or to the left main and all its eventual branches.

Right coronary artery

The right coronary artery (RCA) is an artery originating above the right cusp of the aortic valve. It travels down the right atrioventricular groove, towards the crux of the heart. It branches into the posterior descending artery and the right marginal artery. Although rare, several anomalous courses of the right coronary artery have been described including origin from the left sinus of valsalva. At the origin of the RCA is the conus artery. In addition to supplying blood to the right ventricle (RV), the RCA supplies 25% to 35% of the left ventricle (LV).

In 85% of patients (Right Dominant), the RCA gives off the posterior descending artery (PDA). In the other 15% of cases (Left Dominant), the PDA is given off by the left circumflex artery. The PDA supplies the inferior wall, ventricular septum, and the posteromedial papillary muscle.

The RCA also supplies the SA nodal artery in 60% of patients. The other 40% of the time, the SA nodal artery is supplied by the left circumflex artery.

Volume of Blood Flow to the Heart

The blood flow through the heart depends on the demand of the body. 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.,
normally, the extra oxygen needed during exercise is supplied by a faster and a more voluminous blood flow through the coronary arteries.

Heart rate directly affects blood flow; increased blood flow is a direct result of a faster heart rate. Blood flow to the heart automatically speeds up as the heart beats quicker because the coronary arteries are fed directly by the outflow of the heart. Blood flow to the heart is crucial, obstruction of blood flow to the LCA or the RAC can cause ischemia and can lead to a heart attack and be fatal.

Heart Disease

Heart disease in simplest terms means that the heart is not working normally. Babies can be born with heart disease which is a condition referred to as congenital heart disease. Heart disease may involve the valves in the heart, or the heart may not pump well and cause heart failure. The heart rhythm can also be affected, causing arrhythmias. Coronary heart disease (CHD) which is also called coronary artery disease (CAD) is the most common type of heart disease. This condition results from a buildup of plaque on the inside of the arteries, which reduces blood flow to the heart and increases the risk of a heart attack and other heart complications. Other forms of heart disease include:

- irregular heartbeat (arrhythmias)
- congenital heart defects
- weak heart muscles (cardiomyopathy)
- heart valve problems
- heart infections
- cardiovascular disease

Coronary artery disease (CAD)

CAD is a condition in which plaque, which is made up of fat, cholesterol, calcium and other substances in the blood, builds up inside the coronary arteries which supply oxygen-rich blood to the heart muscle. This plaque build-up is caused by atherosclerosis. Atherosclerosis develops slowly over time. From a young age, cholesterol-laden plaque can start to deposit in the blood vessel walls. Over time, the plaque burden builds up, inflaming the blood vessel walls and raising the risk of occlusions or blood clots that can lead to a heart attack. The plaques release chemicals that promote the process of healing but make the inner walls of the blood vessel sticky. Then, other substances, such as inflammatory cells, lipoproteins, and calcium that travel in your bloodstream start sticking to the inside of the vessel walls.

Eventually, a narrowed coronary artery may develop new blood vessels that go around the blockage to get blood to the heart. However, during times of increased exertion or stress, the new arteries may not be able to supply enough oxygen-rich blood to the heart muscle. In some cases, a blood clot may totally block the blood supply to the heart muscle, causing heart attack.

Atherosclerosis

Atherosclerosis is a condition that develops when plaque builds up in the walls of the arteries. This buildup narrows the arteries, making it harder for blood to flow through. Atherosclerosis is the underlying disorder that causes CAD. Atherosclerosis thickens the walls of the coronary arteries. The atherosclerotic thickenings occur as bulges due to plaque formation 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.
Fat and cells collect in bulges just below the surface of the lining of arteries. These bulges that are created 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.

Atherosclerosis usually begins in childhood or adolescence and then gradually worsens over time. Any medium or large artery in the body can be affected. For the most part atherosclerosis causes no clinical problems. Many people have atherosclerosis throughout their bodies but develop no serious medical symptoms, and the disease is only discovered postmortem.

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 non-heart arteries, a person can develop portal artery disease (PAD).

- In the aorta, it can lead to aneurysms.
- In the carotid arteries, PAD can cause strokes.
- In the legs, it can cause episodic pain when walking 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.

**PLAQUE FORMATION**

Atherosclerosis derives its name from the Greek words ‘sclerosis’ meaning hardening and ‘athere’ meaning gruel (accumulation of lipid). The phenomenon is characterized by accumulation of cholesterol, infiltration of macrophages, proliferation of smooth muscle cells (SMC), and accumulation of connective tissue components and formation of thrombus. The development of plaque occurs slowly over time and in stages. Atherosclerotic plaques begin with the appearance of fatty streaks along artery walls. As the disease progress foam cells develop followed by smooth muscle cells start moving into the expanding plaques. The last stage blood clots start to form and can cause major health issues.

Stage 1: Fatty Streaks- accumulation of cholesterol

As atherosclerosis begins, the first notable changes are the appearance of fatty streaks in the artery walls. Excess fat accumulates creating these streaks.
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 2: Foam Cells—macrophages

Foam cells are formed when the body sends macrophages to the location of a fatty deposit on the blood vessel walls. Foam cells develop as white blood cells are attracted to these unhealthy accumulations of lipids. Phagocytes which are white blood cells attack and 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 3: Smooth Muscle Cells—SMC

Yellow fatty streaks containing foam cells continue to grow and thicken. Soon smooth muscle cells from deeper in the arterial walls move into the enlarging plaque. 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.

Stage 4: Blood Clots—thrombus begin to form

As the disease progresses, the endothelial cells covering the bulge begin to tear, letting blood come in contact with the underlying collagen and other extracellular molecules. Extracellular molecules are stimulants of blood clotting. Small blood clots and clumps of platelets form along the tear 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 occlusion of an 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).
**Blood Clots**

A blood clot is a normal function of blood cells that is used to repair damaged blood vessel walls. Blood clots become a problem when the blood "clots" are in an artery or vein inappropriately. As atherosclerotic plaques bulge form they narrow the space in the arteries for blood to flow. Under some conditions, these plaques also generate blood clots and vasospasms.

**Vasospasms**

Vasospasm is a condition in which a blood vessel's spasm leads to vasoconstriction. This can lead to tissue ischemia and tissue death (necrosis). Along with physical resistance, vasospasm is a main cause of ischemia. Like physical resistance, vasospasms can occur due to atherosclerosis. Vasospasm is the major cause of chest pain (angina).

The rupture of a plaque can also cause vasospasms in that region. The resulting vasospasm narrows the artery suddenly and causes ischemia downstream. The results together of 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).

**Symptoms of Coronary Artery Disease:**

As the coronary arteries narrow, they can't supply enough oxygen-rich blood to the heart — especially when it's beating hard, such as during exercise. Initially, the decreased blood flow may not cause any coronary artery disease symptoms. As the plaques continue to build up in the coronary arteries, signs and symptoms may appear. Coronary artery disease signs and symptoms, including:

1. **Angina:** Chest pain, pressure or tightness in chest. The pain, referred to as angina, is usually triggered by physical or emotional stress. It typically goes away within minutes after stopping the stressful activity. In some people, especially women, this pain may be fleeting or sharp and felt in the abdomen, back or arm.
2. **Shortness of breath.** If the heart muscle can't pump enough blood to meet the body's needs, shortness of breath may occur or extreme fatigue with exertion.
3. **Myocardial Infarction or Heart attack.** A completely blocked coronary artery may cause a heart attack. The classic signs and symptoms of a heart attack include crushing pressure in the chest and pain in your shoulder or arm, sometimes with shortness of breath and sweating. Women are somewhat more likely than men are to experience less typical signs and symptoms of a heart attack, such as nausea and back or jaw pain. A heart attack may occur without any apparent signs or symptoms.

**Key Risk Factors for Heart Disease**

- High blood pressure
- High cholesterol
- Smoking
- Diabetes and prediabetes
- Overweight or obese
- Sedentary life style
- Having a family history of early heart disease
- Having a history of preeclampsia during pregnancy
- Poor diet
- Age (55 or older for women)
Some risk factors, such as age and family history of early heart disease, can't be changed. For women, age becomes a risk factor at 55. After menopause, women are more apt to get heart disease, in part because their body's production of estrogen drops. Women who have gone through early menopause, either naturally or because they have had a hysterectomy, are twice as likely to develop heart disease as women of the same age who have not yet gone through menopause.

Another reason for the increasing risk is that middle age is a time when women tend to develop risk factors for heart disease. Family history of early heart disease is another risk factor that can't be changed. If your father or brother had a heart attack before age 55, or if your mother or sister had one before age 65, you are more likely to get heart disease yourself. Preeclampsia is another heart disease risk factor. This condition may occur in some women during pregnancy, characterized by high blood pressure, sometimes with fluid retention and proteinuria.

**Impact of Heart Disease**

Coronary artery disease is the most common form of heart disease. CAD Impacts both men and women as the leading cause of death. 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 CAD. CAD is the number one killer in the developed world, with over 7.4 million deaths attributed to CAD in 2012 according to World Health Organization. In the United States, it is estimated that one in seven deaths is due to heart disease. The American Health Association reports that heart disease is the primary cause of death in women, taking more lives than all cancers combined. 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.

**Atherosclerosis of the Coronary Arteries**

Atherosclerosis develops in a patchy pattern and is unevenly distributed within the arterial walls. The specific coronary arteries affected by the disease vary in each individual, but there are common features. Most frequently plaques are found at branch points, places where the blood flow naturally becomes turbulent.

The narrowing of coronary arteries develops slowly, as blood flow through the arteries decrease; new small collateral arteries have time to grow into the fields of the atherosclerotic arteries to help increase 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. As the disease progress the coronary arteries continue to narrow despite the small collateral arteries growth, eventually causing ischemia and angina. Initially, these symptoms occur only with exercising or emotional stress; later, the symptoms begin to occur even when the patient is at rest. Atherosclerosis can also cause a sudden medical emergency. The degeneration of a plaque can cause clots into the bloodstream and can also trigger local vasospasm. These can lead to significant reduction of blood flow, and the resulting damage can range from temporary to permanent and even cause a fatal event.

**Risk Factors for Atherosclerosis**

Atherosclerosis can occur in any individual, but some people have a genetic predisposition for developing the disease. Dietary fat is a major contributing factor in developing atherosclerosis. Cholesterol that is carried in the blood is a critical causative agent. Low-density lipoprotein or LDL cholesterol: Low-density lipoprotein cholesterol commonly referred to as ‘bad’ cholesterol. Elevated LDL levels are associated with an increased risk of developing atherosclerosis. Lipoproteins, which are combinations of fats (lipids) and proteins, are the form in which lipids are transported in the blood. High blood levels of LDL cholesterol can cause and worsen atherosclerosis. Other factors that contribute to atherosclerosis are cigarette smoking, high blood pressure, type 2 diabetes, age, gender, physical inactivity, and obesity.

**Heart Disease Associated Terms:**

The most common term is Coronary artery disease (CAD) but often referred to as:
Coronary heart disease (CHD)
Ischemic heart disease (IHD)
Coronary atherosclerotic disease
Atherosclerotic heart disease

Atherosclerosis often is also referred to as:

- Hardening of the arteries
- Narrowing of the arteries

Main types of coronary artery disease are:

Acute Coronary Syndromes
Three types of heart attack occur because of sudden rupture of plaque inside the coronary artery. These depend on the location of blockage, amount of time blood flow is blocked, and damage that occurs. Emergency medical care is critical for these life-threatening conditions.

Advanced Ischemic Heart Disease
Patients with aggressive heart conditions are at risk for heart failure and arrhythmias. They may have already had at least one coronary artery bypass surgery, multiple stents or angioplasty procedures, or still suffer from chest pain even after receiving optimum treatment.

Bifurcation Blockage
Fatty build-up is more likely to occur in the Y-junction where vessels branch off from the main coronary artery because of changes in blood flow. Narrowing in this region is called bifurcation blockage, and it is treated using special techniques to prop up the vessel.

Microvessel Disease
This type occurs when tiny blood vessels narrow when they should widen. This starves the heart muscle of oxygen and causes chest pain that can impact quality of life. This disease affects women and can be treated by medications to ease pain and lifestyle changes.

Total Coronary Occlusion
A complete blockage in a coronary artery can lead to heart attack because of restricted blood flow to the heart muscle. When the blockage is more than three months old, it is called a chronic coronary occlusion.

Chronic stable Angina
Stable angina is chest pain or discomfort that most often occurs with activity or stress. Angina last a short period of time and goes away with rest.

Three types of acute coronary syndromes are:

- Unstable Angina
- Heart Attack (Myocardial Infarction)
- Sudden cardiac death

1. Unstable angina: Unstable angina is chest pain that happens suddenly and becomes worse over time. It occurs seemingly without cause—can occur at rest or even during sleep. An attack of unstable angina may lead to a heart attack. For this reason, an attack of unstable angina should be treated as an emergency, and patient should seek immediate medical treatment.

   Risk Factors: Diabetes, obesity, a family history of heart disease, high blood pressure, high low-density lipoprotein (LDL) cholesterol, low high-density lipoprotein (HDL) cholesterol, being male, using any form of tobacco, leading a sedentary life. Men 45 and older and women 55 and older are more likely to experience unstable angina.

   Symptoms: The main symptom of unstable angina is chest discomfort or pain. Patient may describe the angina as squeezing or as sharp chest pain. Pain may radiate to extremities or back. Shortness of breath, nausea, anxiety, sweating, dizziness or unexplained fatigue is also symptoms of unstable angina. Patients taking nitroglycerin to
enhance blood flow, during stable angina attacks may find that the medication doesn’t work during unstable angina attacks.

Diagnosis: Physical exam, blood work and other test will be used to diagnosis unstable angina. Some tests that may be performed include: Blood tests to check for cardiac biomarkers (troponin) and enzymes creatine kinase (CK) that leak from the heart muscle if it has been damaged. Electrocardiogram to see patterns in the heartbeats that indicate reduced blood flow. Echocardiography is used to produce images of the heart to check for angina-related problems. Stress tests, computed tomography angiography, coronary angiography and heart catheterization may also be used to evaluate the health of the heart muscle and its arteries. Because coronary angiography can help doctors visualize any artery narrowing and blockages, it’s one of the most common tests to diagnose unstable angina.

Treatment: Treatment for unstable angina depends on the severity. Patient’s physician may recommend blood thinner such as heparin or clopidogrel to improve blood flow. Physician may use other medications to reduce angina symptoms, including those that reduce: blood pressure, cholesterol, anxiety and arrhythmias.

Patients with blockage or severe narrowing in an artery may need more invasive procedures. These include angioplasty, where they open up an artery that was previously blocked. A stent may also be used to keep the artery open. In severe instances, heart bypass surgery may be needed, which reroutes blood flow away from a blocked artery to help improve blood flow to your heart.

Prevention (nonmedical): Healthier lifestyle can improve heart health and reduce the risk of future unstable angina episodes. Healthy diet and eating habits can keep cholesterol in check. Maintain proper LDL and HDL levels. Regular exercise, losing weight and quitting tobacco use can also reduce risk.

Stable Angina vs Unstable Angina

Although stable and unstable angina may have similar symptoms, they differ in terms of severity of pain and when the symptoms occur. Stable angina is chest discomfort, shortness of breath that happens with a predictable, reliable amount of exertion or stress, and when that pattern has been present for more than four weeks. Stable angina usually starts when you exert yourself or feel stressed. If you stop what you are doing, the pain or discomfort usually stops too. Activity—exercise, eating a big meal, or having an argument—makes your heart rate go up and your blood pressure higher, so your heart works harder. To do the work, the heart needs more oxygen. If it is not getting enough, it can cause the pain and discomfort of angina. When your pattern of angina has been stable for several months, it may be referred to chronic stable angina.

Unstable angina is when symptoms of chest pressure, shortness of breath (or any of the others described above) occur for the first time, or have been happening for less than two weeks. Also, if a change in the usual pattern of angina occurs with exertion, that also is unstable angina. Unstable angina can happen any time, they can occur at rest sitting in front of the television or during sleep. It’s hard to ignore. If the symptoms stop, they usually return again soon.

Stable angina can also become unstable. For instance, if the patient usually has chest discomfort every time they walk two blocks, that would be considered stable angina. However, if that pattern of chest discomfort changes over the course of a short period of time, then the angina has become unstable. For example, if discomfort comes on with less activity (after walking only half of a block instead of two) or is occurring more frequently than previously, that would be an example of stable angina that has become unstable.

Things to consider when dealing with stable vs unstable angina

Stable Angina
➢ is pain or discomfort similar to past episodes of angina with similar amounts of exertion and usually resolves in less than five minutes.
➢ is chest pain or other symptoms that usually stop after you take medication or stop to rest.
➢ is triggered by activities that make the heart work harder—physical and emotional exertion or stress, extreme temperatures, or a big meal.

Unstable Angina

➢ can happen anytime. You could be taking a nap or having a cup of coffee.
➢ may feel different than the pain or discomfort of stable angina.
➢ is often more painful or severe and lasts longer than stable angina—more than a few minutes.
➢ may not go away with rest or use of angina medication.

2. Heart Attack (Myocardial Infarction): “Myo” means muscle and cardial refers to the heart. Infarction means death of tissue due to lack of blood supply. Heart attack is myocardial infarction, and it causes permanent damage to the heart muscle.

Risk Factors: Age, men age 45 or older and women age 55 or older are more likely to have a heart attack than are younger men and women. Other risk factors include: tobacco use, high blood pressure, high blood cholesterol or triglyceride levels, family history of heart attack, sedentary lifestyle, stress, illegal drug use (cocaine or amphetamines), history of preeclampsia or a history of an autoimmune condition, such as rheumatoid arthritis or lupus.

Symptoms: The symptoms of a heart attack can vary. Sometimes heart attacks are sudden and excruciating, but often, they start out slow with mild discomfort. According to the National Heart, Lung, and Blood Institute, one study found that one-third of people who had a heart attack had no chest pain. These people were likely to be older, female, or diabetic. Some people have no symptoms. This is called a silent heart attack. The most common symptoms of a heart attack include: pressure, tightness, pain or squeezing or aching sensation in the chest. The sensation may also be present in the arms and spread to the neck, jaw or back. Nausea, indigestion, heartburn, abdominal pain, shortness of breath, fatigue, sudden dizziness and fatigue are common during a heart attack as well.

Diagnosis: In an emergency setting with symptoms of a heart attack physical exam will include blood pressure, pulse, and temperature along with the use of a heart monitor. Electrocardiogram (ECG) is used to check the electrical activity of the heart via electrodes attached to the skin. Impulses are recorded as waves displayed on a monitor or printed on paper. Because injured heart muscle doesn't conduct electrical impulses normally, the ECG may show that a heart attack has occurred or is in progress. Blood test will be performed to check for certain heart enzymes that slowly leak out into the blood if your heart has been damaged by a heart attack. Additional test may include chest X-rays, echocardiogram, coronary catheterization (angiogram), exercise stress test (days or weeks after episode), cardiac CT or MRI.

Treatment: Heart attack treatment at a hospital

The immediate goal of treatment is to prevent heart damage. With each passing moment after a heart attack, more heart tissue loses oxygen and is damaged or dies. The main way to prevent further heart damage or tissues death is to restore blood flow as quickly as possible.

The use of medications plays a key role in helping promote blood flow. Aspirin may be suggested by a 911 operator in an emergency call situation if a heart attack is suspected. Aspirin reduces blood clotting, thus helping maintain blood flow through a narrowed artery. Thrombolyitics also called clotbusters, help dissolve a blood clot that's blocking blood flow to the heart. The earlier a patient receives a thrombolytic drug after a heart attack, the greater the chance of survival and with less heart damage. Antiplatelet agents may be given to help prevent new clots and
keep existing clots from getting bigger. These include medications, such as clopidogrel (Plavix) and others, called platelet aggregation inhibitors. Blood-thinning medications like Heparin may be used to make the blood less “sticky” and less likely to form clots. Pain relievers may be used to reduce discomfort. Nitroglycerin maybe used to help reduce angina and can help blood flow to the heart by dilating the blood vessels. Beta blockers help relax the heart muscle, slow heart rate and decrease blood pressure, allowing your heart to work easier. Beta blockers can limit the amount of heart muscle damage and prevent future heart attacks. Ace inhibitors lower blood pressure and reduce stress on the heart.

In addition to medications, surgery or other procedures may be needed. A coronary angioplasty and stenting might be necessary. After a cardiac catheterization is performed to locate the blockage, the coronary angioplasty and stenting may be performed. A catheter is placed through the artery in the leg or groin to the blocked artery in the heart. The catheter equipped with a special balloon will be inflated to open a blocked coronary artery. A metal mesh stent may be inserted into the artery to keep it open long term, restoring blood flow to the heart. Depending on the condition of the patient, a stent coated with a slow-releasing medication to help keep the artery open may be used.

In some cases a coronary artery bypass surgery may necessary, if possible three to seven days after the heart attack so that the heart has time to recover. Emergency bypass surgery may be performed at the time of the heart attack. Bypass surgery involves sewing veins or arteries in place beyond a blocked or narrowed coronary artery, allowing blood flow to the heart to bypass the narrowed section. Once blood flow to the heart is restored and the patient’s condition is stable, patients are likely to remain in the hospital for several days.

Prevention: Lifestyle affects the patient’s heart health. Preventing heart attacks and recovering from a heart attack requires lifestyle changes. Key factors to preventing and recovering from a heart attack include: Avoid smoking, control blood pressure and cholesterol levels, regular medical checkups, exercise regularly, maintain a healthy weight, eat a heart-healthy diet, manage diabetes, control stress, and alcohol use only in moderation.

Heart-healthy diet: Saturated fat, trans fats and cholesterol rich diet can narrow arteries to the heart, and too much salt can raise blood pressure. Eating a heart-healthy diet includes lean proteins, such as fish and beans, plenty of fruits and vegetables and whole grains.

3. Sudden cardiac death: Sudden cardiac death (SCD) is a sudden, unexpected death caused by loss of heart function (sudden cardiac arrest). Sudden cardiac death is the largest cause of natural death in the United States, causing about 325,000 adult deaths in the United States each year.

Risk Factors: A family history of coronary artery disease, smoking, high blood pressure, high blood cholesterol, obesity, diabetes, sedentary lifestyle, drinking too much alcohol (more than one to two drinks a day). Other factors that contribute to increase risk of sudden cardiac arrest include: A previous episode of cardiac arrest or a family history of cardiac arrest. Having a prior heart attack increases risk. A personal or family history of other forms of heart disease, such as heart rhythm disorders, congenital heart defects, heart failure and cardiomyopathy. Age — the incidence of sudden cardiac arrest increases with age, especially after age 45 for men and age 55 for women. Being male — men are two to three times more likely to experience sudden cardiac arrest. Caffeine or amphetamine use. Poor diet with nutritional imbalance, such as low potassium or magnesium levels.

Cause: Abnormal heart rhythms or arrhythmias are the most common cause of sudden cardiac death. Ventricular fibrillation is the most common life-threatening arrhythmia, which is an erratic, disorganized firing of impulse from the ventricles. The result is an inability of the heart to pump blood and death occurs within minutes if not treated immediately.

Symptoms such as racing heartbeat, feeling dizzy or feeling faint may occur when experiencing sudden cardiac arrest. Loss of consciousness, with no heartbeat (or pulse) occurs. Chest pain, shortness of breath, nausea, or vomiting may have been experienced within one hour prior to the event. In fifty percent of the cases, sudden cardiac arrest occurs without prior symptoms.
Sudden Cardiac Arrest: Sudden cardiac arrest (SCA) is a condition in which the heart suddenly and unexpectedly stops beating. If this happens, blood stops flowing to the brain and other vital organs. If not treated within minutes SCA usually results sudden cardiac death.

SCA is caused by problems with the heart’s electrical system that controls the rate and rhythm of the heartbeat. Irregular heartbeats also known as arrhythmias can cause the heart to beat too fast, too slow, or with irregular rhythm. In some cases the arrhythmias can cause the heart to stop pumping blood to the body which causes SCA. Unlike in the event of a heart attack which occurs if blood flow to part of the heart muscle is blocked, the heart doesn’t suddenly stop beating. SCA, however, may occur after or during recovery from a heart attack. Individuals with known heart disease are at high risk of SCA. SCA can also occur in individuals who appear healthy and have no known heart disease or other risk factors for SCA.

Most individuals who have SCA event die from it—often within minutes. Rapid treatment of SCA with a defibrillator can be lifesaving. A defibrillator sends an electric shock to the heart to try to restore its normal rhythm. Automated external defibrillators (AEDs) can be used to save the lives of people who are having SCA. These portable devices often are found in public places, such as shopping malls, golf courses, businesses, airports, airplanes, casinos, convention centers, hotels, sports venues, and schools.

The most life-threatening form of arrhythmia is Ventricular fibrillation (V-fib), which causes most sudden cardiac arrests. During v-fib, the ventricles of the heart don't beat normally. Instead, they quiver very rapidly and irregularly. When this happens, the heart pumps little or no blood to the body. V-fib is fatal if not treated within a few minutes.

Other problems with the heart's electrical system also can cause SCA. SCA can occur if the rate of the heart's electrical signals becomes very slow and stops. SCA also can occur if the heart muscle doesn't respond to the heart's electrical signals.

Certain diseases and conditions can cause the electrical problems that lead to SCA. Examples include coronary heart disease, severe physical stress, certain inherited disorders, and structural changes in the heart.

Arrhythmia: An irregular rhythm of the heart caused by the malfunction of the heart's electrical system. Tachycardia, bradycardia, skipped heartbeat and fluttering heart are all examples of an arrhythmia. Arrhythmias can be life-threatening or harmless. Most arrhythmias are harmless, but some can be serious or even life threatening. During an arrhythmia, the heart may not be able to pump enough blood to the body. Lack of blood flow can damage the brain, heart, and other organs.

Heart's Electrical System

To understand arrhythmias, it helps to understand the heart's internal electrical system. The heart's electrical system controls the rate and rhythm of the heartbeat. With each heartbeat, an electrical signal spreads from the top of the heart to the bottom. As the signal travels, it causes the heart to contract and pump blood.

Each electrical signal begins in a group of cells called the sinus node or sinoatrial (SA) node. The SA node is located in the heart's upper right chamber, the right atrium. In a healthy adult heart at rest, the SA node fires off an electrical signal to begin a new heartbeat 60 to 80 times a minute.

From the SA node, the electrical signal travels through special pathways in the right and left atria. This causes the atria to contract and pump blood into the heart's two lower chambers, the ventricles.
The electrical signal then moves down to a group of cells called the atrioventricular (AV) node, located between the atria and the ventricles. Here, the signal slows down just a little, allowing the ventricles time to finish filling with blood.

The electrical signal then leaves the AV node and travels along a pathway called the bundle of His. This pathway divides into a right bundle branch and a left bundle branch. The signal goes down these branches to the ventricles, causing them to contract and pump blood to the lungs and the rest of the body. The ventricles then relax, and the heartbeat process starts all over again in the SA node.

A problem with any part of this process can cause an arrhythmia. For example, in atrial fibrillation, a common type of arrhythmia, electrical signals travel through the atria in a fast and disorganized way. This causes the atria to quiver instead of contract.

There are many types of arrhythmia. Most arrhythmias are harmless, but some are not. The outlook for a person who has an arrhythmia depends on the type and severity of the arrhythmia. Even serious arrhythmias often can be successfully treated. Most people who have arrhythmias are able to live normal, healthy lives.

The four main types of arrhythmia are premature (extra) beats, supraventricular arrhythmias, ventricular arrhythmias, and bradyarrhythmias.

1. **Premature (Extra) Beats**

   The most common type of arrhythmia is premature beats. Innocuous most of the time and often don't cause any symptoms. If symptoms do occur, they usually feel like fluttering in the chest or a feeling of a skipped heartbeat. Most of the time, premature beats need no treatment, especially in healthy people.

   Premature beats that occur in the atria of the heart are called premature atrial contractions, or PACs. Premature beats that occur in the ventricles are called premature ventricular contractions, or PVCs. In most cases, premature beats happen naturally. However, some heart diseases can cause premature beats. They also can happen because of stress, too much exercise, or too much caffeine or nicotine.

2. **Supraventricular Arrhythmias**

   Supraventricular arrhythmias are tachycardias that start in the atria or atrioventricular (AV) node. Types of supraventricular arrhythmias include atrial fibrillation (AF), atrial flutter, paroxysmal supraventricular tachycardia (PSVT), and Wolff-Parkinson-White (WPW) syndrome.

   Atrial Fibrillation (AF) involves a very fast irregular rhythm where single fibers in the heart twitch or contract. According to the National Institutes of Health (NIH), about 2.2 million Americans have atrial fibrillation. During AF, the heart's electrical signals don't originate in the SA node. Instead, they begin in another part of the atria or in the nearby pulmonary veins. The electrical signals travel irregularly. They may spread throughout the atria in a rapid, disorganized way. This causes the walls of the atria to quiver very fast (fibrillate) instead of beating normally. As a result, the atria aren't able to pump blood into the ventricles the way they should.

   In AF, electrical signals can travel through the atria at a rate of more than 300 per minute. Some of these abnormal signals can travel to the ventricles, causing them to beat too fast and with an irregular rhythm. AF usually isn't life threatening, but it can be dangerous if it causes the ventricles to beat very fast. The two major complications of AF are stroke or heart failure.
In AF, blood can pool in the atria, causing blood clots to form. If a clot breaks off and travels to the brain, it can cause a stroke. Blood-thinning medicines that reduce the risk of stroke are an important part of treatment for people who have AF. About 15% of strokes happen in patients with AF.

Heart failure occurs if the heart can't pump enough blood to meet the body's needs. AF can lead to heart failure because the ventricles are beating very fast and can't completely fill with blood. Thus, they may not be able to pump enough blood to the lungs and body.

Damage to the heart's electrical system causes AF. The damage most often is the result of other conditions that affect the health of the heart, such as high blood pressure, coronary heart disease, and rheumatic heart disease. Inflammation also is thought to play a role in the development of AF. Other conditions also can lead to AF, including an overactive thyroid gland (too much thyroid hormone produced) and heavy alcohol use. The risk of AF increases with age. Sometimes AF and other supraventricular arrhythmias can occur for no obvious reason.

**Atrial Flutter**

Atrial flutter is similar to AF. However, the heart's electrical signals spread through the atria in a fast and regular—instead of irregular—rhythm. Atrial flutter is much less common than AF, but it has similar symptoms and complications.

**Paroxysmal Supraventricular Tachycardia**

PSVT is a very fast heart rate that begins and ends suddenly. PSVT occurs because of problems with the electrical connection between the atria and the ventricles. In PSVT, electrical signals that begin in the atria and travel to the ventricles can reenter the atria, causing extra heartbeats. This type of arrhythmia usually isn't dangerous and tends to occur in young people. It can happen during vigorous physical activity.

A special type of PSVT is called Wolff-Parkinson-White syndrome. WPW syndrome is a condition in which the heart's electrical signals travel along an extra pathway from the atria to the ventricles. This extra pathway disrupts the timing of the heart's electrical signals and can cause the ventricles to beat very fast. Patients with this syndrome may feel dizzy, have chest palpitations or, have episodes of fainting.

3. **Ventricular Arrhythmias**

Ventricular arrhythmias start in the heart's lower chambers, the ventricles. They can be very dangerous and usually require emergency medical care. Ventricular arrhythmias include ventricular tachycardia and ventricular fibrillation (v-fib). Coronary heart disease, heart attack, a weakened heart muscle, and other heart complications can cause ventricular arrhythmias.

**Ventricular Tachycardia**

Ventricular tachycardia is a fast, regular beating of the ventricles that may last for only a few seconds or for much longer. A few beats of ventricular tachycardia often don't cause problems. However, episodes that last for more than a few seconds can be dangerous. Ventricular tachycardia can turn into other, more serious arrhythmias, such as v-fib.

**Ventricular Fibrillation**
V-fib occurs if disorganized electrical signals make the ventricles quiver instead of pump normally. Without the ventricles pumping blood to the body, sudden cardiac arrest and death can occur within a few minutes. V-fib may occur during or after a heart attack or in patients whose heart is already weak because of another condition.

Torsades de pointes (torsades) is a type of v-fib that causes a unique pattern on an EKG test. Certain medicines or imbalanced amounts of potassium, calcium, or magnesium in the bloodstream can cause this condition. Patients who have long QT syndrome are at increased risk for torsades. People who have this condition need to be careful about taking certain antibiotics, heart medicines, and over-the-counter products.

4. **Bradyarrhythmias**

Bradyarrhythmias occur if the heart rate is slower than normal. If the heart rate is too slow, not enough blood reaches the brain. This can cause patients to faint. In adults, a heart rate slower than 60 beats per minute is considered a bradyarrhythmia. Some individuals normally have slow heart rates, especially those who are very physically fit. For them, a heartbeat slower than 60 beats per minute isn’t dangerous and doesn’t cause symptoms. But in other people, serious diseases or other conditions may cause bradyarrhythmias.

Bradyarrhythmias can be caused by:

- Heart attacks
- Conditions that harm or change the heart's electrical activity, such as an underactive thyroid gland or aging
- An imbalance of chemicals or other substances in the blood, such as potassium
- Medicines such as beta blockers, calcium channel blockers, some antiarrhythmia medicines, and digoxin

**Arrhythmias in Children**

Children’s heart rates normally decrease as they get older. A newborn's heart beats between 95 to 160 times a minute. A 1-year-old's heart beats between 90 to 150 times a minute, and a 6- to 8-year-old's heart beats between 60 to 110 times a minute.

A baby or child's heart can beat fast or slow for many reasons. Like adults, when children are active, their hearts will beat faster. When they're sleeping, their hearts will beat slower. Their heart rates can speed up and slow down as they breathe in and out. All of these changes are normal.

Some children are born with heart defects that cause arrhythmias. In other children, arrhythmias can develop later in childhood. Doctors use the same tests to diagnose arrhythmias in children and adults.

Treatments for children who have arrhythmias include medicines, defibrillation (electric shock), surgically implanted devices that control the heartbeat, and other procedures that fix abnormal electrical signals in the heart.

**Coronary Artery Disease (CAD) Nonpreventable and Preventable Risk Factors:**
Coronary artery disease can be a silent killer. In most patients, atherosclerosis builds over years and even decades. Although it most likely begins before people are out of their teenage years, the coronary effects of atherosclerosis usually do not show up until later in life. The most common symptom of CAD is chest pain, shortness of breath and fatigue. These symptoms are the most common cause of patient visits to the doctor. CAD can be symptomless and silent for years. Even those patients who have been diagnosed with CAD 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 apparent signs and symptoms of coronary artery disease, there are some characteristics and risk factors present in most patients with the disease.

Nonpreventable Risk Factors:

Age

In men, the risk for coronary artery disease (CAD) increases starting around age 45. In women, the risk for CAD increases starting around age 55. Most people have some plaque buildup in their heart arteries by the time they're in their 70s. However, only about 25 percent of those people have chest pain, heart attacks, or other signs of CAD.

Gender

CAD is slightly more common in men than in women; in the United States, about 9.0% 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. Some risk factors may affect CAD risk differently in women than in men. For example, estrogen provides women some protection against CAD, whereas diabetes raises the risk of CAD more in women than in men.

Also, some risk factors for heart disease only affect women, such as preeclampsia, a condition that can develop during pregnancy. Preeclampsia is linked to an increased lifetime risk of heart disease, including CAD, heart attack, heart failure, and high blood pressure. (Likewise, having heart disease risk factors, such as diabetes or obesity, increases a woman’s risk of preeclampsia.)

Family History

A family history of early CAD is a risk factor for developing CAD, specifically if a father or brother is diagnosed before age 55, or a mother or sister is diagnosed before age 65.

Ethnicity

African Americans have a higher risk of developing, and a higher death rate from, CAD than other ethnic groups. 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 others and are less likely to receive invasive treatment. Americans of Asian Indian origin are 2 to 3 times as likely as European Americans to develop coronary artery disease. 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 according to the American Heart Association.
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. Those 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.

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:

There are many known CAD risk factors. You can control some risk factors. Risk factors you can control include:

- High blood cholesterol and triglyceride levels (a type of fat found in the blood)
- Hypertension—High blood pressure
- Diabetes and prediabetes
- Overweight and obesity
- Smoking
- Sedentary lifestyle
- Unhealthy diet
- Stress

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 II 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. Lowering cholesterol may slow, reduce, or even stop the buildup of plaque in the arteries. It also may reduce the risk of plaque rupturing and causing dangerous blood clots.

HYPERTENSION

Hypertension causes inflammation, which can damage the lining of arteries and increases fatty deposits contributing to the development of atherosclerosis and CAD. For those individuals 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.

<table>
<thead>
<tr>
<th>Blood Pressure Classification</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
</tr>
<tr>
<td>Stage I Hypertension</td>
<td>140-159</td>
<td>or 90-99</td>
</tr>
<tr>
<td>Stage II Hypertension</td>
<td>&gt;160</td>
<td>or &gt;100</td>
</tr>
</tbody>
</table>
Treating hypertension is an important factor in preventing CAD and includes the following strategies:

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

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. According to the American Heart Association, around 65% of people with diabetes die of some form of heart or blood vessel disease.

Patients with type II 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 II 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.

OBESITY
The terms "overweight" and "obesity" refer to body weight that’s greater than what is considered healthy for a certain height. The most useful measure of overweight and obesity is body mass index (BMI). BMI is calculated from your height and weight. Obesity can lead to many health issues including CAD.

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. Obese patients are also at increased risk for developing metabolic syndrome and diabetes.

Exercise alone will not usually lead to significant weight loss. A reduced-calorie diet combined with regular exercise is the best approach to weight loss. 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.

Every pound counts, 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 the goal of reducing weight.

SMOKING
People who smoke have a risk of developing CAD as great as 4 times higher as 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.

Quitting smoking can be a key to reducing the likelihood of developing CAD. 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 approach to quitting smoking includes the following components:
Sedentary Lifestyle

Physical inactivity or sedentary lifestyle is a risk factor for CAD. Patients with a sedentary lifestyle are also more likely to be obese or overweight, which contributes to the risk of developing CAD. The four main types of physical activity are aerobic, muscle-strengthening, bone-strengthening, and stretching. Aerobic activity is the type that benefits the heart the most. Initially, patient goals for physical activity should begin modest 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 benefits increase as the intensity and duration of the exercise increase. 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:

- Reducing blood pressure
- Lipid metabolism
- Insulin sensitivity
- Burn calories

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.

Examples of Aerobic Activities

Depending on your level of fitness, they can be light, moderate, or vigorous in intensity:

- Pushing a grocery cart around a store, fast pace walk in a shopping mall
- Gardening, such as digging or hoeing that causes your heart rate to go up
- Walking, hiking, jogging, running
- Water aerobics or swimming laps
- Bicycling, skateboarding, rollerblading, and jumping rope
- Dancing and aerobic dancing
- Racquetball, tennis, soccer, hockey, and basketball

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.

OTHER RISK FACTORS

Stress may be a contributing factor for developing CAD. High stress may cause people to overeat, start smoking, or smoke more than they otherwise would. Heart rate and blood pressure increase during times of stress.

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).

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. According to the American Heart Association the guidelines place emphasis on foods and an overall eating pattern rather than on percentages of food components such as fat.

Heart Healthy Diet Recommendations:

1. Portion size Control

The amount eaten is just as important as the food eaten. Plate overloading, taking seconds and eating until feeling stuffed can lead to eating more calories and weight gain. Portions served in restaurants are often more than anyone needs.

Using a small plate or bowl to help control portions will help control amount eaten. Eat larger portions of low-calorie, nutrient-rich foods, such as fruits and vegetables, and smaller portions of high-calorie, high-sodium foods, such as refined, processed or fast foods. This strategy can shape up any diet as well as the heart and waistline.

Keeping track of the number of servings can also reduce caloric intake. A serving size is a specific amount of food, defined by common measurements such as cups, ounces or pieces. For example, one serving of pasta is 1/2 cup, or about the size of a hockey puck. A serving of meat, fish or chicken is about 2 to 3 ounces, or about the size and thickness of a deck of cards. Judging serving size is a learned skill.

2. Eat more vegetables and fruits

Vegetables and fruits are good sources of vitamins and minerals. Vegetables and fruits are also low in calories and rich in dietary fiber. Vegetables and fruits contain substances found in plants that may help prevent cardiovascular disease. Eating more fruits and vegetables may help you eat less high-fat foods, such as meat, cheese and snack foods.

Featuring vegetables and fruits in your diet can be easy. Keep vegetables washed and cut in your refrigerator for quick snacks. Keep fruit in a bowl in your kitchen so that you'll remember to eat it. Choose recipes that have vegetables or fruits as the main ingredients, such as vegetable stir-fry or fresh fruit mixed into salads.

<table>
<thead>
<tr>
<th>Fruits and vegetables to choose</th>
<th>Fruits and vegetables to limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh or frozen vegetables and fruits</td>
<td>Coconut</td>
</tr>
<tr>
<td>Low-sodium canned vegetables</td>
<td>Vegetables with creamy sauces</td>
</tr>
<tr>
<td>Canned fruit packed in juice or water</td>
<td>Fried or breaded vegetables</td>
</tr>
<tr>
<td></td>
<td>Canned fruit packed in heavy syrup</td>
</tr>
<tr>
<td></td>
<td>Frozen fruit with sugar added</td>
</tr>
</tbody>
</table>

Grain products to choose

Grain products to limit or avoid
3. Select whole grains

Whole grains are good sources of fiber and other nutrients that play a role in regulating blood pressure and heart health. You can increase the amount of whole grains in a heart-healthy diet by making simple substitutions for refined grain products. Or be adventuresome and try a new whole grain, such as whole-grain farro, quinoa or barley.

4. Limit unhealthy fats

Limiting saturated and trans fats it is an important step to reduce blood cholesterol and lower risk of coronary artery disease. A high blood cholesterol level can lead to a buildup of plaques in the arteries, which can lead to atherosclerosis, which can increase risk of heart attack and stroke.

The American Heart Association offers these guidelines for how much fat to include in a heart-healthy diet:

<table>
<thead>
<tr>
<th>Type of fat</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fat</td>
<td>Less than 7% of your total daily calories, or less than 14 g of saturated fat if you follow a 2,000-calorie-a-day diet</td>
</tr>
<tr>
<td>Trans fat</td>
<td>Less than 1% of your total daily calories, or less than 2 g of trans fat if you follow a 2,000-calorie-a-day diet</td>
</tr>
</tbody>
</table>

The best way to reduce saturated and trans fats in one’s diet is to limit the amount of solid fats — butter, margarine and shortening — you add to food when cooking and serving. You can also reduce the amount of saturated fat by trimming fat off meat or choosing lean meats with less than 10 percent fat.
Also use low-fat substitutions when possible for a heart-healthy diet. For example, baked potato with low-sodium salsa or low-fat yogurt rather than butter on top, or use sliced whole fruit or low-sugar fruit spread on toast instead of margarine.

Check the food labels on packages like cookies, crackers and chips. Many of these snacks — even those labeled "reduced fat" — may be made with oils containing trans fats. One clue that a food has some trans fat in it is the phrase "partially hydrogenated" in the ingredient list.

Choose monounsaturated fats, such as olive oil or canola oil when using fats. Polyunsaturated fats, found in certain fish, avocados, nuts and seeds, also are good choices for a heart-healthy diet. When used in place of saturated fat, monounsaturated and polyunsaturated fats may help lower total blood cholesterol. But moderation is essential. All types of fat are high in calories.

An easy way to add healthy fat (and fiber) is to use ground flaxseed. Flaxseeds are small brown seeds that are high in fiber and omega-3 fatty acids. Studies have found that flaxseeds may help lower cholesterol in some people. Grind the seeds in a coffee grinder or food processor and stir a teaspoon of them into yogurt, applesauce or hot cereal.

Fats to choose

- Olive oil
- Canola oil
- Vegetable and nut oils
- Margarine, trans fat free
- Cholesterol-lowering margarine, such as Benecol, Promise Activ or Smart Balance
- Nuts, seeds
- Avocados

Fats to limit

- Butter
- Lard
- Bacon fat
- Gravy
- Cream sauce
- Nondairy creamers
- Hydrogenated margarine and shortening
- Cocoa butter, found in chocolate
- Coconut, palm, cottonseed and palm-kernel oils

5. Choose low-fat protein sources

Lean meat, poultry and fish, low-fat dairy products, and eggs are some of the best sources of protein. But be careful to choose lower fat options, such as skim milk rather than whole milk and skinless chicken breasts rather than fried chicken patties.

Fish is another good alternative to high-fat meats. And certain types of fish are rich in omega-3 fatty acids, which can lower blood fats called triglycerides. You'll find the highest amounts of omega-3 fatty acids in cold-water fish, such as salmon, mackerel and herring. Other sources are flaxseed, walnuts, soybeans and canola oil.

Legumes — beans, peas and lentils — also are good sources of protein and contain less fat and no cholesterol, making them good substitutes for meat. Substituting plant protein for animal protein — for example, a soy or bean burger for a hamburger — will reduce your fat and cholesterol intake.

Proteins to choose

- Lean meat
- Poultry
- Fish
- Low-fat dairy products
- Eggs

Proteins to limit or

- Butter
- Lard
- Bacon fat
- Gravy
- Cream sauce
- Nondairy creamers
- Hydrogenated margarine and shortening
- Cocoa butter, found in chocolate
- Coconut, palm, cottonseed and palm-kernel oils
• Low-fat dairy products such as skim or low-fat (1%) milk, yogurt and cheese
• Eggs
• Fish, especially fatty, cold-water fish, such as salmon
• Skinless poultry
• Legumes
• Soybeans and soy products, such as soy burgers and tofu
• Lean ground meats

• Full-fat milk and other dairy products
• Organ meats, such as liver
• Fatty and marbled meats
• Spareribs
• Hot dogs and sausages
• Bacon
• Fried or breaded meats

6. Reduce sodium intake

Eating a lot of sodium can contribute to high blood pressure, a risk factor for CAD. Reducing sodium is an important part of a heart-healthy diet. The Department of Health and Human Services recommends:

➢ Healthy adults have no more than 2,300 milligrams (mg) of sodium a day (about a teaspoon of salt)
➢ People age 51 or older, African-Americans, and people who have been diagnosed with high blood pressure, diabetes or chronic kidney disease have no more than 1,500 mg of sodium a day

Although reducing the amount of salt you add to food at the table or while cooking is a good first step, much of the salt you eat comes from canned or processed foods, such as soups and frozen dinners. Eating fresh foods and homemade soups and stews can reduce the amount of salt you eat.

Be wary of foods that claim to be lower in sodium because they are seasoned with sea salt instead of regular table salt — sea salt has the same nutritional value as regular salt. Another way to reduce the amount of salt is to choose condiments carefully. Many condiments are available in reduced-sodium versions, and salt substitutes can add flavor to your food with less sodium.

Low-salt items to choose    High-salt items to avoid
- Herbs and spices
- Salt substitutes
- Reduced-salt canned soups or prepared meals
- Reduced-salt versions of condiments, such as reduced-salt soy sauce and reduced-salt ketchup
- Table salt
- Canned soups and prepared foods, such as frozen dinners
- Tomato juice
- Soy sauce

7. Plan ahead: Create daily menus

Create daily menus using the six strategies listed above. When selecting foods for each meal and snack, emphasize vegetables, fruits and whole grains. Choose lean protein sources and healthy fats, and limit salty foods. Portion sizes and variety are also important in planning daily meals.

For example, if you have grilled salmon one evening, try a black-bean burger the next night. This helps ensure that the body gets all of the nutrients it needs. Variety also makes meals and snacks more interesting.

8. Occasional treat

A candy bar or handful of potato chips won't derail your heart-healthy diet. The occasional treat is ok, but don't let it turn into an excuse for giving up on your healthy-eating plan. If overindulgence is the exception, rather than the rule, things will balance out over the long term. What's important is eating healthy foods most of the time. With planning and a few simple substitutions, heart healthy diet will reduce the risk of CAD.

Coronary Artery Disease: Diagnosis and Evaluation

Chest Discomfort

Diagnosis and Evaluation of CAD

Chest Discomfort

Frequently the chief complaint of a person with CAD is chest discomfort. When a patient presents with a nonacute history of chest discomfort, the workup for possible CAD can proceed in a precise manner.

In contrast, when a patient comes to a medical facility with very recent or ongoing chest discomfort, a preplanned triage should be set in motion. In the emergency department, clinic, or office, the clinician must quickly identify those patients who have potentially life-threatening conditions such as MIs, aortic dissection, pulmonary embolus, or tension pneumothorax from non-life-threatening causes of chest pain or discomfort (Lee, 2008). Conditions such as heartburn, swallowing disorders, pancreatic and gallbladder problems, muscle and bone problems, and lung disorders can all cause symptoms similar to those caused by the heart (Mayo Clinic, 2012).

Classic Angina

The classic symptom of CAD is chest discomfort or angina. It is important to identify several key indicators including quality, location, duration, and triggers of the angina.

Quality: Squeezing Pressure

With CAD patients often describe the angina as a feeling of constriction, heaviness, pressure, and tightness in the chest. Other terms frequently used to describe the chest discomfort are aching, choking, crushing, smothering, and squeezing. Patients typically hold a clenched fist over their chest when describing the feeling of angina.

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

**Duration: Length of Angina**
The angina of stable angina is temporary, lasting just a few minutes and coming in a wave that worsens, reaching a peak and then subsiding. Unstable angina lasts longer, typically 10 to 20 minutes. The angina of MI has a variable duration, often lasting longer than 30 minutes.

**Triggers: Exercise**
Angina can be triggered by exercise, sexual activity, exposure to cold weather, emotional stress (anger, fright, frustration), or a large meal. Any stress that changes the hearts rhythm that may lead to tachycardia may trigger angina.

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

**Occurrence: Predictable or Unpredictable**
The angina of stable angina comes predictably when the patient engages in a certain level of exercise. The angina of unstable angina arises unpredictably, sometimes at an increasing rate or at increasingly lower levels of exercise. The angina of an MI happens unpredictably.

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

**Accompanying Symptoms: Variable**
The angina of stable angina can be accompanied by other symptoms, such as shortness of breath. The angina of an acute coronary syndrome can be accompanied by a variety of other symptoms, including shortness of breath, lightheadedness, nausea, or sweating (Lee, 2008).

**Uncommon Descriptors: Not Often Used To Describe Classic CAD Angina**
Patients typically do not describing classic angina in the following manner:

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

Angina is the complaint that usually triggers a workup for CAD. However, not all people with CAD get angina. Even during an MI, some people do not experience significant chest discomfort.

Heart Ischemia: **Anginal Equivalents**
A few complaints other than chest discomfort can also be caused by heart ischemia. Clinicians suspect heart ischemia when patients complain of episodes of shortness of breath, fatigue, nausea, vomiting, sweating or faintness that are triggered by exercise, stress or cold, but are then relieved by rest.

When they are caused by heart ischemia, these symptoms behave like angina and are called anginal equivalents. Among patients with CAD, angina is more often reported by men than by women. Instead of classic angina, women often report anginal equivalents. Anginal equivalents are also more common for CAD in older adults and in people with diabetes. It is of upmost importance that a clinical practitioner can distinguish angina caused by heart ischemia from other types of chest pain or discomfort.

Other Causes of Chest Pain:

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

### CAUSES OF CHEST PAIN

<table>
<thead>
<tr>
<th>Origin</th>
<th>Causes</th>
</tr>
</thead>
</table>
| Cardiovascular | • Aortic aneurysm  
              | • Aortic dissection  
              | • Myocardial ischemia  
              | • Pericarditis        |
| Pulmonary   | • Pneumonia  
              | • Pneumothorax  
              | • Pulmonary embolism  |
| Musculoskeletal | • Chest wall injury  
                  | • Costochondritis  
                  | • Herniated intervertebral disc  
                  | • Spinal arthritis  |
CAUSES OF CHEST PAIN

<table>
<thead>
<tr>
<th>Origin</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>• Esophageal spasm</td>
</tr>
<tr>
<td></td>
<td>• Esophageal tear</td>
</tr>
<tr>
<td></td>
<td>(Mallory-Weiss)</td>
</tr>
<tr>
<td></td>
<td>• Esophagitis</td>
</tr>
<tr>
<td></td>
<td>• Gallbladder disease</td>
</tr>
<tr>
<td></td>
<td>• Pancreatitis</td>
</tr>
<tr>
<td></td>
<td>• Ulcer disease</td>
</tr>
<tr>
<td>Infectious</td>
<td>• Herpes zoster</td>
</tr>
<tr>
<td>Neurologic</td>
<td>• Panic attack</td>
</tr>
</tbody>
</table>

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 clinical practitioner to the possibility of an increased risk for atherosclerotic coronary artery disease include:

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

Ischemia

Medical history may reveal that atherosclerosis of the coronary arteries has already shown signs of existence. A patient with CAD may already have had episodes of heart ischemia, such as a prior heart attack (MI).

Peripheral Artery Disease

Atherosclerosis is a whole-body disease. Patients with CAD will often have indications of atherosclerosis in arteries other areas of the body. 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 II 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 according to the American Heart Association.

**Metabolic Syndrome**

A combination of metabolic abnormalities that sometimes accompany abdominal obesity, such as insulin resistance, dyslipidemia, elevated fasting blood levels of glucose, and elevated blood pressure, and are associated with an increased risk of cardiovascular disease. Having metabolic syndrome increases a patient’s risk of developing type II diabetes. The risk of developing serious atherosclerotic vascular disease with coronary artery blockage also increases.

**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 predisposition is the existence of close 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**

Smoking and poor diets increase the risk of CAD. Assessment should include taking a careful history of current or previous smoking as well as asking about dietary habits. A person who smoking one or more packs of cigarettes a day for several years doubles their chance of dying from CAD; Quitting smoking can reduce this extra risk. As well, 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**

Physical exam will include weight, vital signs, skin, head & neck, thorax, abdomen and extremities. A patient with CAD who presents to the emergency department with acute coronary syndrome can show many abnormalities on physical examination. A patient with CAD who comes to the clinic or office for a check-up may have only a few signs of the disease. During a routine physical examination, the following findings would be expected.

**WEIGHT**

Patients with excess intra-abdominal or visceral fat 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 regular checkup, the pulse may have a normal rate and rhythm in a patient with coronary artery disease. Tachycardia is not uncommon when a patient is suffering from an episode of myocardial ischemia. Bradycardia during an acute coronary syndrome can be an unfavorable sign. Patients with CAD often have hypertension (BP $\geq 140/90$), and the higher the blood pressure, the greater the risk of CAD. Hypotension during a myocardial infarction is a negative sign. The respiration rate is usually normal during a regular checkup. The patient will breathe more rapidly under the stress of heart ischemia.

**SKIN**

During a regular checkup no unusual sweating is expected, but acute coronary syndromes, especially myocardial infarctions, are often accompanied by profuse sweating. If the patient is a heavy smoker evidence of nicotine stains on their fingers or teeth maybe present.

**HEAD AND NECK**

During a regular checkup a funduscopic exam of the back of the eye (retina) may reveal presence of CAD. The blood vessels of the retina may show the effects of hypertension or atherosclerosis. 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**

If the patient’s chest discomfort can be reproduced with clinician pressing on some point along the chest wall, the pain is unlikely to be angina. Some patients with MI may have a broad region of the chest become tender. During a regular exam, the lungs of a patient with coronary artery disease can be clear and unremarkable. With heart attack, however, 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 abnormal sounds such as rales can be heard.
A regular office checkup physical exam of a patient with CAD may find no apparent heart problems. If the patient has a history of a heart attack, 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**

During the examination of the abdomen, blood flow will be assets. Changes in the sounds of blood flow (bruits) may indicate a narrowed blood vessel in the abdomen. This is a sign of hardening of the arteries (atherosclerosis) in the large blood vessels that run through the abdomen. Also, an abdominal aortic aneurysm usually indicates atherosclerosis. 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 Test**

A patient being evaluated for CAD will have 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).

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Optimal Levels</th>
<th>Unhealthy Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>&lt;200 mg/dl</td>
<td>&gt;240 mg/dl</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>≥60 mg/dl</td>
<td>&lt;40 mg/dl for men &lt;50 mg/dl for women</td>
</tr>
</tbody>
</table>
### Fasting Blood Lipid Levels

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Optimal Levels</th>
<th>Unhealthy Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL cholesterol</td>
<td>&lt;100 mg/dl</td>
<td>&gt;160 mg/dl</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>&lt;150 mg/dl</td>
<td>&gt;200 mg/dl</td>
</tr>
</tbody>
</table>

### 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 heart attack, specific heart proteins referred to as 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

<table>
<thead>
<tr>
<th>Marker</th>
<th>Normal Levels</th>
<th>Duration of Elevation after MI</th>
</tr>
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## CARDIAC MARKERS

<table>
<thead>
<tr>
<th>Marker</th>
<th>Normal Levels</th>
<th>Duration of Elevation after MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine kinase(CK)</td>
<td></td>
<td>Peaks at 12 to 18 hours; remains elevated up to 72 hours.</td>
</tr>
<tr>
<td></td>
<td>38–190 units/L (men)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10–150 units/L (women)</td>
<td></td>
</tr>
<tr>
<td>Myoglobin</td>
<td>0.0–0.09 mcg/ml</td>
<td>Peaks within 6 to 7 hours; returns to baseline within 24 hours.</td>
</tr>
<tr>
<td>Troponin I</td>
<td>&lt;0.1 mcg/ml</td>
<td>Peaks within 14 to 20 hours; returns to baseline in 10 to 15 days.</td>
</tr>
</tbody>
</table>

Source: Adapted from McLaughlin, 2014.

### Imaging Studies
Full ranges of imaging techniques are used in diagnosis of Heart Disease.

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

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

A type of echo called Doppler ultrasound shows how well blood flows through the heart’s chambers and valves. Echo can detect blood clots inside the heart, fluid buildup in the pericardium (the sac around the heart), and problems with the aorta, the main artery that carries oxygen-rich blood from the heart to the body (NHLBI, 2011d).

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

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

**Coronary Calcium Scan Image**
(A) The position of the heart in the body and the location and angle of the coronary calcium scan image; (B) scan image showing calcification in a coronary artery. (In the CT scan, the patient’s back is at the bottom of the image, and the patient’s sternum is at the top.) Source: NHLBI, 2012.

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

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

**Cardiac Catheterization Angiography**

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

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

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

**New STEMI Guidelines**

Percutaneous coronary intervention (PCI), often with stenting, is performed emergently in patients having an acute ST elevation MI (STEMI). Thrombolytics are also used for STEMIs when PCI is not available.

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

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

There has been recent discussion and research regarding the current recommendation of treating new or presumed new left bundle branch block with thrombolytics and catheter lab intervention. The data suggests that most patients with LBBB do not have an occluded artery at cardiac catheterization and are therefore needlessly exposed to the risks of fibrinolytic therapy. (NIH, 2012b)
The new STEMI guidelines also stress the importance of therapeutic hypothermia for selected unconscious patients who survive out-of-hospital ventricular fibrillation. There is increased emphasis on the importance of cardiac rehabilitation and postprocedural care.

**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).

**Coronary Artery Disease Management:**

Time is of the essence, patients with CAD may have mild symptoms that occur with stable angina that can be monitored and treated over time. Other patients may present with chest pain, shortness of breath, profuse sweating, extreme fatigue, or other acute symptoms that may need to be seen in an emergency department and evaluated immediately.

**Emergency Treatment**

In an emergency situation the American Heart Association’s recommends using the “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)

Patients should be educated on the importance of early action. Unless the patient has 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 rapidly by emergency responders to an emergency department whenever experiencing an episode of angina.

**BEFORE THE HOSPITAL**

Time is of the essence, 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 angina or a sudden onset of shortness of breath should treat the symptoms as a possible heart attack 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 emergency department 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 shortness of breath should be implemented.

It is important for the team of providers in the emergency department 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. It is thought that patients with STEMI's...
usually have a completely blocked artery, whereas patients whose infarctions do not produce ST-elevations have an incompletely blocked artery (AHA, 2013).

Goals of Emergency Treatment
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.
- Emergency cardiac surgery may also be performed for patients who are unable to undergo percutaneous interventions.

Cardiac Procedures and Surgery: Coronary Revascularization
In order to improve heart function due to blockage and increase blood flow to ischemic areas procedures such as coronary revascularization are performed. 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: Patients with significant narrowing of 1 but not more than 3 major coronary arteries and with a normally functioning left ventricle are good candidates for the procedure. Other clinical indications for PCI include the following:

- Acute ST-elevation myocardial infarction (STEMI)
- Non-ST-elevation acute coronary syndrome (NSTE-ACS)
- Unstable angina
- Stable angina
- Anginal equivalent (eg, dyspnea, arrhythmia, or dizziness or syncope)
- High risk stress test findings

CABG: CABG is indicated for patients with more than two arterial constrictions, with weakened left ventricles, or with diabetes. Other clinical indications for PCI include the following:

- Disease of the left main coronary artery (LMCA)
- Disease of all 3 coronary vessels (LAD, LCX and RCA)
- Diffuse disease not amenable to treatment with PCI
- High-risk patients such as those with severe ventricular dysfunction (low ejection fraction), or diabetes mellitus
- Relieving severe symptoms of angina, dyspnea, fatigue not responding to first-line therapy
- Multivessel CAD

In patients with stable angina, medical therapy is recommended as first-line therapy unless one or more of the following indications for cardiac catheterization and PCI or coronary artery bypass grafting (CABG) are present:

- Severe symptoms
- A change in symptom severity
- Failed medical therapy
- High-risk coronary anatomy
- Worsening left ventricular (LV) dysfunction

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.)

CORONARY ARTERY BYPASS GRAFT (CABG)

The most common open-heart operation performed in the United States is coronary artery bypass (CABG). Nearly 1400 CABG procedures are performed daily in the US and over 500,000 procedures performed each year. CABG may be contraindicated in patients at higher risk for complication such as elderly patients, patients with end-stage kidney disease, lung disease, and peripheral vascular disease.

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 operation 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.

There are several types of coronary artery bypass grafting (CABG).

Traditional Coronary Artery Bypass Grafting

Traditional CABG is used when at least one major artery needs to be bypassed. During the surgery, the chest bone is opened to access the heart.

Medicines are given to stop the heart; a heart-lung bypass machine keeps blood and oxygen moving throughout the body during surgery. This allows the surgeon to operate on a still heart.
After surgery, blood flow to the heart is restored. Usually, the heart starts beating again on its own. Sometimes mild electric shocks are used to restart the heart.

**Off-Pump or Beating Heart Coronary Artery Bypass Grafting**

This type of CABG is similar to traditional CABG because the chest bone is opened to access the heart. However, the heart isn’t stopped, and a heart-lung bypass machine isn’t used. Off-pump CABG sometimes is called beating heart bypass grafting.

Beating heart surgery often allows patients to be discharged from the hospital more sooner 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.

**Minimally Invasive Direct Coronary Artery Bypass Grafting**

This type of surgery differs from traditional CABG because the chest bone isn't opened to reach the heart. Instead, several small cuts are made on the left side of the chest between the ribs. This type of surgery mainly is used to bypass blood vessels at the front of the heart.

Minimally invasive bypass grafting is a fairly new procedure. It isn't right for everyone, especially if more than one or two coronary arteries need to be bypassed.

The goals of CABG may include:

- Improving your quality of life and reducing angina and other CHD symptoms
- Allowing you to resume a more active lifestyle
- Improving the pumping action of your heart if it has been damaged by a heart attack
- Lowering the risk of a heart attack (in some patients, such as those who have diabetes)
- Improving your chance of survival

**CABG Postoperative Care**

A collaborative team approach for patients postoperative is necessary for CABG patients. 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

The most common post-CABG complication is atrial fibrillation (AF). AF occurs in 20% to 50% of patients post-CABG. 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 amiodarone. 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.

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)

**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)

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 of CAD

➢ 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

Nitroglycerin, 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.

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, dietician, 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).

Complications 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


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).

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 Program
Benefits of exercise program:
- 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

Cardiac exercise programs are supervised and designed to the abilities of the patient, and these programs increase exercise levels 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 Changes
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.
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).
**Stop Smoking**

Smoking injures cells throughout the body. Smoking contributes to the development of atherosclerotic cardiovascular disease, insulin resistance, type II 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/m², 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).

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.

**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:**

- Stop 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)

**Complications and Comorbidity**

**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.

**Conclusion**

Heart disease is the leading cause of death for both men and women worldwide. Over 600,000 people die of heart disease in the United States every year—that’s 1 in every 4 deaths. Coronary arterial disease (CAD) is the most common type of heart disease, killing more than 370,000 people each year. Prevention of CAD begins with understanding the risk factors and implementing lifestyle changes that decrease preventable risk factors. Weight loss, improved diet, medications, and regular physical exercise are all key factors of the initial treatment program. Drugs and interventional procedures are used to treat those components of CAD that do not improve greatly 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. Advances in medicine and technology have greatly improved the early detection and survival rate of patients with CAD. It is vital for all healthcare professionals to understand the key components of treating these with patients.

**Post Test**

1. Heart disease is the leading cause of death for both men and women worldwide.
   - A. True  B. False
2. Coronary arterial disease (CAD) is the most common type of heart disease.
   - A. True  B. False
3. CAD is a condition in which plaque, which is made up of fat, cholesterol, calcium and other substances in the blood, builds up inside the coronary arteries.
   - A. True  B. False
4. Atherosclerosis is a condition that develops when plaque builds up in the walls of the arteries. This buildup narrows the arteries, making it harder for blood to flow through. Atherosclerosis is the underling disorder that causes CAD.
   - A. True  B. False
5. Coronary artery disease signs and symptoms, including: apnea, increased energy, and weight loss.
   - A. True  B. False
6. Three types of acute coronary syndromes are: unstable angina, heart attack (Myocardial Infarction) and sudden cardiac death.
   - A. True  B. False
7. Arrhythmia: An irregular rhythm of the heart caused by the malfunction of the hearts electrical system. Tachycardia, bradycardia, skipped heartbeat and fluttering heart are all examples of an arrhythmia.
   - A. True  B. False
8. The classic symptom of CAD is chest discomfort or angina. It is important to identify several key indicators including quality, location, duration, and triggers of the angina.
   - A. True  B. False
9. There are many known CAD risk factors. You can control some risk factors. Risk factors you can control include: age, gender and family history.
10. The two types of coronary revascularization procedures are percutaneous coronary interventions (PCI) and coronary artery bypass grafts (CABG).
   A. True   B. False