

# Coronary artery disease:



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*The number one cause of death in the United States, coronary artery disease (CAD) affects one in three Americans. Globally, 7 million people with CAD die each year, making it one of the world's top health problems. We fill you in on treatment and prevention.*

By Andrew Herman, MS, RN  
Direct Care Nurse • AmLake VA • Tacoma, Wash.

# The plaque plague



CAD—the narrowing of the small blood vessels that supply blood and oxygen to the heart—may cause myocardial infarction (MI), which affects 785,000 Americans annually, 500,000 of whom will have a second MI. During the past decade, early cardiac catheterization with percutaneous coronary intervention (PCI) has decreased mortality from MI in the developed world by over 50%.

Monitoring and treatment techniques for the compromised heart pump are extensive, but are no substitute for a healthy lifestyle that includes wise food consumption, regular exercise coupled with adequate weight control, and avoidance of abuse of substances such as tobacco and alcohol. Let's take a closer look.

## Plaque attack

To understand CAD, we must first take a look at atherosclerotic plaque. Most plaques will remain asymptomatic for many years, but some types are prone to thrombosis. The most common form of vulnerable plaque has a large lipid core with a thin cap covering (see *Types of atherosclerotic plaque*). In areas of lipid deposits, apoptosis (dead cells) can also make plaque unstable. New microvessels develop around the area in an attempt to bring circulation to the viable cells that remain; these neovessels are fragile and can cause hemorrhage beneath the plaque, along with dislodgement.

As the plaque shears away from the vessel wall, circulating platelets adhere to

it. Proteins in the subendothelium emerge then cause the platelets to change shape and expose their IIb/IIIa receptor site. Fibrinogen connects the platelets together at this site, and an obstructive white clot begins to form. Diminished blood flow causes a shift of electrolytes in the surrounding heart cells, the pH drops, and normal electrical conduction is altered (see *Picturing CAD*).

So how can plaque cause an MI? Early in life, a thin layer develops along the interior of blood vessels, which is made up of wandering cells of the reticuloendothelial, or mononuclear phagocyte, system that originate in the liver, spleen, lung, spinal cord, and brain. Over time, these cells fill with lipids, and the layer grows thicker. By early adulthood, the lipids begin to form outside the cells and become an intermediate lesion that can still be clinically silent. The lipid-filled cells eventually form a cap over an ever-widening lipid core that can grow large enough to cause turbulence of blood flow within the vessel. Any subsequent tearing or defect of the cap can cause activation of passing platelets and formation of an obstructive clot, which brings on chest pain and other symptoms.

### Mind on MI

Individuals most likely to have an MI are men older than age 45 and woman older than age 55. A history of heart disease in parents and siblings, high BP, diabetes,

tobacco smoking, obesity, and a sedentary lifestyle are other risk factors.

The classic symptoms of MI are left-sided chest pain, with radiation of pain to the left arm or jaw. However, in patients with diabetes, women, individuals over age 65, and renal patients, other signs may be seen, such as right-sided chest pain with radiation to the neck, back, shoulder, or epigastric areas. The patient may experience nausea, shortness of breath, diaphoresis, weakness, or dizziness. There may be a sensation of numbness in the hands or a feeling of impending doom. Most patients will present with a group of symptoms during an MI, but one in three won't have chest pain. Chest pain of varying duration and intensity is more suggestive of an active infarction.

The out-of-hospital survival rate for a patient experiencing MI is less than 5%. Any delay in seeking treatment can result in numerous complications, such as chronic dysrhythmias, congestive heart failure, pericarditis, or rupture of the heart. The patient should call 911 if signs or symptoms don't improve after 5 minutes. People vary widely in their decision to seek treatment and may wait anywhere between 1 and 24 hours after the onset of symptoms. Individuals most likely to seek treatment quickly are those who recognize their signs and symptoms or live in close proximity to a medical facility. Delaying factors include denial of symptoms, advanced age, worries

## Types of atherosclerotic plaque

A computed tomography scan can be used to measure the density of atherosclerotic plaques. From this, three general classifications of plaque are derived:

- exclusively calcified (the most dense and stable)
- calcified and noncalcified
- exclusively noncalcified.

Plaques can also be measured for percentage of macrophage, collagen, or smooth muscle cell content. The number of blood vessels within a plaque may be counted, which is significant because bleeding beneath a plaque can make it more prone to rupture. The most vulnerable plaques have large lipid cores, a thin cap, and active inflammation around the deposit.

about expense, living alone, or attempts to self-treat.

Approximately 7% of patients will slip into a systolic BP of less than 90 mmHg over a period of 4 to 24 hours due primarily to disease of the left anterior descending (LAD) artery. This decreased cardiac output will give the patient cool extremities, low urinary output, altered mentation, pulmonary edema, jugular venous distension, and an S3 heart sound. The heart rate may increase in an attempt to maintain cardiac output, but this only furthers the problem as the heart's demand for oxygen is increased. The period of diastole is also shortened, which means that less blood is being delivered to the heart muscle.

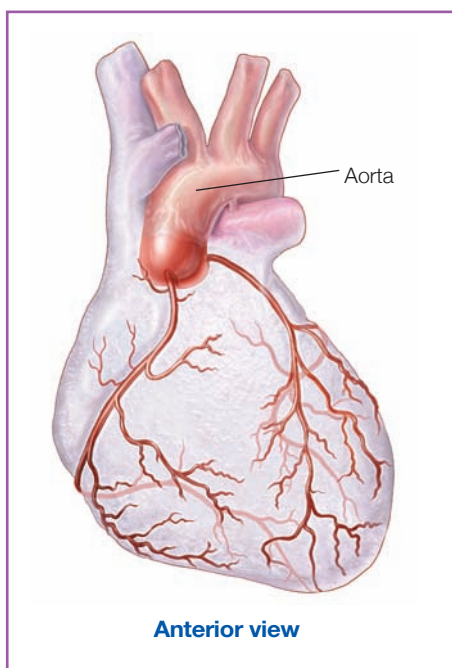
To detect MI, we can use a standard, five-wire bedside ECG monitor that can display two leads. We should first display an inferior lead that looks toward the bottom of the heart, such as lead II, which lines up closely with the electrical path of the heart and prominently displays P-waves and QRS complexes. We could also display lead V1, which is best for evaluating wide QRS complexes of greater than 0.12 seconds and for distinguishing ventricular tachycardia from supraventricular tachycardia.

We then want to examine the ST segment for depression or elevation. Depression of the ST segment of 1 mm or more suggests possible ischemia (partial blockage) of an artery. Spasm or inflammation of a coronary artery can also depress the ST segment. Elevation of the ST segment of 0.5 mm or more

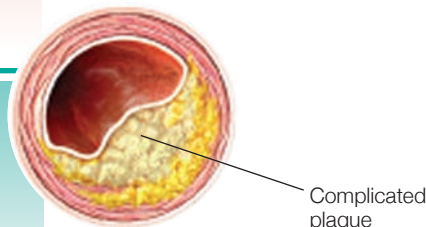
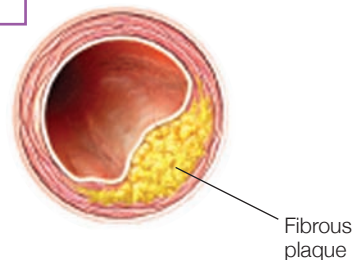
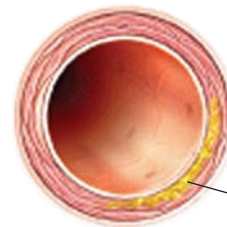
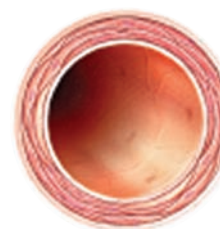
## Picturing CAD

### Coronary arteries

Coronary arteries supply blood to heart tissue. They originate from the aorta.



#### Normal coronary artery



### How it happens

CAD results when atherosclerotic plaque fills the lumens of the coronary arteries and obstructs blood flow to the heart, diminishing the supply of oxygen and nutrients to the heart tissue.

### What to look for

- Angina
- Nausea and vomiting
- Cool extremities
- Diaphoresis
- Pallor



points to possible infarct (complete blockage) of an artery. However, there are other possible causes of ST elevation, such as pericarditis, acute abdomen, and hyperkalemia.

We turn to our 12-lead ECG to determine what areas of the heart are being affected. ST segment elevation along leads V1 to V4 would, significantly, indicate infarct along the anteroseptal wall—the strongest area of heart muscle that’s supplied by the LAD. ST elevation in leads V5 to V6 would suggest infarct of the lateral wall of the heart fed by the left circumflex artery. For a closer look at the left circumflex area, we could move our V4 to V6 lead wires to the V7 through V9 leads positioned along the patient’s back. An inferior lead, such as II, III, or aVF, is used to observe activity on the right side of the heart, or a right-sided ECG may be done to specifically view the area supplied by the right ventricular branch.

As cardiac myocytes sustain insult and damage from interrupted circulation, biomarkers are released into the bloodstream that can be detected with blood tests. A series of blood draws are needed because biomarker levels in the blood fluctuate. CK-MB is creatine kinase specific to heart muscle. CK-MB levels increase 3 to 12 hours after the onset of chest pain, peak at 24 hours, and return to baseline after 48 to 72 hours. Two CK-MB readings of greater than 5 U/L suggest an active MI. Renal disease, trauma, and cardiac surgery may also raise the CK-MB level. Testing CK-MB can detect ischemia earlier than a 12-lead ECG, but it’s less specific than the troponin blood test.

The troponin I test can find even trace amounts of heart damage; for this reason, its use as the main cardiac biomarker has increased markedly. Levels increase 3 to 12 hours after the onset of chest pain, peak at 24 to 48 hours, and return to baseline over 5 to 14 days. A troponin I level of greater than 0.1 ng/mL suggests an active MI. Troponin

is slower, however, in detection of a repeat MI. And troponin levels can be raised by other noncardiac conditions, such as sepsis and pulmonary embolus.

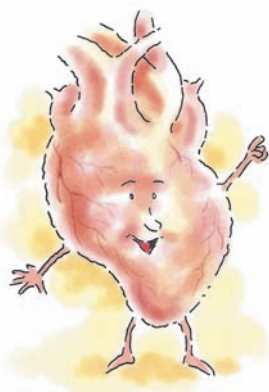
Biomarkers are usually drawn at admission and at 6 and 12 hours afterward. An MI is normally ruled out after three consecutive, negative biomarker results. However, an additional troponin draw may be necessary because its appearance can be delayed past 12 hours. Bedside blood testing devices exist for troponin and CK-MB that can display results in less than 20 minutes.

### Action STAT

It’s important to learn the time of onset of chest pain or other symptoms because this may affect the mode of treatment used. Give oxygen by nasal cannula to keep the patient’s oxygen saturation at greater than 95% because increased oxygen levels to the myocardium can lessen ischemic pain.

Aggregated platelets set into motion other clotting mechanisms. Thromboxane A2 (TXA2) is a substance secreted by platelets that attracts neighboring platelets. Aspirin blocks production of TXA2. Oral aspirin, 324 mg, may be given unless the patient has an aspirin allergy, a history of peptic ulcer, or a recent gastrointestinal (GI) bleed, stroke, or surgery.

Nitroglycerin tabs or spray can be given sublingually at 0.4 mg every 5 minutes up to three doses in an attempt to eliminate chest pain. BP must be checked before each dose because nitro is a potent vasodilator. If chest pain persists, a nitro drip may be hung at 10 to 20 mcg/minute and can be increased by 10 mcg every 3 to 5 minutes until the pain is gone or the patient’s systolic BP approaches 90 mmHg; the max dose is 200 mcg/minute. Nitro must be given with extreme caution in right-sided MI because the infarcted right ventricle is very sensitive to fluid levels and often needs additional I.V. fluids to maintain cardiac output.



### did you know?

A subtle but useful finding on the ECG is the deep V shape greater than 0.03 seconds that precedes the QRS complex. The Q-wave signals an area of dead heart tissue that can no longer depolarize. This patient has had a previous MI.

Morphine, 2 to 4 mg, can be given by slow I.V. push every 5 to 15 minutes for chest pain unrelieved by nitroglycerin. However, morphine also has a vasodilative effect that diverts fluid volume to the peripheral circulation away from the heart so must also be given with caution in right-sided MI.

### Treatment treatise

Multiple medications may be offered to cardiac patients because agents target platelet and coagulation activity at different points.

Clopidogrel is an antiplatelet aggregation drug that inhibits the action of the adenosine diphosphate (ADP) receptor on the platelet surface that helps activate the IIb/IIIa receptor site. Clopidogrel is an alternative for patients with an aspirin allergy or can be given along with aspirin for up to 1 year post-MI to help maintain circulation to the heart. It can be given after PCI to prevent stent obstruction. The loading dose is 300 to 600 mg by mouth, followed by 75 mg/day for 3 months to 1 year depending on the type of stent. MI patients who are treated with fibrinolytics may benefit from concurrent clopidogrel treatment. Prasugrel can be offered as a substitute for patients who respond poorly to clopidogrel. It exhibits a more robust inhibition of the ADP receptor but also has a greater risk of bleeding.

Ticagrelor is a new antiaggregation agent that exerts a short-term effect on the platelet ADP receptor. The patient is at less risk for bleeding after therapy; however, it must be taken with low-dose aspirin to achieve the desired effect. Ticagrelor also has multiple interactions with other drugs and is contraindicated in patients with a history of intracranial bleeding or severe liver disease.

Abciximab and eptifibatid block the binding of fibrinogen to the IIb/IIIa receptor. These drugs are considered for patients with moderate-to-high biomarker levels. They have a faster onset of action than

### did you know?

Fish oil is rich in the omega-3 fatty acids eicosapentaenoic acid, or EPA, and docosahexaenoic acid, or DHA, which exert an anti-inflammatory effect on cardiac myocytes. Consumption of 1 g/day of fish oil can reduce the resting heart rate, increase the stability of plaque, and reduce the risk of cardiovascular events.

clopidogrel and are given along with heparin during PCI and stent placement. Renal failure patients will need an adjusted (lower) dose.

Heparin binds with antithrombin to increase its inhibitory effects on thrombin. This, in turn, prevents conversion of fibrinogen into fibrin clot. Heparin is given during PCI to prevent further clot formation. Its starting dose is based on patient weight. Heparin isn't cleared by the kidneys. Its effects can be reversed if necessary with 1 mg of protamine sulfate I.V. per every 100 units of heparin.

Patient response to heparin is inconsistent and the treatment window very narrow. The activated partial thromboplastin time (aPTT) must be frequently measured and kept within 1.5 to 2.5 times control. Thirty percent of patients on a heparin drip for 2 to 4 days will exhibit a moderate drop in platelet count that usually self-resolves. Three percent of patients will develop antibodies to heparin, which will activate thrombin and can cause a 50% drop in platelet count. Heparin in all forms (drip, I.V. line flushes) must then be stopped and another type of anticoagulant substituted.

Low-molecular-weight heparin (LMWH) is made from a segment of the heparin molecule. LMWH has an increased bioavailability and a longer half-life than unfractionated heparin. It provides more consistent anticoagulation and doesn't require frequent blood testing. Its dose, also weight-based, can be delivered by subcutaneous injection once or twice a day. LMWH is associated with less

If your patient has had an MI, there are several treatments available.



memory jogger

## CAD risk factors

To remember the risk factors for CAD, just think about the word **RISKS**:

**R**ising LDL and triglyceride levels—LDLs should be < 130 mg/dl, triglycerides < 200 mg/dl.

**I**nadequate control of hypertension, diabetes, and obesity—Diet and exercise lifestyle changes are the first step to regaining control.

**S**ex—CAD is more common in men until after age 75.

**K**infolk—Heredity is a nonmodifiable risk factor.

**S**moking—The sooner stopped, the better.

risk of bleeding or development of antibodies. Protamine sulfate, however, can only partially correct the action of LMWH. Renal patients will require a 50% lowering of the dosage.

Direct thrombin inhibitors (DTIs), such as bivalirudin and ximelagatran, block the action of thrombin by binding directly to it. DTIs can be used as an alternative for patients who can't tolerate heparin. The main disadvantage of this class of medication is that it carries a high risk of bleeding. aPTT must be monitored and kept between 1.5 and 2.5 times control. Patients receiving DTIs must have intact renal function, and DTI treatment has been associated with an elevation of liver enzymes.

Warfarin reduces the production of vitamin K in the liver and makes it impossible for vitamin-K-dependent coagulation factors 2, 7, 9, and 10 to function. Complete

anticoagulation is achieved after 8 to 15 days as the coagulation factors already in circulation gradually taper off. Warfarin is given to patients who have a clot in the left ventricular circulation during MI or who have developed disease of the mitral valve. It can also be given along with low-dose aspirin for up to 4 years following an MI to prevent reinfarction.

Disadvantages of warfarin include its narrow therapeutic window and variable patient response. It has multiple interactions with various foods and medicines. The prothrombin time (PT) and international normalized ratio (INR) must be frequently measured and checked; more often if the patient is ill, has changed his or her food or drug routine, or has shown upward spikes in PT/INR results. Warfarin use is a common cause of GI bleeding. Older patients being treated with warfarin are at increased risk for intracranial bleed. The antidote is 5 to 10 mg of vitamin K or, in more serious cases, fresh frozen plasma.

Fibrinolytics are designed to dissolve fibrin clot by converting plasminogen to plasmin. These medications are considered for a patient with infarction if signs and symptoms began less than 3 hours before administration and PCI will be delayed beyond 2 hours. Use is contraindicated if signs and symptoms began more than 24 hours ago or if the patient is scheduled for coronary artery bypass graft surgery. Other contraindications include recent surgery or trauma, peptic ulcer, severe hypertension, or brain cancer. The greatest risk of fibrinolytic use is intracranial bleeding. These agents are more frequently used in areas of the world where cardiac catheterization isn't available. Their use, however, often results in reocclusion of the artery and failure to restore blood flow.

PCI is considered if the onset of signs and symptoms began less than 3 hours before the patient presented and if fibrinolytics are contraindicated. Balloon inflation of an obstructed artery within 6 hours of

symptom onset yields the best results. The American Heart Association has proposed a door-to-balloon time of less than 90 minutes.

Insertion of the procedural catheter is usually through the femoral or radial arteries. There has been less access site bleeding with the radial artery approach because it's more easily accessible and compressible. It's superior to the brachial artery approach because the hand can remain perfused via the ulnar artery and the patient is able to ambulate sooner after the procedure. After balloon inflation, a stent is inserted to keep the artery open, or a rotational cutter is used to shave the plaque. Radiation is then applied to the area with brachytherapy to prevent reocclusion.

Success of PCI is related to the degree of vessel obstruction and the condition of the vessel. Renarrowing of the artery can occur if the inner lining of the vessel is torn, and a clot can develop at the balloon site. Other possible complications include bleeding, dysrhythmias, infarction, stroke, and renal failure. Metal mesh, drug-eluting stents are placed to reduce blockage from inflammation and clotting. Concurrent antiplatelet therapy is added to prevent stent thrombosis. Some protocols call for clopidogrel and aspirin treatment for up to 2 years.

### Tell your patient

Patients on anticoagulants should use caution and try to avoid trauma from falls and bleeding. Antifall measures include the use of nonslip rugs, night-lights, and extra handrails. Patients should also use an electric razor and a soft toothbrush to prevent cuts and bleeding gums.

Patients taking warfarin should know the vitamin K content of foods and consume a consistent amount of vitamin-K-containing foods and beverages each day to avoid wide swings in INR test results. Foods high in protein can lessen the effect of warfarin, and numerous medications

## Prevention strategies

cheat

sheet

- Participate in a regular exercise program.
- Follow a low-cholesterol diet.
- Maintain a normal weight for height.
- Quit smoking.
- Avoid/control diabetes.
- Control BP.

Exercise, healthy eating, and quitting smoking can reduce CAD risk.



can potentiate the drug. Over-the-counter products containing aspirin should be avoided. Warfarin reacts unpredictably to alcohol intake and the risk of bleeding may increase.

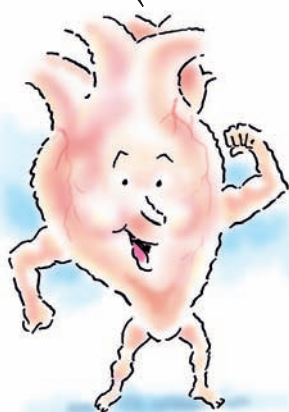
Warfarin should be taken at the same time each day. Tablets of different milligram strengths are color-coded for safety. Changing the brand of warfarin can alter the patient's response. Each manufacturer makes the pill in a different shape to help identify the brand. Older patients will require a decrease in dosage due to diminished liver function. Patients older than age 65 are at greater risk for an adverse drug reaction due to the potential for decreased cognition and compliance.

### Winning prevention strategies

An exercise program can significantly lessen the risk of CAD. Patients who've had an MI and have undergone PCI should postpone exercise for 1 month until cleared by their healthcare provider. Exercise for the cardiovascular patient can include walking or doing 30 minutes of moderate aerobic activity for most days of the week. The intensity is sufficient if the patient feels mildly exerted afterward. Walking is economical and can be divided into short sessions to fit lifestyle and encourage compliance. Sedentary persons, once cleared by their healthcare provider, can begin exercising at 10 to 15 minute intervals and then increase to 30 minutes/day.



Controlling diabetes and BP helps keep me healthy.



Besides a regular exercise program, other important prevention strategies to teach your patient include:

- following a low-cholesterol diet. Cholesterol is a key component of atherosclerotic plaque. Genetics plays some role in high serum cholesterol levels, but environmental factors such as dietary habits are also important contributors.
- maintaining a normal weight for height. Obesity usually contributes to higher CAD risk. After age 30, volumes of food consumed must decrease because individuals in their 30s and older require fewer calories to maintain adequate body weight.
- quitting smoking. The incidence of MI is significantly lower in nonsmokers and risk also decreases for people who quit smoking. It's also advisable to avoid second-hand environmental tobacco smoke.
- avoiding/controlling diabetes. The risk of CAD is two times greater in men and three times greater in women diagnosed with diabetes. The hyperinsulinemia associated with diabetes contributes to adverse remodeling of vascular structures.
- controlling BP. Uncontrolled BP may enlarge the heart over time. An enlarged heart can have less stamina than the normal-sized heart. High BP is also associated with accelerated atherosclerosis.

### Just say no to CAD

With CAD, an ounce of prevention is worth a pound of cure. The direct care nurse is the soldier on point who can inform patients and families about proper exercise and sensible dietary patterns that can delay the development of atherosclerotic plaque. ■

### Learn more about it

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