

# Attacking anterior-wall myocardial infarction in time

Responding to a dangerous myocardial infarction is all about time. Make sure you have the skill and knowledge you need to beat the clock.

**IN THE UNITED STATES**, between 1.2 and 1.5 million people suffer a myocardial infarction (MI) every year. And among MIs, anterior-wall MIs are the most serious and have the worst prognosis. Typically, they are larger than other MIs, and they can result in significant ventricular wall-motion abnormalities and a significantly lower ejection fraction.

Patients who survive anterior-wall MIs remain at high risk for recurrent cardiac events, and 10% die in the first year. Most deaths occur in the first 3 to 4 months and result from advanced atherosclerotic coronary artery disease (CAD). But early recognition of the signs and symptoms and prompt treatment can improve outcomes.

The primary risk factors for atherosclerotic CAD and anterior-wall MI are hyperlipidemia, diabetes mellitus, hypertension, and cigarette smoking, all of which can be modified, and male gender and a family history of atherosclerotic CAD. Although atherosclerotic CAD and anterior-wall MI occur in more men than women in all age-groups, the gender gap narrows with increasing age.

## Myocardial ischemia, injury, and infarction

When a coronary artery occlusion blocks the blood supply, the affect-

ed area of the myocardium progresses through three stages: ischemia, injury, and infarction. Myocardial ischemia and injury are reversible. MI is not. (See *Coronary artery circulation at a glance*.)

The causes of occlusion leading to ischemia, injury, and infarction include:

- coronary artery thrombosis
- coronary artery spasm from causes including cocaine use
- decreased coronary blood flow because of arrhythmias, pulmonary embolism, hypotension, or shock
- increased myocardial workload

because of emotional stress, increased blood volume, or exertion.

Anterior-wall MI is categorized as a ST-segment elevation MI (STEMI), the most common type of MI. These MIs usually begin in the subendocardium, which is the area with the greatest oxygen demand and the least blood supply. The infarction progresses outward until it involves the full thickness of the myocardium; myocardial necrosis is usually complete.

## Recognizing MI

The World Health Organization has three criteria for a diagnosis of MI:

- a patient history of severe, prolonged chest pain
- unequivocal electrocardiogram (ECG) changes that include abnormal and persistent Q waves
- changes in serial cardiac biomarker levels that indicate myocardial injury and infarction.

## Signs and symptoms

The risk of death from an anterior-wall MI is greatest in the first 24 to 48 hours after symptoms begin, so early diagnosis and treatment are critical to preserve myocardial function and prevent complications. The first symptom is usually deep, sub-

By Rose M. Coughlin, MSN, RN, APRN-BC



## LEARNING OBJECTIVES

1. Describe the signs and symptoms of anterior-wall myocardial infarction (MI).
2. Recall the diagnostic techniques used to detect anterior-wall MI.
3. Describe the medical management of anterior-wall MI.
4. Discuss the nursing management of anterior-wall MI.

## Coronary artery circulation at a glance

sternal, visceral pain described as aching or pressure that radiates to the back, jaw, left side of the neck, or left arm. MI can occur any time of the day, but most occur within 3 hours of awakening, and the pain is continuous for 30 minutes or more. Other signs and symptoms include:

- cool, pale, diaphoretic skin
- dyspnea or orthopnea
- epigastric discomfort with nausea and vomiting
- fatigue
- impaired cognitive function
- Levine's sign (clenched fist held over the sternum)
- palpitations
- peripheral or central cyanosis
- restlessness and apprehension
- syncope or near-syncope.

Women, diabetics, African Americans, and the elderly may experience different signs and symptoms. In women, common signs and symptoms include unusual fatigue, sleep disturbances, shortness of breath, indigestion, and anxiety. Many women describe chest discomfort as aching, tightness, pressure, sharpness, burning, fullness, or tingling. Women may also experience weakness, cold sweating, dizziness, pain or pressure in the back or high chest, pain or discomfort in one or both arms, irregular heart rate, and nausea.

According to the American Heart Association, 3 to 4 million Americans—especially women, diabetics, and African Americans—have *silent ischemia*, a mild discomfort that may go unnoticed. In the elderly, symptoms may differ and be blamed on arthritis. Elderly people with dementia may have difficulty communicating that they are having pain.

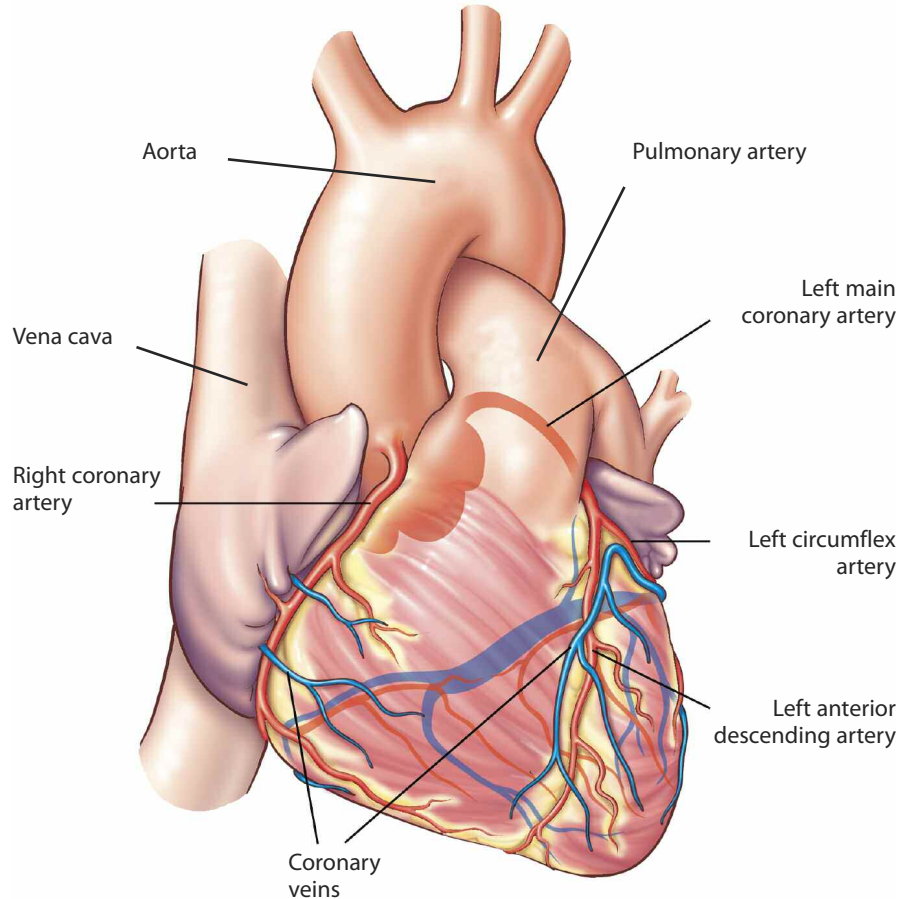
### Reading the 12-lead ECG

A 12-lead ECG should be done within 10 minutes of the patient's arrival in the emergency department (ED). Ischemia, injury, and infarction cause characteristic changes on a 12-lead ECG. Changes in the leads facing the damaged myocardium are called *indicative changes*. Changes in the

The left main coronary artery divides into the left anterior descending (LAD) artery and the left circumflex artery. The LAD supplies blood flow to the anterior two-thirds of the intraventricular septum, the anterior left ventricle, the lateral left ventricle, bundle of His, and bundle branches. The left circumflex artery wraps around to the posterior wall of the heart and supplies blood flow to the left atrium, the sinoatrial node in 45% of hearts, the atrioventricular node in 10% of hearts, and the lateral and posterior wall of the left ventricle.

The right coronary artery supplies blood to the right atrium, right ventricle, inferior left ventricle, and posterior intraventricular septum.

The coronary veins deliver oxygen-poor blood to the right atrium.



Courtesy of The Cleveland Clinic, Cleveland, Ohio

leads that don't face the damaged tissue are called *reciprocal changes*:

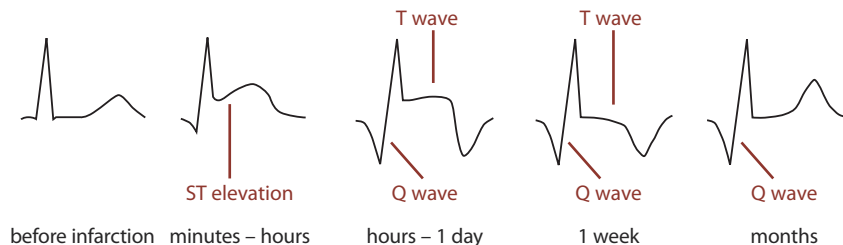
- *Ischemia* produces symmetrically inverted T waves in the indicative leads and tall T waves in the reciprocal leads.
- *Injury* produces ST-segment elevation in the indicative leads and ST-segment depression in the reciprocal leads.
- *Infarction* produces pathologic Q waves (waves that are 0.4 second wide or one-quarter of the R-wave height) in the indicative leads and tall R waves in the reciprocal leads. ST-segment elevation indicates

myocardial injury and requires immediate intervention. This elevation appears in the early hours of the infarction and lasts from several hours to several days. For several weeks after the ST segment returns to baseline, the T wave may remain inverted. Over time, the T wave resumes its upright position, but the Q-wave changes remain. (See *T wave changes as MI progresses*.)

If the initial ECG doesn't indicate STEMI, but the patient remains symptomatic and the clinicians still suspect STEMI, serial or continuous 12-lead monitoring should be used to detect ST-segment elevation. Be-

## T wave changes as MI progresses

These five waveforms show the progression of an MI as reflected by the T wave.



cause symptoms of MI can be subtle, an ECG should be performed on any patient older than age 45 who is experiencing new epigastric pain or discomfort.

The location of an MI depends on which coronary artery is occluded. The size and location of the infarction determines the immediate and long-term effects. In an anterior-wall MI, the left anterior descending artery, which supplies blood to the large muscular anterior wall of the left ventricle and the anterior two-thirds of the intraventricular septum, becomes occluded. (See *Linking MI location and ECG changes*.)

When a patient has an anterior-wall MI, you'll see the indicative changes in leads V<sub>1</sub> through V<sub>4</sub> and the reciprocal changes in lateral leads I and aVL and inferior leads II, III, and aVF. In leads V<sub>1</sub> through V<sub>4</sub>, you'll see that the normal R-wave progression is lost. The higher or more proximal the occlusion, the more muscle damage that occurs.

Anterior-wall MIs can be categorized as anteroseptal, anterolateral, true anterior, and extensive anterior infarcts. Anteroseptal infarcts involve the anterior part of the intraventricular septum and produce changes in leads V<sub>1</sub> through V<sub>3</sub>. Anterolateral infarcts result from the occlusion of the left main coronary artery, and changes appear in leads V<sub>5</sub>, V<sub>6</sub>, I, aVL, and sometimes V<sub>4</sub>. A true anterior infarct doesn't involve the septum or the lateral wall and causes abnormal Q waves or ST-segment elevation in leads V<sub>2</sub> through V<sub>4</sub>. An extensive anterior infarction affects the anterior wall plus the anteroseptal or anterolateral wall and causes

abnormal Q waves or ST-segment elevation in any or all of the precordial leads V<sub>1</sub> through V<sub>6</sub>, I, and aVL.

### Biomarkers and echocardiography

Serum cardiac biomarkers are used to detect myocardial injury and infarction. Measurements of creatine kinase (CK) and CK-MB have been the standard serum markers of MI. These enzymes are released with tissue necrosis. Blood levels increase 4 to 6 hours after MI and return to normal in 24 to 48 hours. However, troponin T has become the preferred cardiac biomarker because it's more specific for MI. The level of this biomarker rises 3 to 5 hours after MI and remains elevated up to 21 days.

Echocardiography may also be performed to compare areas of the left ventricle that are contracting normally with those that are not. The echocar-

diogram can help identify which coronary arteries are occluded and which portion of the heart is affected.

### Treating MI

Treatment goals include relieving pain, providing adequate oxygenation to the myocardium, preventing platelet aggregation, restoring coronary blood flow, and salvaging the functional myocardium. (See *Hospital quality measures for MI*.)

Immediate treatment for chest pain consists of:

- Morphine sulfate 2 to 4 mg I.V. in increments of 2 to 8 mg repeated at 5- to 15-minute intervals
- Oxygen if oxygen saturation is less than 90%
- Nitroglycerin 0.4 mg sublingual every 5 minutes for three doses, after which the need for I.V. nitroglycerin is assessed
- Aspirin 162 to 325 mg chewed for rapid buccal absorption.

The acronym MONA can help you remember morphine, oxygen, nitroglycerin, and aspirin, but remember, too, that MONA doesn't reflect the therapeutic sequence. Aspirin and oxygen come before nitroglycerin and morphine. Nitroglycerin may be given as a translingual spray (Nitromist); administer 1 to 2 sprays under the

### Linking MI location and ECG changes

The location of the infarction and the electrocardiogram (ECG) leads that reflect the damage both depend on which coronary artery is blocked.

Infarction site	Coronary artery	Indicative leads	Reciprocal leads
Anterior wall	Left anterior descending	V <sub>1</sub> -V <sub>4</sub>	II, III, aVF
Lateral wall	Left circumflex	I, aVL, V <sub>5</sub> , V <sub>6</sub>	II, III, aVF
Inferior wall	Right coronary artery (RCA)	II, III, aVF	I, aVL
Posterior wall	RCA or left circumflex	None. Posterior leads V <sub>7</sub> -V <sub>9</sub> may be used.	V <sub>1</sub> -V <sub>2</sub>



## Hospital quality measures for MI

In 2004, The Joint Commission and the Centers for Medicare & Medicaid Services worked together to establish *hospital quality measures* to improve the quality of care for hospitalized patients and allow hospitals to be compared to one another to improve performance.

The measures for MI include:

- giving aspirin within 24 hours before or after hospital arrival
- giving a fibrinolytic within 30 minutes of hospital arrival
- performing percutaneous coronary intervention (PCI) within 120 minutes of hospital arrival
- giving smoking cessation advice or counseling during the hospital stay, if necessary
- prescribing an angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker for left ventricular systolic dysfunction at discharge
- prescribing a beta blocker at discharge
- prescribing aspirin at discharge.

The American College of Cardiology (ACC) and the American Heart Association (AHA) also recommend assessing low-density lipoprotein cholesterol levels and prescribing antilipemic drug therapy at discharge as necessary. The ACC and AHA recommend PCI within 90 minutes of first medical contact, not 120 minutes, and aspirin as soon as possible.

tongue every 5 minutes for a maximum of three doses in 15 minutes. Don't rinse the patient's mouth for at least 5 to 10 minutes. If a patient has a systolic blood pressure of less than 90 mm Hg or one that's 30 mm Hg or more below baseline with marked bradycardia or tachycardia, or known or suspected right ventricular infarction, avoid using any nitrates. Also, if the patient has taken sildenafil or vardenafil within the last 24 hours or tadalafil within the last 48 hours, don't give nitrates because the hypotensive effects will be exaggerated.

Within 24 hours of the onset of MI symptoms, the patient should receive a beta blocker, such as metoprolol or carvedilol. Contraindications to beta-blocker use include heart failure, low-output state, increased risk for cardiogenic shock, heart block, and active asthma. For secondary prevention, beta-blocker therapy will continue after discharge because it decreases mortality after MI by 30%.

Angiotensin-converting enzyme (ACE) inhibitors, such as enalapril and captopril, reduce the risk of death if given orally in the first 24 hours of ST-segment elevation to patients with anterior-wall MIs. Ideally, you should give these drugs after fibrinolytic therapy is completed and blood pressure is stabilized. ACE inhibitors help reduce afterload by reducing the work of the heart and decreasing ventricular remodeling that occurs with MI. Before giving an ACE inhibitor, determine the patient's baseline creatinine level and blood pressure. If a patient can't tolerate ACE inhibitors, administer angiotensin receptor blockers instead.

Clopidogrel, an oral antiplatelet drug, should be given with aspirin and continued for at least 14 days. During hospitalization, patients should not receive any non-steroidal anti-inflammatory drugs except aspirin because of the higher risk of morbidity and mortality.

Ideally, patients should be treated with percutaneous coronary intervention (PCI) within 90 minutes of

the first medical contact. PCI can restore coronary blood flow in 90% to 95% of MI patients, and early PCI reduces mortality rates. Using a stent with PCI is better than not using one because stenting reduces the need for subsequent target-vessel revascularization. If the hospital doesn't have PCI capability, the patient can't be transferred within 90 minutes, and the patient is eligible for fibrinolytic therapy, administer it.

Fibrinolytic therapy reduces the risk of death and salvages the myocardium. Fibrinolytic drugs include alteplase (t-PA), streptokinase, anistreplase, reteplase (r-PA), and tenecteplase. Ideally, the patient should receive a fibrinolytic within 30 minutes of first medical contact. Contraindications include previous hemorrhagic stroke, active internal bleeding, suspected aortic dissection, and intracranial neoplasm. Anticoagulant therapy is recommended for patients who have received PCI or fibrinolytic therapy.

### Complications of anterior-wall MI

An anterior-wall MI may produce varying degrees of atrioventricular (AV) or fascicular heart block—such

as first-degree AV block, type II second-degree AV block, third-degree AV block with ventricular escape, and bundle-branch block. Bradycardia or heart block with anterior-wall MI is a poor prognostic sign.

Other complications include severe left ventricular dysfunction, resulting in heart failure and cardiogenic shock, ventricular septal rupture, and ventricular free-wall rupture.

### Monitoring the patient

Nursing care of the anterior-wall MI patient in the coronary care and step-down units includes the following:

- managing and alleviating chest pain
- assessing and reducing anxiety
- monitoring laboratory test results, especially potassium and magnesium levels because low levels can lead to arrhythmias
- monitoring the ST segment continuously to help detect silent ischemia or recurrent ischemia and to determine the effectiveness of reperfusion therapy
- monitoring the patient for signs of arrhythmias
- monitoring arterial oxygen saturation by pulse oximetry
- creating an individualized plan for

the patient's physical activity, keeping in mind that hemodynamically stable patients with uncomplicated MI shouldn't be confined to bed for more than 12 hours

- assessing the patient for signs and symptoms of heart failure, such as crackles, increased jugular venous pressure, and pedal edema
- assessing heart sounds for new murmurs, indicating valve involvement; muffled heart sounds, indicating cardiac tamponade; and pericardial friction rub, indicating pericarditis
- assessing the patient to ensure the efficacy of the drug regimen
- giving stool softeners daily to prevent straining.

### Patient teaching

Discharge planning and patient teaching should begin early in the hospital stay.

- Explain the need for a diet that's low in saturated fat and cholesterol and that includes omega-3 fatty acids, fruits, vegetables, soluble fiber, and whole grains.
- Explain that the patient's lipid status will be evaluated and drugs may be prescribed.
- Encourage eligible patients to engage in moderate-intensity aerobic activity, such as walking, jogging, or cycling, at least 30 to 60 minutes a day on most—preferably all—days of the week.
- If the patient's blood pressure is 140/90 mm Hg or higher (or

130/80 mm Hg or higher in a patient with chronic kidney disease or diabetes), recommend lifestyle changes, such as weight control, and explain that antihypertensive drugs may be needed.

- Strongly encourage the patient and family to stop smoking and avoid secondhand smoke.
- Tell the patient to take one nitroglycerin dose sublingually for chest pain and to call 911 immediately if the pain doesn't improve or gets worse within 5 minutes.
- Make clear that if symptoms of MI develop, the patient should be transported to the hospital by ambulance.
- Advise family members to take cardiopulmonary resuscitation training and become familiar with using an automated external defibrillator.

### Matter of time

Preventing death from MI is a matter of time. The patient needs to seek treatment quickly, and you need to act fast to recognize the signs and symptoms and intervene appropriately. ★

### Selected references

Alspach JG. *Core Curriculum for Critical Care*. 6th ed. Philadelphia, Pa: W.B. Saunders Co; 2006.

American College of Cardiology/American Heart Association Task Force on Practice Guidelines. ACC/AHA clinical performance measures for adults with ST-elevation and non-ST elevation myocardial infarction. *J Am Coll Cardiol*. 2006;47(1):236-265.

American College of Cardiology/American Heart Association Task Force on Practice Guidelines. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. *J Am Coll Cardiol*. 2004;44:671-719.

Antman EM, Armstrong PW, Green LA, et al. 2007 focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. <http://content.onlinejacc.org/cgi/content/full/jacc.2007.10.001>. Accessed December 18, 2007.

Bajzer CT. Acute myocardial infarction. The Cleveland Clinic Disease Management Project. [www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute/acute.htm](http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute/acute.htm). Accessed November 20, 2007.

DeLuca G, Suryapranata H, Ottervanger JP, Antman EM. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. *Circulation*. 2004;109:1223-1225.

Field JM, ed. *Advanced Cardiovascular Life Support Provider Manual*. Dallas, Texas: American Heart Association; 2006.

Sole ML, Klein DG, Moseley MJ. *Introduction to Critical Care Nursing*. 4th ed. St. Louis, Mo: Elsevier Saunders; 2005.

Tschopp D, Brener S. Complications of acute myocardial infarction. The Cleveland Clinic Disease Management Project. <http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/complications/complications.htm>. Accessed November 20, 2007.

Woods SL, Sivarajan Froelicher ES, Underhill Motzer S, Bridges EJ. *Cardiac Nursing*. 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2005.

**Rose M. Coughlin, MSN, RN, APRN-BC, is a Clinical Nurse Specialist on the Cardiothoracic Stepdown Units at the Cleveland (Ohio) Clinic. The planners and authors of this CNE activity have disclosed no relevant financial relationships with any commercial companies pertaining to this activity.**

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