STEMI Alert!

Rapid Response to Acute Myocardial Infarction
A NATIONAL EPIDEMIC

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Purpose and Goals

Heart disease is the leading cause of death in the United States. A complication of heart disease is acute MI, which itself has a significant mortality rate. The goal of this course is to assist healthcare professionals in recognizing the signs and symptoms of acute ST-segment elevation MI (STEMI) and implementing immediate interventions. Cardiac pathophysiology is reviewed, and complications associated with STEMI as well as post-MI treatments are explained.

Introduction

Acute MI is one condition in a spectrum of illnesses known as acute coronary syndrome. The cause of acute MI can be traced to several causes, but the main perpetrator of a "heart attack" is coronary artery disease (CAD). Acute MI is an emergency diagnosis that requires quick thinking on the part of the nurse and immediate intervention from the nurse as well as the physician and other healthcare staff. The majority of MI-associated deaths are a result of fatal arrhythmias, such as ventricular fibrillation, that can be stopped with prompt intervention, including emergency cardiopulmonary resuscitation (CPR), defibrillation, and advanced cardiac life support (ACLS).

Treatments for acute MI are ever changing and have resulted in a decrease in MI-related mortality in the past decades. Thrombolytic therapy, a revolutionary treatment a few years ago, has been replaced with percutaneous coronary intervention (PCI) as the gold standard for care because of its proven effectiveness in treating acute MI. Because of the time-dependent benefits of medical intervention in the event of acute MI, and because most patients delay seeking treatment, the National Heart, Lung, and Blood Institute (NHLBI) initiated the National Center of Continuing Education, Inc. to design courses to ensure dosage recommendations and treatments are precise and agree with the highest standards of practice. However, as a result of accumulating clinical experience and continuing laboratory studies, dosage schedules and/or treatment recommendations are often altered or discontinued. In all cases the advice of a physician should be sought and followed concerning initiating or discontinuing all medications or treatments. The planner(s), author(s) and/or editor(s) of each course have attested to no conflict of interest nor bias on the subject. The National Center of Continuing Education, Inc. does not accept commercial support on any course nor do they endorse any products that may be mentioned in the course. Any off-label use for medications mentioned in a course is identified as such.

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Heart Attack Alert Program (NHAAP) to make healthcare providers aware of the American College of Cardiology/American Heart Association’s guidelines for treating patients. The NHAAP also made great efforts to educate patients about the symptoms of an acute MI and appropriate actions to take. The medical community continues these efforts today.

Patients experiencing an acute MI may present with symptoms in any environment, including the hospital emergency room, a physician’s office, an ambulatory clinic, and any other department within the hospital. With this in mind, it is important for nurses in a variety of settings to be familiar with the basics of an acute MI. They should be able to recognize the signs and symptoms of acute MI and be prepared to take immediate action. This course provides nurses with essential information regarding an acute MI. It explains signs and symptoms, describes ST elevation on a 12-lead electrocardiogram (EKG), summarizes nursing interventions and pharmacologic management, and outlines associated emotional and psychosocial issues related to acute MI. Information on patient education is also presented.

The reader should keep in mind that certain adjunctive treatments may vary slightly depending on the policies of individual healthcare facilities, but the information provided is based on recommendations from the American Heart Association (AHA) and other leading medical groups, information from drug manufacturers, research in current literature, and the experiences of the authors.

A Possible Scenario

During change of shift, Marcy and Tessa sat at the nursing station on the ortho unit. Marcy, the day-shift nurse, quickly reported off to Tessa on the patient in room 701: “Mr. Olsen is a 62-year-old male patient who was admitted to our floor Tuesday after a left knee replacement. He has a dry and intact dressing on his knee. He has a fentanyl PCA and his pain seems to be under control. He has been pretty sleepy all evening and really has not had any complaints. I should probably mention he is a large guy. He’s 6’1” and he weighs about 110 kg. He is a smoker, but his anxiety level is okay for now. I also read in his chart earlier this evening in the history and physical that he has a family history of heart disease. I guess it’s a good thing that he’s healthy except for a bad knee. I cannot think of anything else to tell you about this gentleman. It should be a quiet night for you.”

After gathering report on every patient, Tessa performed her assessments and passed her evening meds. During that time, Mr. Olson described some vague knee pain, but overall said he was feeling good. Around 3 a.m., when everything was quiet, Mr. Olsen put on his call signal and indicated that he was having pain. Tessa assumed he was referring to his knee pain. “He probably needs a couple Norco to reduce his pain enough to sleep,” Tessa thought to herself.

She stopped by the medication dispenser and withdrew a couple hydrocodone tablets on her way to his room. Mr. Olsen looked extremely anxious. When Tessa inquired about the intensity and location of his pain, he stated that his knee felt fine, but he was experiencing a tight, squeezing sensation in the center of his chest and rated the pain a 9 on a scale of 1–10. He indicated that just before he called the nurse’s station, he awoke with serious discomfort. Tessa quickly assessed the patient and noted his blood pressure was 167/89, his pulse was 90, and his respirations were 22. His skin was pale, cool, and clammy. A quick chest auscultation revealed clear and equal breath sounds and normal heart sounds.

Tessa realized that the symptoms were most likely unrelated to his knee surgery and recalled the classic signs and symptoms of acute MI. She also remembered the importance of early treatment and immediately called Mr. Olsen’s attending physician. The doctor ordered a 12-lead EKG. He ordered nitroglycerin sublingual 0.4 mg q 5 minutes × 3 as long as the patient’s blood pressure remained elevated. He also ordered 162 mg aspirin to be chewed. After reviewing the faxed EKG, the physician called Tessa and told her that the cardiologist and the cardiac catheterization lab team were on their way to take the patient for an urgent cardiac catheterization because the 12-lead EKG showed significant ST-segment elevation in leads II, III, and AVF. Tessa recalled her EKG training and knew that Mr. Olson was having an inferior MI. The doctor ordered STAT lab draws for cardiac enzymes, as well as a complete blood count, a coagulation study, and a basic metabolic panel. He also ordered 5 mg morphine IV to be administered immediately.

Within 15 minutes, the cath lab team arrived to take Mr. Olson for percutaneous coronary intervention, where the cardiologist placed a stent to open what was a 95% occlusion of the right coronary artery. Afterward, Mr. Olson was transferred to the cardiac care unit for further monitoring. Twenty-four hours later, he was transferred to a telemetry unit and continued his recovery until he was discharged from the hospital.

The Statistics

Mr. Olsen’s case is not unique or unusual in the hospital setting. Although managing an acute MI usually isn’t a daily occurrence for nurses on a busy medical-surgical ward, it is not uncommon for nurses to care for patients who suffer an acute MI during their hospitalization for another problem or disease. With the prevalence of cardiovascular disease, nurses need to be aware of CAD-related, and possibly fatal, conditions.

A large number of men and women in the United States suffer from cardiovascular disease. Almost three fourths of Americans between the ages of 60 and 79 and about one third of men and women between ages 40 and 59 suffer from CAD. In 2008, nearly 400,000 U.S. men, and about that many U.S. women died from complications of CAD, according to the AHA. Cardiovascular disease is also of concern for children. The obesity rate and cholesterol levels are rising in children, with an alarming one in three U.S. children classified as obese.

Unfortunately, CAD often has lethal effects. Although mortality rates for heart disease declined by about 27% between 1997 and 2007 due to increased awareness and education and improved therapies, heart disease remains a leading cause of death in America. In fact, the disease claims more than 2,000 lives each day in the United States. Recent reports reveal that almost 800,000 Americans will have a new coronary attack and more than 400,000 with have a recurrent attack each year. For too many in this group, the attack will be a
STEMI—recognized as the most lethal type of myocardial infarction. The AHA also reports that heart disease is the leading cause of death of American women, contrary to the popular belief that it is breast cancer.

The majority of the deaths associated with acute MI are the result of lethal arrhythmias. Some patients may respond to prompt defibrillation and advanced cardiac life support (ACLS). Studies have also shown that early treatment with thrombolytic therapy, cardiac catheterization, or PCI reduces mortality, decreases infarct size, and improves left ventricular function. For these reasons, the National Heart, Lung, and Blood Institute initiated the National Heart Attack Alert Program (NHAAP) which emphasizes the importance of early intervention to both healthcare providers and laypersons.

Nurses working in every specialty should be familiar with the signs and symptoms of an acute MI, its pathophysiology, and treatment methods to facilitate early intervention. Nursing care is not limited to nurses in the emergency department or the cardiac care unit.

Pathophysiology of MI

The nurse who is familiar with the pathophysiology of acute coronary syndrome (ACS) and MI will be able to better understand the signs and symptoms and treatment of the condition. Myocardial infarction, or heart attack, is defined as irreversible necrosis of myocardial tissue as a result of inadequate blood flow for a critical period of time. The heart is a muscular pump responsible for circulating oxygenated blood to all cells in the body and returning deoxygenated blood to the lungs (see Figure 1). To perform effectively, the myocardium is in constant need of an oxygen supply, which is provided by two main coronary arteries and their branches:

• Left main coronary artery (LMCA)—supplies blood to the chambers on the left side of the heart through its branches, the left anterior descending (LAD) artery and the circumflex (Cx) artery. The LAD provides blood to the front of the left side of the heart. The circumflex (or “circ”) circles the heart to supply blood to the side and posterior portions of the heart.

• Right coronary artery (RCA)—supplies blood to the right side of the heart, including the sinoatrial (SA) node and the atrioventricular (AV) node. These nodes, particularly the SA node, work to regulate the heart’s rate.

When the blood supply to the heart is compromised for more than 20 minutes, irreversible tissue damage and necrosis occur, that is, a myocardial infarction. Although any condition that disrupts blood flow to the heart can cause tissue damage, atherosclerosis is the most common cause. Atherosclerosis is the buildup of fatty deposits or plaque in the walls of the arteries. The plaque in the arteries can break through the endothelium and come in contact with blood flow. The rough surface of the plaque activates the body’s clotting mechanisms, and a thrombus forms. The thrombus, or clot, occludes the vessel and prevents blood flow to the portion of the myocardium supplied by that vessel. Lack of blood flow means lack of oxygen and tissue damage occurs.

Healthcare providers classify MI as a non-ST-segment elevation MI (NSTEMI or non-STEMI) or a STEMI.

• STEMI—complete occlusion of a coronary vessel characterized by elevation of the ST segment in certain leads on a 12-lead EKG (see section on 12-lead EKG interpretation for details about specific diagnostics) and positive cardiac enzymes.

• NSTEMI—incomplete occlusion of a coronary vessel and no ST-segment elevation on a 12-lead EKG; however, cardiac enzymes are positive.

The severity of MI depends on several factors: the amount of occlusion, the length of time blood supply is compromised, and whether the patient has collateral circulation to the area supplied by the occluded vessel. MI can also be classified by the direct damage to the heart:

• Transmural MI—tissue necrosis extends the full thickness of the cardiac wall, from the endocardium to the epicardium.

• Nontransmural MI—partial thickness tissue damage, either contained in the endocardium or extending only to the myocardium.

Signs and Symptoms of Acute MI

Unfortunately, the majority of patients who experience an acute MI do so outside the hospital, such as at home or in the community, and away from immediate medical care. Some research has shown that most patients do not seek medical care for 2 hours or more after the onset of symptoms, and a fairly large number of people wait 12 or more hours. Reasons for delay may include patients’ failure to recognize the seriousness of the problem; denial of symptoms or attributing symptoms to other illnesses, such as heartburn or anxiety; and limited access to a healthcare facility.

People can have difficulty recognizing the symptoms of MI because they can vary by individual. For example, men suffering from acute MI more commonly complain of chest pain, whereas women may complain of pain between their scapulae. The most common and cardinal symptom of an acute MI is chest pain, and some people, such as people with diabetic neuropathy and the elderly, may suffer no symptoms, a condition called silent MI.

Despite the variance in symptoms, MI presents with a few "classic" symptoms:

• Chest pain is described as squeezing or pressure in the area of the sternum (the patient may describe the pain as "an elephant sitting on my chest"). The pain lasts longer than 30 minutes, and rest, nitroglycerin, and a change in posture or position do not resolve the pain.

• The pain radiates to the neck, jaw, shoulder, arms, or back.

• In addition to pain, the patient experiences dyspnea, diaphoresis, nausea, or vomiting. Some patients may complain of discomfort that feels like "heartburn."

• Syncope or near syncope that cannot be attributed to any other condition.

• Feeling of impending doom.

It is important to note that some patients will experience all of these symptoms; others may experience only one or a few. Contrary to what many people believe, symptom onset occurs most frequently at rest and in the early hours of the morning.
Figure 1
*Coronary Artery Circulation*

Figure 2
*ECG Complex*
Showing when each part of the conductive system is depolarized.
Although prolonged chest pain, dyspnea, and diaphoresis are considered the classic symptoms of an acute MI, they can be associated with other disease processes as well. Also, not every patient with chest pain is experiencing an acute MI. Therefore, along with a thorough physical examination and chest x-ray, evidence-based practice dictates health care professionals to obtain a 12-lead EKG and cardiac enzymes as soon as the patient reports symptoms.

**12-Lead EKG Interpretation**

The 12-lead EKG is a tool used to record the electrical activity of the heart. The EKG is a reliable way to determine whether a patient is suffering an acute MI and whether it is a STEMI or non-STEMI. All patients suspected of having an acute MI should be given a 12-lead EK within 10 minutes of arrival to the hospital or, if the patient is already admitted, when symptoms of MI first present. Because of the evolving process of acute MI, it may be necessary to take several recordings as the patient experiences changes in symptoms.

EKGs provide much information about a patient's cardiac status, and interpreting them can be quite complicated. This course focuses on EKG changes related to acute MI, in particular changes to the ST segment. The ST segment begins after the QRS complex and ends at the beginning of the T wave (see Figure 2). The ST segment is typically isoelectric and remains at baseline or no more than 1 mm above or below baseline in normal sinus rhythm. The T wave represents ventricular repolarization that ends the cardiac cycle.

Lack of oxygen to the myocardial tissue can present in several ways on an ECG. Ischemia indicates hypoxic tissue and occurs as a result of partial occlusion of a cardiac artery, which reduces blood supply to the myocardium. In cases of ischemia, the EKG will show inversion of the T wave and ST-segment depression. Such signs on an ECG with patient complaints of pain or discomfort indicate a non-STEMI. Ischemia is usually reversible; however, during this time the patient is prone to arrhythmias and progression to injury or infarction (see Figure 3).

Infarction is necrosis or death of myocardial tissue caused by complete occlusion of a cardiac artery, causing transmural damage. The first sign of infarction is a tall, peaked T wave, although this change typically occurs before the patient presents to the hospital, so this sign is not often detected in the clinical setting. According to AHA guidelines, 2 mm elevation in leads V2 and V3 (2.5 mm in men < 40 years old and 1.5 mm in all women) or 1 mm in all other leads indicates a STEMI. The T wave may be inverted or not visible because of the size of the ST elevation. STEMI represents an acute process that requires immediate reperfusion (see Figure 4).

Infarction also can be noted in Q waves on the EKG. Normally, a Q wave is the first negative deflection on the QRS, and small Q waves may be normal in leads I, II, III, AVF, AVL, V5, and V6. It is the appearance of “new” Q waves on the EKG that represents damage to the myocardium. By definition, a significant or pathological Q wave is one third of the amplitude of the entire QRS complex or ≥ 0.04 second (1 mm) in width. Q waves are also considered relevant when they occur in two or more contiguous leads (see Figure 5). It is important to also realize that not all infarctions have Q waves present on the EKG. In about one third of infarctions, ST and T wave changes happen without associated Q waves. These are referred to as non-Q wave infarctions. These patients are at a greater risk for reinfarction, and many will develop Q waves up to 12 weeks following the MI.

Changes in the EKG are seen in the leads that look at the part of the myocardium where the infarction is occurring. It is helpful to become familiar with each of the 12 leads and the parts of the heart they represent since the location of the infarction can affect treatment decisions. Figure 6 depicts the type of MI, the affected artery, and area of infarction as shown on a 12-lead EKG.

Remember that acute MI is a dynamic event. Serial EKGs should be compared against previous EKGs to detect changes in morphology, and patient history and symptomology must be considered in conjunction with the EKG. Nurses must be sure to keep doctors informed of the patient’s condition and ensure doctors review each EKG promptly.
Guideline for interpreting a 12 lead EKG.

<table>
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<tr>
<th>Type</th>
<th>Artery Involved</th>
<th>EKG Patterns</th>
<th>Clinical Findings</th>
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<tr>
<td>Lateral Wall Infarction</td>
<td>Left Circumflex and Left Anterior Descending</td>
<td>Pathologic Q wave, ST segment elevation, T wave inversion in Leads I, AVL, V5, V6</td>
<td>If localized here, there are few complications. It is usually associated with an anterior MI. Monitor closely for posterior wall involvement.</td>
</tr>
<tr>
<td>Inferior Wall Infarction</td>
<td>Right Coronary Artery</td>
<td>Pathologic Q wave, ST segment elevation, T wave inversion in Leads II, III, and AVF</td>
<td>Bradycardia, nausea and vomiting</td>
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<tr>
<td>Anterior Wall Infarction</td>
<td>Left Anterior Descending</td>
<td>Pathologic Q wave, ST segment elevation, T wave inversion in Leads I, AVL, V1-V4</td>
<td>Symptoms associated with LV failure such as congestive heart failure, pulmonary edema, cardiogenic shock, and arrhythmias</td>
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<tr>
<td>Posterior Wall Infarction</td>
<td>Right Coronary Artery and Left Circumflex Artery</td>
<td>Tall upright R waves and ST segment depression in Leads V1-V3</td>
<td>Bradycardia, heart block, and conduction defects</td>
</tr>
<tr>
<td>Right Ventricular Infarction</td>
<td>Proximal Segment of the Right Coronary Artery</td>
<td>ST elevation in leads III, AVF and ST elevation in V4r-V6r</td>
<td>Symptoms associated with RV failure such as jugular venous distention, decreased cardiac output, increased systemic venous pressure.</td>
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**Cardiac Enzymes**

As soon as the patient exhibits signs and symptoms of an acute MI, draw the following labs: a complete blood count, a metabolic panel, and coagulation studies. In addition to these tests, it is vital that every patient experiencing symptoms of acute MI have lab work to determine if cardiac enzymes are present in the blood.

When damage occurs to the muscle, including the myocardium, the enzyme creatine kinase (CK) is released into the bloodstream. CK levels will begin to rise shortly after irreversible injury has occurred, which can indicate the extent of the infarction. Because CK is can indicate damage to any muscle, blood is tested for the presence of other cardiac markers. CK-MB (creatine kinase MB) provides a more definitive indication of myocardial cell damage or death.

CK-MB levels will rise during an MI or when other myocardial damage has occurred, such as damage associated with defibrillation with high joules, cardiac contusion, myopericarditis, or cardiac surgery. CK-MB is usually released 40–60 minutes after myocardial injury; however, blood levels may remain within normal limits for 8–12 hours. For this reason, the CK-MB levels may be drawn 3 to 4 times in a 24-hour period.

The usefulness of CK-MB as an early diagnostic marker is somewhat limited because of the amount of time necessary to run the test. However, CK-MB can be helpful in determining the extent of myocardial damage. Keep in mind CK-MB may not be a useful marker in certain postoperative patients because small amounts of this isoenzyme are also found in skeletal muscle.

Of all cardiac markers, cardiac troponin-T (cTnT) or cardiac troponin I is the gold standard in determining whether an acute MI is taking place. Troponin is a cardiac-specific protein that is released when the heart muscle is damaged. If troponin levels are increased in a patient with chest pain, then it is likely the patient has suffered an acute MI. It is important to note that troponin alone cannot be used to diagnose or rule out MI. Patients with chronic conditions such as heart failure may have "troponin leaks," in which a positive troponin is obtained; however, no MI is taking place. Therefore, the nurse must remember that labs are used in conjunction with other diagnostic tests, such as EKG and echocardiogram, to confirm a diagnosis of MI.

**Management of an Acute Myocardial Infarction**

Patients who are suspected of having an acute MI should be treated promptly. The AHA recommends that patients suspected of acute MI receive initial assessment and an EKG within 10 minutes of arrival to the emergency department. The goals in early treatment are reperfusion of myocardial tissue, reduction of infarct size, and improvement or preservation of left ventricular function.

**Nursing Assessment**

Nurses play a vital role in management of an acute MI. They must respond rapidly and efficiently to patients who are experiencing symptoms of acute MI. Nurses must quickly work to assess their patient; administer sublingual nitroglycerin and aspirin, if indicated; obtain a
Acute Coronary Syndromes

Symptoms suggestive of ischemia or infarction

EMS assessment and care and hospital preparation:
- Monitor, support ABCs. Be prepared to provide CPR and defibrillation
- Administer aspirin and consider oxygen, nitroglycerin, and morphine if needed
- Obtain 12-lead ECG; if ST elevation:
  - Notify receiving hospital with transmission or interpretation; note time of onset and first medical contact
  - Notified hospital should mobilize hospital resources to respond to STEMI
- If considering prehospital fibrinolysis, use fibrinolytic checklist

Concurrent ED assessment (<10 minutes)
- Check vital signs; evaluate oxygen saturation
- Establish IV access
- Perform brief, targeted history, physical exam
- Review/completed fibrinolytic checklist (Figure 2); check contraindications (Table 5)
- Obtain initial cardiac marker levels, initial electrolyte and coagulation studies
- Obtain portable chest x-ray (<30 minutes)

Immediate ED general treatment
- If O₂ sat <94%, start oxygen at 4 L/min, titrate
- Aspirin 160 to 325 mg (if not given by EMS)
- Nitroglycerin sublingual or spray
- Morphine IV if discomfort not relieved by nitroglycerin

ECG interpretation

ST elevation or new or presumably new LBBB; strongly suspicious for injury
ST-elevation MI (STEMI)

6 Start adjunctive therapies as indicated (see text)

Do not delay reperfusion

7 Time from onset of symptoms <12 hours?

>12 hours

≤12 hours

ST depression or dynamic T-wave inversion; strongly suspicious for ischemia
High-risk unstable angina/ non-ST-elevation MI (UA/NSTEMI)

9 Troponin elevated or high-risk patient (Tables 3, 4 for risk stratification). Consider early invasive strategy if:
- Refractory ischemic chest discomfort
- Recurrent/persistent ST deviation
- Ventricular tachycardia
- Hemodynamic instability
- Signs of heart failure

10 Start adjunctive treatments as indicated (see text)
- Nitroglycerin
- Heparin (UFH or LMWH)
- Consider: PO β-blockers
- Consider: Clopidogrel
- Consider: Glycoprotein IIb/IIIa inhibitor

11 Reperfusion goals:
- Therapy defined by patient and center criteria (Table 1)
- Door-to-balloon inflation (PCI) goal of 90 minutes
- Door-to-needle (fibrinolysis) goal of 30 minutes

12 Admit to monitored bed
- Assess risk status (Tables 3, 4)
- Continue ASA, heparin, and other therapies as indicated
- ACE inhibitor/ARB
- HMG CoA reductase inhibitor (statin therapy)
- Not at high risk: cardiology to risk stratify

13 Normal or nondiagnostic changes in ST segment or T wave
- Low-intermediate-risk ACS

14 Consider admission to ED chest pain unit or to appropriate bed and follow:
- Serial cardiac markers (including troponin)
- Repeat ECG/continuous ST-segment monitoring
- Consider noninvasive diagnostic test

15 Develops 1 or more:
- Clinical high-risk features
- Dynamic ECG changes consistent with ischemia
- Troponin elevated

16 Abnormal diagnostic noninvasive imaging or physiologic testing?

Yes

No

If no evidence of ischemia or infarction by testing, can discharge with follow-up

Figure 7 Evidence-based Practice for Acute MI
12-lead EKG; and notify the physician. AHA provides guidelines for treatment of acute MI that help to improve mortality rates (see Figure 7). These guidelines have become the standard of care for many hospitals nationwide.

When assessing a patient with suspected MI, the nurse’s priority is airway, breathing, and circulation as well as level of consciousness and cardiac arrhythmias. Since the incidence of sudden death is very high during the first hour of an MI, it is essential to monitor the patient closely and be prepared for an emergency.

Emergency departments often have devices that can detect cardiac enzymes in blood within seconds after labs are drawn. However, nurses in other areas of the hospital may not have access to this rapid-acting diagnostic equipment. Although nurses may draw stat labs, the results are not readily available. Immediately obtain a 12-lead EKG and assess the patient’s signs and symptoms. Show the EKG to the physician immediately for interpretation, and inform the doctor of your physical assessment. Completing these tasks while labs are pending will help prevent delay of diagnosis.

One of the most important assessments a nurse can conduct for a patient with suspected MI is a pain assessment. Chest pain can occur with pulmonary edema, congestive heart failure, pericarditis, pneumothorax, and unstable angina. Therefore, it is helpful to use a systematic method for assessing chest pain. Areas to consider include precipitating factors, quality, region, and radiation.

Precipitating factors—Ask the patient the following questions: What brought on the symptoms? What time did they begin? What were you doing at the time? What did you do to relieve the pain and did it help? The nurse should also document if the patient was doing any particular activity and if cessation of the activity helped the pain to decrease.

Quality—MI-associated chest pain is often very intense; therefore, it is important to obtain an objective description of the patient’s discomfort. Ask the patient to describe the pain to determine whether the pain feels like pressure, burning, squeezing, or aching. Ask the patient to rate the pain on a scale of 0–10, with 0 being no pain and 10 being the worst pain ever experienced.

Be sure to document the quality of the pain on your initial assessment to establish a baseline. This baseline will help you determine whether the patient experiences any changes in condition later, and it will help nurses in other units if the patient transfers to their care.

Region and radiation—Chest pain associated with MI often begins in the center of the chest and radiates to other areas. Common areas include the left arm, the neck, the jaw, and the back. Ask the patient to point to where it hurts. Also, keep in mind that the patient may only have pain in the arms, neck, or jaw. Some patients who complain of a toothache have been found to actually be suffering an acute MI.

Pharmacological Therapies

Many healthcare personnel are familiar with the acronym MONA (morphine, oxygen, nitroglycerin, aspirin) in the treatment of acute MI. In recent years, the application of MONA has changed slightly based on evidence-based practice from the AHA.

In addition to healthcare professionals, the general public has become aware of the benefits of aspirin during acute MI. Aspirin decreases platelet aggregation and reduces the likelihood of a partially dissolved clot forming again. Research indicates that giving an initial 160–325 mg dose, either chewed or dissolved in water, significantly reduces mortality. Aspirin is simple to administer and easily forgotten. This should be one of the first things given to the patient, if emergency personnel have not already given it or if the patient did not take it at home at the onset of chest pain.

Sublingual nitroglycerin should be given for pain relief and coronary artery dilation. The recommended dose is 0.4 mg sublingual q5 minutes three times, or until pain subsides. A translingual spray is available and is given as 1–2 sprays under the tongue q5 minutes three times, or until pain subsides. Nitroglycerin should be given with caution if the patient is hemodynamically unstable. One major side effect of nitroglycerin is hypotension because of arterial vasodilatation. The patient may also complain of a severe headache as a side effect of nitroglycerin. A continuous nitroglycerin infusion is usually initiated in the cardiac intensive care unit. Nitroglycerin infusion should be titrated to keep the patient free of chest pain while maintaining a systolic blood pressure >90.

Chest pain which persists after three doses of sublingual nitroglycerin and administration of oxygen should be treated with an analgesic. Morphine is the analgesic of choice because it also is a coronary artery vasodilator. Prompt pain relief may reduce the incidence of heart failure, arrhythmias, and other complications.

In the past, oxygen was often prescribed for patients presenting with chest pain, even in cases with normal oxygen saturation. Research has indicated that supplemental oxygen for patients with suspected MI can have harmful effects. Current AHA guidelines recommend 4L/min oxygen if a patient’s oxygen saturation is less than 95%.

The doctor may also order heparin or a low molecular weight heparin, such as enoxaparin sodium (Lovenox) to medically manage patients undergoing acute MI. Enoxaparin sodium is an antithrombotic that has been shown to reduce complications in both STEMI and non-STEMI patients. The dose is 1 mg/kg SC q12 hours. If your patient is scheduled for a heart catheterization, ask the physician if the dose should be held before the procedure to prevent complications of bleeding. Unfractionated heparin may be used until the thrombus is no longer occluding blood flow. Heparin can be administered IV or SC. A loading dose of heparin is 60U/kg IV bolus, with 12U/kg/hr titrated to reach a goal PTT of 30–70 seconds. When administering heparin, nurses need to monitor the patient for heparin-induced thrombocytopenia, a life-threatening complication of heparin administration. Nurses should monitor the patient’s platelet count and watch for signs of bleeding for the duration of heparin administration.

In addition to administering medications, nurses also need to obtain IV access in at least two sites, initiate cardiac monitoring, and keep the patient’s family updated on the patient’s condition as the medical team determines the most suitable treatment for the patient.
Interventional Therapies

Treatment of acute MI involves reperfusion, either mechanically or pharmacologically. PCI, a mechanical reperfusion intervention, has emerged as the preferred reperfusion treatment for acute MI. PCI is performed in the cardiac catheterization (cath) lab by the cardiologist and a cath lab team of nurses and technicians. In percutaneous coronary angioplasty (PTCA), the cardiologist inserts a diagnostic catheter into the femoral artery and guides the catheter up to the vessels of the heart. Once the catheter is in place, dye is injected and x-rays taken that show the dye being pumped through the cardiac vessels. Any occlusions will be seen as the dye fails to penetrate the blocked artery’s lumen.

If the doctor determines the blockage is treatable, a catheter with a balloon is guided to the affected vessel. The balloon is inflated, pushing the atherosclerotic plaque against the artery wall. The result is a smooth artery lining and patent vessel. The doctor may also opt to place a stent at the site of the lesion. A cardiac stent is a fine, wire-mesh tube placed in the artery to prevent vessel closure or restenosis.

After the heart cath, the patient will be on bedrest for a prescribed amount of time. Monitor the patient’s cardiac status for signs and symptoms of vessel reocclusion and complications of reperfusion (e.g., EKG changes, chest pain, change in vital signs, arrhythmias). Bleeding is a main complication of a heart cath. During bedrest, do not allow the patient to bend the affected leg. Closely monitor the insertion site for signs of bleeding. If the patient’s groin site starts to bleed, hold pressure for at least 15 minutes until bleeding stops. Redress the site, maintain bedrest, and closely monitor for additional bleeding. A sand bag can be applied to the site if it is oozing slightly. Minutes count during an acute MI. The AHA has set the door-to-balloon-inflation goal time at 90 minutes. Whether nurses work in a clinic, an emergency department, or on a med-surg floor, they work together with other members of the healthcare team to meet this goal time. Doing so can help save heart muscle and even save a patient’s life.

In some hospitals, such as at rural hospitals, a cath lab is not available; other times, mechanical reperfusion is delayed beyond the 90-minute goal. In these cases, the patient may be treated with fibrinolytics, such as tenecteplase (TNKASE), streptokinase, alteplase (Activase), or reteplase (Retavase). All of these drugs, which are administered intravenously, limit the progression of the MI by dissolving the thrombus in the coronary artery and restoring blood supply to the ischemic myocardium.

Each thrombolytic agent has a different dosing regimen. Streptokinase is infused as 1.5 million units IV over 60 minutes. Tenecteplase is given as a single bolus over 5 seconds. Reteplase is given as two 10-unit boluses, 30 minutes apart. Each reteplase dose should be given over 2 minutes. Alteplase is given as a bolus dose of 15 mg over 2 minutes, then 0.75 mg/kg over the next 30 minutes, and finally 0.50 mg/kg is given for the next 60 minutes for a total dose of 100 mg.

Since thrombolytic agents have a profound effect on the clotting system, side effects include bleeding problems. The most serious is intracranial hemorrhage or stroke. Others include gastrointestinal bleeding, genitourinary bleeding, gingival bleeding, and bleeding on the skin surface from cuts, scratches, and other IV sites.

Not every patient is a candidate for thrombolytic therapy. For example, the patient in the case scenario had kidney surgery 48 hours prior to experiencing an MI. Because of an increased risk for bleeding, thrombolytics were withheld and angioplasty was performed instead. Healthcare professionals should follow facility guidelines to determine whether a patient is a candidate for thrombolysis.

Heparin is another important drug in treating an acute MI. Whereas a thrombolytic agent will cause the clot to dissolve, heparin will keep the blood from clotting again and reoccluding the coronary artery. Heparin is most often given as a continuous IV infusion and the goal is to keep the partial thromboplastin time (PTT) 1.5–2 times normal.

Other pharmacologic agents that have been shown to be effective in reducing mortality in patients experiencing an MI include beta blockers, angiotensin-converting-enzyme (ACE) inhibitors, calcium channel blockers, and, for people who cannot tolerate ACE inhibitors, angiotensin II receptor blockers. The purpose of these drugs is vasodilation of coronary and peripheral vessels, decreased heart rate, reduced myocardial oxygen consumption, and overall decrease the extent of myocardial damage.

In some cases, such as if PCI is unsuccessful or if a patient has severe blockages in the large coronary vessels, coronary artery bypass (CABG; pronounced “cabbage”) surgery may be indicated. CABG surgery is the most common type of open-heart surgery in the United States and is performed by a cardiothoracic surgeon. During CABG surgery vessels taken from other areas of a patient’s body, typically the left internal mammary artery or the saphenous vein, and grafted onto the heart to reroute blood around occluded cardiac vessels. The outlook after CABG surgery is good for most patients, who can experience symptom relief for up to 15 years in some cases. Patients who undergo CABG will be transferred to the ICU postoperatively and will later recuperate on a telemetry unit.

Reperfusion and Resolution of the MI

The nurse plays an important role in determining if reperfusion of the coronary artery has occurred. The only way to verify reperfusion with certainty is the use cardiac catheterization to view the artery. This requires an invasive procedure and may pose some risk to the patient. Other noninvasive markers of reperfusion may be used.

A reduction of 50% or more in the ST segment indicates a return in blood flow to the injured myocardium. This is usually observed in the EKG lead with the greatest degree of ST elevation. Keep in mind that even if reperfusion occurs, the ST segment may not return to baseline due to myocardial damage.

Another clinical reperfusion marker is the resolution of chest pain. When blood flow is restored to the myocardium after thrombolysis, relief from chest pain is usually rapid and occurs with 30 minutes of the first noted improvement of the pain level. It is important to objectively assess the patient’s pain level using the pain scale before, during, and after administration of a thrombolytic agent.
Complications Associated With Myocardial Infarction

Amazing advances have been made in the treatment of acute MI over the past several years; however, it is still one of the leading causes of death. This is due to the serious complications usually associated with an infarction. These include coronary artery reocclusion, heart failure and cardiogenic shock, and arrhythmias. Nurses play an important role in assessing the patient for signs and symptoms of complications and assisting with early intervention.

Coronary Artery Reocclusion

A small number of patients will experience reocclusion of the artery after thrombolytic therapy even when preventative measures are taken. This happens because, although, the clot in the artery has been dissolved, the atherosclerotic plaque is still present and if anticoagulation is inadequate, another thrombus may form. About 50% of the reocclusions occur within the first 24 hours following thrombolytic therapy. Symptoms such as chest pain, nausea, diaphoresis, and ST segment elevation will usually be similar to those experienced with the original MI. With this in mind, it is crucial to monitor the patient closely and be aware of changes indicative of reocclusion. Since readministration of a thrombolytic agent is not recommended, the patient will need to have a PTCA or CABG if angioplasty is not an option or unsuccessful.

Heart Failure and Cardiogenic Shock

Congestive heart failure following a myocardial infarction can range from mild to severe, depending on the extent of ventricular damage. Heart failure occurs when myocardial tissue is damaged and the ventricle no longer works as an efficient pump. In right-sided failure, the compromised right ventricle causes fluid to back up in the peripheral circulation; in left-sided heart failure, fluid backs up in the pulmonary circulation. The nurse should monitor for signs of heart failure, including shortness of breath; hypoxia; production of pink, frothy sputum; hypotension; oliguria; confusion or changes in level of consciousness; and tachycardia.

Treatment of heart failure depends on the severity. Expect to administer supplemental oxygen, diuretics, a continuous nitroglycerin infusion, morphine, inotropic agents to improve cardiac contractility, and an angiotensin-converting enzyme (ACE) inhibitor. These patients will be quite ill and may require transfer to the critical care unit. Mechanical ventilation may also be required.

Patients with heart failure can rapidly decline into cardiogenic shock. Cardiogenic shock occurs when 40% or more of the myocardium has been affected by the infarction. Because the heart is incapable of contracting with sufficient force to pump enough blood, the vital organs and peripheral tissues cease to function as a result of ischemia. The patient may experience the following symptoms: pulmonary congestion, diaphoresis, cool extremities, and mental confusion. Treatment for cardiogenic shock is aggressive and can include fluid replacement, inotropic drugs, and an intra-aortic balloon pump (an invasive device used to decrease ventricular workload and improve coronary artery perfusion).

Unfortunately, death occurs in about 85% of patients who develop cardiogenic shock. Therefore, a very important nursing intervention is helping the patient and family work through end of life issues.

Arrhythmias

Successful thrombolysis can cause a variety of cardiac arrhythmias, such as ventricular tachycardia, premature ventricular contractions, accelerated idioventricular rhythm, and sinus bradycardia. These are generally accepted as normal consequences of coronary reperfusion, and treatment is not necessary unless the patient becomes unstable.

Ventricular fibrillation. The majority of sudden cardiac deaths are because of ventricular fibrillation, or v fib. This arrhythmia results in an ineffective quivering of the ventricles and no cardiac output. Treatment includes basic life support (airway, breathing, and circulation), defibrillation, and advanced cardiac life support. The sooner the ventricular fibrillation is treated, the greater the chance of survival for the patient.

Nurses in all areas of health care should be able to recognize the signs of cardiac arrest and intervene appropriately.

Ventricular tachycardia. Patients who have suffered an acute MI may experience ventricular tachycardia, or v tach. This ventricular arrhythmia can be benign or it can be life threatening. Patients may be asymptomatic or they may experience shortness of breath, chest discomfort, palpitations, and syncope. Patients may be given an antiarrhythmic, such as lidocaine, procainamide, or amiodarone, to reestablish sinus rhythm. If the patient is unstable, electrical cardioversion may be conducted in an attempt to convert the myocardium to a sinus rhythm. This arrhythmia is most common in patients who have experienced an anterior or anterolateral MI.

Sinus bradycardia and heart block. Bradycardia is a slowing of the heart rhythm. The patient may experience hypotension and syncope which usually responds to oxygen and atropine. Heart blocks occur as a result of problems in the atrioventricular (AV) node of the conduction system. Electrical impulses are not conducted from the atrium to the ventricles, which can cause a decrease in cardiac output. To correct the arrhythmia, patients may need transcutaneous (external) pacing or surgery to insert a transvenous (internal, temporary) pacemaker.

Emotional and Psychosocial Issues

When an individual experiences an acute MI, healthcare professionals focus most of their attention on meeting the eminent physical needs of the patient. However, an acute MI usually occurs suddenly and without warning and can be an extremely stressful experience for the patient and family. Studies have shown that patients often ignore symptoms when they first begin or contribute them to heart burn or muscle pain. They may also delay seeking medical care because they are in denial, refusing to believe they could be having a heart attack. When symptoms become more serious, patients often appear apprehensive and fearful. They may have a feeling of impending doom and ask questions such as “Am I going to die?”

The emotional stress can have a profound effect on physiological functions as well. During times of anxiety and
knowledge about this life-threatening event. The nurse plays a large part in keeping the patient and family informed through each stage of the process. Using a calm, collected approach provides the patient a feeling of security and reassurance. Although it is not appropriate to start discussing lifestyle changes in the initial treatment period, it is very helpful to explain in simple, concise terms what can be expected in each phase of the process. Remember that in times of increased stress, individuals do not retain information as well. It may be necessary to repeat the same information or answer the same questions. Try to anticipate care and provide information prior to procedures. For example, explain serial labs to the patient and provide information about upcoming tests before the patient undergoes them. Knowing what is going to happen next helps relieve patients’ anxiety.

Caring for the Family

Sudden onset of an illness such as an acute MI usually finds the healthcare providers working quickly to meet the patient’s needs, while the family and significant others wait anxiously nearby. Many patients who experience an MI are between the ages of 35 and 60. This age group may have children living at home and may be employed full time. Family stressors include not only anxiety about the patient’s condition but also role changes that need to be made.

Family members may experience emotional and physical distress, feelings of helplessness, disorganization, and difficulty in mobilizing resources. While the patient is receiving an analgesic and experiencing some relief, the family may continue to be very anxious and upset. Since all families respond and cope with stress in different ways, the role of the nurse is to assess each unique family situation and intervene as appropriate. Some families may do well if they can be at the bedside constantly with the patient. Others may create more anxiety for the patient; nurses need to encourage these family members to deal with their apprehension away from the bedside to avoid upsetting the patient. No matter what kind of family dynamics are present, it is still essential to keep the family informed using simple, concise, and honest communication.

Information Sharing

It would be helpful if patients came to the hospital with an instruction manual describing what to expect when they are having a heart attack; however, most individuals and families have no knowledge about this life-threatening

Patient and Family Education

The patient and family will need education about the MI. The education should begin as soon as the patient comes in contact with the healthcare system. Initial information should be simple and concise and focus on what to expect. It may also be necessary to educate the patient and family about thrombolytic agents and PCI so that informed decisions. Nurses need to remember to use layman’s terms when describing medications and procedures. For example, the nurse may want to describe the thrombolytic agent as a “clot-buster” or medicine used to dissolve clots in the arteries of the heart. The nurse should also explain that these drugs “thin the blood” and may cause bleeding complications. Most important, the nurse should offer emotional support and attempt to relieve anxiety.

It is appropriate in most cases to begin more detailed education once the patient’s condition has stabilized and some of the initial feelings of fear and anxiety have subsided. This is a good time to begin explaining exactly what happened. It may be helpful to use pictures and diagrams of the heart and provide educational information that the patient can take home and review.

The nurse should take into account the patient’s learning style, learning challenges, and home language. Give information in a way that will be most beneficial to the patient. Provide written materials in the patient’s native language and use a translation service or professional translator if needed. To address individual learning needs, the nurse can offer the patient choices such as “Would you like to read a booklet about what to expect after your heart attack or would you prefer to watch a 15 minute video?” Always remain open to queries and don’t give the patient and family a sense that they are asking “dumb questions.”

The next step in the patient education process usually occurs once the patient transfers from an intensive care unit to a monitored floor or intermediate care area. Patients and families may be less anxious at this point and may begin making plans for discharge. Patients may also begin to ask questions about lifestyle changes. Keep in mind that each individual may have different ideas about what caused the MI. One patient
may attribute it to smoking; another patient may think it was brought on by workplace stress. The nurse should discuss the patient’s perceptions and talk about making lifestyle changes specific to these perceptions, keeping in mind that patients will usually have more motivation to change the things that are most meaningful to them.

Discharge teaching is an essential part of nursing care. The nurse should give the patient verbal and written instructions about medications, smoking cessation, exercise and daily activities, ability to return to work, and dietary changes. Most patients will also be discharged with medication prescriptions, including anticoagulants, beta blockers, ACE inhibitors, and an antilipemic. It is helpful to provide verbal and written instructions about the medications. It is also valuable to assess if the patient has the necessary resources to acquire medications. If not, a social worker may be able to assist the patient. The patient and family also should be given specific instructions regarding what to do if chest pain reoccurs.

Most health care facilities offer an outpatient cardiac rehabilitation program. The nurse should be familiar with available resources and promote development of a healthy lifestyle. A hospital case manager can assist patients in establishing a rehab plan.

Looking to the Future

New treatments and protocols have improved the outlook for patients suffering from acute MI. Fibrinolytics, PCI, advanced cardiac life support, and time goals for treatment have decreased mortality for patients undergoing a heart attack. Nurses are a vital part of the team that produces such remarkable results. Clinicians continue to work on improving currently used treatments and research new treatments, such as cell therapy, which can replace damaged myocardial cells. As medical professionals learn more about the causes of acute MI, information can be relayed to patients, reducing their risk and helping them to recognize symptoms and receive prompt treatment.

Prevention is also a target for government agencies, healthcare organizations, and healthcare professionals. Multigeneration initiatives can help patients develop healthy lifestyles, receive regular checkups, and seek treatment when symptoms start. One such initiative is the One in a Million Hearts Initiative, a joint effort of the U.S. Health and Human Services Department, the Centers for Disease Control and nonprofit groups, such as the AHA. The goal of this program is to prevent 1 million heart attacks and strokes in 5 years. As part of the group's effort, they promote the ABCS—appropriate aspirin therapy, blood pressure control, cholesterol management, and smoking cessation. For more information about the One in a Million Hearts initiative, visit http://millionhearts.hhs.gov/index.html.

Nurses are a large part of the treatment and prevention process. Nurses must keep many issues in mind for a patient experiencing an acute MI. Genentech, a medical company, coined the phrase “time is muscle.” Whether the patient is on a med-surg unit, presenting to the emergency department, or in a physician’s office, it is essential for nurses to recognize signs and symptoms of an acute MI and begin intervention as quickly as possible. The sooner the myocardium is reoxygenated, the greater the chance of a positive outcome for the patient. It is vital for the multidisciplinary team to work together not only to treat patients promptly but also to promote a heart-healthy lifestyle.

References


