Standard Medicine

Competency Integrates assessment findings with principles of epidemiology and pathophysiology to formulate a field impression and implement a comprehensive treatment/disposition plan for a patient with a medical complaint.

TOPIC 26

CARDIOVASCULAR EMERGENCIES: ACUTE CORONARY SYNDROME

INTRODUCTION

Acute coronary syndrome (ACS) is a hot-button issue of modern prehospital medicine. This topic is at the forefront of discussion not only because of its sweeping mortality—cardiovascular disease is the leading cause of death in the United States—but also because it is a condition in which the swift actions of EMS providers really do have an impact on patient outcomes.

ACS is an umbrella term used to cover any cluster of clinical signs and symptoms related to diminished blood flow to the heart. In general, ACS is a supply-and-demand issue. The heart requires a constant supply of blood through the coronary arteries to deliver nutrients and oxygen to its muscle mass. When this demand cannot be met, the myocardial cells suffer damage. ACS is commonly associated with atherosclerosis and its buildup of plaque inside arterial walls, but it can also be related to other pathologies that decrease the flow of blood, such as vasospasm and aneurysm.

The spectrum of ACS can range widely, from simple chest pain with exertion (angina) to full-blown myocardial infarction (MI) with resultant muscle death and the precipitation of pulmonary edema from left heart failure. Although these disorders are grouped together, the paramedic will use specific diagnostic tools, such as 12-lead ECG, to rapidly identify and aggressively treat specific underlying disorders.

Prehospital providers can have a significant impact on patients suffering from ACS. As part of the larger health care team, paramedics represent an important link in the early identification and initial treatment of ACS. Prehospital actions are essential to maximizing patient outcome; the responsibilities of prehospital providers are likely to increase as treatment of ACS becomes more sophisticated.

This topic discusses the pathophysiology, assessment, and treatment of ACS. (Note that Topic 27 will discuss the use of diagnostic 12-lead ECG in greater detail.)

TRANSITION *highlights*

- *Inclusion of the term acute coronary syndrome as an umbrella term for ischemic cardiac events.*
- *Pathophysiology of acute coronary syndrome.*
- *Increased emphasis on recognition of myocardial infarction and on emergency department door-to-intervention time.*
- *Changes in philosophy of oxygen administration to patients with acute coronary syndrome.*
- *Inclusion of aspirin and nitroglycerin administration for the acute care of acute coronary syndrome.*
- *Assessment findings indicative of cardiac arrest, as well as findings suggestive of a patient who may go into cardiac arrest.*
- *Importance of prehospital interventions and why they are successful in reversing cardiac arrest.*

EPIDEMIOLOGY

It has been estimated that more than 62 million Americans have some form of cardiovascular disease that commonly leads to coronary artery disease (CAD). Through previous research, the American Heart Association has found that approximately 7 to 8 million people each year will seek treatment in an emergency department in the United States for chest discomfort. Of those patients, approximately 2 million will actually suffer from a cardiac-related condition that involves the coronary arteries. About 1.5 million will suffer an actual heart attack, in which the coronary artery is occluded and a portion of the heart muscle begins to die. Of those patients, 500,000 will die from this heart attack, 250,000 of whom will die within one hour following the onset of the signs and symptoms.

In a sobering light, about every 25 seconds an American will suffer a coronary event, every 34 seconds a heart attack will occur, and about once every minute someone will die from sudden cardiac arrest. These statistics are definite indications of the

significance of cardiac-related emergencies and underscore the importance of having a thorough knowledge base regarding cardiac emergencies.

PATHOPHYSIOLOGY

As stated earlier, the pathophysiology of ACS is related to a supply-and-demand problem. ACS occurs when the necessary supply of blood cannot perfuse heart tissue. This problem is most commonly associated with vascular disease. Therefore, to better understand the pathophysiology of ACS, one must first understand the basics of vascular disease.

Arteriosclerosis is a condition that causes the smallest of arterial structures to become stiff and inelastic. This is often referred to as "hardening of the arteries." A form of arteriosclerosis is *atherosclerosis*. Atherosclerosis is a systemic arterial disease that is derived from the Greek word *athere*, meaning "gruel" or "porridge," and *scleros*, which means "hard."

Atherosclerosis is an inflammatory disease that starts in the intimal (innermost) lining of the blood vessels, where endothelial cells become damaged. Common risk factors that are thought to cause this endothelial injury include smoking, diabetes, hypertension, high levels of low-density lipoproteins (LDL), and low levels of high-density lipoproteins (HDL). Once injury occurs, intimal dysfunction and inflammation progress through the following five basic pathophysiologic events (▶ Figure 26-1):

- Intimal damage allows the migration of blood platelets and other substances in the blood (serum lipoproteins) into the vascular wall. This irritates and inflames the vascular wall.
- As a result of the irritation and inflammation, different types of cells migrate to the location, as do smooth muscle cells of the tunica media layer (muscular middle layer of the blood vessel).
- As these cells proliferate, longitudinal fatty streaks develop in the lumen of the blood vessel. The blood vessel weakens as intima and media are deprived of nutrients from the expanding plaque.
- In an attempt to "close off" the fatty streaks, smooth muscle cells produce collagen and migrate over the fatty streak to form a fibrous cap.
- Fibrous caps, however, are not stable and may rupture. This causes the body's clotting mechanism to activate with development of a thrombus (clot), which may occlude the blood vessel.

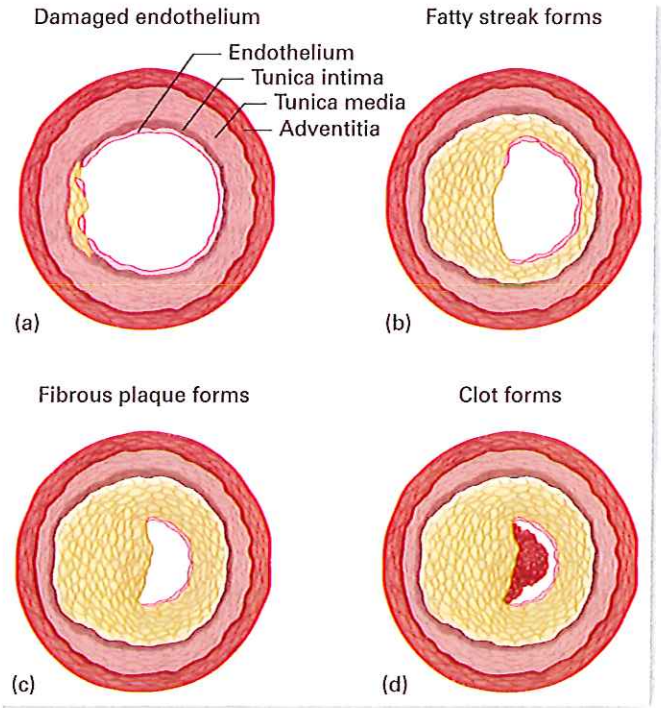

Figure 26-1 The process of artery occlusion (atherosclerosis): (a) The endothelium (inner wall) of the artery is damaged. (b) Fatty streaks begin to form in the damaged vessel walls. (c) Fibrous plaques form, causing further vessel damage and progressive resistance to blood flow. (d) The plaque deposits begin to ulcerate or rupture; platelets aggregate and adhere to the surface of the ruptured plaque, forming clots that may block the artery.

The buildup of fatty deposits on the inside of the coronary arteries (atherosclerosis) is called *coronary artery disease*. The narrowing of a coronary blood vessel (also known as *remodeling*) increases the resistance to blood flow through the artery and decreases the amount of blood flow to the distal heart muscle. In addition, the fatty deposits will reduce the coronary arteries' ability to dilate (become larger) and deliver additional blood flow to the heart when needed, such as during an increase in heart rate or more forceful pumping action, as needed during stress or exercise.

Although CAD is a vascular problem in and of itself, it sets up the body for an increase risk of additional emergencies, which is the real focus of this topic: acute coronary syndrome.

Acute Coronary Syndrome

As discussed previously, ACS results from any of a variety of conditions in which the coronary arteries are narrowed or occluded by fat deposits (plaque), clots, or spasm. The word *acute* refers to a sudden onset, *coronary* refers to a condition affecting the coronary arteries, and *syndrome* indicates a group of signs and symptoms produced by the condition.

Two conditions that are part of any ACS are *angina* (stable and unstable) and *myocardial infarction* (heart attack). When the myocardial cells that make up the heart muscle do not receive an adequate amount of oxygenated blood, they become hypoxic. This condition, most commonly referred to *myocardial ischemia*, represents a state in which inadequate oxygen is delivered to the heart muscle. Ischemia can be caused by narrowing of the coronary arteries by plaque or spasms, clot formation inside the coronary artery blocking the blood flow, an increase in the work of the heart that demands more blood flow than can be supplied through the coronary arteries, or any combination of these conditions.

Angina Pectoris

Angina pectoris (literally, "pain in the chest") is a condition and a symptom commonly associated with CAD and can manifest itself as one of the ACSs (▶ Figure 26-2). Angina typically occurs when an increased workload is placed on the heart from an increase in the heart rate or the contractile function of the heart or when an increase in systemic vascular resistance causes the ventricles to work harder to keep blood moving.

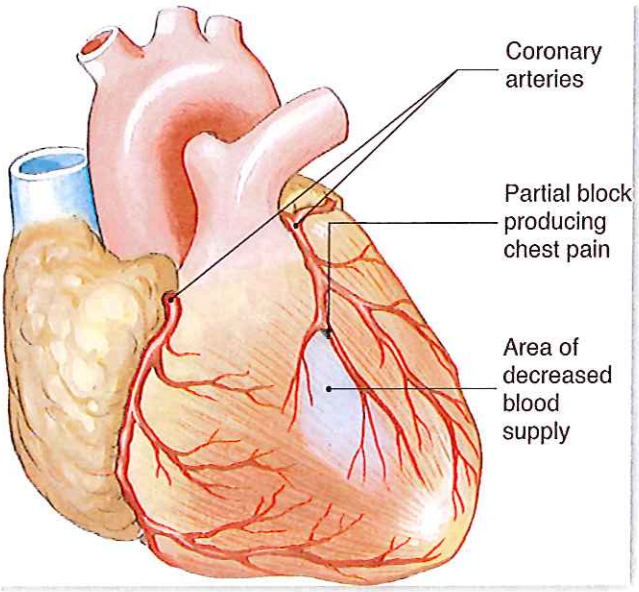

Figure 26-2 Angina pectoris, or chest pain, results when a coronary artery is blocked, depriving an area of the myocardium of oxygen.

In any instance, angina pectoris is a symptom of inadequate oxygen supply to the heart muscle, which is often caused by partial blockage of the coronary arteries that, in turn, produces cellular ischemia (reduced delivery of oxygenated blood) and tissue hypoxia (oxygen deficiency in the tissues).

Generally, angina pectoris occurs during periods of stress, either physical or emotional, at a time when the oxygen requirements of heart muscle are not met by oxygen delivery. Once the stress is relieved or the patient rests, oxygen requirements are reduced, and the pain will usually go away. Although more uncommon, angina may also occur in patients with valvular heart disease, enlarged hearts (cardiac hypertrophy), and hypertension. The pain is commonly felt under the sternum and may radiate to the jaw, or down either arm, to the back, or to the epigastrium. The pain usually lasts for about 2 to 15 minutes.

Many patients will be able to tell you that they have had angina as part of their past medical history and will have nitroglycerin prescribed for this condition. Prompt relief of the symptoms after rest and administered nitroglycerin is a key finding typical of angina.

Angina pectoris has two subcategories: *stable angina* and *unstable angina*. Stable angina patients will experience episodes of chest pain of a more or less predictable nature. Commonly, the patient will know that certain levels of physical exertion (e.g., running versus walking) or extreme emotional or mental distress will precipitate the pain. Just as stable angina has a predictable onset, its resolution with rest and/or nitroglycerin is also predictable.

Unstable angina, conversely, is a subcategory of angina pectoris in which the onset of pain cannot be predicted. Unstable angina commonly occurs unexpectedly, in patterns that are not reliable. Although the underlying pathophysiology for unstable angina is still a diminishing of blood flow to distal capillary beds of the myocardium, it may occur during sleep, in the absence of physical exertion, or concurrently with other medical conditions, such as infections or inflammations of the body. In addition, a change in the usual pattern of anginal episodes suggests unstable angina.

Variant angina (Prinzmetal angina) is a type of unstable angina in which a coronary artery spasm is the cause for diminished blood flow, but like unstable angina in general, the onset cannot be predicted. Variant angina is highly correlated with secondary lethal arrhythmias, MI, and sudden cardiac death.

Myocardial Infarction

In CAD, plaque builds up between the intimal and medial layers of the coronary vessel. Although the vessel may remodel and partially obstruct the flow of blood, the plaque is shielded from passing blood flow by the thin lining of the vessel and the fibrous cap formed over the plaque. This shielding is very important and represents a key risk factor associated with CAD, because when this shielding ruptures and plaque is exposed to flowing blood, the clotting cascade begins and a clot is formed.

The vessels of some CAD patients are well protected by a thick fibrous cap, but in other cases, this cap is thin and quite vulnerable to rupture. Why some caps rupture while others remain intact is thought to be related to dilation and constriction of the blood vessel, but is still an important question in the treatment of vascular disease. Most important, though, the rupture of a fibrous cap initiates a chain of events that can rapidly lead to the death of the patient if not recognized immediately and treated aggressively.

Topic 7, "Anatomy and Physiology: The Blood," discussed the manner in which hemostasis protects the blood vessel when a leak occurs. Specifically, in a process called the clotting cascade, platelets plug the hole in the blood vessels and coagulation forms a lasting clot. The events of an MI result from the same physiology.

When a fibrous cap ruptures, flowing blood is exposed to the plaque built up between the layers of an artery. Although the wall of the blood vessel may remain intact, the flowing blood interprets this plaque to represent a loss of blood vessel integrity and takes steps to repair the hole. Plaque, in essence, signals the start of a clotting cascade.

Just as in the creation of any clot, the first step in the creation of a coronary thrombus is platelet aggregation. Exposure of plaque and exposed collagen signals circulating platelets and other clotting factors to attach themselves and congregate in the exposed area. As platelets stick together, they "activate" and secrete their own chemical markers. Chemicals such as thromboxane enhance the attraction among platelets and further assist in gathering other circulating platelets to plug the theoretical hole.

As the clotting cascade continues, activated platelets chemically signal other distinct changes and coagulation begins. Thromboxane A_2 activates additional platelets, and glycoprotein IIb/IIIa receptors are activated to create and activate fibrin. The creation of fibrin serves to further stabilize the clot by linking platelets with a meshlike fibrin net. As the coagulation process continues, the clot grows in size.

It is important to remember that in this case, there is no actual hole in the vessel. The aggregation of platelets and the creation of a clot obstruct blood flow through the already narrowed vessel. Remember also that vasoconstriction is an important element of hemostasis and, therefore, the vessel may narrow further simply as part of the hemostatic process. In trying to defend itself from perceived blood loss,

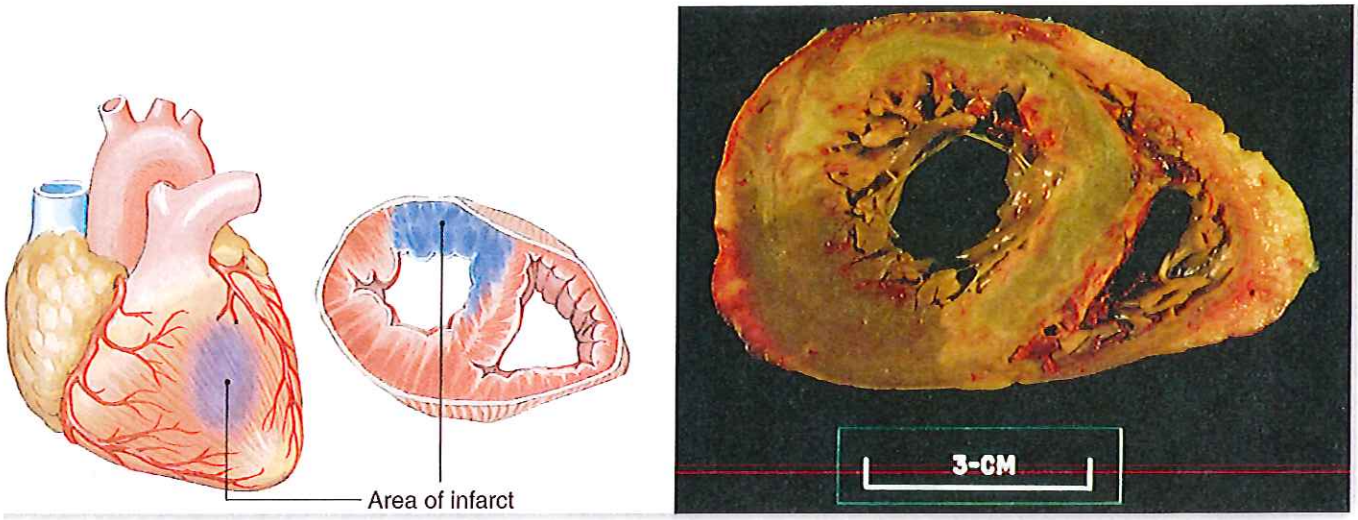

Figure 26-3 (a) Cross section of a myocardial infarction. (b) A heart with normal and infarcted tissue.

the body is actually obstructing its own blood supply. If the clot grows large enough, blood flow through this diseased artery will actually stop. When this blood flow is obstructed, myocardial cells downstream from the occlusion will become ischemic and eventually die unless the clot is resolved (▶ Figure 26-3).

Tissue will die in stages. Infarcted tissue consists of a necrotic core surrounded by marginal zones that can either recover normal function or become irreversibly damaged. One of the greatest determinants of MI size is the presence of any collateral blood flow. Collateral blood flow refers to an area of the heart that is also receiving blood from a blood vessel that is not occluded. This collateral blood flow is an important determinant of infarct size and of whether the marginal zones become irreversibly damaged.

Infarcted tissue does not contribute to tension generation during systole; therefore, it can alter ventricular systolic and diastolic functions, which in turn changes stroke volume and cardiac output, often detrimentally. Infarcted tissue can also interrupt normal electrical activity within the heart, leading to potentially fatal dysrhythmias.

If the MI affects the left ventricle, which is most common, the drop in cardiac function can lead to pulmonary vascular congestion and pulmonary edema. Infarcted tissue can also precipitate abnormal cardiac rhythms and conduction blocks that further impair global cardiac functioning and may become life threatening in some cases.

Reduced cardiac output and arterial pressure can activate baroreceptor reflexes that attempt to compensate for the drop in pressure by activation of numerous neurohumoral compensatory mechanisms, such as the sympathetic nervous system, as well as fluid retention systems, such as the renin–angiotensin and aldosterone systems. Renal hypoperfusion and sympathetic nervous system stimulation promote renin release—renin is converted into angiotensin II—and aldosterone release, which enhance renal retention of sodium and water and promote vasoconstriction.

These negative feedback mechanisms cause the heart to work harder, thus increasing cardiac output. Retention of fluid by the kidneys drives up pressure by placing more fluid into the vascular system (explaining the elevated blood pressure and tachycardia seen in many MI patients). The pain and/or anxiety associated with an MI further stimulates the sympathetic nervous system and results in ongoing peripheral systemic vasoconstriction and cardiac stimulation as catecholamines (such as adrenaline, norepinephrine, and dopamine) are released from the adrenal medulla.

Although the increased sympathetic tone and fluid retention help to maintain arterial pressure, they also lead to a large increase in myocardial workload and necessitate a greater myocardial oxygen need in an environment of diminished supply. This supply–demand mismatch can lead to more myocardial hypoxia, enlargement of the infarcted region, precipitation of cardiac arrhythmias, and potentially a worsening in cardiac output. In addition, the sympathetic nervous system activation is responsible for the diaphoresis, anxiety, and nausea/vomiting commonly experienced by the patient.

After several weeks, if the patient survives the heart attack, the body remodels the infarcted tissue with a noncontractile fibrotic scar that does not contribute to ventricular strength. Long-term adaptation to the muscle loss includes development of compensatory hypertrophy or dilation, congestive heart failure, chronic dysrhythmias, and increased risk of sudden death. Table 26-1 provides an overview of the important body system

TABLE 26-1 Effects of Myocardial Infarction on Body Systems

Neurohumoral effects	• Enhanced sympathetic tone • Increased circulating catecholamines • Higher levels of angiotensin II and aldosterone • Increased arginine vasopressin
Cardiopulmonary effects	• Tachycardia • Dysrhythmias • Diminished stroke volume • Increased oxygen requirements • Pulmonary vascular congestion
Peripheral vascular effects	• Increased vascular resistance (vasoconstriction) • Elevations in blood volume • Possible systemic edema

changes secondary to an acute myocardial infarction (AMI).

ASSESSMENT FINDINGS

At any level of cardiac care, an MI is diagnosed in essentially three ways: symptoms and patient history, electrocardiogram, and cardiac enzymes. This is very important to EMS providers, for it demonstrates that even without major technological advancements, diagnosis of ACS-related syndromes is a *prehospital* capability. Although sophisticated testing will be used later to discern a more precise diagnosis, identification of at least certain types of MI is within the skill set of paramedics.

Symptoms and History

There is no such thing as a "typical" acute myocardial infarction. In fact, research has shown that there is no single symptom or historical finding that can fully rule out an AMI. As such, paramedics should "cast a wide net" and err on the side of being inclusive rather than exclusive. Some findings, though, certainly make AMI more likely.

The classic feature of an AMI is chest pain or discomfort. This discomfort is typically present in the anterior chest but can radiate to the arm (classically, the left arm), the jaw, the shoulder, and the abdomen. The pain of AMI is commonly dull in nature and is often described as squeezing or pressure.

It is important to remember that we are describing common findings—but uncommon findings do not rule out an AMI. In fact, many patients who experience an AMI do not report any chest pain. Often the patient complains of dyspnea. Shortness of breath has been found to be a very common finding after the age of 65. Nausea/vomiting is also a common symptom. Other common findings include syncope, anxiety, and diaphoresis. Interestingly, it is estimated that 10 percent of AMI patients will present with cardiac arrest as their first symptom.

The presence of risk factors for CAD also increases the likelihood of the symptoms being related to ischemia or infarction. Such risk factors include a past history of coronary or vascular disease, family history of coronary or vascular disease, smoking, hypertension, hypercholesterolemia, and diabetes mellitus.

Patients with classic angina will have a history of substernal chest pain or discomfort. The pain is usually described as a pressure or heaviness on the center of the chest; it usually occurs with activity and is relieved by rest. It may radiate to the left arm or jaw. The pain is frequently associated with dyspnea, nausea and vomiting, and diaphoresis. Depending on the significance of the myocardial ischemia, the patient may also display tachycardia and changes in blood pressure, inspiratory crackles (rales), or "cardiac asthma." ▶ Figure 26-4 lists common findings in patients with ischemic/infarction episodes.

Of special concern is the presentation of myocardial ischemia or infarction in women. Almost all common descriptions of the way ACS presents clinically have been taken from male cases. This is in part because medical literature at one time described only male patients with ACS, as they were primarily the ones who suffered cardiac events. However, as women have gained a more prominent presence in the workforce and have higher rates of smoking, poor nutrition habits, higher stress levels, and so

The most classic features of MI include chest discomfort that is dull in nature and typically radiates into the left arm or jaw.

DISTINGUISHING ANGINA PECTORIS FROM MYOCARDIAL INFARCTION

	Angina Pectoris	Myocardial Infarction
Location of Discomfort	Substernal or across chest	Same
Radiation of Discomfort	Neck, jaw, arms, back, shoulders	Same
Nature of Discomfort	Dull or heavy discomfort with a pressure or squeezing sensation	Same, but maybe more intense
Duration	Usually 2 to 15 minutes, subsides after activity stops	Lasts longer than 10 minutes
Other symptoms	Usually none	Perspiration, pale gray color, nausea, weakness, dizziness, lightheadedness
Precipitating Factors	Extremes in weather, exertion, stress, meals	Often none
Factors Giving Relief	Stopping physical activity, reducing stress, nitroglycerin	Nitroglycerin may give incomplete or no relief

Figure 26-4 Both myocardial infarction and less serious angina can present symptoms of severe chest pain. Treat all cases of chest pain as cardiac emergencies.

forth, they are now suffering from heart attacks at rates approaching those among men. In fact, because the incidence of AMI in women is greater at older ages than in men, women are almost twice as likely to die from AMI or its complications within the first few weeks following the event.

A woman may present with different signs and symptoms from a man when she is experiencing a cardiac event; however, the event is just as dangerous and can be as deadly. Therefore, the paramedic must be sure to recognize some of the more subtle signs and symptoms that women suffering from ACS experience.

The list of symptoms in Table 26-2 is common for women suffering from cardiac ischemia or infarction. Although you will see that some descriptions are the same as for men, many others are not.

Because the death rate for women is higher than that for men when heart attack occurs, the paramedic should have a high index of suspicion of ischemia/infarction when gathering a history from the female patient. Err on the side of the patient and provide emergency care for a potential MI, despite a presentation of "atypical" signs of ischemia or infarction. Finally, note that diabetics and the elderly (Table 26-3) are also high-risk groups who may present with atypical findings.

TABLE 26-2 Symptoms in Women with Cardiac Ischemia or Infarction

- "Classical" findings (not necessarily *common* findings)
  - Dull substernal chest pain or discomfort
  - Dyspnea or respiratory distress
  - Nausea, vomiting
  - Diaphoresis
- "Nonclassical" or "atypical" findings (not necessarily *uncommon* findings)
  - Neck ache
  - Pressure in the chest
  - Pains in the back, breast, or upper abdomen
  - Tingling of the fingers
  - Unexplained fatigue or weight gain (water weight gain)
  - Insomnia

TABLE 26-3 Special Considerations in Geriatric Cardiac Events

History of diabetes mellitus	A geriatric patient with diabetes has long-term damage to the nerve endings in the body. This causes the typical pain from an MI to be perceived poorly, if at all, by the diabetic patient. Therefore, the diabetic patient experiencing an MI may complain only of respiratory distress or dizziness when standing, or even excessive weakness and dyspnea on exertion. It is important for EMS providers to identify the patient with diabetes as potentially having an acute coronary event and to treat him appropriately.
History of trauma	If the geriatric patient is a trauma patient, there must be a high index of suspicion for cardiac involvement as well. Geriatric patients who are traumatized can slip quickly into cardiac arrest and do not respond well to typical interventions. Geriatric patients with head trauma, chest trauma, abdominal trauma, or extremity trauma with severe bleeding are especially susceptible to cardiac arrest.
History of asthma	If a patient with a history of asthma goes into cardiac arrest, the cause may be acute bronchoconstriction that led to hypoxemia, acidosis, and cardiac arrest. Until the bronchoconstriction is reversed, the patient will not regain a pulse or start to breathe again.
History of chronic obstructive pulmonary disease (COPD)	Elderly patients commonly have some form of COPD (emphysema or chronic bronchitis). The arrest may have been caused by an exacerbation of the COPD, which led to hypoxemia, acidosis, and then arrest. Remember also that COPD disorders can weaken the lung tissue and cause the development of a pneumothorax and collapse of the lung. (This too may precipitate a cardiac arrest.) Be alert for the presence or the development of a pneumothorax during positive pressure ventilation, which may cause a bleb on the lung tissue to rupture.

Electrocardiography (ECG)

AMI often causes predictable changes to cardiac conduction, and these changes are frequently noticeable through the use of 12-lead electrocardiography. Topic 27 will discuss in great detail how a 12-lead ECG is used to diagnose AMI; however, it is important that we touch on its general importance here.

A 12-lead ECG is used to rule in a specific type of AMI based on the findings of ST segment elevation. This type of MI is called an *ST elevation myocardial infarction* (STEMI). ST elevation identification is important because it has been found to be a reliable positive finding that can be used (in the proper context) at all levels of cardiac care to rule in AMI. In many cases, paramedics will combine symptoms and history with ECG findings to rapidly diagnose an AMI and initiate specific treatment strategies. Of course, ECG findings cannot be used alone to diagnose anything. As always, paramedics must assess the patient and not the monitor.

Twelve-lead ECG findings cannot not rule out an AMI. In fact, the majority of AMIs diagnosed do not initially present with ST elevation. Rather, paramedics should use 12-lead ECG to rule *in* AMI, but continue to use other assessment findings to categorize the level of ACS risk.

Cardiac Enzymes

The third way that an AMI is diagnosed is through the use of cardiac biomarkers (frequently referred to as *cardiac enzymes*). When the heart is in distress, it releases very specific biochemical markers that can be measured. In many cases, but not always, these chemicals are enzymes. In most cases, these markers are measured through a blood test with very specific diagnostic equipment. In the past, these tests were limited to the hospital setting, but with the advance of modern technology, this is no longer necessarily true. Many EMS systems use portable point-of-care tests that can enable paramedics to test for these markers in the prehospital setting.

Cardiac biomarkers can be used effectively to identify cardiac damage, but unfortunately there are limitations. Some cardiac markers do not appear until the ACS event has significantly progressed (sometimes they do not appear for as long as 12 to 24 hours) and

TABLE 26-4 Cardiac Biomarkers

Marker	Description	Timing	Limitation
Troponin	Very specific and sensitive marker released as myocardial tissue is damaged.	Released after 2–4 hours but levels peak after 12 hours	Can be mimicked by severe pulmonary embolism, heart failure, and myocarditis.
Creatine kinase (CK-MB)	CK is an enzyme released when muscle is damaged. Its MB band is somewhat more specific to cardiac muscle.	Released after 3–4 hours but peaks after 10–24 hours	Also released when skeletal muscle is damaged. Can be mimicked by trauma.
Glycogen phosphorylase isoenzyme BB	Enzyme with isoform (BB) that is specific to cardiac muscle. Released as a result of ischemia. Increased levels can be diagnostic in myocardial infarction and unstable angina.	Released after 1–3 hours but peaks after 7 hours	Relatively new test. Minimal research validation.
B-natriuretic peptide (BNP)	Enzyme released from dysfunctioning myocardium. Commonly used biomarker for heart failure, but can also signal ACS.		Not specific to ACS. Can be mimicked by valvular heart disease, ventricular hypertrophy.
Myoglobin (Mb)	Protein found in skeletal and cardiac muscle. Secreted rapidly as a result of damage. Typically not specific enough to rule in an AMI, but lack of its presence can help rule out AMI.	Released after 2 hours	Low specificity for myocardial infarction. Can be mimicked by skeletal muscle damage.
Lactate dehydrogenase (LDH)	Enzyme secreted by conversion of lactate. Can be somewhat specific to heart muscle.	Released after 72 hours	High LDH levels can also be present in other conditions that cause tissue breakdown or destruction of blood cells. Mimics include cancer, meningitis, and HIV.

often they are not specific to AMI. For example, chest trauma can frequently produce many of the same chemical markers as AMI. However, when examined in context with other assessment findings, such as history and ECG, cardiac biomarkers can be a valuable diagnostic tool. Table 26-4 reviews key cardiac biomarkers.

EMERGENCY MEDICAL CARE

The treatment of ACS must be geared toward identifying and eliminating the occlusion to blood flow. Time is of the essence. Every 10 minutes of an occluded coronary artery lead to 1 percent patient mortality; therefore, rapid identification and treatment are essential. In many ways, the treatment of ACS should be approached in a manner similar to trauma care. There are very few meaningful on-scene treatments that should delay transport to definitive treatment. Rapid transport is the goal.

Definitively, AMI is generally treated in one of two ways. The first method is percutaneous coronary intervention (PCI). In this method, a catheter is inserted through a large blood vessel and is navigated to the area of the clot. The catheter can then dilate the vessel and eradicate the clot; a stent is then inserted through the catheter to keep the vessel open. This process not only is generally considered the safest alternative, but also requires extensive technology and is limited to facilities with catheterization capabilities.

The second method to treat an AMI is the use of thrombolytic or fibrinolytic therapy, in which medications such as streptokinase or tissue-type plasminogen activator (tPA) are used to chemically lyse the clot. Fibrinolytics are effective, but not specific to clots in the coronary arteries. Therefore, administration can lead to bleeding in other locations within the body and cause stroke. In some places, paramedics have been used to effectively administer fibrinolytic medications.

It is important for EMS provider to remember that both these therapies are generally available only in the hospital environment. Therefore, treatment of ACS should not interfere with rapid transport. Although prehospital administration of fibrinolytics is feasible, it is a controversial topic. As a result (and for the sake of the limited scope of this text), the treatments discussed here will focus on more universally accepted therapies.

Rapid Identification

The first element of treatment must be rapid identification. As stated previously, paramedics should cast a wide net and be inclusive when it comes to ruling out ACS. In general, there are three categories of cardiac patients. The first category is STEMI patients. This group can be rapidly and reasonably conclusively ruled in and therefore require the most aggressive treatment. The criteria for this group are positive symptomatology and history and an ST elevation on the ECG.

The second group can be categorized as ACS patients. These patients will have positive symptomatology and history, but lack ST elevation on the ECG. For the purposes of being inclusive, they fit the profile and should be treated as if they were having an MI until proven otherwise.

The final group would be non-ACS patients; this group would be populated by patients where AMI would be very unlikely. Chest pain secondary to traumatic injury would be a likely criteria for inclusion. This group should have the

least number of patients, as it will be very difficult to conclusively rule out ACS given positive symptoms.

Treatment of STEMI

The treatment of STEMI patients will include the most aggressive interventions. In this group, we can reasonably conclude that an AMI is present; as such, the cost–benefit analysis shifts. In many systems, the presence of STEMI allows for bypass of the emergency department in favor of direct transport to the catheterization laboratory. By doing so, door-to-balloon time (the time from the patient entering the hospital to successful catheterization) can be decreased significantly.

The presence of STEMI may also initiate specific protocols regarding the number and placement of intravenous lines in anticipation of either PCI or thrombolytic medications. As always, follow local protocols. At a minimum, identification of STEMI should lead to immediate communication of the finding to the receiving hospital to initiate a larger team effort of cardiac care and to speed the initiation of more advanced treatment options.

Treatment Basics Revisited

Paramedics wield a wide array of medications and therapies for the treatment of their patients, but when dealing with ACS, it is vital that basic-level treatment is not forgotten. Steps as simple as calming the patient down and limiting exertion will help minimize cardiac oxygen demand and therefore limit the scope of infarction.

Position the patient. Semi- or high Fowler positioning will assist the patient in breathing and minimize the negative effects of fluid accumulating in the lungs. If positive pressure ventilation is being provided or frank hypotension is present, the patient will need to be placed supine for ongoing management.

In some cases, airway management will be necessary, but in most cases simply assuring a normal oxygenation level is most important. Provide oxygen if the patient is dyspneic, hypoxemic, has obvious signs of heart failure, or has a SpO_2 reading of less than 94 percent. Initiate oxygen therapy via nasal cannula at 4 lpm and titrate oxygen therapy to maintain a SpO_2 reading of 94 percent or greater. If the breathing is inadequate, provide positive pressure ventilation at 10 to 12/min with high-flow supplemental oxygen.

Of all the medications that are potentially available, aspirin still makes the most difference. Aspirin blocks thromboxane A_2 and helps prevent the activation of platelets and the further creation of a clot. The American Heart Association calls aspirin the "single most effective medication used to treat myocardial infarction." Administer 325 mg nonenteric-coated aspirin. Instruct the patient to chew the aspirin to promote a more rapid absorption. Aspirin should be administered even if the patient is on a daily regimen (unless he has taken the aspirin within the last hour).

Advanced Treatment

Immediate advanced level care may be treatment of cardiac dysrhythmias. Often ACS leads to electrical dysfunction and can cause bradycardias, tachycardias, and other arrhythmias.

Nitroglycerin is a commonly used medication to treat ACS. Nitroglycerin assists the ACS patient in two ways. First, theoretically, it dilates coronary arteries and can increase flow through otherwise obstructed vessels. Second, nitroglycerin is a primarily venous vasodilator and, as a result, increases venous pooling of blood and reduces preload. This reduction decreases the workload of the heart and reduces myocardial oxygen demand.

If the systolic blood pressure is greater than 90 mmHg or is less than 30 mmHg from the patient's baseline systolic blood pressure, administer one nitroglycerin tablet every 3 to 5 minutes. Be sure the systolic blood pressure remains above 90 mmHg following the administration. Do not administer nitroglycerin if any of the following is present:

- A systolic blood pressure <90 mmHg, or 30 mmHg or more below the patient's baseline systolic blood pressure
- Extreme bradycardia (< 50 bpm)
- Tachycardia in the absence of heart failure (>100 bpm)
- Right ventricular failure
- Erectile dysfunction medication use by the patient within the previous 72 hours

Nitroglycerin infusions have become a reliable administration route for both the treatment of ACS and the treatment of acute pulmonary edema. Infusion pumps offer an exact and controllable dose that is easily titrated to a desired effect.

Other advanced treatments can include additional medications to prevent platelet aggregation and clotting. Some EMS systems administer clopidogrel (Plavix), which works by inhibiting the glycoprotein IIb/IIIa pathway of coagulation. It is effective in minimizing clots that cause ACS, but can also cause a high risk of hemorrhage. Eptifibatide (Intergilin) is a similar medication that is less commonly used prehospitally.

Finally, analgesics may be necessary when chest pain is unrelieved by nitroglycerin. Although for many years morphine sulfate was the standard, recent studies have shown concern regarding the potentially cardiac toxic properties of narcotics. In related studies, morphine was shown to actually drop cardiac output; as a result, its utility in ACS care has, in some places, come into question. As always, follow local protocol.

Besides specific treatments, constant reassessment is essential. Remember that a frequent side effect of AMI is cardiac arrest. Paramedics must be hypervigilant and prepared for this possibility.

TRANSITIONING

REVIEW ITEMS

1. Aspirin is given in acute coronary syndrome to _____.
   a. block thromboxane A_2 and minimize the clotting process
   b. alleviate pain
   c. thin the blood
   d. dilate coronary vessels and increase blood flow
2. Which of the following best describes the difference between myocardial ischemia and myocardial infarction?
   a. In ischemia, the heart muscle dies.

b. In infarction, the heart muscle dies.
c. Ischemia arises from total occlusion of a coronary artery.
d. Infarction occurs when myocardial oxygen needs are not being met adequately.

3. Which of the following is a modifiable risk factor for coronary artery disease?
   a. tobacco use
   b. age and gender
   c. family history of cardiovascular disease
   d. confirmed diagnosis of diabetes mellitus

4. Your 86-year-old female patient is complaining of dyspnea, weakness, swollen ankles, and diffuse chest discomfort. She has a history of coronary bypass and hypertension. She is allergic to penicillin. Currently, her mental status is normal. Her vital signs are blood pressure 180/100 mmHg, heart rate 108 beats per minute and irregular, respiratory rate 18/minute. Given this scenario, what should your *first* action be?
   a. Administer 365 mg baby aspirin orally.
   b. Administer oxygen via a nasal cannula at 4 lpm.
   c. Assist with the administration of her prescribed nitroglycerin.
   d. Assist with the administration of her oral antihypertensive medication.

5. You arrive on scene to find a 45-year-old male patient with a chief complaint of chest pain. The patient states that the pain began while he was mowing the lawn but subsided after he sat on the couch for a few minutes. The patient has a cardiac history, and he states, "This just happened last week while mowing the lawn." Based on this information, from what do you think the patient is most likely suffering?
   a. acute myocardial infarction
   b. Prinzmetal angina
   c. stable angina
   d. congestive heart failure

APPLIED PATHOPHYSIOLOGY

A 48-year-old male patient is complaining of chest pain that he describes as dull and located substernally, with radiation to his neck. He rates the pain a 6 on a scale of 1 to 10 and complains of nausea and lightheadedness. His skin is cool and diaphoretic. His heart rate is 96 beats per minute, blood pressure is 124/82 mmHg, respiratory rate is 14, SpO_2 is 92 percent. He has taken two of his own nitroglycerin tablets prior to your arrival, without any relief.

1. Should you suspect that this patient is suffering from an ischemic or an infarction episode? Support your answer with the appropriate clinical findings provided.

2. Why might the administration of nitroglycerin to this patient be beneficial?

3. Describe the anticipated physiologic benefit(s) for each of the interventions that may be administered to a patient suffering from a myocardial infarction. Also list the appropriate dosage for each medication:
   a. Oxygen
   b. Aspirin
   c. Nitroglycerin

4. Discuss the differences in pathophysiology and presentation of the following acute coronary syndrome manifestations:
   a. Stable angina pectoris
   b. Unstable angina pectoris
   c. Myocardial infarction

5. Describe the pathophysiologic changes that occur to the lumen of the blood vessel resulting in atherosclerosis and occlusion of coronary blood vessels.

CLINICAL DECISION MAKING

You are summoned to a retirement community for an unknown medical emergency. On your arrival you meet a 68-year-old man sitting beside a ladder he was attempting to climb so he could clean out his gutters. On further questioning, you learn that he experienced a sensation of vertigo and nausea while climbing the ladder. A nearby family member stated that the patient was caught as he stumbled off the ladder and that no trauma occurred. The patient has also been experiencing "heartburn" for two days without relief. He last ate two hours earlier. His medical history includes hypertension, diabetes mellitus, a previous MI, and a "mini stroke" two years ago.

1. Based on the scene size-up characteristics, identify the clues that point to the field impression of either an ischemic or an infarction episode.

2. What medical condition does this patient have that could mask the common finding of an acute coronary syndrome?

The primary assessment reveals the patient to be alert and well oriented. His airway is clear; his breathing is rapid at 22/minute, respirations are slightly shallow, vesicular sounds are present, and slight inspiratory crackles (rales) are noted. Peripheral pulses are also present and noted to be rapid and slightly irregular. His blood pressure is 132/92. The neck veins are obviously engorged, and the SpO_2 reading on room air is 87 percent. His blood sugar is currently 142 mg/dL.

3. What are the life threats to the patient, if any, that you are currently aware of?

Further assessment reveals the patient's pupils to be equal and reactive, and the SpO_2 has increased to 94 percent with supplemental oxygen. Breath sounds are unchanged, and jugular venous distention is still present. His blood pressure is now 140/98, heart rate is 112 and irregular, and respirations have increased to 28/minute. The patient is starting to complain of chest "pressure" as well. During the ongoing management of the patient, you elect to administer the therapies listed below.

4. For each one, discuss in specific detail (1) the reason the intervention is warranted, (2) the expected outcome of that intervention, and (3) how you would assess to determine if the desired effect is occurring (i.e., the treatment worked).
   a. Position patient in high Fowler position
   b. Administer oxygen
   c. Administer aspirin
   d. Administer nitroglycerin