Recognizing and Managing Angina Pectoris

By Bryan E. Bledsoe, DO, EMT-P, and Dwayne E. Clayden, EMT-P

One of the major causes of death and disability in the United States today is ischemic heart disease. A common manifestation of ischemic heart disease is angina pectoris, a name that literally means "pain in the chest.

Angina has been documented in the medical literature for many years. In fact, the famous English physician William Heberden described angina in the late 18th century as "a disorder of the breast which occurs on walking, with stress, or after meals." Then, in the late 19th century, Canadian physician Sir William Osler proposed that angina pectoris was caused by a spasm or narrowing of the coronary arteries. But today we define angina pectoris as a cramping pain in the chest caused by an imbalance between myocardial oxygen supply and demand.

Angina is most commonly caused by atherosclerotic heart disease. It occurs whenever myocardial oxygen demand is increased and the coronary arteries are unable to provide sufficient blood to meet this demand. However, some patients with only mild coronary artery disease suffer from angina pectoris. Interestingly, there is an enigmatic group of angina patients who have completely normal coronary arteries. In these patients, coronary artery spasm appears to be the cause of the reduced coronary artery blood flow.

Case Presentation

Medic 26, an advanced life support ambulance, is dispatched to a suburban residence to assist a patient suffering chest pain. On arrival, the paramedics find a moderately obese male in his late 50s who is in acute distress. Quickly performing the primary assessment, the caregivers detect no problems with the patient's airway, breathing or circulation, and they begin administering oxygen using a non-rebreather mask.

The patient states that he recently began a new exercise regimen in an attempt to lose weight. Prior to the call, he was riding a stationary exercise bicycle and approximately five minutes into his exercise routine developed tightness in his chest. The patient rates the pain as an eight on a scale of one to ten. He reports that the tightness does not radiate, nor is it alleviated by stopping exercise or changing position.

The patient is moderately anxious but not diaphoretic. His vital signs are: blood pressure—168/108; pulse—100; and respirations—20/min. The paramedics hook the patient up to non-invasive monitors. The EKG monitor shows a sinus tachycardia with occasional unifocal premature ventricular contractions (PVCs), and the pulse oximeter shows an oxygen saturation (SpO₂) level of 99 percent.

Per standing orders, the paramedics establish an IV of D₅W KVO (keep vein open) and administer a 0.4 mg nitroglycerin (NTG) tablet sublingually. Shortly thereafter, the patient reports a decrease in his chest pain from an eight to a four, and the paramedics note that the PVCs have stopped. The blood pressure is now 148/96.
The paramedics package the patient and transport him to the ambulance, where a second dose of 0.4 mg NTG is administered sublingually, approximately five minutes after the first dose. This promptly results in complete relief of the patient's chest tightness. The trip to the hospital is uneventful.

Once in the emergency department, the patient is re-examined by the ED physician. A 12-lead EKG, obtained shortly after arrival, is entirely normal. Because of the patient's history, however, he is admitted to the coronary care unit to rule out a myocardial infarction.

The patient's next two hospital days are unremarkable. Serial EKGs and cardiac enzymes remain normal, but he has a slight recurrence of the chest tightness following a meal on the second day.

On the third hospital day, the patient is taken to the cardiac catheterization lab, where he undergoes coronary angiography. The angiogram reveals a 95-percent blockage of his left anterior descending coronary artery, and percutaneous transluminal coronary angioplasty (PTCA) is carried out successfully. The patient tolerates the procedure well and is discharged the following day. He is now on a medically supervised diet and exercise regimen, and he takes an aspirin a day to prevent additional problems.

**Pathophysiology**

*To meet the metabolic demands of the myocardial tissues, the heart must receive a constant supply of oxygenated blood. This blood is supplied to the heart through the coronary arteries, which originate in the aorta just above the leaflets of the aortic valves.*

The left coronary artery, with its two major branches—the anterior descending artery and the circumflex artery—supplies the left ventricle, the interventricular septum and part of the right ventricle. The right coronary artery, with its two major branches—the posterior descending artery and the marginal artery—supplies a portion of the right atrium and part of the right ventricle (see Figure 1). Blood flow to the heart is provided during relaxation of the cardiac muscle (diastole).

**Atherosclerosis** is a progressive degenerative disease of the medium-sized and large arteries that commonly affects the aorta and its branches, the coronary arteries and the cerebral arteries, among others.
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Glossary of Terms

Atherosclerosis — A condition in which lipid (fat) deposits are made in the medium-sized and large arteries, resulting in hardening of the artery walls and diminished blood flow.

Atherosclerotic Heart Disease — Heart dysfunction brought on by the deposit of plaque in the arteries and the resultant lack of blood flow.

Beta Receptors — Areas in the autonomic nerve pathway where inhibitory responses occur when agents such as norepinephrine and epinephrine are released.

Coronary Angiography — A process by which pictures are taken of the coronary vessels of the heart after injection of a radiopaque substance.

Coronary Artery Bypass Graft (CABG) — A surgical process in which a shunt is placed that permits blood to travel from the aorta to a branch of the coronary artery at a point past an obstruction.

Epigastrum — The region above the stomach.

Ischemic Heart Disease — A disease condition created by insufficient blood supply to the heart muscle.

Myocardial — Refers to the middle layer of the heart walls, which are composed of heart muscle.

Nitrate — A salt of nitric acid which dilates blood vessels and reduces blood pressure.

Percutaneous Transluminal Coronary Angioplasty (PTCA) — A method of treating localized coronary-artery narrowing by inserting a special double-lumen catheter surrounded by a balloon into the artery and inflating the balloon to dilate the narrowed vessel.

Plaques — A patch on the surface of a mucous surface. Atherosclerotic patches are caused by fat or lipid deposits.

Sternotomy — Surgery in which a cut is made through the sternum.

Vasospasm — Spasm of a blood vessel.

Others. It results from the deposition of fats (lipids and cholesterol) in the walls of the affected vessels. This results in damage to the artery wall, ultimately causing the formation of plaques. These plaques can become large enough to reduce the amount of blood transported through the artery. In severe cases, plaques can completely block the artery.

As mentioned, angina occurs whenever myocardial oxygen demand is increased and the coronary arteries are unable to supply sufficient blood to meet those demands. This usually occurs when plaques in the coronary arteries restrict the amount of blood that can be delivered to the myocardial tissues. At rest, the amount of blood delivered is usually adequate. However, during periods of physical or emotional stress, the heart rate and cardiac contractile force are increased, resulting in the need for increased oxygen and other nutrients. If the coronary arteries are unable to deliver the required amount of blood, the myocardium will become ischemic, resulting in the pain typically associated with angina.

To complicate matters, ischemia causes the left ventricle to dilate, creating a further increase in myocardial oxygen demand. Ischemia also reduces coronary blood flow, worsening the situation. The combination of increased myocardial oxygen demand and decreased coronary blood flow results in the classic syndrome called angina pectoris.

There are several types of angina, each of which may be identified through patient history or method of onset. After a period of rest, the heart rate and contractile force usually decrease to a point where the myocardium again receives an adequate amount of blood. The return of adequate blood supply to the myocardium relieves the ischemia and subsequently alleviates the pain. This type of angina, which occurs following exertion or exercise and is relieved by rest or NTG, is referred to as stable angina. It is predictable and recurrent, often causing the patient to change or curtail activities to prevent the pain. Patients who do not undergo physical or emotional stress remain symptom-free.
As coronary atherosclerosis worsens, however, episodes of angina tend to occur more frequently. Eventually, the pain will occur without exertion or stress, becoming what is called unstable angina. Unlike stable angina, which occurs when the heart’s metabolic demand increases, unstable angina occurs without any detectable increase in myocardial demand. Instead, the problem is almost exclusively related to decreased supply.

Unstable angina is the most severe form of angina and is often a warning of impending myocardial infarction (MI), hence its alternative names of pre-infarction angina or crescendo angina. The pain of unstable angina may be similar to that of stable angina, although it tends to be more intense, occur more frequently and last longer than stable angina pain—often up to several hours.6

In those angina patients who have only mild coronary artery disease, the condition is usually associated with abnormal spasm of the coronary arteries. This type of angina, called variant angina, vasospastic angina or Prinzmetal’s angina, occurs without any increase in myocardial oxygen demand. Instead, vasospasm superimposed on normal or diseased arteries can induce transient, or brief, myocardial ischemia, which causes the pain and symptoms characteristic of angina.

Last, there is a group of angina patients who have no evidence of coronary artery disease. This unusual condition is referred to as Syndrome X. Although the exact cause of Syndrome X is not known, it appears to be caused by an imbalance between myocardial oxygen demand and supply.7

**Assessment**

Approximately 80 percent of all patients with angina pectoris are men. They are typically in their 50s or early 60s, often overweight and have a sedentary lifestyle. Patients with angina will rarely use the word “pain” to describe their discomfort. Instead, they describe it as a heaviness, pressure, tightness or a choking sensation.

The discomfort is usually located substernally but occasionally may be found in the epigastrium or in the left arm. While the pain may or may not radiate, if it does, common locations are the left arm, jaw and neck. The most common clinical feature of angina is that it is induced by exertion or emotion and is relieved by rest or NTG administration. The typical angina chest discomfort lasts only a few minutes and rarely more than 30 minutes. (See Table 1 for a comparison of the signs and symptoms of angina pectoris and acute myocardial infarction.)

Assessment of the patient with angina pectoris should be the same as for any patient suffering chest pain. First, a primary assessment should be conducted to detect any immediate problems with the airway, breathing or circulation. Next, the secondary assessment should be started with a brief history. If possible, determine if there is a relationship between the chest discomfort and exertion or emotional stress.

Also, note if the patient has any risk factors for heart disease, including hypertension, smoking, diabetes mellitus, elevated serum cholesterol, a sedentary lifestyle or a family history of ischemic heart disease. The presence of any of these factors enhances the likelihood of underlying coronary artery disease.

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Continued on page 79
Physical examination of the patient with angina is often normal. However, particular emphasis should be placed on examination of the chest and heart, listening for extra beats and for the presence of rales or other abnormal breath sounds. Also, look at the neck for signs of jugular vein distension and at the extremities for signs of edema, which signal cardiac compromise.

An accurate set of vital signs should be determined early in the patient encounter. Repeat the vital signs often, especially after administration of a medication or a change in the patient’s status. Also, always be alert for subtle indicators of underlying heart disease, including the presence of a sternotomy scar from a prior coronary artery bypass graft (CABG), a pacemaker generator or the presence of a NTG patch (see Figure 2).

An advanced assessment should also include non-invasive monitoring techniques, such as an EKG and pulse oximetry. It is often helpful to obtain an EKG when the patient is suffering pain and compare it to a tracing obtained when the patient is pain-free, as some angina patients will have ventricular ectopic activity during periods of ischemia. This often resolves when the ischemia is corrected.8

ALS units with 12-lead EKG monitoring capability may note transient ST-segment depression or T-wave inversion during periods of pain. These usually return to normal when symptoms abate. If available, pulse oximetry should be used to determine the SpO2 level and to guide further therapy.

Management
Since it is difficult to determine which patients with chest pain are suffering from angina and which are suffering an MI or other problem, any patient with chest pain should be assumed to be suffering an MI until proven otherwise. As soon as the primary assessment is completed, administer high-flow oxygen, preferably via a non-rebreather mask. This will increase the amount of oxygen available to the ischemic myocardium and will aid in reducing ischemia and pain in the angina patient.
Table 1.

Angina vs. MI

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Angina Pectoris</th>
<th>Acute Myocardial Infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous attacks or similar pain</td>
<td>Usually</td>
<td>Occasionally</td>
</tr>
<tr>
<td>Character of pain</td>
<td>Squeezing</td>
<td>Squeezing, increases with time</td>
</tr>
<tr>
<td>Onset of pain</td>
<td>Following stress or exercise</td>
<td>Precipitating factor not necessary</td>
</tr>
<tr>
<td>Duration of pain</td>
<td>Two to 10 minutes, up to 20 to 30 minutes</td>
<td>Greater than 30 minutes</td>
</tr>
<tr>
<td>Location of pain</td>
<td>Retrosternal</td>
<td>Retrosternal</td>
</tr>
<tr>
<td>Associated findings</td>
<td>Occasionally dyspnea</td>
<td>Nausea, vomiting, dyspnea, diaphoresis</td>
</tr>
<tr>
<td>Exam findings</td>
<td>Often normal</td>
<td>Congestive heart failure, shock, restlessness</td>
</tr>
<tr>
<td>Response to NTG</td>
<td>Partial or complete alleviation</td>
<td>Minimal if any</td>
</tr>
<tr>
<td>EKG changes</td>
<td>None or minimal</td>
<td>Often diagnostic</td>
</tr>
<tr>
<td></td>
<td>(inverted T-waves or ST-segment depression)</td>
<td>(ST-segment elevation in affected leads)</td>
</tr>
</tbody>
</table>

As soon as possible, insert an IV or saline flush to provide for rapid intravenous access should the patient deteriorate or develop life-threatening dysrhythmias. Provide continuous monitoring of the EKG, pulse, blood pressure and SpO2.

In addition to oxygen, there are a number of medications that can be used to treat angina pectoris (see Table 2). These medications—nitrates, beta blockers, calcium channel blockers and narcotics—typically work to decrease the stress on
Always look for subtle indicators of underlying heart disease. That could mean any combination of the clues in this photo:

- an NTG patch
- a sternotomy scar
- or the presence of a pacemaker generator.

Although a bulge in the skin from a pacemaker would normally be located on the chest, don't be surprised to find it elsewhere—as it is here, in the mid-abdominal area.

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the heart, decrease pain and thus decrease the patient’s anxiety and stress.

Nitrate
Initial prehospital treatment of angina by ALS personnel should include the administration of a nitrate preparation, such as NTG, which is the most common nitrate preparation used in the emergency setting. (EMTs may assist patients in administering their own prescribed medication but currently may not initiate such treatment.) NTG, a potent vasodilator, is often used to alleviate anginal chest pain and is effective in the treatment of angina through two different mechanisms.

First, it relaxes the smooth muscle in the walls of the veins, causing dilation. This reduces the amount of blood being returned to the heart (preload), resulting in decreased ventricular volume, decreased ventricular pressure and decreased ventricular-wall stress. These actions effectively decrease cardiac work, lessen myocardial oxygen demand and improve myocardial perfusion.

Second, NTG relaxes the smooth muscle within the coronary arteries, causing vasodilation. This helps counteract any vasospasm and increases myocardial blood flow. Thus, the preparation both reduces myocardial oxygen demand and increases myocardial blood supply.

Most patients with pre-existing angina are on some form of nitrate therapy. This may be in the form of a short-acting NTG tablet, a long-acting preparation or a skin patch that delivers a steady supply of the drug through the skin. Since NTG preparations deteriorate fairly quickly when exposed to air, NTG that is old or that has been improperly stored is often ineffective. To be safe, always administer fresh NTG in the field, and once a bottle of NTG tablets is opened, label it with the date opened; some services make it a policy to open a new bottle of NTG for each patient and to leave it at the hospital with the patient. NTG that has been exposed to air for an extended period of time should be replaced following the manufacturer’s guidelines.

Initially, NTG should be administered at 0.4 mg sublingually either as a tablet (Nitrostat) or as a spray (Nitrolingual Spray). Effects of the drug can be seen within two minutes and may last for 15 to 20 minutes, and the dose can be repeated at five-minute intervals until the pain is relieved, the blood pressure falls, or a total of three tablets have been administered.

Patients on long-term nitrate therapy, such as an NTG patch, can develop a tolerance to the drug and may need the additional therapy of sublingual NTG to alleviate the anginal chest pain. This should only be administered after the blood pressure has been assessed and it is determined that the patient is not hypertensive. In some cases of unstable angina, NTG may be administered as an IV drip to ensure constant delivery of the drug.

Calcium Channel Blockers
A second class of drugs, calcium channel blockers, also play a major role in angina therapy. Blockers such as nifedipine (Procardia) and diltiazem (Cardizem) are
Many cases of angina are precursors to a full-blown MI and warrant comprehensive care.

effective in angina because they relax vascular smooth muscle, causing vasodilation. These drugs decrease the systemic vascular resistance, thus reducing the pressure against which the heart must pump (afterload) and effectively reducing myocardial work.

In addition, the calcium channel blockers cause vasodilation of the coronary arteries, increasing blood flow and reversing spasm. They also cause a decrease in myocardial oxygen demand due to a direct effect on the heart itself.

Nifedipine is preferred in patients who are hypertensive (many anginal patients are), since it has a greater vasodilatory effect than diltiazem and tends to decrease blood pressure to a higher degree. The initial dose of nifedipine is 10 mg capsule sublingually or by mouth, repeated in 10 to 20 minutes if the blood pressure remains stable. For non-hypertensive patients, the standard dose of diltiazem is 30 mg to 60 mg by mouth.

There are three methods of administering oral nifedipine. The first is to let the capsule dissolve under the tongue; the second is to cut the capsule or pierce it with a sterile needle and place it under the tongue; and last, the patient can bite or crush the capsule and swallow. While the first method was once preferred due to its slow administration rate, there is no real evidence that any one method is better.

**Beta Blockers**

Beta blocker drugs block beta receptors and are often used in conjunction with nitrates to prevent angina attacks. Beta receptors are activated by hormones such as epinephrine and norepinephrine. When released, these hormones tend to increase myocardial oxygen demand because they increase both the rate and strength of the cardiac contraction.

The administration of beta blockers causes a decrease in heart rate and cardiac contractile force, which decreases myocardial work. However, beta blockers have a limited emergency use in the treatment of angina since they cannot be used in the presence of coexisting congestive heart disease.

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1. **Angina pectoris** literally means:
   a. Heart pain
   b. Chest pressure
   c. Pain in the chest
   d. Near death

2. **Angina pectoris** is defined as:
   a. Any chest pain of cardiac origin
   b. A cramping pain in the chest caused by an imbalance between myocardial oxygen supply and demand
   c. Chest pain due to ischemia caused by spasms of the coronary arteries
   d. Chest pain associated with a myocardial infarction

3. Blood is supplied to the heart through the coronary arteries, which originate at the:
   a. Left ventricle
   b. Circumflex artery
   c. Aorta
   d. Vena cava

4. Blood flows to the myocardium during the _____ stage of the cardiac cycle.
   a. Systole (contraction)
   b. Diastole (relaxation)

5. Lipids and cholesterol are deposited on the walls of an atherosclerotic vessel, resulting in:
   a. Plaque formation
   b. Hardening of the vessel wall
   c. Dilation of the vessel
   d. Frequent spasms of the vessel

6. **Angina pectoris** typically occurs:
   a. At rest
   b. At night or when the patient is lying supine
   c. When a coronary blood vessel is completely occluded
   d. During periods of physical or emotional stress

7. Unstable angina is characterized by:
   a. Anginal pain not relieved by nitroglycerin (NTG)
   b. Anginal pain occurring at rest or without exertion
   c. Anginal pain unrelated to coronary blood flow
   d. Radiation of pain to the arms or back

8. Angina occurring without an increase in myocardial oxygen demand is called:
   a. Variant angina
   b. Vasospastic angina
   c. Prinzmetal's angina
   d. All of the above

9. The most common clinical feature of angina is:
   a. Chest pain
   b. Onset of chest pain with exertion
   c. Onset of chest pain with exertion and relief with relaxation
   d. Elevated blood pressure

10. The single most important BLS treatment for an angina patient is:
    a. Administration of supplemental oxygen
    b. Calming and reassuring the patient
    c. Rapid transport to a coronary care unit
    d. Elevation of the lower extremities

11. The oxygen-delivery device of choice for the angina patient is the:
    a. Nasal cannula
    b. Simple face mask
    c. Non-rebreather mask
    d. Positive-pressure demand valve

12. Nitrostat is a trade name for NTG, which is a:
    a. Beta blocker
    b. Calcium channel blocker
    c. Nitrile
    d. Vasodilator

13. NTG relieves angina pain by:
    a. Blocking pain receptors
    b. Increasing preload on the heart
    c. Increasing myocardial oxygen demand and reducing myocardial blood supply
    d. Reducing myocardial oxygen demand and increasing myocardial blood supply

14. Which of the following statements regarding NTG tablets is true?
    a. Once open, a bottle of NTG has a shelf life of one year.
    b. NTG does not expire as long as it is stored in its amber bottle.
    c. NTG deteriorates fairly quickly when exposed to air.
    d. None of the above

15. A patient states that she takes Inderal. You recognize this as a:
    a. Beta blocker
    b. Calcium channel blocker
    c. Nitrile
    d. Antidepressant

**Questions 16 through 20 are mandatory for paramedics. EMTs may answer the questions, but credit will not be applied to their scores.**

16. During periods of ischemia, angina patients may exhibit:
    a. Ventricular ectopic activity
    b. First-degree heart block
    c. Sinus bradycardia
    d. Junctional escape beats

17. Effects of NTG are usually seen:
    a. Immediately
    b. Within two minutes
    c. Within five to 10 minutes
    d. Within 20 minutes

18. NTG should be administered until:
    a. Pain is relieved
    b. The systolic blood pressure falls below 100 mmHg
    c. A total of three tablets have been administered
    d. Any of the above

19. One of the reasons morphine is effective for angina patients is that it:
    a. Reduces the afterload
    b. Blocks beta receptors in the myocardium
    c. Decreases the force of cardiac contractions (inotropic effect)
    d. Causes vasodilatation

20. The drug of choice for lowering the blood pressure of a hypertensive angina patient is:
    a. Cardiazem (diltiazem)
    b. Procainamide (nifedipine)
    c. Procainamide (nifedipine)
    d. Calm (verapamil)
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Answers

Mark your answers to the test by checking the appropriate boxes (☐) below. Each question has only one answer. EMTs: Answer questions 1 through 15. Paramedics are required to answer all questions.

1. ☐ a. ☐ b. ☐ c. ☐ d.
2. ☐ a. ☐ b. ☐ c. ☐ d.
3. ☐ a. ☐ b. ☐ c. ☐ d.
4. ☐ a. ☐ b. ☐ c. ☐ d.
5. ☐ a. ☐ b. ☐ c. ☐ d.
6. ☐ a. ☐ b. ☐ c. ☐ d.
7. ☐ a. ☐ b. ☐ c. ☐ d.
8. ☐ a. ☐ b. ☐ c. ☐ d.
9. ☐ a. ☐ b. ☐ c. ☐ d.
10. ☐ a. ☐ b. ☐ c. ☐ d.

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heart failure or reactive airway disease, such as asthma or chronic obstructive pulmonary disease. Common beta blockers include propranolol (Inderal), nadolol (Corgard), and metoprolol (Lopressor).

Narcotics
Narcotic preparations, such as morphine, are occasionally used in the treatment of anginal chest pain, especially when the pain does not respond to nitrate therapy. Morphine is useful in the treatment of angina for several reasons. First, it causes vasodilation of the veins, resulting in a decrease in the preload and in decreased myocardial work. It also acts on the central nervous system to alleviate the chest pain.

In addition, due to its action on the central nervous system, morphine has a calming effect on the patient, decreasing anxiety and fear and further lessening myocardial oxygen demand. In the acute setting, morphine is usually administered intravenously in 2 mg to 5 mg boluses until the patient's pain is significantly improved or the blood pressure begins to fall.

Table 2.
Common Medications Used in the Treatment of Angina Pectoris

<table>
<thead>
<tr>
<th>Oral Short-Acting Nitrates</th>
<th>Oral Long-Acting Nitrates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin (Nitrostat)</td>
<td>Isosorbide dinitrate (Isordil)</td>
</tr>
<tr>
<td>Isometric dinitrate</td>
<td>(Dilatrate-SR)</td>
</tr>
<tr>
<td>Isosorbide mononitrate</td>
<td>(Ismo)</td>
</tr>
</tbody>
</table>

Topical Nitrates Preparations
Nitroglycerin ointment (Nitro-Bid)
Nitroglycerin patch (Nitro-Dur)

Calcium Channel Blockers
Nifedipine (Procardia)
Diltiazem (Cardizem)
Verapamil (Calan, Verelan, Isoptin)
Nicaldipine (Cardene)

Beta Blockers
Propranolol (Inderal)
Nadolol (Corgard)
Metoprolol (Lopressor)

Narcotics
Morphine sulfate

Anti-Platelet Agents
Aspirin (Ecotrin, Bayer)
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Conclusion
Angina pectoris is a common manifestation of atherosclerotic heart disease and one to which prehospital caregivers will most certainly be called at some time in their careers.

One of the most important things to remember about angina pectoris is that all cases of chest pain should be treated as though they were an MI until proven otherwise. Always perform a primary assessment, and proceed to the secondary assessment with particular emphasis on the chest exam and the vital signs. Always administer high-concentration oxygen unless there is a medical contraindication, and if you are a paramedic, consider the administration of NTG or a similar nitrate preparation. If this is ineffective, consider administration of a narcotic or calcium-channel blocker.

Remember, many cases of angina are precursors to a full-blown MI and warrant comprehensive care.

References