Angina Pectoris

Ischemic heart disease is a major cause of death and disability in the Canada today. A common manifestation of ischemic heart disease is angina pectoris. Angina pectoris literally means “pain in the chest.” The famous English physician William Heberden described angina in the late eighteenth century as “a disorder of the breast which occurs on walking, with stress, or after meals.” In the late nineteenth century, Sir William Osler proposed that spasm or narrowing of the of the coronary arteries was the cause of angina pectoris. 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, there are certain patients who suffer from angina pectoris who have only mild coronary artery disease. Interestingly, there is an enigmatic group of patients who suffer angina, yet who have completely normal coronary arteries. In these patients coronary artery spasm appears to be the cause of reduced coronary artery blood flow.

In this article we will present the pathophysiology of angina pectoris. This will be followed by a discussion of assessment and management of the patient with angina with particular emphasis on the prehospital aspect of emergency care.

Case Presentation

Medic 26, an advanced life support ambulance, is dispatched to a suburban residence to assist a patient suffering chest pain. Upon arrival they find a moderately obese male in his late fifties who is in acute distress. Paramedics quickly perform the primary assessment. They detect no problems with the airway, breathing, or circulation. Oxygen administration is begun using a non-rebreather mask. The paramedics learn that the patient had recently begun a new exercise regimen in an effort to lose weight. He began riding a stationary exercise bicycle and approximately 5 minutes into his exercise routine developed tightness in his chest. He rates the pain as an eight on a scale of one to ten. He reports that the tightness does not radiate. It is not alleviated by stopping exercise or changing position. He is moderately anxious, but not diaphoretic. Vital signs are blood pressure 168/108 mmHg, pulse of 100 beats per minute, and respirations of 20 breaths per minute. Non-invasive monitors are placed. The ECG monitor shows a sinus tachycardia with occasional unifocal PVCs. The pulse oximeter shows an SpO2 of 99%.

Per standing orders, the paramedics establish an IV of 5% dextrose in water TKO and administer a 0.4 mg nitroglycerin tablet sublingually. Shortly thereafter the patient reports a decrease in his chest tightness from eight to four. Paramedics note that the PVCs have stopped. They promptly recheck the blood pressure following the nitroglycerin administration. It is now 148/96 mmHg.

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The patient is then packaged and transported to the ambulance. Once in the ambulance a second dose of 0.4 mg nitroglycerin is administered sublingually, approximately 5 minutes after the first. This promptly results in complete relief of the patient's chest tightness. The trip to the hospital is uneventful.

In the emergency department the patient is examined by the emergency department physician. A 12 lead ECG obtained shortly after arrival is entirely normal. However, because of the history, the patient is admitted to the coronary care unit to rule out a myocardial infarction. The patient's next two hospital days are unremarkable. Serial ECGs and cardiac enzymes remain normal. He has a slight recurrence of the chest tightness on the second day following a meal. On the third hospital day the patient is taken to the cardiac catheterization lab where coronary angiography is carried out. The angiogram reveals a 95% blockage of his left anterior descending coronary artery. Percutaneous transluminal coronary angioplasty (PTCA) is successfully carried out. The patient tolerates the procedure well and is discharged the following day. He is now on a medically-supervised diet and exercise regimen. In addition, he takes an aspirin a day to prevent additional problems.

**Pathophysiology of Angina Pectoris**

The heart must receive a constant supply of oxygenated blood to meet the metabolic demands of the myocardial tissues. Blood is supplied to the heart through the coronary arteries. These vessels originate in the aorta just above the leaflets of the aortic valves. The left coronary artery supplies the left ventricle, the interventricular septum, and part of the right ventricle. The two major branches of the left coronary artery are the anterior descending artery and the circumflex artery. The right coronary artery supplies a portion of the right atrium and part of the right ventricle. The two major branches of the right coronary artery are the posterior descending artery and the marginal artery. Blood flow to the heart is provided during cardiac relaxation (diastole).

Atherosclerosis is a progressive degenerative disease of the medium-sized and large arteries. It commonly affects the aorta and its branches, the coronary arteries, and the cerebral arteries, among others. It results from the deposition of fats (lipids and cholesterol) in the walls of the affected vessels. This results in damage to the wall of the artery ultimately causing the formation of plaques. These plaques can become large enough to reduce the amount of blood which can be transported through the artery. In severe cases the artery can become completely blocked either by an additional plaque or a blood clot.

As mentioned previously, 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 due to fixed atherosclerotic lesions in the coronary arteries which 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. This increased work results 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. This dilation causes a further increase in myocardial oxygen demand. In addition, ischemia 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.

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. Angina, which occurs following exertion or exercise, and is relieved by rest or nitroglycerin, is referred to as stable angina. It is predictable and recurrent, often causing the patient to change or curtail his or her activities to prevent pain. As long as the patient does not undergo physical or emotional stress, they remain symptom-free.

As coronary atherosclerosis worsens, episodes of angina tend to occur more frequently. Eventually, pain will occur without exertion or stress. Angina which occurs at rest, or without exertion, is called...
unstable angina. Unlike stable angina, which occurs when the heart's metabolic demand increases, unstable angina occurs without any detectible 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. Because of this, unstable angina is also referred to as “pre-infarction angina” or “crescendo angina.” The pain of unstable angina may be similar to that of stable angina. However, it tends to be more intense, occur more frequently, and last longer than stable angina pain—often up to several hours.

Some patients who experience angina have only mild coronary artery disease. In these patients the cause is usually associated with abnormal spasm of the coronary arteries. This type of angina, called variant angina, vasospastic angina or Frinzmetal's angina, occurs without any increase in myocardial oxygen demand. Vasospasm, superimposed upon normal or diseased arteries, can induce transmural myocardial ischemia causing the pain and symptoms characteristic of angina.

Lastly, there is a group of patients who suffer from angina who do not have any 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 that there is an imbalance between myocardial oxygen demand and supply.

Clinical Presentation and Assessment

Approximately 80% of all patients with angina pectoris are men. The typical patient is a male in his fifties or early sixties. He is often overweight and lives a sedentary lifestyle. The patient will usually not use the word “pain” to describe the discomfort of angina. Instead, they will describe their chest discomfort as a heaviness, pressure, tightness, or choking sensation. The discomfort is usually located substernally. Occasionally the pain is located in the epigastrium or in the left arm. The pain may or may not radiate. Common locations of pain radiation are the left arm, jaw, or neck. The most common clinical feature of angina is that it is induced by exertion or emotion, and relieved by rest or nitroglycerin. The chest discomfort of angina usually lasts only a few minutes and rarely over 30 minutes.

Assessment of the patient with angina pectoris should be the same as for any patient suffering from chest pain. First, the primary assessment should be carried out to detect any immediate problems with the airway, breathing or circulation. Following completion of the primary assessment, you should begin the secondary assessment by eliciting a brief history. If possible, try and determine if there is a relationship between the chest discomfort and exertion or emotional stress. The likelihood of underlying coronary artery disease is enhanced if the patient has any of the common risk factors for heart disease. These include hypertension, smoking, diabetes mellitus, elevated serum cholesterol, a sedentary lifestyle, or a family history of ischemic heart disease.

The physical examination of the patient with angina is often normal. Particular emphasis should be placed on examination of the chest and heart. Listen for extra beats and for the presence of rales or other abnormal breath sounds. Also, look at the neck for signs of jugular venous distention and at the extremities for signs of edema. 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. Always be alert for subtle indicators of underlying heart disease. These include such things as the presence of a sternotomy scar from a prior coronary artery bypass graft (CABG), a pacemaker generator, or the presence of a nitroglycerin patch.

Advanced life support units should utilize noninvasive monitoring techniques when caring for patients with a possible cardiac complaint. The most essential monitor is the ECG. It is often helpful to obtain an ECG strip when the patient is suffering pain and compare it to a tracing obtained when the patient is pain-free. Some angina patients will have ventricular ectopic activity during periods of ischemia. This often resolves when ischemia is corrected. ALS units with the capability of 12 lead ECG monitoring 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 and to guide further therapy.

Prehospital Management

It is difficult to determine which patients with chest pain are suffering from angina and which are suffering a myocardial infarction or other problem. Thus, any patient with chest pain should be assumed to be suffering a myocardial infarction until proven otherwise. As soon as the primary assess-
ment is completed, administer high-flow oxygen, preferably via a non-rebreather mask. Supplemental oxygen administration will increase the amount of oxygen available to the ischemic myocardium. This will aid in reducing ischemia and pain in the angina patient. 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 ECG, pulse, blood pressure, and SpO2.

Nitrates
In addition to oxygen, initial prehospital treatment of angina should include the administration of a nitrate preparation. Nitroglycerin (Nitrostat) is the most common nitrate preparation used in the emergency setting. Nitroglycerin is a potent vasodilator and is often used to alleviate anginal chest pain. Nitroglycerin relaxes smooth muscle and is effective in the treatment of angina through two different mechanisms. First, nitroglycerin 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 in decreased ventricular wall stress. These actions effectively decrease cardiac work, lessen myocardial oxygen demand, and improve myocardial perfusion. Nitroglycerin also relaxes the smooth muscle within the coronary arteries causing vasodilation. This helps counteract any vasospasm present and increases myocardial blood flow. Thus, nitroglycerin 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 nitroglycerin tablet, a long-acting preparation, or a skin patch which delivers a steady supply of the drug through the skin. Nitroglycerin preparations deteriorate fairly quickly when exposed to air. Thus, nitroglycerin which is old, or which has been improperly stored, is often ineffective. Always administer fresh nitroglycerin in the field. Once a bottle of nitroglycerin is opened it should be labeled with the date opened. Nitroglycerin which has been exposed to air for an extended period of time should be replaced following the manufacturer's guidelines.

The initial dose of nitroglycerin should be 0.4 mg administered sublingually either as a tablet (Nitrostat) or as a spray (Nitrolingual Spray). Effects are seen within 2 minutes and may last for 15 to 20 minutes. Nitroglycerin can be repeated at 5 minute intervals until the pain is relieved, the blood pressure falls, or a total of three tablets have been administered. In some cases of unstable angina, nitroglycerin may be administered as an IV drip to assure constant delivery of the drug.

Calcium Channel Blockers
In addition to the nitrates, a second class of drugs, the calcium channel blockers, play a major role in angina therapy. Calcium channel blockers, such as nifedipine (Procardia) and diltiazem (Cardizem), are 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). This effectively reduces myocardial work. In addition, the calcium channel blockers cause vasodilation of the coronary arteries increasing blood flow and reversing spasm. The calcium channel blockers also cause a decrease in myocardial oxygen demand due to a direct effect on the heart itself.

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

Beta Blockers
Beta blockers are drugs which block

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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 20-30 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>ECG changes</td>
<td>None or minimal (inverted T-waves or ST-segment depression)</td>
<td>Often dianostic (ST segment elevation in affected leads)</td>
</tr>
</tbody>
</table>
beta receptors and are often used in conjunction with the nitrates to prevent attacks of angina. Beta receptors are activated by hormones such as epinephrine and norepinephrine. These hormones, when released, tend to increase myocardial oxygen demand because they increase both the rate and strength of the cardiac contraction. When administered, beta blockers cause a decrease in heart rate and cardiac contractile force which decreases myocardial work. Beta blockers have a limited use in the emergency setting in the treatment of angina. They cannot be used if there is coexisting congestive heart failure or reactive airway disease such as asthma or COPD. 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 it does not respond to nitrate therapy. Morphine is useful in the treatment of angina for several reasons. First, morphine causes vasodilation of the veins. This causes a decrease in the amount of blood returned to the heart (preload) and 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 which helps decrease anxiety and fear and further lessens myocardial oxygen demand. In the acute setting, morphine is usually administered intravenously in 2-5 milligram boluses until the patient's pain is significantly improved or the blood pressure begins to fall.

Summary
Angina pectoris is a common manifestation of atherosclerotic heart disease. It occurs whenever coronary blood flow cannot keep up with myocardial oxygen demand. This usually occurs during periods of physical or emotional stress. As myocardial work increases, so does myocardial oxygen demand. If the coronary arteries cannot supply an adequate amount of blood to the myocardium, it becomes ischemic and results in the chest pain typical of angina. Most cases of angina can be relieved by rest or nitroglycerin and are referred to as stable angina. People with more severe atherosclerotic heart disease will develop chest pain at rest, without any detectable increase in myocardial oxygen demand. This condition is called unstable angina. Finally, there is a smaller group of angina patients who suffer angina purely from spasm of the coronary arteries.

In the prehospital setting, angina pectoris, like all cases of chest pain, should be treated like myocardial infarction until proven otherwise. Always perform a primary assessment. Proceed to the secondary assessment with particular emphasis on the chest exam and the vital signs. Noninvasive monitors, such as ECG and pulse oximetry, are helpful. Always administer high-concentration oxygen unless there is a medical contraindication. Paramedics should consider the administration of nitroglycerin or a similar nitrate preparation. If this is ineffective, consider administration of a narcotic or a calcium-channel blocker. Remember, many cases of angina are precursors to a full blown myocardial infarction and warrant comprehensive care.

References

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TABLE 2 - Common Medications used in the Treatment of Angina Pectoris

<table>
<thead>
<tr>
<th>Oral Short-Acting Nitrates</th>
<th>nitroglycerin (Nitrostat)</th>
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</thead>
<tbody>
<tr>
<td>isosorbide dinitrate (Isordil)</td>
<td></td>
</tr>
<tr>
<td>Oral Long-Acting Nitrates</td>
<td>isosorbide dinitrate (Dilatrate-SR)</td>
</tr>
<tr>
<td>isosorbide mononitrate (Ismo)</td>
<td></td>
</tr>
<tr>
<td>Topical Nitrate Preparations</td>
<td>nitroglycerin ointment (Nitro-Bid, Nitro-Dur)</td>
</tr>
<tr>
<td>Calcium Channel Blockers</td>
<td>nifedipine (Procardia)</td>
</tr>
<tr>
<td>diltiazem (Cardizem)</td>
<td></td>
</tr>
<tr>
<td>verapamil (Isoptin)</td>
<td></td>
</tr>
<tr>
<td>Beta Blockers</td>
<td>Propranolol (Inderal)</td>
</tr>
<tr>
<td>Nadol (Corgard)</td>
<td></td>
</tr>
<tr>
<td>Metoprolol (Lopressor)</td>
<td></td>
</tr>
<tr>
<td>Narcotics</td>
<td>Morphine Sulfate</td>
</tr>
<tr>
<td>Anti-Platelet Agents</td>
<td>Aspirin (Ecotrin, Bayer)</td>
</tr>
</tbody>
</table>
Angina Pectoris

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

2. Angina pectoris is defined as:
   a. Any chest pain of cardiac origin
   b. A cramping 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 nitroglycerine (NTG)
   b. Anginal pain occurring at rest or without exertion
   c. Anginal pain unrelated to coronary blood flow
   d. Radiation of pain to arms or back

8. Angina occurring without an increase in myocardial oxygen demand is called:
   a. Variant angina           c. Prinzmetal’s angina
   b. Vasospastic 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           c. Non-rebreather mask
    b. Simple face mask        d. Positive-pressure demand valve

12. Nitrostat is a trade name of NTG, which is a:
    a. Beta blocker           c. Calcium channel blocker
    b. Nitrate                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. Nitrate
    d. Antidepressant

Questions 16 through 20 are ALS questions.

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           c. Within five to 10 minutes
    b. Within two 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 vasodilation

20. The drug of choice for lowering the blood pressure of a hypertensive angina patient is:
    a. Cardizem (diltiazem)   c. Procacid (nifedipine)
    b. Nitrostat (NTG)       d. Isopin (verapamil)

Answers in next issue.