Lafe and Steve, both EMTs, are working at a rural station staffing a BLS ambulance on a snowy December day. Mary, a paramedic, is staffing the ALS fly car. The BLS ambulance is dispatched, nonemergency, to an elderly female complaining of foot pain.

They arrive on scene and meet a 62-year-old female, Mrs. Berry, who is conscious, alert and oriented, sitting up on her couch. She does not appear to be in any distress, but says her foot hurts. She says she woke one hour ago and shortly thereafter experienced an acute onset of throbbing, constant nonradiating pain in her right foot that has been getting worse since. She rates the pain as a 5 on a scale of 0–10, and says nothing makes it better or worse.

While Steve talks with Mrs. Berry about her past medical history, Lafe inspects her foot. He notes the right foot is pale, with a delayed capillary refill, and cool to the touch compared to the left foot. Lafe also notes the pedal pulse on the right side is present but barely palpable, while the pedal pulse on the left side is strong. Mrs. Berry can still move her toes and feels Lafe lightly touching them.

When asked, she tells Steve she has a history of insulin-dependent diabetes, high blood pressure, an irregular heartbeat and coronary artery disease, with a stent placement two years ago. She says she also had “the same thing done in my leg, sort of, about a month ago. The doctor put a catheter in my groin and used a balloon in my artery in my leg. I stayed at the hospital overnight and came home the next day.”

“So you had a balloon angioplasty performed on your right leg?” Steve asks.

“I think that’s what the doctor called it,” Mrs. Berry replies.

Lafe inspects her medications, which include insulin, diltiazem and Plavix (clopidogrel), an antiplatelet medication used to prevent formation of blood clots. Noting the Plavix was only a sample and is now empty, Lafe asks, “Have you been taking your Plavix?” Mrs. Berry replies, “I’ve not gotten my prescription filled since those free ones ran out three weeks ago.”
Lafe reports Mrs. Berry's vital signs: a pulse of 84 beats a minute and irregular, blood pressure of 162/90 mmHg, respiratory rate of 14 per minute with good tidal volume, and pulse oximetry of 95% on room air.

Questions:
• What risk factors does the patient have for acute limb ischemia?
• What signs and symptoms are present that suggest the patient is experiencing acute limb ischemia?
• What is the appropriate prehospital management of the patient with acute limb ischemia?

Pathophysiology
Acute limb ischemia is defined as a sudden decrease in limb perfusion leading to failure to meet the oxygen and nutrient requirements of limb tissue, resulting in tissue ischemia. Ischemic tissue is characterized by hypoxia (decreased oxygen levels) and the accu-
mutation of metabolic wastes such as lactic acid. If uncorrected, it can result in rapid (less than 24 hours) tissue death and the need for amputation. Events that can lead to acute limb ischemia include an acute thrombosis at the site of an atherosclerotic plaque or bypass graft, an arterial embolism that originates in the heart or elsewhere, arterial dissections or aneurysms, and trauma. These etiologies of acute limb ischemia are worth a closer look.

**Thrombosis**—A thrombus is a blood clot that forms in a blood vessel. Thrombosis (the formation of a clot) most often occurs at the site of a blood vessel injury, but under certain conditions clots can form without injury. Occlusion of a blood vessel by a thrombus is the most common cause of acute limb ischemia, accounting for more than 80% of cases in the lower limbs and about half of cases in the upper limbs.²

In the setting of acute limb ischemia, thrombosis is most likely to occur at the site of an atherosclerotic plaque, but can also occur in arterial dissections and aneurysms and at the site of bypass grafts. Atherosclerosis is a disease of the medium- and large-size arteries. In the lower limbs, the aortoiliac, femoral and popliteal are the most commonly affected arteries. Atheroma formation has two major effects on the arterial wall. First, it weakens the arterial wall and increases the risk of injury such as plaque rupture, aneurysm and dissection. Second, as they increase in number and size, atheromas progressively take up more space in the artery wall, decreasing the lumen size and blood flow distal to the lesion. This process by itself can reduce blood flow enough to result in claudication. Claudication is pain, discomfort or tiredness in the legs or feet that is relieved by rest; it indicates the patient suffers from peripheral arterial disease. Does this sound familiar? It should, as

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**Venous Thromboembolism**

Thrombus formation can occur on the venous side of the circulation as well, and while more common it is generally less acute in consequence. Deep vein thrombosis (DVT) occurs in the lower extremities and is associated with risk factors such as smoking, oral contraception, immobility (after surgery or a long car ride or flight), pregnancy, obesity and cancer. Emboli from a DVT can lead to life-threatening pulmonary embolus (PE). See Figure 1.

Deep vein thrombosis (DVT) occurs in the lower extremities and is associated with risk factors such as smoking, oral contraception, immobility (after surgery or a long car ride or flight), pregnancy, obesity and cancer. Emboli from a DVT can lead to life-threatening pulmonary embolus (PE). See Figure 1.

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this process is the same that occurs in angina and a transient ischemic attack, two pathologies with which EMTs and paramedics are very familiar.

As atherosclerosis progresses, the arterial wall is further degraded, and the fibrous cap covering the atheroma can rupture. Plaque rupture results in the rupture of the inner layer of the arterial wall, the tunica intima, and activation of the clotting cascade and thrombus formation at the site of rupture. As a thrombus grows and blocks an artery, blood flow to the distal tissue is reduced, and ischemia can develop. Untreated, it will grow to completely obstruct the artery, leading to tissue death and loss of the limb. Again, this process is identical to what can occur in a coronary artery, resulting in acute myocardial infarction, and in a cerebral artery, resulting in stroke. In acute limb ischemia, this process occurs in the limbs.

Thrombosis can also occur at the site of a bypass graft. It is not a vessel injury, but turbulent or stagnant blood flow at the graft site that results in activation of the clotting cascade and thrombus formation. Any patient who has had a recent endovascular or surgical revascularization procedure is at risk of thrombosis. Specific procedures include placement of an artificial or natural bypass graft, stent placement and balloon angiography.

Thrombosis can also develop secondary to vascular trauma from a blunt or penetrating mechanism of injury. Blunt force trauma can create shearing forces that stretch and twist an artery, resulting in damage to the vessel wall and thrombus formation with little or no external sign of trauma.

**Arterial embolism**—Most arterial embolisms (about 85%) originate in the heart, and the majority of these (60%–70%) are the result of acute myocardial infarction. A left ventricle weakened by an AMI will experience a decrease in stroke volume and ejection fraction, the fraction of blood pumped out of the ventricle with each heartbeat. This allows blood to pool in the left ventricle, increasing the risk of thrombus formation. When the thrombus breaks free and enters the arterial system, it is called an embolus. An embolus is a thrombus (or any other foreign body) that is carried by the circulatory system to a site in the body other than its origin. A blood clot acting as an embolus is termed a thromboembolus. When an embolus lodges in and occludes a peripheral artery, blood flow to the tissues distal to the occlusion is prevented, and ischemia develops. If it’s left uncorrected, tissue death will occur.

Arterial embolisms of cardiac origin can also result from atrial fibrillation and occur in patients with prosthetic heart valves. Atrial fibrillation results in impaired movement of blood through the atria, which allows blood to pool there, increasing the risk of clot formation. Mechanical valves create turbulent blood flow that also promotes clot formation. These can also embolize and travel downstream, leading to a stroke or PE, depending on if the patient with acute arterial occlusion and limb ischemia will present with some or all of the “five Ps.”
An embolus originates in the left or right atrium. This is why patients with atrial fibrillation and mechanical valves are placed on lifelong anticoagulants such as Coumadin (warfarin).

Noncardiac arterial embolisms include thromboembolisms released from aneurysms or dissections and atheroembolisms released from atheromas. An atheroembolism is an embolus that consists of cholesterol and other debris dislodged from an atheroma.

**Aneurysm and dissection**—Both aneurysms and dissections of peripheral arteries can result in distal limb ischemia. An aneurysm is a localized dilation of a weakened vessel wall. The most common cause of aneurysm formation is severe atherosclerosis. Common sites of formation that lead to arterial emboli are the abdominal aorta and aortoiliac, femoral and popliteal arteries in the lower limbs and the subclavian artery in the upper limbs. A thrombus can form in the aneurysm, and thromboembolisms can be released from the developing thrombus. If the thrombus grows large enough, it can totally occlude the vessel.

A dissection occurs when a tear in the inner layer of the aorta (tunica intima) allows high-pressure blood to enter the arterial wall and force the layers apart, dissecting the layers along the length of the artery (see Figure 2). While aortic dissections are the most feared, they can also occur in many smaller arteries with severe consequences. As with an aneurysm, a thrombus can form in the area of the injury, and thromboembolisms can be released from the developing thrombus.

**Tissue death and limb loss**—The time limits for tissue and limb viability vary and depend on factors such as the degree of vessel occlusion and the presence of collateral circulation. Patients with chronic arterial insufficiency will develop collateral circulation over the years to augment blood vessels that cannot adequately supply tissues with arterial blood. As such, these patients have a “backup” blood supply to the affected tissues should total occlusion occur. The patient with no history of chronic arterial insufficiency, and who subsequently does not have a robust collateral circulation, is less “prepared” for an acute occlusion and limb ischemia.

**History and Clinical Exam Findings**

Ask patients presenting with signs and symptoms of acute limb ischemia about past episodes of claudication. Patients with claudication are said to have chronic arterial insufficiency and are at increased risk of acute arterial occlusion. Chronic arterial insufficiency can progress to ischemic pain with rest, and patients may report a history of foot
pain while at rest or sleeping that tends to be unrelieved with limb elevation or analgesics. Standing often relieves this pain. Patients may report that they sleep in a chair or with their legs dangling over the side of their bed in an effort to improve perfusion pressures to the distal tissues.\(^1\) Other risks include recent surgeries (especially vascular procedures), IV drug use, a history of phlebitis or pulmonary embolism, major illnesses such as cancer, and a history of coagulation abnormalities.

In addition, patients with a past medical history of conditions related to peripheral artery disease are at risk of developing acute limb ischemia. These include cardiac disease, AMI, cardiac dysrhythmias such as atrial fibrillation, transient ischemic attack, stroke and renal disease. Risk factors that increase the risk of atherosclerosis (and therefore acute limb ischemia) include high cholesterol, hypertension, smoking and diabetes.\(^3\) If acute limb ischemia is suspected, it is important to consider life-threatening causes of the emergency such as aortic abdominal aortic aneurysm (AAA), aortic dissection and AMI.

The patient with acute arterial occlusion and limb ischemia will present with some or all of the "five Ps": pain, pallor, pulselessness, parasthesias and paralysis.
Some sources list six Ps, also including polar for the cold skin that accompanies a severe occlusion.\textsuperscript{2} The presentation of these findings can vary widely, and while a patient presenting with a cold, pale, pulseless foot with reduced sensation is easy to identify as having acute limb ischemia, the patient who complains only of foot pain will be harder to identify. To think a limb-sacrificing event such as acute limb ischemia can present with an initial complaint as simple as “my foot hurts” should grab any healthcare provider’s attention!

Perform a careful assessment of the peripheral pulses and compare the pulse presence and strength between the brachial, radial, ulnar, femoral, posterior tibial and dorsalis pedis arteries.\textsuperscript{4} In addition, assess the abdominal aorta, palpating for the presence of a pulsatile mass that may indicate an abdominal aortic aneurysm as the cause of the occlusion. While abdominal examination of the very thin and very obese can be challenging due to false positives and negatives respectively, up to a 76% sensitivity has been reported for palpating pulsatile masses in larger aneurysms.\textsuperscript{5} Numerous classification systems exist to rate the presence and quality of pulses in patients with suspected arterial occlusion, but there is no consensus.\textsuperscript{6}

Describing the peripheral artery exam is standardized to facilitate communication with other healthcare providers. Most commonly a 0–4 grading scale is used, with 0 being absent, 1 diminished, 2 normal, 3 increased and 4 bounding.\textsuperscript{7} An EMT might report a patient’s left popliteal pulse was “a 1, slightly diminished compared to the right.”

Assess the skin for signs of poor perfusion. Early during an ischemic event, the signs may be as subtle as a delayed capillary refill. As ischemia progresses, the skin will become pale or even white, and possibly mottled or cyanotic. Late skin findings characteristic of occlusion include ulcer formation and necrosis.

With decreased perfusion, the affected limb will become cold, and the patient will experience pain that may be described as steady, throbbing and severe. Paresthesia, a “pins and needles”-like sensation, may be felt and should be considered a positive neurologic finding. The ability to distinguish light touch is a sign that the ischemic tissue is still viable. Two-point discrimination, the ability to detect vibration, and proprioception (the ability to sense the relative position of the affected limb) are often diminished prior to the total loss of sensation. Total loss of sensation and paralysis are ominous findings and precede impending necrosis and loss of the limb.\textsuperscript{2}

Explore any history of trauma to the affected limb. Any mechanism of injury
that produces twisting, crushing or shearing forces to an underlying artery should increase the suspicion of arterial injury and clot formation. Inspect for injuries such as abrasions, bruising, swelling, penetrations and other signs of trauma. If possible, perform a 12-lead ECG to rule out AMI as an etiology of suspected acute limb ischemia.

**Treatment**

Early recognition of acute limb ischemia and rapid transport to an emergency department are vital for increasing potential for limb salvage. A heightened index of suspicion may be required to identify subtle presentations such as mild extremity pain.

BLS care of the patient with acute limb ischemia includes placing the limb in a dependent position, administering oxygen by nasal cannula and keeping the limb warm. Placing the limb in a dependent position allows gravity to help increase perfusion pressures.

If a BLS provider carries baby aspirin as part of their protocol for acute coronary syndrome, consider contacting medical control, giving a report and determining if they would like to administer aspirin. Aspirin, with its antiplatelet properties, can inhibit thrombus formation in the patient with evolving acute limb ischemia. Paramedics should also contact medical control if aspirin is not already approved via protocol for acute limb ischemia.

As with stroke, maintenance of adequate blood pressure is important to ensure the best possible opportunity for perfusion distal to a developing arterial occlusion. Treat cardiac dysrhythmias resulting in hypotension immediately and use intravenous fluids and vasopressors as needed to maintain adequate blood pressure. Aggressively manage limb pain from ischemia with intravenous analgesics, as the pain can be severe.

**Case Conclusion**

Lafe immediately radios dispatch and requests that the paramedic, Mary, respond to the scene in the ALS fly car. They administer oxygen via nasal cannula at 2 lpm, and Steve explains to Mrs. Berry that they are concerned she may have a blood clot in her leg and want to transport her to the hospital. Mrs. Berry agrees, and the crew starts to gather her belongings, place her on the stretcher and prepare her for transport. Steve ensures her right leg is as low as the stretcher will allow and wraps her leg in a blanket to keep it warm.
The paramedic, Mary, arrives on scene, introduces herself to Mrs. Berry and asks what's going on. Steve gives Mary a quick report of their history and clinical exam findings and says, "We're thinking she may have a blood clot at the angioplasty site in her leg." "Agreed," replies Mary. "Nice pick-up, guys."

The EMTs and paramedic briefly discuss their transport options. A university-based cardiac and level 1 trauma center is a 60-minute drive away but would require bypassing a smaller community hospital. Mary decides the university hospital has the resources to best handle the patient. They place Mrs. Berry in the ambulance and set out for the university hospital after a discussion with medical control.

Mary turns up the heat in the ambulance to keep Mrs. Berry warm and places her on the cardiac monitor, noting she is in atrial fibrillation. A 12-lead ECG shows right foot pain described as throbbing, constant and progressively worse over the past hour. Her right foot is pale with delayed capillary refill, cool to the touch, and has a diminished pedal pulse.

What is the appropriate prehospital management of the patient with acute limb ischemia?

The BLS crew recognized the situation, called for ALS support and promptly started packaging the patient for transport. The seriousness of the situation, and the potential for loss of limb, was recognized by the EMTs and paramedic, and the best destination and transport modality considered. The leg was kept in as dependent a position as possible, and kept warm with a blanket and control of the ambulance temperature. The paramedic considered AMI as a potential cause of a thromboembolus and administered aspirin to help prevent any further clot formation.

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no acute ST-segment elevation changes. An 18-gauge intravenous catheter with a saline lock is placed in her left antecubital region, and Mary considers the need for pain control, asking Mrs. Berry to again rate her pain. Mrs. Berry says her pain is still a 5 on a scale of 0–10, but she’d rather not have pain control because “I don’t like taking drugs and don’t want to get addicted to painkillers. I can handle it.”

As they leave the hospital, Mary, Lafe and Steve note that Mrs. Berry is on a heparin drip to anticoagulate her, and the ED staff is preparing to transfer her to the cardiac catheterization lab for angiography and possible percutaneous transluminal angioplasty to reopen any occluded arteries.

REFERENCES

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