INTRODUCTION

The body’s cells are highly specialized and complex. Most of them are fixed: unable to move toward nutrition or away from toxic wastes. They depend on the circulatory system for constant delivery of food and fuel and constant carrying away of garbage. Suppose a person needed someone to run to the grocery store and to flush the toilets and take out the trash. How long would the person last if this service were interrupted? Massage can promote circulatory health, but it can also interrupt or interfere with this service. For a massage therapist to make informed choices about to whom and when to give massage, he or she must have a strong understanding of the interaction between bodywork and the circulatory system.
**GENERAL FUNCTION: THE CIRCULATORY SYSTEM**

The body depends on *diffusion*, the random distribution of particles throughout an environment, for the exchange of nutrients and wastes. For diffusion to happen, a medium must allow substances to move freely within small areas. What could be better than the combination of blood and lymph? People contain about 23 liters of liquid. In every milliliter of it particles are flowing this way and that, chemicals are reacting, and life is happening.

The circulatory system, through the medium of the blood, works to maintain homeostasis, which is the tendency to maintain a stable internal environment. It does this in a number of different ways:

- **Delivery of nutrients and oxygen.** The blood carries nutrients and oxygen to every cell in the body. If for some reason the blood can’t reach a specific area, cells in that area starve and die. This is the situation with many disorders, including stroke, heart attack, pulmonary embolism, renal infarction, and decubitus ulcers.

- **Removal of waste products.** While dropping off nutrients, the blood, along with lymph, picks up the waste products generated by metabolism. These include carbon dioxide and more noxious compounds that can cause problems if left to stew in the tissues. Again, if blood and lymph supply to an area is limited, the affected cells can drown in their own waste products and be damaged or even die.

- **Temperature.** Superficial blood vessels dilate when it’s hot, and they constrict when it’s cold. Furthermore, blood prevents the hot places (the heart, the liver, working muscles) from getting too hot by flushing through and distributing the heated blood throughout the body. By helping us to keep a steady temperature, the circulatory system helps to maintain a stable internal environment.

- **Clotting.** This is an often overlooked but truly miraculous function of the circulatory system without which people would quickly die. Every time a rough place develops in the endothelium of a blood vessel, a whole chain of chemical reactions results in the spinning of tiny fibers that catch cells to plug any possible gaps. Unfortunately, in certain circumstances, this reaction is sometimes more of a curse than a blessing.

- **Protection from pathogens.** Without white blood cells we would have no defense against the hordes of microorganisms that try to gain access to the body’s precious (and precarious) internal environment. For a closer look at what actually happens to those would-be invaders, see the introduction to Chapter 6.

- **Chemical balance.** The body has a very narrow margin of tolerance for variances in internal chemistry. A person can actually die if his or her blood gets even 15% too alkaline or too acidic. Happily, blood components, including red blood cells, are supplied with enzymes and other buffers specifically designed to keep pH balance within the safety zone.

**Structure and Function: The Blood**

**Red Blood Cells (Erythrocytes)**

Almost all of the blood cells, red and white alike, are made in the red bone marrow. Red blood cells are created at the command of a hormone secreted by the kidneys called *erythropoietin*. Red blood cells are constantly being produced and dying, at about 2 million per second. They comprise 98% of blood cells. Their life span is about 4 months, and during that time they do a single job: they deliver oxygen to the cells and carbon dioxide to the lungs. They are so devoted to this task that they have given up their nuclei to make more room to carry their cargo.

Red blood cells are tiny; 1 mL of blood holds about 5 million of them. They are built around an iron-based molecule called *hemoglobin*. This molecule (there are 250 million of them in each red blood cell) is extremely efficient at carrying oxygen and slightly less so at carrying carbon dioxide; most of that is dissolved in plasma. Another key quality to healthy red blood cells is their shape: they are discs that are thinner in the middle than around the edges. They are very smooth and should be flexible enough to bend and distort themselves to get through the tiniest capillaries. If for some reason they are not round, smooth, and flexible, big problems ensue.

**White Blood Cells (Leukocytes)**

Leukocytes aren’t really white; they’re more or less clear. Unlike red blood cells, which are all identical, different classes of white blood cells fight off different types of invaders in different stages of infection. Types of white blood cells include neutrophils, basophils, eosinophils, monocytes, and lymphocytes (Figure 5.1).

**Platelets (Thrombocytes)**

Thrombocytes are not whole cells at all but fragments of huge cells that are born in the red bone marrow. They are usually smooth, but when they are stimulated, they quickly become spiky and sticky. Thrombocytes travel the system looking for leaks or rough places in the blood vessels. If they find one, a series of chemical reactions causes tiny
threads of fibrin, a protein, to be woven from plasma proteins in the injured area. These fibers act as a net to catch not only passing thrombocytes but passing red blood cells as well, forming a crust on the skin or a clot (thrombus) internally. This is a good thing; it’s very important, and it’s usually not a problem because other chemicals whose job is to melt clots circulate in the blood. Under certain circumstances the clotting mechanisms work harder than the clot-destroying mechanisms. This ultimately can become life threatening.

Structure and Function: The Heart

The heart is divided by the septum into left and right halves. The right half pumps blood to the lungs (the pulmonary circuit), and the left half pumps to the rest of the body (the systemic circuit). Each half of the heart is further divided into top and bottom. The small top chambers, where blood returning from the lungs and body enters, are called the atria (the singular form is atrium from the Latin for entrance hall), and the larger bottom chambers are the ventricles (from the Latin for belly). The two-part “lub-dup” of the heartbeat is the closing of the valves that separate the chambers from each other and the ventricles from the arteries leaving the heart.

The cardiac muscle of the atria is much thinner and weaker than that of the ventricles. This is because the atrial contraction has to push blood only a few centimeters downhill into the ventricles. The cardiac muscle of the ventricles is thicker and stronger than that found in the atria because the ventricular contraction pushes blood into the circulatory system—through the pulmonary circuit to the lungs from the right ventricle, and through the systemic circuit to the rest of the body from the left ventricle. The differences in the workload of various parts of the heart have great implications for the seriousness of myocardial infarctions (heart attacks); the location of the damaged tissue determines how well the heart will function without it.

Structure and Function: Blood Vessels

The vessels leaving the heart are called arteries and arterioles; the vessels going toward the heart are called venules and veins; the vessels that connect them are called capillaries. Ideally this should be a closed system. That is, although white blood cells are free to come and go through capillary walls, the red blood cells should never be able to leave the 60,000 miles of continuous tubing that constitutes the circulatory system. If they do leak out, it’s because the system has an injury, and a blood clot should be forming.

Arteries and veins share the basic properties of most of the tubes in the body. They have an internal layer of epithelium (it’s called endothelium here because it’s on the inside); this layer is called the tunica intima, or inside coat. The middle layer (tunica media) is made of smooth muscle; and the external layer of tough, protective connective tissue is called the tunica externa, or the adventitia. This combination of tissues makes these tubes strong, pliable, and stretchy.

Capillaries are delicate variations of basic tube construction. As the arteries divide into smaller arterioles, their outer layers get thinner and thinner. Finally all that is left is one layer of simple squamous epithelium wrapped with smooth muscle cells: the capillaries. This construction is ideal for the passage of substances back and forth, be-
cause most diffusion happens readily through single-cell layers. But because capillaries lack the tougher muscle and connective tissue layers of the larger tubes, they are much more vulnerable to injury.

Blood cells leave the heart through the thick-walled arteries, crowd into arterioles, and line up one by one to squeeze through the capillaries. Once they’ve dumped their cargo of oxygen and picked up the carbon dioxide, they have more breathing room; now they’re in the venules. Again the three-ply construction design is present, but with a difference. Much of the venous system operates against gravity. Blood flows upward in the legs, the arms, and the trunk, partly by indirect pressure exerted by the heart on the arterial system but also with the help of hydrostatic pressure and muscular contraction. To help the blood move along without backing up in the system, small epithelial flaps or valves line the veins. The smooth muscle layer here is thinner and weaker than in the arteries, which have to cope with much higher pressure coming directly from the heart. Veins get wider, bigger, and stronger as they approach the heart, but they are never as strong as arteries. Fortunately, the force with which blood moves through them is never as strong either.

When blood returns from the body to the heart (the systemic circuit), it goes to the lungs to be oxygenated (Figure 5.2).

The chapter on cardiovascular conditions is the most self-referential portion of this book. Most of the conditions discussed here are caused by or are complications of (or both) other conditions in this chapter.

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**Figure 5.2.** The right side of the heart pumps blood to the lungs in the pulmonary circuit; the left side of the heart pumps blood through the rest of the body in the systemic circuit.
Circulatory System Conditions

Blood Disorders

- Anemia
- Embolism, thrombus
- Hematoma
- Hemophilia
- Leukemia
- Malaria
- Myeloma

Sickle cell disease
Thrombophlebitis, deep vein thrombosis

Vascular Disorders
- Aneurysm
- Atherosclerosis
- Hypertension

Heart Conditions
- Raynaud syndrome
- Varicose veins
- Heart attack
- Heart failure

Blood Disorders

Anemia

**Definition: What Is It?**

Anemia is the condition of having either an insufficient supply of red blood cells, an insufficient or somehow functionally impaired supply of hemoglobin, or both. In any case, anemia by itself is not a diagnosis; it’s a description. The diagnosis comes when one determines why a shortage of red blood cells or hemoglobin has developed.

**Demographics: Who Gets It?**

Anemia is a common problem, affecting about 3.4 million people in the United States. Most anemia patients are women or people with a chronic disease, such as cancer, infection, or bone marrow suppression.

**Etiology: What Happens?**

Several kinds of anemia are possible, each with different guidelines for treatment and the appropriateness of massage. Some of the most common varieties are discussed here, with descriptions of the process and how massage might positively or negatively affect them.

**Idiopathic anemias** These conditions, which have no well-understood cause, may be due to poor nutritional uptake because of how stress affects gastric juices, or to other more mysterious factors. But once other pathologies are ruled out, these anemias (which are usually comparatively mild) may respond well to massage.

**Nutritional anemias** These anemias occur because the body is missing something vital in its diet and no amount of massage, no matter how brilliantly administered, can replace it. However, most of these conditions are not negatively affected by massage. The only exception to this rule is advanced pernicious anemia.

- **Iron deficiency anemia.** Iron is at the center of the hemoglobin molecule. An insufficiency of iron in the diet leads to insufficiency of hemoglobin, and then to pathologically small or
thin red blood cells and “iron-poor blood.” Iron deficiency anemia is the most common variety of anemia around the world and in the United States. It can come about from a diet that is poor in iron, an inability to absorb iron from digestion, chronic bleeding, or lead poisoning. Women are at increased risk for iron deficiency anemia because they need about twice as much iron as men but consume fewer calories. Pregnant women have a radically increased need for iron, and about half of them in this country are anemic.

- **Folic acid deficiency anemia.** Folic acid is a nutrient found in green leafy vegetables that is critical for the formation of red blood cells. If a person doesn’t get enough, it’s impossible to produce the 2 million red blood cells per second that it takes to keep up with the cells that are dying off. Folic acid is water soluble. That means it can’t be stored for later use; a steady fresh supply is necessary. Folic acid anemia is usually related to dietary insufficiency or malabsorption, often due to alcoholism or celiac disease.

- **Pernicious anemia.** Of all of the nutritional anemias, this is the most serious because it can lead to irreversible damage to the central nervous system. Vitamin $B_{12}$ is necessary for the formation of red blood cells. Vitamin $B_{12}$ is available only from animal food sources, including eggs and dairy products. Very few people in the United States have a $B_{12}$ deficiency, yet pernicious anemia is relatively common. This is because a chemical in gastric secretions, **intrinsic factor**, is necessary for absorption of vitamin $B_{12}$. Without it a person may take in all the $B_{12}$ he or she requires, but the body has no access to it and cannot produce enough erythrocytes to keep up with its needs. Age, genetic predisposition, stomach surgery, autoimmune disease, alcoholism, tapeworms, celiac disease, and Crohn disease may all interfere with intrinsic factor activity. The only treatment for pernicious anemia is to supplement vitamin $B_{12}$ in some form that the body can absorb it (this may involve injections). This pales in comparison to the alternative, however. $B_{12}$ is also critical to the maintenance of the central nervous system. Without it, a person experiences the slow onset of paralysis, loss of proprioception, and brain damage.

- **Other nutritional deficiencies.** Anemia can be the result of shortages of several other substances, notably copper and protein. Massage does not improve this condition, and if it is very advanced (with extreme shortness of breath, fatigue, and low stamina), massage could possibly tax the already overworking heart in a dangerous way. Anemia this advanced is relatively rare, however.

**Hemorrhagic anemias** Hemorrhagic anemias are those brought about by blood loss. Usually it’s from a slow leak, but occasionally it’s from some trauma; this would be acute hemorrhagic anemia. The most common causes of hemorrhagic anemia are gastric or duodenal ulcers, chronic kidney problems leading to the loss of blood in the urine, heavy menstruation, and large wounds. Obviously, bleeding ulcers and large wounds contraindicate rigorous circulatory massage. Heavy menstruation isn’t negatively affected unless the therapist works deeply in the abdomen during flow, but it is a sign that something may be wrong, and the client should consult with her health care team to rule out some other disorder, such as endometriosis or fibroid tumors. Blood in the urine contraindicates circulatory massage until the causes can be identified and addressed.

**Hemolytic anemias** These anemias are characterized by the premature destruction of healthy red blood cells. In addition to the basic symptoms of anemia, **splenomegaly** (enlarged spleen) and jaundice may be present. Another sign of hemolytic anemia is the presence of higher than normal numbers of **reticulocytes** in the blood. These are immature red blood cells that cannot carry as much oxygen as fully developed red blood cells. High numbers of reticulocytes are released from the bone marrow when the supply of erythrocytes is getting dangerously low.
Causes of hemolytic anemia include genetic predisposition, allergic reactions to certain drugs, and infection. Two types of hemolytic anemia, sickle cell disease and malaria, are discussed later in this chapter.

Aplastic anemia In aplastic anemia bone marrow activity is sluggish or even nonexistent. The production of every kind of blood cell is slowed or suspended. When this moment’s 2 million cells report to the spleen for destruction, insufficient new red blood cell replacements are available. Likewise, no new white blood cells are manufactured, so resistance to infection is impaired. And finally the stream of thrombocytes has dried up too, making persons with aplastic anemia prone to uncontrolled bleeding. This sometimes shows as frequent bloody noses or bleeding from the gums.

Aplastic anemia can be caused by autoimmune attack against bone marrow, renal failure, folate deficiency, certain viral infections, exposure to some types of radiation, and some environmental toxins, namely benzene and some insecticides.

Myelodysplastic anemia is a closely related problem, but in this case the bone marrow makes multitudes of abnormal cells rather than being suppressed altogether. This can be a precancerous condition that indicates a risk of leukemia or myeloma.

Secondary anemias Anemia is a frequent complication of other disorders. Sometimes a direct cause-and-effect relationship is obvious, and sometimes the association is a less clear but still present. A partial list of the conditions that anemia frequently accompanies includes the following:

- **Ulcers.** Gastric, duodenal, and colonic ulcers can all bleed internally. This may not be very obvious, but it results in a steady draining of red blood cells, which can impair general oxygen uptake and energy levels. This is a type of hemorrhagic anemia, discussed earlier.

- **Kidney disease.** The capillaries inside the kidneys have a tremendous workload. Not only do they filter toxins and maintain water balance, but they do it under tremendous mechanical pressure from the renal arteries. Sometimes the kidney capillaries are damaged and leak red blood cells into the urine. Again, it’s a leak rather than a gusher, but just as that dripping faucet drives up the water bill, a leaking kidney slowly but surely drains away viable red blood cells. Furthermore, the kidneys secrete the hormone that stimulates bone marrow to produce red blood cells (erythropoietin). When the kidneys are not functioning well, erythropoietin levels drop and red blood cell production goes down.

- **Hepatitis.** The liver contributes vital proteins to the blood, and it is responsible, with the spleen, for breaking down and recycling the iron from dead erythrocytes. If liver function is disrupted for any reason, the quality and amount of hemoglobin available to new red blood cells may decline.

- **Acute infectious disease.** Anemia is sometimes an indicator that the body is under attack. It is a frequent follower of pneumonia, tuberculosis, or other infection. Infectious disorders often cause iron to be used elsewhere in the body, reducing the amount of hemoglobin available to new red blood cells. Anemia in these cases usually clears up spontaneously once the primary condition has been resolved.

- **Leukemia, myeloma, lymphoma.** In these conditions masses of nonfunctional white blood cells are produced in bone marrow or in lymphatic tissues. These white blood cells essentially crowd out functional red blood cells.

**Signs and Symptoms**

No matter what the cause of anemia is, some signs and symptoms are consistent. These include the following:
Pallor. Pallor is present because of a reduced number of red blood cells, or a reduced amount of hemoglobin to carry the oxygen that gives the red blood cells their color. Pallor is visible in the skin and in mucous membranes, gums, and nail beds. In dark-skinned people, pallor shows as an ashy-gray appearance to the skin.

Dyspnea. This is shortness of breath. Dyspnea is a symptom of anemia because with less oxygen-carrying capacity, a person has to breathe harder and more often just to keep up. Another sign of anemia is a higher than normal respiratory rate.

Fatigue. Often this is the first noticeable symptom of anemia. Less oxygen is available to go around, so muscles wear out sooner and stamina is nonexistent. Unfortunately, a host of other conditions may make a person feel worn out, so this is rarely enough to go on for a diagnosis.

Rapid heartbeat. Another term for this is tachycardia. It allows oxygen-poor blood to travel faster through the body.

Intolerance to cold. Oxygen is needed for muscle contraction and for heat production (shivering). Someone who is in short supply of oxygen runs out of steam in a hurry.

**Massage?**

The appropriateness of massage for clients with anemia varies according to the cause. Massage has been seen to increase red blood cell count at least temporarily, but this is probably due to the action of pushing sluggish red blood cells back into circulation; these effects don’t tend to last very long. Massage certainly doesn’t provide missing nutrients, but the resulting parasympathetic response may improve uptake in the digestive system.

Anemia contraindicates massage when it accompanies other disorders that may be negatively affected. Specifically, if pernicious anemia has resulted in a decrease in sensation or if the anemia is due to acute infection, circulatory massage is inappropriate.

Very advanced anemia of any kind is fairly rare. However, if the blood is especially low in hemoglobin or oxygen, the heart has to work extremely hard to pump the diminished supply through the body. For this reason, very advanced anemia of any kind contraindicates vigorous circulatory massage.

<table>
<thead>
<tr>
<th>MODALITY</th>
<th>RECOMMENDATIONS FOR ANEMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Contraindicated when severe; otherwise supportive.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Supportive.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Indicated; work liver, spleen, thyroid, lymphatic points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Indicated, especially in conjunction with Chinese herbs. Treat LV, SP, BL, K meridians and extensions systemically.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Indicated with caution for underlying conditions; can help reinforce parasympathetic state.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Supportive.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.
Embolism, Thrombus

**Definition: What Are They?**

An *embolism* is a traveling clot or collection of debris, and *thrombus* is a lodged clot (Figure 5.3). Thrombi that form on the venous side of the systemic circuit are discussed in the section of this chapter on thrombophlebitis and deep vein thrombosis. Emboli and thrombi that form on the arterial side of the systemic circuit are part and parcel of the whole interrelated cardiovascular disease picture. Having them can cause heart trouble, and heart trouble can cause them.

**Etiology: What Happens?**

Blood leaves the left ventricle of the heart via the aorta and goes to its destination through smaller and smaller vessels: arteries, arterioles, and finally capillaries. Nutrient–waste exchange happens at the capillary level, and then the vessels get bigger and bigger as they lead toward the right atrium: from venules to veins to the vena cava. The same telescoping action happens in the pulmonary circuit: blood leaves the right ventricle through the huge pulmonary artery, and vessels going into the lungs get smaller and smaller. Oxygen and carbon dioxide are exchanged in capillaries in the lungs, and the freshly oxygenated blood goes back toward the heart through venules, veins, and finally the large pulmonary vein.

Platelets constantly flow through the circulatory system looking for rough spots, which indicate injury. If they find any disruption in the walls of the blood vessels, they quickly develop spikes and stick to that spot. Then they release the chemicals that cause blood proteins to weave fibers, making a net to catch other blood cells, and a clot is formed. Clots can also form in places where blood doesn’t flow quickly; clotting factors accumulate enough to thicken the fluid even without an injury to initiate the action. This kind of stasis thrombosis happens frequently enough that it now has a name: “coach class syndrome”.

The construction of the circulatory system is such that clots cannot pass through capillaries; they form and remain either on the arterial side of the systemic circuit or on the venous side. The damage that ensues depends on the origin of the clot, its size, and where it finally gets stuck.

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**Embolism, Thrombus in Brief**

**Pronunciation:** EM-bo-lizm, THROM-bus

**What are they?**

Thrombi are stationary clots; emboli are clots that travel through the circulatory system. Emboli are usually composed of blood but may also be fragments of plaque, fat globules, air bubbles, tumors, or bone chips.

**How are they recognized?**

If venous thrombi break loose, or *embolize*, they can only land in the lungs, causing pulmonary embolism. Symptoms include shortness of breath, chest pain, and hemoptysis, or coughing up sputum that is streaked with blood. Many venous thrombi and pulmonary emboli are silent: they create no symptoms until the damage has been done.

Clots that form on the arterial side of the system can lodge anywhere their artery carries them. Some of the most common (and dangerous) locations for arterial clots to land are in the coronary arteries (heart attack), the brain (transient ischemic attack or stroke), and the kidneys (renal infarction).

**Is massage indicated or contraindicated?**

Any disorder associated with the potential for lodged or traveling clots contraindicates circulatory massage.

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**Figure 5.3.** A. thrombus is a lodged clot; an embolus is a moving clot.
**Pulmonary Embolism**

The lungs are the one and only destination for clots or debris anywhere on the venous side of the systemic circuit, unless the heart has a structural defect called a patent foramen ovale, which allows communication between the left and right sides. When something, often a sudden movement after prolonged immobility, knocks any debris in any vein loose, it jets toward the heart in increasingly big tubes. It goes through the right atrium and ventricle, enters the pulmonary artery, and ends up in the lungs (Figure 5.4). Most pulmonary emboli are fragments that lodge in multiple places in both lungs simultaneously. The extent of damage can vary from a temporary loss of a tiny bit of lung function to total circulatory collapse when suddenly little or no blood returns to the heart from the lungs.

Every year about 650,000 people in the United States have a pulmonary embolism. Many of them resolve spontaneously, but about 200,000 people die of pulmonary embolism every year. This condition is usually related to thrombophlebitis or deep vein thrombosis as a complication of immobility, surgery, or trauma.

**Risk factors for pulmonary embolism** Risk factors for pulmonary embolism include other types of cardiovascular disease, recent trauma, extended bed rest, and any kind of surgery, although surgeries for femur and hip fractures or any gynecological problem have a particularly high embolism rate. Pulmonary embolism is the third leading cause of death in hospitals.
Women who are pregnant or who have recently given birth are at high risk for pulmonary embolism, as the weight of the uterus on the femoral vessels can cause blood to pool and thicken in the legs, and hormonal changes associated with pregnancy and childbirth can also thicken the blood.

Other risk factors include being overweight, smoking, and taking hormones for birth control or as hormone replacement therapy.

**Signs and symptoms of pulmonary embolism** Some studies estimate that up to 80% of pulmonary embolism cases show no discernible signs or symptoms until after lung damage has occurred. Classic symptoms of pulmonary embolism include difficulty breathing, chest pains, and hemoptysis (coughing with bloody sputum), but many people don’t show this pattern. Other symptoms that may or may not be present are shortness of breath, lightheadedness, fainting, dizziness, rapid heartbeat, and sweating. Chest pain and chest wall tenderness along with back, shoulder or abdominal pain are also possible.

**Complications of pulmonary embolism** A person with a history of pulmonary embolism is at increased risk for having another episode. Further, if a significant amount of lung function is lost, pulmonary hypertension and right-sided heart failure may develop as the heart tries to push blood through the damaged, restricted pulmonary circuit.

**Treatment of pulmonary embolism** Treatment of non–life-threatening pulmonary embolism is usually an aggressive course of thrombolytics and anticoagulants and attention to the cause of the deep vein thrombosis. If it is a major embolism, surgery may be required to remove it from the lung.

**Prevention of pulmonary embolism** Pulmonary embolism is the leading cause of death from surgical complication. Because this condition is very difficult to diagnose early, emphasis has shifted from treatment to prophylaxis, or prevention. Preventive measures include identifying patients who are at particularly high risk for developing clots, administering low-dose anticoagulants starting shortly before surgery, elevation of the legs, external compression on the legs, and early walking following surgery.

**Arterial Embolism**

This is one of the many complications of atherosclerosis. Emboli in the arteries can also be a complication of bacterial infection, *atrial fibrillation* (uncoordinated flutter of the atrium instead of a rhythmic, strong contraction), or rheumatic heart disease, which can produce clots inside the heart. Emboli can be made of some foreign object in the bloodstream such as a bit of plaque, a bone chip, an air bubble, or a knot of cancer cells.

The main difference between arterial and venous emboli is the final resting place. If the clot is on the arterial side of the systemic system, it could wind up virtually anywhere except the lungs. Therefore, the damage it causes is very different. The brain, the heart, the kidneys, and the legs are statistically the most common sites for arterial emboli to lodge.

**Signs and symptoms of arterial embolism** Symptoms of emboli in organs may be nonexistent until the affected tissue has significant loss of function. This is particularly dangerous in the kidneys, where many tiny clots can come to rest somewhere in the renal arteries, leading to progressive renal failure. If clots lodge in the legs, however, symptoms are sharp, tingling pain followed by numbness, weakness, coldness, and blueness. Left untreated, this tissue may become necrotic in a matter of hours; immediate medical attention is necessary.

If the embolus lodges in the brain and the symptoms are short-lived, it is called a transient ischemic attack. A more serious brain embolism can cause an ischemic stroke. And finally, if it lodges in a coronary artery, it’s called a heart attack.
Treatment

If a person has a tendency to form clots easily, anticoagulant medications may be prescribed to circumvent the complications of heart attack or stroke.

Massage?

The tendency to form thrombi or emboli systemically contraindicates rigorous circulatory massage. Lodged thrombi are a medical emergency. If a person knows an embolism is present in either the venous or arterial side of the system, he or she should be under treatment. Clients who take anticoagulant medications require extra care from massage therapists. These clients have two significant issues: they have a history of excessive clotting, and they are taking medication that limits their ability to clot, which means they bruise and bleed easily. Both of these contraindicate the use of techniques that push a lot of fluid or challenge the tissue to the point that bruising might occur.

<table>
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<td>Polarity</td>
<td>S: Indicated; can help reinforce a parasympathetic state. R/D: Systemically contraindicated.</td>
</tr>
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<td>Shiatsu</td>
<td>Systemically contraindicated.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Systemically contraindicated; later, be aware that anticoagulant use requires gentle touch.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Systemically contraindicated.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

Hematoma

Definition: What Is It?

The term \textit{hematoma} refers to extensive bleeding and pooling of blood in hollow areas. This can happen in several locations. The leakage of blood from damaged superficial capillaries is called a bruise, or \textit{ecchymosis}. The familiar injury that happens when a person hammers his thumb or catches her finger in the door is called a subungual hematoma. Inside the cranium hematoma can be a result of traumatic brain injury or a ruptured aneurysm. Blood can also accumulate between muscle sheaths as a result of trauma or a complication of hemophilia.

Signs and Symptoms

Bruises are reddish or purplish (or black and blue) in the acute stage. They fade to yellowish green in the subacute stage, when the macrophages have migrated in to clean up the debris. The processes for cleaning up capillary leaks deeper than the skin, for instance in a gastrocnemius that has been kicked, are invisible but otherwise identical.

Larger bleeds can involve extensive inflammation along with discoloration. They can occur when an arteriole inside a muscle or between deep muscle layers is injured. It pours blood into an area until local pressure closes it off. A large acute hematoma feels like hot half-congealed gelatin under the muscle layers, and it is quite painful to the touch. They happen most often in large fleshy areas such as the calf, thigh, or buttocks.
**Treatment**

Small bruises require no medical intervention, although they respond well to alternating hot and cold applications, which stimulate macrophages to come into the area and flush away wastes from the tissue damage.

Bruises under fingernails or toenails require no attention if they discolor less than 25% of the nail area. Larger subungual hematomas are recommended for medical attention to drain the bleeding and to check for damage to the nail bed that may cause permanent disfiguration to the nail.

Large intermuscular bleeds can be complicated. If they’re caught relatively early, they can be aspirated or drained, but if they’re left too long, they are likely to congeal from the concentration of clotting factors in the blood. At that point only time, hydrotherapy, and gentle movement will help to break up the pooled blood into a form that the body can reabsorb.

Hematomas can complicate into more serious problems. A bleed on the anterior lower leg can cause acute compartment syndrome, which can lead to nerve damage and other problems. Another complication of hematoma occurs when the leaked blood calcifies into a mass that looks like a bone chip embedded in soft tissue; this is myositis ossificans.

**Massage?**

Hematomas and bruises contraindicate local massage in the acute stage because of pain and the possibility of disturbing blood clots. In the subacute stages (at least 2 days after the injury occurs) the local blood vessels are generally sealed off. Gentle massage may be appropriate around the edges of the lesions, always within the tolerance of the client. This can be coupled with alternating hot and cold applications to speed the turnover of circulation and the reabsorption of leaked blood.

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### Hematoma in Brief

**Pronunciation:** he-mah-TOE-mah

**What is it?**
A hematoma is a deep bruise (leakage of blood) between muscle sheaths or in other soft tissues.

**How is it recognized?**
Superficial hematomas are simple bruises. Deep bleeds may not be visible, but they are painful, and if extensive bleeding has occurred, a characteristic gel-like feel develops in the affected tissue.

**Is massage indicated or contraindicated?**
Massage is at least locally contraindicated for acute hematomas because of the possibility of blood clots and pain. In the subacute stage (at least 2 days later), when the surrounding blood vessels have been sealed shut and the body is breaking down and reabsorbing the debris, gentle massage around the perimeter of the area and hydrotherapy can be helpful.

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### Modality Recommendations for Hematoma

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Locally contraindicated; otherwise supportive.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Indicated in subacute stage.</td>
</tr>
<tr>
<td>Polarity</td>
<td>S: Indicated. R/D: Locally contraindicated; light work above/below site to client comfort when subacute.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Locally contraindicated; otherwise supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Locally contraindicated; work heart, solar plexus, and endocrine points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Locally contraindicated; indicated elsewhere. Treat LV and SP systemically. Avoid pressure at the local site.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Locally contraindicated; in subacute stage use local caution and stay within pain tolerance.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Locally contraindicated; otherwise supportive.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.
Hemophilia

**Definition: What Is It?**

Hemophilia is a genetic disorder characterized by the absence of some plasma proteins that are crucial in the clot-forming process. It is actually a collection of genetic disorders, each one of them affecting a different clotting factor in the blood. Their presentations are all much the same, however, so the only people who need to know which type of hemophilia is present are the patients themselves and the people who care for them.

**Demographics: Who Gets It?**

In this country hemophilia affects about 18,000 men. The incidence of the most common variety is about 1 in 5,000 males, and the disease appears equally among races and socioeconomic status. It is identified in about 400 boys every year. The disease is carried on the X chromosome, so males with hemophilia pass the chromosome along to their daughters, who become carriers. These female carriers pass the mutation on to about half of their sons. About one-third of hemophilia cases occur as a spontaneous mutation, with no family history of the disorder.

It is possible for a girl to have hemophilia, but she would need positive X chromosomes from both her father and mother, and this is very rare.

**Etiology: What Happens?**

Thrombocytes constantly cruise around the circulatory system looking for signs of damage. When they encounter any kind of rough spot inside a blood vessel, they stick to that spot and secrete a series of chemicals that cause plasma proteins to weave nets of fibers called fibrinogen. These nets catch passing platelets and red blood cells, forming a plug to limit loss of blood through the damaged vessel. The plasma proteins that weave the fibrinogen have been identified as 12 distinct factors. Hemophilia occurs when a genetic mutation causes a deficiency in one or more of these clotting factors.

The most common variety of hemophilia is hemophilia A, which accounts for about 80% of all cases. It is characterized by a deficiency in clotting factor VIII. Hemophilia B, also called Christmas disease, is characterized by insufficient levels of factor IX. Hemophilia B accounts for about 15% of hemophilia cases. Other clotting factor deficiencies are possible, but they are much rarer than hemophilia A or B.

A person who is deficient in clotting factor VIII or IX has difficulty in forming a solid, long-lasting clot. Hemophiliacs don’t bleed faster than average, but they do bleed longer. Hemophilia is rated as mild, moderate, or severe, depending on what percentage of normal levels of clotting factors the patient has. Severe hemophiliacs, who account for 60% of hemophilia patients, have lower than 1% of normal levels of clotting factors.

**Signs and Symptoms**

Hemophilia first appears at birth, when the umbilical cord bleeds excessively, or in early childhood, as babies begin to engage in physical activities that involve minor bangs and bumps. These toddlers are subject to excessive bruising and bleeding with very mild irritation, and small scrapes and lesions tend to bleed for a long time.

As the person matures, he finds that he is prone to subcutaneous bleeding (bruising), intramuscular hemorrhaging (hematomas),
nosebleeds (epistaxis), blood in the urine (hematuria), and severe joint pain brought about by bleeding in joint cavities. Bleeding episodes may follow minor trauma, or they may occur spontaneously.

**Complications**

The leading cause of death for children with hemophilia is intracranial bleeding: even minor head trauma can cause major bleeding episodes in and around the brain.

Bleeding into joint cavities is a significant problem for people with hemophilia. Unless clotting factors are administered very soon after an episode, the blood inside the joint may collect and lead to an inflammatory response that damages cartilage and articulating bones. This condition is called **hemophilic arthritis**, and it occurs most often at the ankles, knees, and elbows.

Bleeding into muscles can cause pain and numbness as nerve endings are compressed. If the pressure is not quickly resolved, the muscle may develop a contracture, with a permanent loss of range of motion. The muscles most at risk for hematomas are in the calf, thigh, upper arm, and forearm. The psoas is also at risk for deep bleeds and stiffness.

Infected blood products used to be another major worry for people with hemophilia. Before screening methods were used consistently, contracting HIV or hepatitis was a significant risk for hemophiliacs. Blood and plasma screening now filters out most viruses, but some pathogens can still get through. It is recommended that hemophiliacs and other people who regularly use blood products be vaccinated for hepatitis A and B.

The development of genetically engineered clotting factor has improved the quality of life for many people with hemophilia, but a small number of patients develop resistance to this product. They may have to use other blood products until the hypersensitivity reaction subsides.

**Treatment**

Treatment protocols for people with hemophilia have taken gigantic leaps forward in the past 30 years. Before 1965 the only treatment available was transfusion of whole blood, a time-consuming and inefficient means of replacing clotting factors for someone with an internal hemorrhage. Consequently, most hemophiliacs were in wheelchairs by their teens, and their life expectancy was much shorter than the norm. In 1965 techniques were developed to isolate the specific missing clotting factors, allowing a much more efficient treatment.

More recently, clotting factors have been manufactured in a form that can be stored at home and self-administered. These recombinant factors radically increase a hemophiliac’s independence and ability to work and travel.

Synthetic or blood-based clotting factors can be administered after an injury takes place to limit bleeding into a joint or between muscles. They can also be taken prophylactically, before a surgical or dental procedure, for instance, to limit anticipated bleeding.

People with mild hemophilia A can also treat themselves with an injected or inhaled form of the hormone desmopressin, which stimulates production of extra clotting factor in response to an injury.

In addition to managing bleeding, people with hemophilia are counseled to exercise (although they must obviously avoid contact sports) and to keep their weight under control; both

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**SIDEBAR 5.1: VON WILLEBRAND DISEASE: AN EQUAL OPPORTUNITY MUTATION**

The kinds of hemophilia that most of us are familiar with involve X-linked genes that affect clotting factors VII or XI. But these types of hemophilia affect a relatively small proportion of the population. It turns out that another type of genetic mutation causes different clotting factor deficiency, and it is **not** on the X chromosome, which means men and women equally can be affected.

The condition is called von Willebrand disease, and it is a deficiency or poor quality of von Willebrand factor, one of the last clotting factors to participate in the cascade of chemical reactions that cause the spinning of fibrinogen to make blood clots. Von Willebrand disease is the most common genetically linked bleeding disorder in the world.

Von Willebrand disease is usually very mild. Signs and symptoms include frequent bloody noses, bleeding from the gums, and in women, heavy menstrual flow. A person may never be diagnosed until he or she has a tooth pulled or goes through childbirth or undergoes some other experience that can lead to prolonged bleeding.

If a client knows he or she has von Willebrand disease, no special precautions need to be taken for massage other than to avoid bruises and other lesions, but these are precautions for all clients.
of these can limit the risk of arthritis and muscle contracture. Physical therapy may be suggested to help rehabilitate injured joints or muscles.

**Massage?**

Severe hemophilia contraindicates rigorous mechanical circulatory massage. Clients and therapists should consult with the client’s medical team about receiving bodywork, and the therapist should use pressure that the client experiences as part of everyday activity.

Energetic and noncirculatory techniques are appropriate and welcome and often have powerful effect on stress and pain sensation.

### Modality Recommendations for Hemophilia

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Contraindicated for deep work.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Supportive.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Indicated for extremely light work: focus on heart, solar plexus, endocrine points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Supportive with gentle techniques; no tapotement or deep pressure.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Systemically contraindicated for deep work.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

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**Leukemia**

**Definition: What Is It?**

Leukemia, or white blood, is a cancer of white blood cells produced in bone marrow. Some overlap has been established between types of leukemia that affect lymphoid cells and lymphoma: cancer associated with lymph nodes. Lymphoma is discussed in Chapter 6.

Dozens of types of leukemia have been identified, but this article focuses on the four most common classifications. These types of leukemia have much in common, but each has some unique features that are examined under individual headings.

**Demographics: Who Gets It?**

Leukemia in various forms is diagnosed in about 35,000 Americans every year, and it causes about 22,000 deaths. Although this disease is the leading cause of death from cancer in children, it is much more common in adults. About 208,000 leukemia patients are living in the United States today.

The overall survival rate has tripled in the past four decades to about 48%. Children with this disease often have a better prognosis than mature adults. For more details about leukemia statistics, see Sidebar 5.2.
**SIDEBAR 5.2: TYPES OF LEUKEMIA: INDIVIDUAL DISCUSSIONS**

The four major classes of leukemia have some characteristics and statistical information that distinguish them from each other. Massage therapists working with clients who have leukemia should become familiar with some of the basic features of their client’s condition.

**Acute Myelogenous Leukemia**

*Basic Information*

Acute myelogenous leukemia (AML) is a rapidly progressive cancer of the myeloid group of white blood cells. The immature cancer cells are called blast cells, leading to the synonym acute myeloblastic leukemia. Other synonyms are acute myelocytic leukemia and acute granulocytic leukemia. The genetic damage that causes AML has been associated with certain specific environmental factors. High doses of radiation, chemotherapy for other types of cancer, and exposure to benzene all increase the risk of contracting AML in later years.

Because this variety of leukemia is aggressive and rapidly progressive, it is important to start treatment as soon as possible. The steps in administering chemotherapy are outlined in the general treatment section.

**Statistics for AML**

About 12,000 new cases of AML are diagnosed every year in the United States, and about 26,000 people are living with this disease. Although it can occur in young adults, this variety of leukemia is associated with age. Most cases occur in people over 50 years old; the average age at diagnosis is 65.

**Chronic Myelogenous Leukemia**

*Basic Information*

Chronic myelogenous leukemia (CML) is a slow-growing cancer of myeloid cells in the bone marrow. It is also called chronic granulocytic leukemia and chronic myeloid leukemia. It involves the myeloid cells: granulocytes (which include neutrophils, eosinophils, and basophils) and monocytes.

CML is characterized by production of nonfunctioning white blood cells, but these are produced relatively slowly and they are released into the bloodstream when they are fully mature. These faulty cells can interfere with and slow down normal immune system activity, but they do not usually bring it to a halt.

CML patients often have an enlarged spleen and pain, as the cancerous cells congregate in this organ. Night sweats, unexpected weight loss, and a decreasing tolerance for warm temperatures are other signs and symptoms common to CML patients.

CML occasionally changes its pattern and becomes more aggressive. This is called an accelerated phase of the disease, and it is treated as if it were AML.

**Statistics for CML**

About 5,000 new cases of CML are diagnosed in the United States every year. Most patients are adults, but a small number of children contract CML. The survival rate for untreated CML is poor: about 15 to 25% of patients survive for 5 years. When the cancer is treated with chemotherapy and/or bone marrow stem cell transplants, the survival rate is much better.

**Acute Lymphocytic Leukemia**

*Basic Information*

Acute lymphocytic leukemia (ALL) is a rapidly progressive, aggressive form of leukemia characterized by DNA mutations in stem cells that produce bone marrow lymphocytes. Synonyms for ALL include acute lymphoid leukemia and acute lymphoblastic leukemia.

ALL is an aggressive cancer of bone marrow lymphocytes. The proliferation of cancerous cells is so overwhelming that all other bone marrow activity is suppressed and immune system function is crippled. Cancerous lymphocytes are released into the blood before they are fully mature. These lymphocytes may gather in lymph nodes, or they may gain access to the central nervous system, where they accumulate and can cause severe headaches, vomiting, and seizures.

Causative agents and risk factors for ALL are not clear, but this disease has been associated with high doses of radiation and exposure to toxic substances during gestation or in early childhood.

**Statistics for ALL**

ALL affects young children more often than adults—until adults become mature. The incidence of this disease peaks among 4-year-olds and then subsides among older children and young adults. It becomes more common among people over 50.

ALL is most common in developed countries and among higher socioeconomic groups. About 4,000 new cases of ALL are diagnosed in the United States each year; about half of them are in children. The remission rate in children is 80%. Among adults the 5-year survival rate is 25% to 35%.

**Chronic Lymphocytic Leukemia**

*Basic Information*

Chronic lymphocytic leukemia (CLL) is a slow-growing version of lymphocytic leukemia. Although it can involve T cells or natural killer cells, most cases involve B-cell malignancies.

Sometimes CLL is so stable and so nonthreatening that no treatment is recommended. If numbers of functioning blood cells drop to dangerous levels, chemotherapy may be recommended, along with radiation to shrink enlarged lymph nodes or other tissues.

**Statistics for CLL**

CLL is found primarily in elderly people. Up to 95% of cases are among patients over 50, and the incidence rises sharply with age. About 10,000 cases of CLL are diagnosed every year.

CLL has the best prognosis of all the types of leukemia: 71% of patients make it to 5 years of remission.
Etiology: What Happens?
Healthy leukocytes come in several shapes and sizes, each with a specific role to play in the effort to keep us free from potential invaders.

Most white blood cells are produced in bone marrow. They develop from nonspecific blood stem cells into whatever type of cell is needed at the moment. Two types of stem cells, myeloid and lymphoid, manufacture white blood cells in bone marrow. Leukocytes are classified as myeloid or lymphocytic, depending on their origin. Leukemia occurs when a mutation in the DNA of one or more stem cells in the bone marrow causes the production of multitudes of nonfunctioning leukocytes. These cells can crowd out the functioning cells in the bone marrow and in the blood. Leukemia can be aggressive and quickly progressive, releasing immature cells into the circulatory system (acute), or it can be slowly progressive, leading to the release of mature but nonfunctioning cells (chronic). In either case, the mutated cells cannot function as part of the immune system, and they live far longer than normal cells, leading to dangerous accumulations of nonfunctioning cells. The four most common varieties of leukemia are as follows:

• Acute myelogenous leukemia (AML) is aggressive cancer of the myeloid cells.
• Chronic myelogenous leukemia (CML) is slowly progressive cancer of the myeloid cells.
• Acute lymphocytic leukemia (ALL) is aggressive cancer of the lymphocytes.
• Chronic lymphocytic leukemia (CLL) is slowly progressive cancer of the lymphocytes.

Each of these is discussed in more detail in Sidebar 5.2.

Unlike many types of cancer, leukemia spreads through the blood rather than the lymph. It can cause tumors in the lymph nodes (although not as readily as lymphoma), and it also affects the liver, spleen, testes, skin, and central nervous system.

The genetic mutations seen with leukemia are usually acquired rather than inherited. Exposure to environmental toxins and radiation are cited most often as contributing factors. Electromagnetic fields are being studied as possible risk factors for leukemia, but results so far are inconclusive. Some forms of leukemia are linked to a congenital problem: Down syndrome and some other genetic anomalies can increase the risk for these diseases.

Untreated leukemia results in death from excessive bleeding or infection.

Signs and Symptoms
Signs and symptoms of all types of leukemia point to bone marrow dysfunction. When the marrow is sabotaged by a genetic mutation that causes overproduction of nonfunctioning cells, functioning cells are produced in smaller numbers if at all. A leading sign of leukemia is fatigue and low stamina due to anemia: low numbers of red blood cells are available to deliver oxygen to working tissues. A person with leukemia bruises easily or may bruise with no particular trauma. Small cuts and abrasions may bleed for long periods. Unusual bleeding or bruising comes about because platelet production is suppressed (thrombocytopenia) and the person has limited ability to make blood clots. Finally, a person with leukemia is susceptible to chronic infections—these can be skin infections such as hangnails or pimples, or they can be respiratory infections such as colds and flu. They can even be chronic urinary tract infections. Whatever the infectious agent is, the person with leukemia has very limited resources to fight it off, because functioning white blood cells are in short supply.

Diagnosis
All types of leukemia are diagnosed by a combination of blood tests, bone marrow biopsies, and examination of cerebrospinal fluid for signs of metastasis. It is important to find out exactly which kinds of cells have been affected and whether the cancer is an acute or chronic va-
riety. Furthermore, each type of leukemia has subtypes that respond differently to various treatment options.

Recent breakthroughs in the study of cell form and lineage have revealed that the lymphocytic leukemias that affect T cells, B cells, and natural killer cells are essentially the same as associated forms of lymphoma; the only difference is in whether the targeted cells are stationary or circulating. This has allowed for more accurate diagnoses and more effective treatments of these blood cancers.

**Treatment**

Leukemia treatment depends to a certain extent on what types of cells have been affected, how aggressive the disease is, and what kinds of treatments the patient has already had. Treatment usually begins with chemotherapy: administration of chemicals that are highly toxic to any cells that reproduce rapidly. Exactly which chemotherapy drugs are used depends on the type of cancer that is present. A course of chemotherapy for leukemia usually takes place in four stages:

- **Induction.** Chemotherapeutic drugs are introduced into the system, usually intravenously. The goal is to suppress cancer cells and to begin a period of remission.
- **Consolidation.** Once the process has begun, chemotherapy continues in high doses for several weeks or months in an effort to establish and maintain remission.
- **Central nervous system prophylaxis.** Some types of leukemia attack the central nervous system, but normal chemotherapeutic agents are blocked from these areas by the blood-brain barrier. To overcome this, chemotherapeutic drugs may be introduced directly into the central nervous system. Radiation therapy may be employed for this as well.
- **Maintenance therapy.** Continuing repeated tests and treatments as necessary are applied to keep the cancer in remission.

If a person doesn’t respond well to chemotherapy or if the cancer keeps recurring (refractory leukemia), it is necessary to explore other treatment options. This can include adding radiation therapy or surgery, especially if cancerous cells have aggressively invaded any particular organ or location.

The treatment options for leukemia (and other types of cancer) are broadening every day. The use of bone marrow transplants with preserved marrow of the patient (*autogenic transplants*) or closely matched donors (*allogenic transplants*) is useful for some leukemia cases, but the incidence of complications is high. It is also possible to harvest stem cells from the bloodstream, bone marrow, or umbilical cords of healthy people and to transplant these “cellular blanks” into leukemia patients so that they can make healthy, functioning blood cells.

New treatments for leukemia also include the use of interferon or other medication to slow the production of cancerous cells and the use of manufactured antibodies that are designed to identify and destroy cancer cells. One new drug has been used successfully to suppress cellular replication for a previously untreatable form of leukemia; more drugs in this category are in development.

The treatment for leukemia, especially the acute varieties, can seem to take as hard a toll on the body as the disease itself. Chemotherapy introduces substances whose function is to kill off any rapidly reproducing cell. Unfortunately, this doesn’t just mean cancer cells; it also means epithelial cells in the skin and the digestive tract and, ironically, healthy blood cells.

The side effects of chemotherapy on epithelial tissues include development of ulcers in the mouth, nausea and diarrhea from gastrointestinal irritation, and hair loss as the epithelial cells in follicles are killed. One of the difficulties with digestive system disturbances is that if the patient can’t eat well, the whole system becomes weaker and less able to cope with the stresses of both the disease and its treatment.
Chemotherapy also exacerbates symptoms of leukemia, as red and white blood cells and platelets are suppressed. Consequently, chemotherapy patients often have anemia, clotting problems, and low resistance to infection—all signs of leukemia itself.

**Massage?**

Leukemia is a type of cancer that can spread through the circulatory system. It involves seriously impaired immunity and a tendency to bleed easily. Some types of bodywork, especially those that enlist the healing energies of the client rather than trying to impose outside forces on blood flow or tissue manipulation, are helpful and supportive for a person going through a difficult, stressful, and often painful process. It is important in this situation to work as part of a well-informed health care team, so that the possibility of secondary infection or other complications can be carefully avoided.

The benefits massage can bring to cancer patients (reinforcing a parasympathetic state, improving immune system function, reducing pain perception) can be enjoyed with a minimum of risk if simple precautions are taken. For more guidelines about massage in the context of cancer and cancer treatments, see Chapter 12.

<table>
<thead>
<tr>
<th><strong>Modality Recommendations for Leukemia</strong></th>
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</thead>
<tbody>
<tr>
<td><strong>Deep Tissue Massage</strong></td>
</tr>
<tr>
<td><strong>Lymphatic Drainage</strong></td>
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<td><strong>Polarity</strong></td>
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<td><strong>Swedish Massage</strong></td>
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<tr>
<td><strong>Trigger Point Therapy</strong></td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

**Malaria**

**Definition: What Is It?**

Malaria is a vector-borne infection of blood cells. The causative agent is one of four single-celled parasites from the *Plasmodium* genus of protozoa, and the vector is the bite of an infected female mosquito from the *Anopheles* species. This species is common all over the world, including in the United States, where it is also the vector for West Nile encephalitis.

**Demographics: Who Gets It?**

Globally, as many as 500 million people are infected with malaria each year. It causes 1.5 to 3 million deaths each year; the average age at death is 4 years. Most malaria cases (up to 90%)
are in sub-Saharan Africa, but it is also common in India, the Middle East, Southeast Asia, Oceania, and Central and South America.

Malaria used to be common in the United States until massive eradication efforts essentially eliminated it by the early 1950s. Nonetheless, between 1,000 and 2,000 cases of malaria are diagnosed here each year. Most of them are in recent travelers to areas where the infection is endemic, but some cases are native to this continent.

**Etiology: What Happens?**

The protozoa, that cause malaria have a complex life cycle, requiring time to mature in both a mosquito and a human host. Four types of *Plasmodium* protozoa have been identified in this disease process: *P. ovale*, *P. vivax*, *P. malariae*, and *P. falciparum*.

When a human is bitten by an infected mosquito (only females take blood meals), an immature form of the parasite is introduced into the bloodstream. It travels to the liver, where it grows for 6 to 9 days. At that time it reenters the bloodstream, where it begins to invade healthy red blood cells.

The protozoa feed on hemoglobin and replicate inside the red blood cells. Finally the infected cells rupture, releasing toxic wastes and more protozoa. Newly released protozoa invade more erythrocytes. When an uninfected mosquito bites this person, immature protozoa enter the insect to begin the cycle again.

The less virulent forms of malaria protozoa are not life threatening, but *P. falciparum* can invade and damage tissues in the central nervous system, liver, and kidneys, leading to potentially fatal complications through liver failure, renal failure, and coma.

While malaria is usually spread through the bite of infected mosquitoes, it can also be transmitted through the placenta from mother to child or through blood transfusions or the use of shared needles.

**Signs and Symptoms**

The signs and symptoms of malaria include extreme fluctuations between fever and chills (these reflect whether red blood cells are being invaded or rupturing), in cycles that may recur over several days.

When enough red blood cells have been invaded, anemia develops. Malaria is a type of hemolytic anemia, in which red blood cells are destroyed faster than they can be replaced (Sidebar 5.3). Jaundice is another complication. It is related to the rapid destruction of erythrocytes that allows bilirubin to accumulate in the skin and mucous membranes.

A typical infection with the less virulent parasites lasts about 2 weeks and then subsides, but some parasites may remain in the liver to launch a new episode months or years later.

**Diagnosis**

In areas where malaria is endemic, diagnosis is based on the presence of alternating fever and chills. In the United States.
many diagnoses are missed because the disease is so uncommon that practitioners often don’t think to look for it; this leads to unnecessary illness and death each year.

The parasites can be identified in blood smears if technicians are skilled and know what to look for. Other tests to identify malaria-related antigens in the blood are in development. Some are available, particularly for use in developing countries where this disease is rampant, but they vary in accuracy and practicality because of costs.

**Treatment**

The treatment options for malaria are limited, and some parasites are developing an alarming resistance to chloroquine, the most common and cost-effective treatment option. This tendency, along with reports of increasing numbers of *P. falciparum* infections, is making the control of malaria an international health priority.

Most malaria patients can be treated successfully if they have full access to all the drugs necessary and if they take the drugs according to prescription. In developing countries, where malaria is most dangerous in young children, it costs about $19 to treat each case, and each household earns approximately $68 dollars per year.\(^7\)

**Prevention**

Travelers to areas where malaria is common can take prophylactic medication that can help to prevent new malaria infections. Basic defense against mosquitoes is also recommended. This includes wearing long sleeves and pants; possibly using clothing treated with permethrin, which repels mosquitoes; and using treated netting over bedding.

A vaccine for malaria is in development but not yet available. Genome studies of both the parasites and the mosquitoes that carry them have revealed many potential ways to interrupt protozoan reproduction.

**Massage?**

A client who is having alternating bouts of high fever and bone-shaking chills is not a good candidate for massage. This person obviously needs to consult a doctor, especially after recent travel to an area where malaria is common. For people whose infection has been treated and who are not at risk for permanent kidney damage, bodywork is a safe and appropriate choice.

### Modality Recommendations for Malaria

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Contraindicated while acute; otherwise supportive.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Contraindicated while acute; otherwise supportive.</td>
</tr>
<tr>
<td>Polarity</td>
<td>S/R/D: Contraindicated while acute; otherwise supportive.</td>
</tr>
<tr>
<td>PNF/MET/Sretching</td>
<td>Contraindicated while acute; otherwise supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Indicated in remission; work lymphatic and pituitary points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Contraindicated when acute. Indicated, especially with Chinese herbs, in remission to strengthen the immune system and blood.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Contraindicated while acute, otherwise supportive when all signs and symptoms have resolved.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Contraindicated while acute; otherwise supportive.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.
Myeloma

Definition: What Is It?

Myeloma (literally, “marrow tumor”) is a blood cancer involving maturing B cells that are found in bone marrow.

Demographics: Who Gets It?

Myeloma is diagnosed about 16,700 times each year in this country. About 58,300 people are living with this disease. Men slightly outnumber women. It is especially prevalent among African Americans, particularly older black men. It is usually diagnosed around age 70; it is rare in people under 45. Myeloma causes about 11,000 deaths per year.

Etiology: What Happens?

The B cells that eventually produce antibodies to help fight off pathogenic invaders spend some time maturing in bone marrow. During this phase it is possible for some cells to undergo a mutation that causes them to do several things: they proliferate into tumors; they secrete cytokines that stimulate osteoclast activity; and they produce faulty antibodies.

Under normal circumstances the bone marrow holds only a few maturing B cells. When the cells are ready, they migrate to lymph tissue, where they operate as normal plasma cells, producing functioning antibodies. But when immature B cells become cancerous in the bone marrow, they rapidly proliferate into tumors. These usually grow in bone tissue (typically the spine, pelvis, ribs, or skull), but occasionally tumors form elsewhere: these are called plastocytomas. Tumors inside bone marrow can interfere with normal blood cell production, leading to the signs and symptoms of other blood cancers: anemia, poor clotting, and reduced resistance to infection. But myeloma cells secrete cytokines that signal osteoclasts to dismantle bone tissue. This makes more room for the growing tumor, and it leads to pathologic thinning or even holes in bone tissue.

Healthy B cells produce many types of functioning antibodies (also called immunoglobulins) that work in different ways to neutralize pathogens. Myeloma cells, on the other hand, produce massive amounts of nonfunctioning antibody molecules, called monoclonal immunoglobulins: monoclonal because they are all alike, and immunoglobulins because they are technically antibodies even though they don’t offer any protective properties. Another name for monoclonal immunoglobulins is M-proteins.

Normal antibodies are Y-shaped proteins, and they are too big to pass through the kidneys into the urinary system. M-proteins have branches that sometimes break off during formation. These fragments, called Bence Jones proteins, are small enough to pass through the filters in the kidney to be excreted in the urine. The good news about this is that myeloma can be detected and to a certain extent tracked through urinalysis. The bad news is that if the disease is rapidly progressive, the kidneys can sustain extensive damage and even fail altogether.

Three types of myeloma have been identified:

- **Multiple myeloma** produces tumors at several sites. It is the most common form, accounting for 90% of myeloma diagnoses.
- **Solitary myeloma** is development of a single myeloma tumor in the bone marrow.
- **Extramedullary plastocytoma** is growth of myeloma tumors outside of bone tissue. These growths can develop in the skin, muscle, lungs, or other areas.
**Signs and Symptoms**

Myeloma can be silent in early stages; it is sometimes found during a routine medical examination. The earliest symptom for most people is pain or even fractures that occur as tumors corrode bone tissue. Other signs include anemia, frequent and persistent infections, kidney problems related to the excretion of Bence Jones proteins, calcium in the blood, and the risk of **amyloidosis**: the accumulation of inflammatory proteins on organs such as the heart or lungs, where they can do significant damage.

**Compare and Contrast 5.1**

**Blood Cancers**

Blood cancers are a confusing collection of conditions because they seem to overlap each other. Indeed, it has been found that some forms of leukemia are essentially the same as some forms of lymphoma, because they affect the same cells. The only difference is that when the cells circulate, it is called leukemia, and when cells are fixed inside lymph nodes, the disease is called lymphoma.

Overall, about 785,000 people in the United States are living with some kind of blood cancer. About 118,000 new diagnoses are made each year (about one every 5 minutes); and about 54,000 deaths from blood cancers occur each year in this country (about one every 10 minutes). The death rates for all types of blood cancers are decreasing, although as always, children have a better survival rates than older adults.  

<table>
<thead>
<tr>
<th>FEATURES</th>
<th>LEUKEMIA</th>
<th>MYELOMA</th>
<th>LYMPHOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cells affected</td>
<td>Any white blood cell: myeloid (monocytes, neutrophils, basophils, eosinophils) or lymphoid (T cells, B cells, natural killer cells)</td>
<td>Nearly mature B cells in bone marrow only</td>
<td>B cells, T cells, natural killer cells in lymph nodes or spleen</td>
</tr>
<tr>
<td>Earliest signs and symptoms</td>
<td>Anemia, thrombocytopenia, poor immune function</td>
<td>Bone pain from corroding tumors in marrow</td>
<td>Painless enlargement of lymph nodes, especially at jaw, axilla, groin</td>
</tr>
</tbody>
</table>

**Diagnosis and Staging**

Several tests can give accurate information about myeloma and the extent of its progression. Urinalysis can show the presence of Bence Jones proteins; a blood test may show M-proteins circulating in the blood; and a bone marrow aspiration or biopsy can show the presence of abnormal cells and tumors. Bone imaging tests (radiography and magnetic resonance imaging) can reveal how much bone has been damaged and where tumors are growing.

Myeloma is rated as stages I to III depending on the extent of myeloma cells and how much bone and/or kidney damage has accrued.

**Treatment**

Myeloma is often not responsive to treatment. If it is slow-growing, and especially if the patient is elderly and in poor health, a period of watchful waiting is recommended to delay difficult procedures as long as possible. A combination of chemotherapy and bone marrow stem cell transplantation is usually suggested, but even with these intrusive interventions the 5-year survival rate is only 33%.
**Massage?**

The guidelines for massage and myeloma are like those for any other blood cancer. The client is facing difficult challenges, and bodywork should support rather than test stability. That said, gentle massage may be recommended to ease the bone pain that affects many of these patients; this must be done with great care to avoid the risk of fractures.

<table>
<thead>
<tr>
<th>MODALITY RECOMMENDATIONS FOR MYELOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Deep Tissue Massage</strong> Supportive for pain management; focus on low back and hips.</td>
</tr>
<tr>
<td><strong>Lymphatic Drainage</strong> Supportive, especially during chemotherapy.</td>
</tr>
<tr>
<td><strong>Polarity</strong> S: Indicated, can support immune system. R/D: Supportive with light touch within client comfort.</td>
</tr>
<tr>
<td><strong>PNF/MET/Stretching</strong> Supportive.</td>
</tr>
<tr>
<td><strong>Reflexology</strong> Indicated; work lymphatic and pituitary points.</td>
</tr>
<tr>
<td><strong>Shiatsu</strong> Indicated to strengthen the immune system. Treat K, TH, SP. Add fire meridians for blood and water meridians for bone/marrow/brain.</td>
</tr>
<tr>
<td><strong>Swedish Massage</strong> Supportive; stay within clientís activity limitations; be cautious of bone weakness.</td>
</tr>
<tr>
<td><strong>Trigger Point Therapy</strong> Supportive, with caution for bone fragility.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

**Sickle Cell Disease**

**Definition: What Is It?**

Sickle cell disease is an autosomal recessive genetic condition that results in production of abnormal hemoglobin, the protein that carries oxygen in red blood cells.

**Demographics: Who Gets It?**

The sickle cell gene is most common in specific populations. Blacks, Hispanics, and people from Italy, Greece, Turkey, and the Middle East are most likely to be carriers. Roughly 2 million people in the United States have the sickle cell trait, and about 72,000 people have sickle cell disease. About 8,000 people are born with sickle cell disease each year, and about 500 people die of complications related to this condition.8

**Etiology: What Happens?**

The gene for sickle cell disease is recessive; this means if a person has only one copy of the gene, he or she has the sickle cell *trait* but not sickle cell disease. If two people who have the sickle cell trait have children together, each child has a 25% chance of inheriting a copy of the gene from each parent. This is the only way sickle cell disease is spread.

Being positive for the sickle cell trait carries no health consequences for the carrier and in fact may be beneficial if that person

**Sickle Cell Disease in Brief**

**What is it?**

Sickle cell disease is a disorder in which the gene that controls hemoglobin production is faulty. The result is short-lived, misshapen red blood cells that cannot pass through tiny blood vessels.

**What does it look like?**

The primary sign of sickle cell disease is the pain that occurs when abnormal erythrocytes block blood vessels. This can lead to organ damage, bone pain, kidney infarction, stroke, lung problems, and blindness. Other signs include a high risk of infection because the spleen is typically disabled and anemia with jaundice because the abnormal red blood cells die off faster than they can be replaced.

**Is massage indicated or contraindicated?**

Sickle cell disease patients often live in chronic pain, even when they are not in a sickle cell crisis. Gentle massage that works for stress relief and circulatory improvement and that is within the activities of daily living of the client is appropriate and helpful when the client is stable, but any rigorous circulatory modalities should be avoided during a sickle cell crisis.
lives in an area where malaria is endemic—interestingly, those areas also happen to be the places where sickle cell genes are most common (Sidebar 5.3). But having two copies of the sickle cell gene means that hemoglobin production is abnormal and red blood cells adopt a characteristic sickle shape (Figure 5.5). This prevents erythrocytes from squeezing through the smallest blood vessels and shortens their lifespan from about 4 months to about 10 days.

The most common form of sickle cell disease comes from inheriting two copies of the sickle gene: this is called the SS form. Other forms come from different genetic anomalies: the inheritance of one S gene and one C gene (for another abnormal form of hemoglobin) leads to SC sickle cell disease. A final form is called S-beta thalassemia sickle cell disease, referring to yet another type of genetic fault.

**Signs and Symptoms**

Having dysfunctional hemoglobin and brittle, fragile red blood cells produces many consequences in the body. The direct symptoms of sickle cell disease include fatigue, shortness of breath, and pallor related to inadequate oxygen-carrying capacity. Jaundice may develop as red blood cells die and bilirubin accumulates in the liver and backs up into the bloodstream.

**Complications**

Sickle cell disease can lead to many serious and potentially life-shortening complications:

- **Sickle cell crises.** An *infarction* is an area of tissue that dies because it is deprived of oxygen. A sickle cell crisis occurs when an infarction damages tissue. One example is *hand-foot syndrome*. This is often the first indicator of sickle cell disease in a young child. The hands and feet are vulnerable to sickle cell crises, and they swell and become extremely painful.

- **Organ damage.** The spleen, as a collection site for dead and damaged red blood cells, is often lost early in the disease process. This leaves the patient vulnerable to serious infections, as the spleen is also a key player in immune system function. Other organs that are frequently damaged include the liver, kidneys, and brain: even young children are vulnerable to ischemic strokes, which can lead to serious learning problems in a growing child.

- **Infection.** As mentioned earlier, the loss of spleen function makes a sickle cell disease patient vulnerable to serious and even life-threatening infections. Pneumonia is the leading cause of death among children with sickle cell disease.

- **Gallstones.** Accumulated bilirubin in the liver can concentrate into crystals that build up in the gallbladder.

*Figure 5.5. Sickle cell disease.*
• **Vision loss.** The accumulation of fragile red blood cells in the arterioles that supply the retina can lead to blurred vision, hemorrhage, and even blindness.

• **Acute chest syndrome.** Damaged cells accumulate in the lungs, leading to inflammation and pneumonia-like symptoms. This puts excessive strain on the right side of the heart and can lead to pulmonary hypertension and right-sided heart failure.

• **Others.** Other complications of sickle cell disease include delayed growth in children; chronic ulcerations of the skin, usually at the lower legs; and **priapism**, a painful and long-lasting erection of the penis that occurs because the vessels that would allow blood to flow out are blocked with damaged red blood cells.

**Treatment**

Sickle cell disease is treated by trying to limit the severity and frequency of sickle cell crises. Mild episodes can be treated at home with over-the-counter pain medications and hot packs, but more severe attacks are often treated in the hospital with intravenous opioid drugs. One cancer drug, hydroxyurea, has been found to limit the frequency and severity of sickle cell crises in adults, but it carries many serious side effects and is not approved for use in children.

The leading cause of death in children with sickle cell disease is pneumonia. This risk is managed with doses of prophylactic antibiotics until age 5, along with careful immunizations for flu and other possible infections.

The life expectancy of a person with sickle cell disease has increased with better treatment options; today a person with this condition can expect to live well into his or her 40s or later.

No cure exists for sickle cell disease, which is an inherited genetic anomaly. However, new discoveries in gene therapy, stem cell transplants, and bone marrow transplants hold some promise for the future of this disease.

**Massage?**

Sickle cell disease is a painful, difficult problem. It involves poor circulation and the risk of organ damage. Clients who have this condition are counseled not to exercise vigorously and to avoid any activities that may trigger a sickle cell crisis.

For these reasons, vigorous circulatory massage is not appropriate for sickle cell disease patients. Energetic and reflexive work, however, may be extremely supportive and helpful. Research into gentle massage as a pain management technique is ongoing, and many specialists recommend warm packs and gentle stroking for people undergoing a difficult time.

### MODALITY RECOMMENDATIONS FOR SICKLE CELL DISEASE

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Systemically contraindicated.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Polarity</td>
<td>S: Indicated; can support immune system. R/D: Supportive with light work within client comfort.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Indicated; work endocrine, heart, lung points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Supportive, especially with K, SP, H, TH meridians and extensions.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Contraindicated when symptoms are present; otherwise stay within activity limitations.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Systemically contraindicated; very light work may be possible.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.
Thrombophlebitis, Deep Vein Thrombosis

Definition: What Is It?

Thrombophlebitis and deep vein thrombosis refer to veins that have become obstructed with blood clots. These clots can form anywhere in the venous system, but they develop most often in the calves, thighs, and pelvis. Thrombophlebitis is clots in superficial leg veins (lesser and greater saphenous), while deep vein thrombosis is much the same problem in deeper leg veins, specifically the popliteal, femoral, and iliac veins.

Demographics: Who Gets It?

The statistics on thrombophlebitis and deep vein thrombosis are difficult to gather because these conditions frequently go unrecognized and untreated. It is safe to say, however, that deep vein thrombosis is a leading cause of mortality in this country. It may happen up to 2 million times per year and is diagnosed in about 600,000 people, leading to about 200,000 deaths. It is estimated that up to 5% of the population will have deep vein thrombosis at some point, although not all cases will be recognized.

Etiology: What Happens?

These conditions are major concerns for well-trained massage practitioners. They involve thrombi, stationary clots in the venous system, where, if they break loose, nothing stops them from traveling up the vena cava, through the right atrium and ventricle, and into the pulmonary artery, causing pulmonary embolism.

The clotting mechanism is a normal part of homeostasis. We form blood clots in specific circumstances, but we also melt them with our own endogenous anticoagulants. Sometimes we form clots faster than we can melt them, and this is where we can run into trouble. In the mid 1800s a pioneer in pathology, Rudolf Virchow, first outlined three key factors in clot formation. The Virchow triad—injury to endothelium, hypercoagulability, and venous stasis, or slowed blood flow—is used today to describe the formation of blood clots in veins.

The triggers for thrombophlebitis and deep vein thrombosis can be many and varied. Any circumstance that involves part of the Virchow triad increases the chances of developing this problem. Here are a few of the most common precipitators of thrombophlebitis or deep vein thrombosis:

- **Physical trauma** is a predisposing factor; being kicked or hit in the leg can damage the delicate venous tissue, which is then prone to clot formation. Any fracture of bones in the leg can also increase risk (Case History 5.1).
- **Varicose veins** are another risk factor, since they too involve damaged tissue and the risk of clot formation. Fortunately, the clots that form in superficial veins tend to embolize, or break loose, much less frequently than those in deeper veins. However, many people with varicose veins also have deep vein thrombosis.
- **Local infection** can cause an inflammatory reaction leading to clot formation. These infections are often related to surgical procedures involving catheters.
- **Reduced circulation** from physical restriction, such as too-tight socks or leg braces, can cause the clotting factors in the blood to accumulate in amounts that cause coagulation without damage to a vessel wall.
• **Immobility**, often linked to sitting for long periods, can contribute to deep vein thrombosis. This phenomenon has given rise to a layman’s term for this condition: “coach class syndrome”. Some experts suggest that up to 50% of cases of deep vein thrombosis are related to recent air travel.3

• **Pregnancy and childbirth** can increase the risk of blood clotting. The weight of the fetus on femoral vessels slows blood return, and hormonal changes can cause the blood to thicken and become more viscous.

• **Certain types of cancer** can lead to thrombophlebitis or deep vein thrombosis, either because of changes in the blood or because of irritation at the site of a catheter.

• **Surgery** is another major risk for deep vein thrombosis. In fact, thrombosis and subsequent pulmonary embolism are the leading cause of death following orthopedic surgery, especially for knee and hip replacements. Heart and any kind of pelvic surgery also hold high risks of thrombosis.

• **High-estrogen birth control pills or hormone replacement therapy** can increase the risk of developing blood clots.

• **Other factors** that increase the risk for deep vein thrombosis include cigarette smoking, hypertension and other cardiovascular diseases, paralysis, and some genetic conditions that lead to excessive coagulation in the blood.

Most blood clots causing deep vein thrombosis or thrombophlebitis form in the lower legs, but they can develop elsewhere with surgery or other trauma. Sudden movement or change in position is often the factor that causes part of a clot to detach and travel to the lungs. Another alarming fact is that a patient who is immobile because of some leg injury is almost as likely to throw a clot from the unjured leg as from the injured side. This is because lack of walking can thicken the blood systemically even where no damage to blood vessels has occurred.

One further twist on where clots travel occurs when a person has a defect in the cardiac septum. Some sources suggest that up to 25% of the adult population has a small hole in the wall between the left and right sides of the heart, usually at the atrium. This condition is called patent foramen ovale.3 If clots from damaged veins that travel to the right side of the heart cross into the left side, they can go on through arteries to the brain as a stroke, to the cardiac muscle as a heart attack, or anywhere else as an arterial embolism.

**Signs and Symptoms**

Thrombophlebitis can show the major signs of inflammation: pain, heat, redness, and swelling. Sometimes itchiness, a hard cord where the vein is affected, and edema with discoloration distal to the area are present (Figure 5.6). Thrombophlebitis that has become a chronic problem may result in poor blood flow to the skin, leading to flaking, discoloration, and skin ulcers. If it is caused by a local infection, fever and general illness may also be present.

Deep vein thrombosis is considered the more dangerous of these two conditions because the clots in deeper veins can be big enough to do serious damage in the lungs and because clots in superficial veins usually melt under the influence of the body’s own anticoagulants before they have a chance to break off and do any damage.

If deep vein thrombosis shows any signs (and it often doesn’t), it may show more swelling and edema than thrombophlebitis, because the clot inhibits more blood flow back to the heart. The backup forces extra fluid out of the capillaries and into the interstitial spaces, thus adding general edema to any swelling of the vein. The capillary exchange may become so sluggish that the edema pits, or leaves a dimple wherever it’s touched. Pitting edema is a red flag for massage therapists. It is an indication that this person’s circulation is absolutely not capable of dealing with the internal changes brought about by massage.

It is important to point out that while the risks of pulmonary embolism from a clot in a superficial vein are minimal, many patients start with a superficial clot and have it intrude into deeper vessels with little or no sign of deep vein thrombosis.10
Diagnosis

Thrombophlebitis and deep vein thrombosis can be diagnosed in various ways, each with inherent benefits and disadvantages. Ultrasound is a fast and noninvasive technique, but it tends to yield a lot of false positives, leading to unnecessary prescriptions of anticoagulants, which can lead to risks of uncontrolled bleeding. Venography—injecting the blood vessel with dye and watching how it moves through the system—can be more accurate, but it is slow and the injection itself can damage delicate tissue. MRI is fast, noninvasive, and accurate, but it is also expensive and not available at all medical facilities.

Treatment

The treatment for both thrombophlebitis and deep vein thrombosis is anticoagulants. These drugs can make a person prone to heavy bleeding, however.

A bedridden patient may be given pneumatic compression to reduce the risk of thrombophlebitis or deep vein thrombosis. A machine mimics the pumping action of exercise by inflating and deflating a tubular balloon around the affected leg. Support hose to prevent the accumulation of postoperative edema are also recommended.

People with thrombophlebitis in superficial veins are at much less risk for embolism than those with deep vein thrombosis. Self-care measures such as hot packs, analgesics, and gentle exercise may be recommended to resolve episodes of vein inflammation.

High-risk patients may have a filter implanted in the vena cava to prevent clots from reaching the lungs.

Thrombophlebitis and deep vein thrombosis may do permanent damage to leg veins, including destruction of valves that assist with blood return to the heart. These patients are at risk for chronic venous insufficiency, which can include permanent edema, skin discoloration or ulcers, and very slow healing in the affected area.
A client who is at high risk for throwing a blood clot and developing pulmonary embolism is obviously not a candidate for circulatory or vigorous massage. The trick is that although thrombophlebitis may show obvious signs, it may not, or the signs may be indistinct. A client may come complaining of an ache deep in her calf that she really wants worked out. This is a reasonable request—except that the massage therapist may be working out a blood clot that will land the client in the hospital with the other 600,000 pulmonary embolism cases this year. This is one of the rare situations in which it is wisest to avoid the area and to refer the client emphatically and immediately to a primary health care provider.
Aortic Aneurysm

**Definition: What Is It?**

An aneurysm is a permanent bulge in the wall of a blood vessel or the heart. They happen most often at the aorta (aortic aneurysm) and in the brain (cerebral aneurysm). Cerebral aneurysms are discussed in the section on stroke in Chapter 4.

The damage may be brought about by any combination of injury, genetically weak smooth muscle tissue, high blood pressure, atherosclerosis, and compromised connective tissue. If an aortic aneurysm ruptures, the person can bleed to death in a very short time.

**Demographics: Who Gets It?**

Most aortic aneurysm patients are men over 60 years old. By some estimates, about 5% of men over 60 will have an aortic aneurysm at some time. About 15,000 aneurysm patients die every year.

**Etiology: What Happens?**

The three-ply construction of the arteries includes the endothelial inside layer, the smooth muscle middle layer, and the tough connective tissue outer layer. Blood pressure in the aorta, the largest artery, is very high. If the walls of the aorta lose their elasticity, they can bulge wide with blood. This bulge is an aneurysm. As the aneurysm grows, the walls stretch and weaken, increasing the risk of rupture and death.

Aneurysms happen most often in the thoracic or abdominal aorta and at the base of the brain. Aneurysms sometimes develop...
in more distal vessels, but those cases are generally much less serious because the blood pressure drops with distance from the heart.

An occasional complication of a major heart attack is an aneurysm in the left ventricle of the heart itself. The damage to myocardium reduces elasticity to the point that chronic pressure causes the whole wall of the ventricle to bulge. This is discussed further in the article on heart failure.

Several factors can contribute to the chances of developing an aneurysm:

- **Compromised smooth muscle.** Atherosclerotic plaques invade and weaken aortal muscle. Aortic aneurysms are a serious and common complication of atherosclerosis and high blood pressure.
- **Smoking.** The damage incurred to endothelium by carbon monoxide from cigarette smoke and a rise in blood pressure from nicotine makes smoking a leading risk factor for aortic aneurysm.
- **Congenitally weak arterial wall muscle.** Sometimes the tissue simply isn’t strong enough to put up with normal blood pressure, and with no warning an aneurysm can rupture. Inherited connective tissue diseases such as Marfan syndrome and Ehlers-Danlos syndrome can contribute to this kind of event.
- **Inflammation.** A few diseases, such as polyarteritis nodosa and bacterial endocarditis, can cause inflammation and weakening of the arterial tissue.
- **Untreated syphilis.** This can lead to damage in the aorta, sometimes decades after the initial infection.
- **Trauma.** Mechanical trauma, such as a car accident in which a person is injured by a steering wheel, may sometimes damage the outer layers of the aorta while leaving the inner one intact. This results in the characteristic bulging and stretching of the most delicate arterial tissue.

### Types of aneurysm
Aneurysms come in a variety of shapes and sizes, some of which are particular to where the lesion occurs (Figure 5.7).

- **Saccular aneurysm.** These usually occur with thoracic or abdominal aortic aneurysms. The aortal wall bulges like a rounded sack, which throbs and pushes against neighboring organs and other structures.
- **Fusiform aneurysm.** This is a common type of aortic aneurysm; in this case the bulge is less round and more tubular, as if the aorta were widened like a sausage for a few inches.
- **Berry aneurysm.** These small aneurysms are usually in the brain (Figure 5.8).
- **Dissecting aneurysm.** Also called false aneurysm, this is the least common and most painful type of aortic damage. The blood pressure actually splits the layers of the aorta.
between the *tunica intima* (innermost layer) and the *tunica media* (muscular layer). In some cases this type of bulge can seal itself off when the blood trapped inside the split coagulates and solidifies. It is possible to have a dissecting aorta *without* an aneurysm.

**Signs and Symptoms**

Aneurysms can be difficult to identify by symptoms because they often aren’t painful until they become a medical emergency. With aortic aneurysms the swelling might create some warning signals; this usually happens when the bulge is pressing on something else or interfering with another organ’s functioning. Thoracic aneurysms sometimes cause difficulty with swallowing (*dysphagia*), chest pain, hoarseness, and coughing that is not relieved with medication, because the protrusion presses on and irritates the larynx. Abdominal aneurysms sometimes show as a throbbing lump near the umbilicus, loss of appetite, weight loss, reduced urine output, and if it’s pushing against the spine, severe backache.

**Diagnosis**

Physical examinations often show signs of aneurysm; the turbulent movement of blood through the wide area of the aorta makes a specific sound called a *bruit* in stethoscopes. Large aneurysms in thin people can be palpated as a pulsating mass. Otherwise aneurysms are diagnosed by ultrasound, computed tomography (CT), and MRI; about three-quarters of the time, this happens when a patient undergoes tests for some other disorder and the aneurysm is found by accident.

**Complications**

For the rare aneurysms that are *not* in the aorta or the brain, no serious complications may develop unless the aneurysm gets large enough to impede blood flow, which can lead to gangrene. But the more typical aneurysm at the very least presses against its neighbors, which can be uncomfortable and can even interfere with function. If blood pools in an aneurysm for any length of time, clots may form and enter the bloodstream again. And of course, a rupture leads to hemorrhaging in the best case and shock followed by collapse of the circulatory system in the worst case. A ruptured cerebral aneurysm is fatal about 50% of the time; ruptured aortic aneurysms are fatal much more often than that.

**Treatment**

Aneurysms *don’t* spontaneously retreat, because the pressure that causes them never lets up. They must be repaired, either with open surgery or with endovascular surgery. Open surgery
involves clamping off the artery above and below the lesion and attaching either a replacement graft or a Dacron substitute to the two ends. This is usually successful, but it has to be done before a rupture.

Endovascular surgery involves inserting a catheter through the femoral artery and threading it up to the aorta to insert a patch or stent at the aneurysm site. This is a much less invasive procedure with a lower risk of surgical complications.

Some aneurysms don’t require immediate intervention. Normal aortic size is about 2 cm; a dangerously distended aneurysm is about 5 to 6 cm. Many doctors recommend checking the growth of small aneurysms by ultrasound every 6 months until the benefits of intervention outweigh the risks.

**Massage?**

Any condition involving damaged blood vessels requires extreme caution for circulatory massage. *Massage changes the internal environment.* It dilates some blood vessels and constricts others. It reroutes circulation, mechanically through compression and friction on the skin, via the parasympathetic nervous system, and by changing hormonal balance (reducing adrenaline), which shifts blood from the skeletal muscles (a sympathetic state) to the internal organs (a parasympathetic state). A client who can’t tolerate having the internal environment shifted in terms of blood vessel dilation, chemical distribution, and autonomic state isn’t a good candidate for circulatory massage.

If a client has been diagnosed with a stable aortic aneurysm and wants to receive massage, the therapist should choose modalities that aim to lower blood pressure and support that parasympathetic state without putting undue mechanical force on the circulatory system.

### Modality Recommendations for Aneurysm

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Contraindicated, especially for deep work in abdomen.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Polarity</td>
<td>S: Indicated.</td>
</tr>
<tr>
<td></td>
<td>R/D: Supportive with light work within client comfort.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Contraindicated unless working as part of health care team.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Contraindicated unless stable or repaired; then use caution and stay within activity limitations. Avoid deep centripetal work.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Supportive with light work only.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

### Atherosclerosis

**Definition: What Is It?**

Arteriosclerosis is hardening of the arteries from any cause. Atherosclerosis is a subtype of arteriosclerosis. It is a condition in which deposits of cholesterol and other substances infiltrate and weaken layers of large and medium-sized blood vessels, particularly the aorta and coronary arteries. It is compounded by the facts that damage to these blood vessels can cause them to spasm and that blood clots form at the site of these deposits. These features contribute to
occlusion of the diameter, or lumen, of the arteries (Figure 5.9) and to the risk of forming and releasing blood clots on the arterial side of the systemic circuit.

Coronary artery disease is atherosclerosis specifically in the coronary arteries that supply the heart muscle (Figure 5.10).

Demographics: Who Gets It?
Random samplings of arteries taken from autopsies of people who died of something other than heart disease reveal that the incidence of atherosclerosis is very high in the United States, although it doesn’t always become symptomatic. In some cultures, generally where dietary diseases are related to famine rather than to excess, atherosclerosis is all but unknown.

Certain populations carry a particularly high possibility of developing atherosclerosis. This is discussed in detail in the discussion of risk factors later in this article. For more information on the statistics on heart disease, see Sidebar 5.4.

Etiology: What Happens?
Development of atherosclerosis is a complex process that is not yet completely understood. It is clear that this multifactorial process varies slightly according to gender, age, race, diet, and other factors. At this point the most widely accepted idea of how atherosclerosis develops includes the following steps, although they may vary from one person to another:

1. **Endothelial damage.** The inside layer of arteries, also called the tunica intima, is made of delicate epithelial tissue and subject to a lot of abuse. A variety of things may hurl the first insult at the tunica intima: constant hypertension in the aorta and arteries surrounding the heart; carbon monoxide from cigarette smoke; high levels of oxidized low-density lipoproteins (LDL) and triglycerides; even a high level of iron in the blood may produce oxygen free radicals that begin endothelial erosion. This occurs most readily at branches or sharp curves in the arteries, where blood flow suddenly changes direction of force.

2. **Monocytes arrive.** These small white blood cells are attracted to any site of damage in the body. The monocytes infiltrate the epithelial layer and turn into macrophages, or big eaters.

3. **Macrophages take up LDL.** The reasons for this are unclear. For a quick overview, LDLs are the “bad guys” of the cholesterol world. Actually, they have an important job, which is to escort usable cholesterol to the cells in the body. But when those cells don’t need any more cholesterol, they stop accepting it. This leaves those LDLs with nowhere to go. They are taken up by the white blood cells in the tunica intima, which are then called foam cells. This is the beginning of the development of fatty streaks that characterize atherosclerosis.
4. **Foam cells infiltrate and damage smooth muscle tissue.** Foam cells secrete growth factors; this causes the smooth muscle cells in the arterial wall to proliferate all around them. The grayish-white lumps of plaque that are inside dissected arteries are made of these extra muscle cells and cholesterol-filled macrophages. Furthermore, these foam cells can release enzymes that damage arterial walls and cause bleeding and clot formation.

5. **Platelets arrive.** Attracted by the changing texture of the arterial wall, platelets come and release their chemicals, which do three counterproductive things:
   a. **Growth factors are secreted,** and they reinforce the proliferation of new smooth muscle cells.
   b. **Clots form,** and they can further restrict the lumen of the artery.
   c. **Vascular spasm** occurs because the chemical that inhibits it can’t get through all the plaque. This leads to a temporary lack of oxygen in the myocardium and the gripping chest pain called **angina pectoris.**

When vascular spasm and clots combine, partial or total occlusion of the artery can occur. Symptoms of this depend on the location of the blockage and the amount of tissue that is affected.

**Risk Factors**

Risk factors for atherosclerosis can be divided into modifiable and non-modifiable types.

**Non-modifiable risk factors**

- **Heredity, genetics.** Higher-than-average incidence of heart disease within families is clearly demonstrable, but a family history is not a death sentence; controlling modifiable factors significantly reduces the chance of developing problems.
Gender. While both men and women are affected by atherosclerosis, the average onset for men is typically around age 45, and for women it is around age 55. This reflects the shift in hormones that occurs after menopause.

Age. The incidence of heart disease rises with age, but it is not a disease exclusively of the elderly.

Kidney disorders. Atherosclerosis can sometimes lead to kidney problems. But if the kidney problems predate the circulatory ones, high blood pressure brought about by kidney failure can be a precipitator for atherosclerosis.

Modifiable risk factors

Smoking. Carbon monoxide from cigarette smoke is extremely corrosive to endothelium. Furthermore, nicotine causes the release of epinephrine and norepinephrine, leading to vasoconstriction and high blood pressure. Studies show that cigarette smokers run a significantly higher risk of dying by heart disease than the general public.

High cholesterol levels. A predictable statistical link has been established between high cholesterol levels and the development of pathological atherosclerosis. Almost 100 million Americans have cholesterol readings above the recommended maximum of 200 mg/dL, and 34.5 million have readings over 240 mg/dL. For more details on cholesterol, see Sidebar 5.5.

High blood pressure. Chronic uncontrolled high blood pressure contributes to endothelial damage, which opens the door to the formation of plaques.

Sedentary lifestyle. Regular moderate cardiovascular exercise, perhaps more than any other factor, can reduce the risk of atherosclerosis. It keeps arteries elastic and pliable; it reduces weight; it raises high-density lipoprotein levels for the reduction of plaques; it reduces the risk of diabetes; and it lowers blood pressure.

Diabetes. People with uncontrolled diabetes are especially susceptible to atherosclerosis because of the way their body metabolizes food. However, if the diabetes is controlled, the risk of atherosclerosis is much lower.

Other risk factors

Continued study into who develops atherosclerosis and what makes them different from the rest of the population has yielded some additional risk factors. It is unclear whether these are modifiable or not, and the exact relationship between these issues and heart disease is not thoroughly understood. However, identifying these issues early and controlling them may improve the outcome for many people.

C-reactive protein is a liver enzyme secreted in the presence of a systemic inflammatory response. It turns out to be a dependable predictor for heart attack, stroke, and other
conditions related to atherosclerosis, although the mechanism is not clearly understood. The link between \textit{C-reactive protein} and a high risk of atherosclerosis opens the door to questions about whether cardiovascular disease might be linked to chronic infection. Clear connections between gingivitis and other long-term, low-grade infections and a high risk of heart disease support this hypothesis.\footnote{17}

- \textit{Homocysteine} is an amino acid in the blood. A small part of the population tends to have very high levels of \textit{homocysteine}, which can contribute to endothelial damage. The exact relationship between homocysteine and atherosclerosis is not yet understood, but people with high levels are usually counseled to try to control this imbalance with folic acid and vitamins B\textsubscript{6} and B\textsubscript{12}.

- \textit{Other risk factors} that continue to be studied include body mass index, levels of fibrinogen, and subtypes of lipoproteins, some of which may be more involved with plaque formation than others. The goal of finding new predictors for heart disease is to be able to identify who is at risk as early as possible and to control those risk factors as carefully as possible.

Other contributors to atherosclerosis are somewhat harder to quantify. The way a person responds to stress, for instance, has a lot to do with his or her health profile. If stress makes him eat poorly, smoke more, and raises his blood pressure, his or her risk of developing atherosclerosis is higher than that of someone who deals with stress in different ways.

\textbf{Signs and Symptoms}

What are the symptoms of atherosclerosis? Until the damage has progressed to dangerous levels, \textit{there are none}! An artery has to be 50\% or more occluded before any tissue dysfunction occurs. This is largely because the body doesn’t depend on any single artery to do a job. Most parts of the body have two or three alternative vessels, or the body can generate new vessels that can be pressed into service if one of them gets clogged up.

Once signs of atherosclerosis begin to develop, they arise from poor delivery of oxygen to the tissues. If the starved cells are in the heart, low stamina and shortness of breath are the earliest signs. More complications develop as other tissues are deprived of oxygen.

\textbf{Complications}

The complications of atherosclerosis are sometimes the first symptoms of the disease. They include but are not limited to these issues:

- \textit{High blood pressure}. Hypertension is both a cause and a result of this disease; it contributes to the original damage to the tunica intima, and it is made worse when the arterial walls are too brittle to adjust to the constant changes in blood volume flowing through them.

- \textit{Aneurysm}. When the wall of an artery is rendered inelastic and defective, it can bulge and become thin, weak, and susceptible to rupture.

\textbf{Sidebar 5.5: A Brief Digression on Cholesterol}

Cholesterol is a fatty substance produced in the liver and available in any animal product. Saturated fats are particularly rich in easily absorbable cholesterol.

Cholesterol by itself has no access to the body’s cells. Just as glucose must be escorted into cells by insulin, cholesterol must be escorted by \textit{lipoproteins}, other chemicals also produced by the liver. When a cholesterol measurement is taken, it is actually the lipoproteins that are being counted.

Three varieties of lipoproteins are involved with the movement of cholesterol: low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglyceride. The LDLs (“bad cholesterol”) deliver cholesterol to the body’s cells. They are bad only when the body’s cells have no more need for their cargo. At that point the LDLs deposit the cholesterol in artery walls. The HDLs (“good cholesterol”) are involved in reverse cholesterol transport. In this process cholesterol is moved out of the arteries and back to the liver for metabolic processing. The third variety, triglycerides, are chemicals that help to convert fats and carbohydrates into energy for muscles. Studies have shown that elevated triglyceride levels contribute to plaque formation, so it is desirable to keep their numbers down.

When a person gets a cholesterol reading, it’s useful to know not just what the overall levels are but in what ratios the fat types occur. An ideal reading would find total levels below 200 mg/dL, with a relatively high proportion of HDLs (over 35 mg/dL) and lower numbers of LDLs and triglycerides (less than 130 mg/dL combined).

Unfortunately, in the United States fewer than half the adult population can make this claim. About 100 million people have cholesterol readings over 200 mg/dL, and about 38\% of those people have levels over 240 mg/dL.\footnote{16}
• **Arrhythmia.** Advanced atherosclerosis can contribute to the development of irregular or uncoordinated beating of the cardiac muscle as blood supply through the coronary arteries is periodically interrupted. Arrhythmia can cause clots to form in the atria when the chamber doesn’t empty completely. These clots can travel anywhere the aorta takes them.

• **Thrombus or embolism, peripheral circulatory damage.** A thrombus is a stationary clot; an embolism is a traveling clot. Thrombi are the link between atherosclerosis and stroke or transient ischemic attack when they travel to the brain, and between atherosclerosis and heart attack when they lodge in the coronary arteries. If a clot lodges in the renal artery, kidney damage occurs. Clots on the arterial side of the system may also end up in peripheral blood vessels, usually in the legs, where they can cause temporary pain and cramping (called *intermittent claudication*), stasis dermatitis, gangrene, and skin ulcers. This condition is called *peripheral vascular disease* (Figure 5.11).

• **Angina pectoris.** The process of developing atherosclerotic plaques also creates a higher risk of short-term vascular spasm, which leads to heart and chest pain.
  
  • **Stable angina pectoris** means that chest pain is predictable with exercise or exertion and subsides during rest.
  
  • **Unstable angina pectoris** means that chest pain varies in intensity, is not associated with exercise, and is unpredictable. Unstable angina is associated with a high risk of heart attack.

• **Heart attack.** When rough plaques form on smooth artery walls, they attract thrombocytes. If a clot or fragment of plaque breaks off in the coronary artery, it travels until its blood vessel becomes too small to let it pass. All of the myocardium that should have been supplied by that artery then dies. This is a myocardial infarction, or heart attack.

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**Diagnosis**

The traditional way to check for *stenosis*, or narrowing of arteries, is to inject them with dye and take a series of radiographs to watch to movement of fluid through the tubes in a procedure called an *angiogram*. Other tests for atherosclerosis include CT, blood tests to look for targeted chemicals, echocardiography, ultrasound, and a test of the *ankle-brachial index*, a comparison of blood pressure and the arm and lower leg to look for evidence of peripheral vascular disease.

**Treatment**

Treatment for atherosclerosis starts simply. If the situation is caught before it gets out of hand, it can often be managed or even reversed by changing eating and exercise habits.

More advanced cases of atherosclerosis often require drugs and/or surgery. The drugs are generally designed to influence blood pressure, cholesterol levels, and platelet activity.

Surgical intervention for atherosclerosis can include *angioplasty, endarterectomy*, or bypass surgery. Angioplasty is a procedure in which the artery may first be treated with a laser, which vaporizes plaques (laser angioplasty), and then a small balloon is inflated to widen the artery (balloon angioplasty). Unfortunately, the scarring that occurs when the balloon is removed (*restenosis*) can be a difficult, even dangerous complication of this procedure; new cells rapidly proliferate where the endothelium was scraped. In an endarterectomy, a tiny rotating drill is inserted into clogged arteries to shave off plaque, and the shavings are trapped and removed. This is sometimes used for carotid arteries when the risk of stroke is high. In bypass surgery, surgeons remove the damaged piece of coronary artery and replace it, often with a
A single, double, triple, or however-many bypass refers to the number of sections of artery being replaced.

With the exception of bypass surgery, these procedures can be done by inserting special tubes into arteries in the arms or legs and guiding the equipment to the target region. Obviously, the amount of shock to the system is less and the recovery process is much easier than it used to be. But new emphasis is being put on maintaining the structural changes.
brought about by intervention; if the patient reverts to former eating and exercise habits, his or her arteries can reach the same sorry state in just a few years.

**Massage?**

It is impossible to tell if a client has a subclinical buildup of plaque. The deciding factor is whether the client can adjust to the changes in internal environment that massage brings about. In other words, a person whose ability to maintain homeostasis is overly challenged by rigorous movement of fluid through the system is not a good candidate for circulatory massage.

If a client is taking *any* kind of medication for circulatory problems, it is important to clearly understand why and to adjust massage modalities to fit the circulatory limitations of the client.

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## Hypertension

**Definition: What Is It?**

Hypertension is a technical term for *high blood pressure*. It is defined as blood pressure persistently elevated above 140 mm Hg systolic and/or 90 mm Hg diastolic.

**Demographics: Who Gets It?**

About 65 million people in the United States have high blood pressure: that’s 1 in 3 adults. It is seen in men more often than women until women reach age 65. Then it evens out and affects both genders equally. African Americans have higher hypertension rates than other races. Age is a predisposing factor; about half of those over 60 years of age have high blood pressure.

Other predisposing factors include obesity, smoking, high cholesterol levels, atherosclerosis, and water retention. A genetic predisposition may raise the risk of high blood pressure, but sometimes it’s hard to know what’s been inherited: high blood pressure genes, or high blood pressure habits.
Etiology: What Happens?

In hypertension, internal and/or external forces put pressure on the blood vessels. To understand how these forces can cause damage it is necessary to take a brief look at exactly what blood pressure is.

A *sphygmomanometer* is an instrument that measures the pressure blood exerts against arterial walls at two moments: ventricular contraction (*systole*) and ventricular relaxation (*diastole*). The blood pressure cuff converts the pressure in the arteries to millimeters of mercury.

Several variables influence blood pressure. Pressure from inside the vessel (which is increased with any buildup of material inside); pressure from outside the vessel (which is increased by having excess fluid pressing all around), blood volume, and blood vessel diameter are key factors. If any of these is out of balance, total body blood pressure may increase, which in turn influences the health and longevity of blood vessels.

Types of High Blood Pressure

Two types of high blood pressure have been identified: *essential*, or not due to some other pathology; and *secondary*, or a temporary complication of some other condition, such as pregnancy, kidney problem, adrenal tumor, or hormonal disorder. Secondary high blood pressure clears up when the precipitating cause is addressed. About 95% of hypertension is essential. In both essential and secondary high blood pressure another condition, *malignant hypertension*, is possible. In this condition the diastolic pressure rises very quickly, over a matter of weeks or months. It is extremely damaging to the circulatory system, a high risk for ischemic or hemorrhagic stroke, and left untreated is often fatal. Malignant high blood pressure is a medical emergency.

The standard scale for hypertension in adults is based on how measurements correspond to the risk of developing cardiovascular disease, stroke, kidney disease, or heart failure. While a reading of 120/80 has traditionally been considered normal, research reveals that the risk of secondary disease increases significantly when the systolic reading is over 115 or when the diastolic reading is over 75. A person’s blood pressure is based on the averages of two or more readings taken at each of two or more doctor visits. Blood pressure ratings are shown in Table 5.1.

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic BP (mm Hg)</th>
<th>Diastolic BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120–139</td>
<td>80–89</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1 (mild)</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage 2 (moderate)</td>
<td>160+</td>
<td>100+</td>
</tr>
</tbody>
</table>
Massage therapists are not generally required to take blood pressure measurements; these numbers are just an indicator of possible trouble. Be aware also that blood pressure can change significantly from hour to hour. It’s fairly common to see it shoot up from anxiety while a person is in a doctor’s office; this is known as “white coat hypertension”.

**Signs and Symptoms**

Hypertension, which is often called the silent killer, has few recognizable symptoms. A few subtle signs are occasionally observed, however, so they are included here: shortness of breath after mild exercise; headaches or dizziness; swelling of the ankles, especially during the daytime; excessive sweating, or anxiety. Any combination of these symptoms is an indicator that a visit to the doctor would be a good idea.

**Complications**

This is a very important list. Having high blood pressure can shorten a person’s life span. Here’s how:

- **Edema.** High blood pressure forces fluid out of the capillaries at the nutrient–waste exchange sites. This adds to overall levels of interstitial fluid, causing edema. In a typically vicious circle, edema further raises blood pressure by putting external force on blood vessels.

- **Atherosclerosis.** Having blood pushing against arteries in an unceasing torrent simply wears out the walls, especially when the arteries have naturally lost some of their resiliency from age. As damage develops, the atherosclerotic process begins. This reinforces high blood pressure by narrowing arterial diameters.

- **Stroke.** Someone with hypertension is more likely to have a stroke than someone who does not have hypertension. The stroke may be from an embolism, or it may be from ruptured arteries in the brain.

- **Enlarged heart, heart failure.** Pushing against narrowed arteries causes the left ventricle to grow considerably, but the coronary arteries do not grow with it to handle the extra load. The muscle fibers also lose elasticity. Therefore, the contractions are actually weaker, because the muscle is not well supplied with blood and it can’t contract fully. This can also cause angina, or heart pain. When the ventricles of the heart are so overtaxed that they simply cannot keep up with the workload, the patient risks heart failure.

- **Aneurysm.** This is the result of high blood pressure causing a bulge in the arteries.

- **Kidney disease.** This complication of high blood pressure demonstrates the circular relationship between hypertension and kidney dysfunction. If problems start with the circulatory system, hypertension causes atherosclerotic plaques to form in the renal arteries, which are subject to huge blood pressure. This causes changes in blood flow to the kidney, which impairs kidney function, leading to kidney damage, systemic edema, and yet more pressure exerted against vessel walls from that edema. If the problem starts in the kidneys, decreased kidney function causes fluid retention. This is often accompanied by extra release of renin, the kidney-based hormone that regulates some electrolyte balance. Excess renin results in vasoconstriction, water and salt retention, increased edema, increased blood volume, and high blood pressure.

- **Vision problems.** Chronic high blood pressure can damage the blood vessels that supply the eyes, causing them to thicken and lose elasticity. Reduction of blood flow to eyes results in permanent visual distortion.
Treatment

Hypertension is a highly treatable disease, but because it has virtually no symptoms until it has progressed to very dangerous levels, it is frequently untreated or incompletely treated (e.g., someone not taking his medication because he feels fine). It is estimated that of the 65 million people who have high blood pressure in this country, 63.4% are aware of their problem; 45.3% are under treatment, and only 29.3% of all high blood pressure patients successfully control their disease. In other words, more than 70% of the people who have high blood pressure don’t control it well enough to prevent other cardiovascular problems.18

Diet is the first way to manage this condition. The National Heart Lung and Blood Institute (NHLBI) has created the DASH (Dietary Approaches to Stop Hypertension) diet: a combination of high-fiber, low-fat foods that provide higher than average levels of calcium, magnesium, and potassium while cutting sodium by about 20%. Following the DASH diet (which is useful for anyone, not just hypertension patients) has been found to be as effective as treatment with one type of blood pressure medication, without side effects or long-term health risks.19

Exercise is also crucial for the development of healthy new blood vessels and for weight control. Losing even a small percentage of body weight for obese or overweight patients can have a profound effect on blood pressure and cardiovascular health.

Medication, if it’s called for, includes diuretics, vasodilators, and beta blockers, which decrease the force of ventricular contraction. Controlling the systolic pressure turns out to be challenging but extremely important, especially in patients over 50 years old.

Medicating high blood pressure is a bit problematic. Because the disease itself has no strong symptoms and because the medicines often have unpleasant side effects (including dizziness, insomnia, impotence, and others), it can be difficult for hypertension patients to be consistent with their medications.

Massage?

If a client knows that he or she has high blood pressure but is not required to take medication for it, circulatory massage is probably fine, especially if the client is physically active or encouraged to become physically active. Massage can help to lower general blood pressure and the stress that contributes to it.20 It is important, however, to rule out kidney disease and other advanced cardiovascular problems. Watch especially for signs that massage is overchallenging the body: clamminess, bogginess, and possible edema in the days after the treatment.

If a client does take medication for high blood pressure, circulatory modalities are strongly cautioned. Techniques that don’t strongly influence fluid flow may be appropriate, but rigorous, fast-paced work may be too much of a challenge for an impaired ability to maintain homeostasis. As always, judgments about massage must be made by comparing the challenges of bodywork with activities of daily living.

Regardless of whether the client is on blood pressure medication, high blood pressure contraindicates deep abdominal massage. This is because it is possible to accidentally trip the vasovagal reaction. Unintentionally overstimulating the vagus nerve can result in amplified parasympathetic reactions. This leads to systemic vasodilation and faintness from lack of blood to the brain. Another possibility is a sympathetic rebound effect. Ordinarily a vasovagal reaction is unpleasant but not dangerous—unless the blood vessels are not equipped to handle a rapid demand to dilate and constrict. Once again, a client’s health is determined by the ability to maintain a stable internal environment during massage.
Raynaud Syndrome

**Definition: What Is It?**

*Raynaud syndrome* is a condition involving the status of the arterioles in the hands and feet, although it can also affect the nose, ears, and lips. Primary Raynaud disease is a vasoconstriction disorder, while secondary Raynaud phenomenon is a complication of an underlying problem.

**Demographics: Who Gets It?**

Raynaud disease, or primary Raynaud syndrome, is unrelated to underlying conditions. It usually affects women between 15 and 40 years of age; this population group makes up about three-quarters of all primary Raynaud syndrome patients. Secondary Raynaud syndrome, also called Raynaud phenomenon, is a symptom or complication of other disorders. Estimates vary, but it is suggested that some variety of Raynaud syndrome affects up to 5% to 10% of the general population.

**Etiology: What Happens?**

In both primary and secondary Raynaud syndrome the arterioles in the extremities develop vasospasm, or contraction of smooth muscle tissue. It occurs in temporary episodes at first, but especially if it is a secondary complication, the vasoconstriction can become permanent.

The chemical changes that occur with a Raynaud syndrome episode are the subject of intense study. The tunica intima is capable of secreting local chemicals that affect the constriction of arterioles and possibly even the viscosity of the blood. A high overlap between people with Raynaud syndrome and people with other vasoconstrictive disorders may eventually point to this condition as a chemical manifestation of hyperreactivity to cold or stress.
Primary causes Raynaud disease is a primary problem, that is, unconnected to underlying pathology. It may be due to emotional stress (the autonomic nervous system routes blood away from the skin during emergencies), cold, or a mechanical irritation, such as operating machinery that influences blood vessel dilation. Pianists and typists are particularly vulnerable. Raynaud disease generally has a very slow onset, and the attacks are less severe than when the symptoms occur as a secondary problem. If a person is prone to Raynaud disease, both the feet and hands tend to be affected.

Secondary causes Occasionally extreme vasoconstriction is a complication of some other disorder. In this case the condition is called Raynaud phenomenon. It generally has a much faster onset than Raynaud disease, the age at onset is typically older, and the risk of serious complications is much higher. Some conditions associated with Raynaud phenomenon include the following:

- Arterial diseases that involve occlusions, such as diabetes, atherosclerosis, and Buerger disease (a rare disease marked by inflammation and blood clots in the arteries)
- Autoimmune connective tissue diseases, such as scleroderma, lupus, and rheumatoid arthritis
- Sensitivity to some drugs, including beta blockers and ergot compounds
- Neurovascular compression, as seen with carpal tunnel syndrome, thoracic outlet syndrome, or crutch use

Signs and Symptoms Raynaud syndrome is usually bilateral. During an attack the skin goes through a characteristic cycle of colors: white as the blood is shunted away from the area (on dark-skinned people the skin looks ashy gray); blue as the cells are starved for oxygen; and red as the attack subsides, the arterioles reopen, and the blood returns to the affected area (Figure 5.12). Some people only shift between blue and red; others show only pallor or blueness (cyanosis) during an episode. It usually affects distal fingers and toes, not the thumb or the rest of the hand. Sometimes only one or two digits are affected, and these may change from one episode to another.

Attacks of Raynaud phenomenon can last anywhere from less than a minute to several hours. Secondary Raynaud can be so extreme and long-lasting that atrophy and ulcerations on the starved skin may develop. Arterioles in the nailbeds can become thickened and distorted; this is identified in a test called a nail fold capillaroscopy. The fingers may taper, and the skin can become thin, smooth, and shiny. Gangrene is a rare but possible complication for these extreme cases.

Figure 5.12. Raynaud syndrome.
**Treatment**

Treatment depends on whether the patient has primary or secondary Raynaud syndrome. Generally a noninvasive approach is taken first, at least for primary Raynaud disease. Quitting smoking, avoiding vasoconstrictors such as nicotine and caffeine, soaking in warm water, dressing appropriately for the weather, protecting the hands when working with cold or frozen foods, making sure that shoes aren’t too tight, even moving to a warmer climate are all suggested before more intrusive intervention is suggested.

Because primary Raynaud disease can be exacerbated by emotional upset, patients are often encouraged to find productive ways to manage stress. This can range from learning biofeedback techniques, to exercising regularly, to receiving massage.

If results are unsatisfactory or if tissue damage from chronically impaired blood flow is a risk, the next step is medication to dilate the blood vessels. Other drugs work to counteract norepinephrine, the stress hormone that initiates vasoconstriction. Unfortunately, medical intervention is often unsuccessful for secondary Raynaud phenomenon. Surgery to interfere with sympathetic motor neuron stimulation of local capillaries may be conducted; this procedure, called a **sympathectomy**, is used only when no other options work, and it tends to be a temporary measure.

**Massage?**

The good news about Raynaud syndrome is that though the primary version of the syndrome is fairly common, the secondary version is rather rare. If Raynaud phenomenon is a part of lupus, scleroderma, or another vascular problem, the guidelines for massage must follow the cautions for the precipitating causes.

If any dangerous underlying causes of the vasoconstriction have been ruled out, primary Raynaud syndrome indicates massage, even during an episode. Massage can work mechanically with local blood vessels and reflexively with the parasympathetic nervous system to stimulate vasodilation.

### Modality Recommendations for Raynaud Syndrome

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep Tissue Massage</td>
<td>Contraindicated when associated with other conditions. Indicated if primary. Work to decrease fascial tension proximal to feet and hands.</td>
</tr>
<tr>
<td>Lymphatic Drainage</td>
<td>Supportive.</td>
</tr>
<tr>
<td>PNF/MET/Stretching</td>
<td>Supportive.</td>
</tr>
<tr>
<td>Reflexology</td>
<td>Indicated; work endocrine, lymphatic, solar plexus points.</td>
</tr>
<tr>
<td>Shiatsu</td>
<td>Indicated. Treat the TH, PC, SP meridians and extensions in legs and arms. Add lots of ROM to joints of limbs.</td>
</tr>
<tr>
<td>Swedish Massage</td>
<td>Supportive if no contraindicated conditions are present.</td>
</tr>
<tr>
<td>Trigger Point Therapy</td>
<td>Locally contraindicated while acute; otherwise supportive.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.
Varicose Veins

**Definition: What Are They?**

Varicose veins are distended, often twisted or ropy superficial veins (*varix* means twisted). They occur when the valves that support blood flow against gravity are damaged. As blood collects in the system, the affected vein is stretched, distorted, and generally weakened (Figure 5.13). Varicose veins can develop at the anus (hemorrhoids), at the esophagus (esophageal varices) or at the scrotum (varicoles). But most often they are in the legs (Figure 5.14), which is the focus of the rest of this discussion.

**Demographics: Who Gets Them?**

Women get varicose veins more often than men; this is largely due to progesterone, which can weaken venous walls, and to a history of pregnancy. Increased blood volume, shifting hormones, and the weight of the fetus on the femoral vein all work together to set the stage for later problems. Varicose veins are very common; about half of people over 50 years old have them.\(^1\)

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Varicose Veins in Brief

**Pronunciation:** VARE-ih-kose vanez

**What are they?**
Varicose veins are distended veins, usually in the legs, caused by venous insufficiency and retrograde flow of blood that should be moving against gravity.

**How are they recognized?**
Varicose veins are ropy, slightly bluish, elevated veins that twist and turn out of their usual course. They are most common in branches of the great saphenous veins on the medial side of the calf, although they are also found on the posterior aspects of the calf and thigh.

**Is massage indicated or contraindicated?**
Massage is locally contraindicated for extreme varicose veins and anywhere distal to them. Mild varicose veins contraindicate deep specific work but are otherwise safe for massage.

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**Figure 5.13.** Varicose veins.

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\(^1\) Non-programmed text.
Etiology: What Happens?

The veins in the legs have a fascinating construction that works to move blood from the toes all the way back to the heart. Small veins pick up the blood from the internal muscle capillaries. These veins tend to run on the superficial aspect of muscles. They feed into larger veins that perforate the muscle bellies and then into the really big deep veins that run under the muscles, close to the bones. When the leg muscles contract, the perforating veins are squeezed, sending their contents to the deep veins. When the leg muscles relax, the perforating veins draw in new blood from the smaller veins. The contraction and relaxation of the leg muscles (especially the soleus—“sump pump of the leg”) is crucial to blood return. The valves inside the perforating veins and the deep veins ensure that blood does not collect in the smaller, weaker superficial veins.

What can damage the valves in the veins? It could be simple wear and tear: being on one’s feet for many hours a day, especially if the leg muscles are not allowed to fully contract and relax during that time, weakens the veins. It could also be a mechanical obstruction to returning blood: knee socks that are too tight, a knee brace, or a fetus that presses on the femoral vein. Systemic problems from kidney or liver congestion have been seen to cause problems too. And finally, it could be simply congenitally weak veins or a structural anomaly at the junction between the great saphenous vein and the femoral vein.

Once a valve sustains damage, blood puts pressure on the next valve down. Vascular incompetence ultimately causes the weakest superficial veins to become distorted, dilated, and twisted off their regular pathway. Deeper veins are protected from this process because they have the external support of muscle tissue.
Signs and Symptoms

Varicose veins look like lumpy bluish wandering lines on the surface of the skin. They are often visible on the back of the calf, where they affect the lesser saphenous vein, but more often they affect the great saphenous vein, where they show up anywhere from the ankle to the groin on the medial side. They may be visible only when the patient is standing.

Varicose veins may itch or cause throbbing pain, especially at the end of the day, when legs feel tired and heavy. They can contribute to edema around the ankles as fluid backs up in the lower leg.

Complications

Although varicosities are seldom more than annoying, they can create some unpleasant or even dangerous complications. Chronically impaired circulation may result in varicose ulcers, which don’t heal until circulation is restored. Skin irritation from poor circulation occasionally leads to a type of dermatitis that isn’t resolved until the varicosity is relieved. Interruptions in blood flow increase the likelihood of annoying night cramps. And stagnant blood in a distended vein may coagulate, raising the possibility of clotting. Most clots that form in varicose veins are superficial and melt easily, however, so they are usually a lesser threat than clots that form in deeper leg veins. Be aware, however, that the presence of grossly distended varicose veins may indicate an increased risk of deep vein thrombosis. This is true especially when the varicosities have a sudden onset or change in size and quality very rapidly.22

Treatment

Mild varicose veins are usually treated with good sense. Using support hose or elastic bandages can give extra help to damaged veins, and avoiding long periods of standing up without full contraction and relaxation of the muscles is often recommended. Clothes that constrict at the leg, the groin, or the waist should be avoided. Reclining with the feet slightly elevated also reduces symptoms.

Whether or not the veins can actually heal is somewhat controversial. If the damage has not progressed too far, relieving the mechanical stresses while strengthening the smooth muscle tissue (with hydrotherapy, for instance) can yield good results.

Surgery for mild varicose veins is not generally recommended as a purely cosmetic intervention. However, varicose veins are a progressive condition; they don’t usually spontaneously reverse, and if they are left untreated, their complications can be serious. Therefore, a certain number of patients eventually seek treatment for health rather than cosmetic concerns.

Several strategies for reducing varicose veins have been developed. These are appropriate when it is clear that the varicosity has not developed as a complication of a hidden obstruction or deep vein thrombosis. The traditional approach is surgery to tie off the proximal and distal sections of the great saphenous and remove the vein: this is called vein stripping. Ambulatory phlebectomy (ministripping) is a similar treatment that removes only small sections of the affected veins. Sclerosing involves injections of chemicals that cause the vein to close down completely; it eventually turns to scar tissue and fades. Applying laser energy or radiofrequency through a catheter to large veins is usually successful. In all of these treatments, the body’s remarkable ability to generate new blood vessels quickly accommodates the closure or removal of the affected vein.

Massage?

Deep, intrusive massage is a local contraindication for varicose veins that are elevated from the skin and that are visibly distorted from their original pathway. Not only is the tissue in-
jured and delicate, but it is inappropriate to push a lot of blood through vessels that may not be able to accommodate it. Heavy massage distal to these veins is cautioned also.

Clients with extreme varicosities who have a high risk of complications should be under a doctor’s care and be checked regularly for signs of deep vein thrombosis.

If the vein is only slightly darkened and not raised or causing any pain, it is still wise to avoid local specific pressure, but otherwise massage is safe. Tiny reddened “spider veins” (telangiectasias) are slightly dilated venules and are safe for massage.

<table>
<thead>
<tr>
<th>MODALITY RECOMMENDATIONS FOR VARICOSE VEINS</th>
</tr>
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<tbody>
<tr>
<td>Deep Tissue Massage</td>
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<tr>
<td>Lymphatic Drainage</td>
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<tr>
<td>Polarity</td>
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<tr>
<td>PNF/MET/Stretching</td>
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<td>Reflexology</td>
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<tr>
<td>Swedish Massage</td>
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<tr>
<td>Trigger Point Therapy</td>
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</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

HEART CONDITIONS

Heart Attack

Definition: What Is It?

A heart attack is damage to some portion of the cardiac muscle as a result of ischemia, which starves and kills some of the muscle cells. The ischemia is usually caused by coronary artery disease, or atherosclerosis of the coronary arteries, which supply the cardiac muscle with oxygen and nutrition. If these arteries are completely occluded by plaque, thrombi, or any combination of the two, some piece of the muscle dies (Figure 5.15). The muscle tissue does not grow back; it is replaced by inelastic, noncontractile scar tissue. The damaged area is referred to as an infarct. Another term for heart attack is myocardial infarction.

Demographics: Who Gets Them?

Coronary artery disease and heart attack is the leading cause of death in the United States, claiming over half a million lives every year, or 1 in 5 deaths. Over a million heart attacks occur every year, and about 40% of them result in death within the year. Over 13 million heart attack or angina survivors are alive in the United States today. Most people are familiar with the high-risk profile for heart attack victims: being sedentary, having hypertension, having high cholesterol levels, smoking, and being overweight. Being a man over 45 or a woman over 55, having a family history of cardiovascular disease,
or being a woman over 35 who takes birth control pills also increases
the chances of a heart attack.

That said, 50% of women and 64% of men who die of heart at-
tack had no previous symptoms or warning signs on record.

**Etiology: What Happens?**

A heart attack occurs when a portion of the cardiac muscle is killed
off from lack of oxygen: an ischemic attack. Usually it comes from a
blockage in the coronary artery that obstructs blood flow. It could
also be from a loosened blood clot or a broken or torn piece of ath-
erosclerotic plaque that blocks the coronary artery. Rarely, a heart
attack may occur when a coronary artery goes into prolonged spasm;
this is seen most often in cocaine or other drug overdose.

Examinations of exactly which plaques pose the greatest risk of
heart attack have revealed that the size of the plaque evidently has
little to do with its chances of breaking off or causing a heart attack.
The more pertinent factor is how stable the plaque is. Older, harder
plaques are relatively stable, but newer, softer plaques have a higher
risk of breaking open and letting go of clots or atherosclerotic frag-
ments that then block the coronary artery.

When a portion of the cardiac muscle is killed off by ischemic
attack, the ability to contract with coordination and efficiency is
badly damaged. If a heart attack is severe enough to trigger ventricular **fibrillations**, the risk
of sudden death is very high.

The seriousness of a heart attack is determined by the size and location of the blockage.
If it is relatively small and blood flow to an area that doesn’t have to work especially hard is
impaired, the heart attack is not a serious one. But if the infarct, or area of damaged tissue,
is large enough to weaken the heart’s ability to contract, or if the damaged tissue contains
the electrical conduction system for the heart, major intervention is necessary to aid in re-
covery.

**Signs and Symptoms**

Heart attacks have a variety of signs and symptoms, some of which are extremely subtle and
some of which are very severe. Some of the most common and dependable signs are these:

- **Pressure, pain in the chest.**
- **Spreading pain.** Pain may spread to the shoulder, arm, neck, and jaw of the left side of
  the body. This is the referred pain pattern for the dermatome shared by the heart.
- **Lightheadedness, nausea, sweating.** These usually occur along with chest pain. When
  they occur **without** chest pain they may still indicate a heart attack, but it is a less com-
  mon presentation.

Other symptoms include shortness of breath with or without chest pain; unexplained nau-
sea, anxiety, or weakness; fainting; palpitations; and cold sweat. Even stomach and abdominal
pain may sometimes be signs of a heart attack.

- **Angina pectoris** (literally, chest pain). This is one of the few early warning signs for the
  risk of heart attack. Not all people have this symptom, but those who do should pay
close attention.
- **Stable angina.** This is the simplest and most common form of angina. It affects about
  6.5 million people and is diagnosed about 400,000 times a year. In this form of angina
the heart can get enough oxygen to perform regular tasks, but any extra effort, such as
 carrying something heavy or running up a flight of stairs, demands too much of the

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**Heart Attack in Brief**

**What is it?**

A heart attack, or myocardial infarction, is damage to the myocardium caused by a sudden obstruction in
blood flow through the coronary arteries, which re-

**How is it recognized?**

Symptoms of most heart attacks include a sensation of
pressure on the chest, spreading pain, lightheaded-
ness, dizziness, and nausea. Sometimes symptoms
vary, and occasionally a heart attack may occur with
no symptoms at all.

**Is massage indicated or contraindicated?**

Patients recovering from recent heart attacks are not
candidates for circulatory massage, although tech-
niques that do not challenge fluid flow may be appro-
priate. When they have completely recovered, they
may be able to receive massage, depending on the
rest of their cardiovascular health.
clogged coronary arteries. The result is moderate to severe chest pain that is relieved with rest and/or angina medication, drugs that help blood vessels to expand.

- **Unstable angina.** This type of angina can occur without unusual physical activity. It often appears in the night, with very extreme but short-lived chest pain. It is caused by vascular spasm at or near the site of atherosclerotic plaques. This variety of angina is a reliable predictor of incipient heart attack.

A heart attack is a dynamic process. The critical blockage of the coronary artery may take place over several hours. This is good news because early intervention can preserve much of
the myocardium: survival rates for heart attacks are better than ever. But this is also bad news, because many people tend to ignore early warning signs and don’t seek attention until symptoms have been present for many hours or even days.

**Complications**

Several complicating conditions are discussed elsewhere in this chapter; diseases of the cardiovascular system are highly interrelated. Furthermore, with heart disease it’s hard to say what comes first, the infarct or the thrombus. In other words, the chronology of these problems is often circular rather than linear.

- **Embolism.** A heart attack can cause blood clots to form in the heart itself. These clots exit through the aorta and travel to wherever the bloodstream takes them; these are arterial emboli, and they can land in the brain, causing a stroke, or the renal arteries, where they can contribute to renal failure. Prolonged bed rest can also promote deep vein thrombosis, which carries a risk of pulmonary embolism. Pulmonary embolism may also occur if clots form on the right side of the heart and are carried out by the pulmonary artery.

- **Atrial and ventricular fibrillations.** These are rapid, incomplete, weak attempts at contraction of the chambers. They occur most often if any part of the sinoatrial node, the heart’s electrical pacemaker, has been damaged. These inefficient contractions allow blood to pool and thicken in the chambers of the heart and may contribute to the risk of stroke from the left side or pulmonary embolism from the right side. Ventricular fibrillations, because they interfere with blood flow to the entire body, may result in death if they are not treated quickly.

- **Aneurysm.** Weakened cardiac tissue can create a bulge in the heart muscle itself similar to aortic aneurysms.

- **Heart failure.** In heart failure the muscle is no longer strong enough to do its work, and the body pays the price. This condition is discussed further elsewhere in this chapter.

- **Shock.** In shock the circulatory system swings reactively from a sympathetic to a parasympathetic state, opening the arteries to a maximum diameter in the process. The main danger with shock is loss of oxygen to the brain from radically decreased blood pressure.

**Diagnosis**

Even with the new technologies that have been developed to deal with cardiovascular disease, the ability to identify and treat heart attacks before damage occurs is extraordinarily limited. Most of the time the best that can be done is to try to limit the damage as quickly as possible.

Screening for high-risk patients is usually conducted by angiogram: a flexible catheter is inserted into the femoral artery and snaked up to the coronary arteries. A contrast dye is injected, and pictures are taken to see how blood flows through occluded arteries. Unfortunately, this is an invasive procedure whose accuracy is somewhat limited. Although it can give useful information about arterial diameter, it doesn’t measure risk of heart attack.

Much emphasis is being put on imaging techniques that may help to identify who is at most risk for having a heart attack and where that risk will come from before the heart is irreversibly damaged. Among the options that are becoming available are high-speed CT of the coronary arteries, contrast echocardiography, blood tests for C-reactive protein (an enzyme associated with inflammation), and MRI that can examine not only where plaques have developed, but also how dense they are and how deeply they penetrate into arterial walls.
The first priority with heart attack patients is to determine where the blockage is and to get rid of it as quickly as possible. This is done with clot-dissolving drugs, which can take effect in 90 minutes or less, and with immediate balloon angioplasty, which can open up most clogged arteries in about an hour. The technical term for this procedure is percutaneous transluminal coronary angioplasty (PTCA). Other immediate-care options include the administration of oxygen and pain management with nitroglycerin and/or morphine.

Later care usually includes more clot dissolvers and nitroglycerin, which works to relax the smooth muscle tissue in the arteries. After the emergency has passed, the main treatment for heart attacks is observation. A barrage of tests is conducted to determine the location and extent of damage to the cardiac muscle. These tests indicate one of three future courses of action: that the infarct was minor and requires no further medical intervention; that prescription anticoagulants are indicated; or that a serious and permanent narrowing of a coronary artery requires surgery to open and repair it. This surgery may be a more complete version of the angioplasty or it may be coronary bypass surgery, in which damaged sections of the coronary artery are replaced with grafts of healthy vessels from elsewhere in the body.

Treatment in heart attack and heart surgery recovery must also embrace the lifestyle changes that will support a healthier future: eating sensibly, exercising regularly, controlling high blood pressure, and quitting smoking are the most important factors.

### Sidebar 5.6: Other Heart Conditions

**Heart Murmurs**
The heart can make several types of noise during its contractions. These are called murmurs. They often, but not always, point to some type of valvular dysfunction in the heart. A client with a persistent heart murmur may have an inefficient pump and may not be able to keep up with rigorous circulatory massage.

**Hypertrophy of the Heart**
This condition, which is seen most dramatically in the left ventricle, is brought about by chronically high systemic pressure in the blood vessels. If the heart has to fight against constricted arteries to push blood through the body, it can grow larger. It may also increase in strength for a while, but as the need for oxygen outgrows the supply from the coronary arteries, the tissue eventually undergoes ischemia, possible myocardial infarction, and eventually congestive heart failure.

**Congenital Heart Problems**
Any of 15 structural problems may affect the heart at birth. Approximately 36,000 babies with these are identified each year, but many more people are identified with minor problems in adulthood. About 1 million people in the United States have been identified as having a congenital heart problem. Defects often involve valve function or a hole in the septum that allows blood to cross from the right to the left side of the heart or vice versa.

**Rheumatic Fever**
Rheumatic fever is an autoimmune complication of exposure to streptococcus in which antibodies attack the heart valves, especially the mitral valve. It affects about 1.3 million Americans and is responsible for close to 6,000 deaths per year. Mitral valve damage affects the way the heart can pump blood through the body. It can lead to arterial emboli or congestive heart failure.

**Infectious Diseases of the Heart**
Different varieties of streptococcus may prey on endocardium. If they find a way in (which can happen from something as innocuous as an abscessed tooth but is more often a complication of open heart surgery), they cause clots that are released into the arterial system.

**Massage?**
Most of these conditions systemically contraindicate circulatory massage. Work that mechanically pushes fluid through the system will hinder rather than help these damaged structures.
Some studies have indicated that taking aspirin regularly can decrease the chance of a repeat heart attack for people with a history of heart disease. Specially coated tablets have been designed to minimize gastrointestinal problems, but this intervention carries a possibility of an increased chance of subdural hemorrhage, so patients must consider carefully all the implications of taking aspirin on a daily basis.

**Massage?**

The appropriateness of massage for heart attack survivors absolutely depends on the individual, the extent of the damage, and how long ago it occurred. Some survivors of mild heart attacks may make themselves healthier than they ever were before, while others will accumulate high levels of plaque on their arteries within just a few years of surgery. The safety of massage depends on how easily the client can withstand the changes in internal environment that this work will bring about. This means that modalities that support rather than challenge homeostasis are generally the safest choices.

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### Compare and Contrast 5.2

**Chest Pain, Chest Pain, Chest Pain**

Not all chest pain means heart attack, although in a culture in which almost 40% of deaths are related to cardiovascular disease, it seems logical to jump to that conclusion. What follows is a comparison of types of chest pain with some indications of what might be heart attack and what probably is not. However, heart attack symptoms are notoriously variable, and it is always a good idea to consult a health care professional when the source of chest pain is not clear.

<table>
<thead>
<tr>
<th>FEATURES</th>
<th>ANGINA</th>
<th>HEART ATTACK</th>
<th>PULMONARY EMBOLISM</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration</strong></td>
<td>Chest pain lasts several minutes, subsides.</td>
<td>Chest pain progressively worsens.</td>
<td>Chest pain progressively worsens.</td>
<td>Chest pain subsides in &lt;1 min.</td>
</tr>
<tr>
<td><strong>Trigger</strong></td>
<td>Usually triggered by activity.</td>
<td>May or may not be immediately triggered by activity.</td>
<td>May or may not be immediately triggered by activity.</td>
<td>May or may not be immediately triggered by activity.</td>
</tr>
<tr>
<td><strong>Activity</strong></td>
<td>Stops when activity stops.</td>
<td>Doesn’t stop when activity stops; continues to worsen.</td>
<td>Doesn’t stop when activity stops; continues to worsen.</td>
<td>Stops when person drinks water, changes position, or takes a deep breath.</td>
</tr>
<tr>
<td><strong>Causes</strong></td>
<td>Caused by transient ischemia; heart muscle temporarily doesn’t get enough oxygen to function.</td>
<td>Caused by permanent ischemia; blockage deprives cells of oxygen, and heart is irrevocably damaged.</td>
<td>Caused by blood clot in lung. Small clot may have little impact. Large clot may lead to pulmonary and circulatory collapse.</td>
<td>Caused by any number of factors, e.g., musculoskeletal injury, gastro-esophageal reflux.</td>
</tr>
</tbody>
</table>
Heart Attack

Bob, Age 49: The wake-up call.

About 15 years ago my mom, who was 60 years old, had bypass surgery. I knew that having a female relative diagnosed with cardiovascular disease at 60 years old put me in a high-risk category for heart problems, especially since I’ve had type 2 diabetes for about 10 years. I know I didn’t eat the healthiest diet in the world. At that time my regular lunch was a quarter-pounder and a bag of fries.

Last summer my mother went through a series of angina attacks followed by an angioplasty and having a stent inserted. After watching her, I began to seriously think about my own condition. I talked to my doctor, and he set me up with a low-cost on-site stress test that just used an exercise bike. He said if I could pass that I’d be okay. I took it, and in the words of the technician, “My heart was not happy with what I was doing to it.” So my doctor scheduled me with a cardiologist for a full treadmill test.

After that first test I went out for what I knew would be my last double quarter-pounder with cheese.

I didn’t last long on the treadmill. When it was over, my blood pressure dropped and I had some really unpleasant symptoms, like dizziness, nausea, and a general feeling of crappiness. My doctor called my wife in from the waiting room, and with both of us together he said, “My recommendation is to put you in the hospital now. We’ll do an angiogram along with anything else that needs to be done.”

“Well,” I said, “I have a couple of things I need to finish up. Can I take care of them and come right back?”

“I’d rather you didn’t.”

I checked right in.

I went into the hospital the Tuesday after Labor Day. On Wednesday I had the angiogram followed by an angioplasty. They found that the main section of the left coronary artery was 100% blocked. They had trouble pushing a wire through it, but when they got that done, they put in the balloon and then a titanium stent. (They said it wouldn’t set anything off, but I can’t get into Target anymore without having all the alarms go off.)

They told me that they found evidence of a recent heart attack—one that was a fraction of an inch away from what they call a widow maker. This was amazing to me, because I have no memory of any chest pains. The only thing I can remember was when I went on a trip with my family at the beginning of the summer. I’d hiked around a little that day. That night in the motel I had a migraine headache. I felt sick and threw up. I took some ibuprofen and went to bed. That’s the only time I can think of that I had any symptoms at all.

Three days after the angioplasty I went home. They started my cardiac rehab right away. I go to the hospital to exercise under supervision. I am next door to an emergency room, and medical staff is in the room with me, so if anything happens, the response time will be really quick. When I first started, I was hooked up to a heart monitor with four patches to measure my blood pressure before, during, and after my exercise. The first time I tried to exercise, they made me quit because I was about to go into ventricular fibrillation. Now I go exercise three times a week. I use the treadmill, the bike, weight machines, and free weights. I walk a lot at work, but I can’t walk at home; it’s too steep around my house. My doctors tell me I can’t ever let my heart rate get over 120 beats per minute. If it gets any higher than that, I run the risk of forming clots around the stent.

I’m on several medications now. Some control my diabetes (I don’t take insulin), and I’m also taking anticoaguants for 3 months, along with beta blockers and calcium channel blockers. And of course I’ve changed my diet. I eat so much chicken I feel like I’m growing feathers. It’s all baked or grilled, and I take off the skin. We have little or no fried food, so no more French fries. Since I’ve made these changes, my blood glucose has been much more under control—I average about 115 now, and normal is anywhere from 80 to 120.

This episode was a real wake-up call for me. I’m the youngest man at my job and the last one they expected to have heart trouble. My identical twin went in for his own stress test and came back fine, but he was a couple of years later than me in developing his diabetes too, so he’ll still have to keep an eye on it. I did some research about my situation, and I found that what I had—silent ischemia—is especially common in diabetic men over 40. I hope any man with type 2 diabetes over 40 or 45 will be sure to get his heart checked. You never know what you might find.
Heart Failure

Definition: What Is It?

Heart failure is a term for the progressive loss of cardiac function that accompanies age and a history of cardiovascular disease. It does not mean that the heart has stopped working altogether (that would be cardiac arrest); it simply means that the heart cannot keep up with the needs of the body.

Demographics: Who Gets It?

The statistics on the incidence of heart failure in the United States are alarming but understandable, considering the changes in the health profile of the population. Heart failure is on a dramatic rise in this country; it affects about 3 million people, and the incidence is expected to rise in coming years. About 400,000 new cases are diagnosed every year. Most of those cases are among people who have survived other cardiovascular crises: heart attacks, coronary artery disease, aneurysm, and others. Where many years ago these problems would have killed these patients, they now survive and their hearts simply wear out from progressive damage to the cardiovascular system.

More men than women have heart failure until age 75, when numbers are about even between genders. It affects African Americans about twice as often as whites. The incidence of heart failure rises dramatically with age: it affects about 1% of the population at age 50 and up to 10% of the population age 75 and older. Acute decompensated heart failure accounts for about 1 million hospitalizations per year.

Etiology: What Happens?

When the heart is asked to pump 2,000 gal of blood each day through vessels that are progressively narrowed and resistant, it goes through a series of changes that ultimately limit its ability to function. Unfortunately, the heart can enter early stages of heart failure with no signs or symptoms.

Heart Failure in Brief

What is it?
Heart failure is a condition in which the heart no longer can function well enough to keep up with the needs of the body. It is usually slowly progressive, developing over a number of years before any changes in function may be noticeable.

How is it recognized?
The symptoms depend on which side of the heart is working inefficiently. Left-sided heart failure results in fluid congestion in the lungs with general weakness and shortness of breath. Right-sided heart failure results in fluid backups throughout the system, which shows as edema, especially in the ankles and legs. Both varieties of heart failure lead to chest pain; cold, sweaty skin; a fast, irregular pulse; coughing, especially when the person is lying down; and very poor stamina.

Is massage indicated or contraindicated?
Heart failure contraindicates circulatory massage, as it has the goal of pushing fluid through a system that is incapable of adjusting to those changes. Energetic work may be more appropriate to help clients lower stress and cope with the challenges of severely restricted circulation.
As resistance in the cardiovascular system increases (usually from atherosclerosis, hypertension, and other manifestations of cardiovascular disease), the heart compensates in various ways. Any of these mechanisms is appropriate for short-term challenges, but over the long term they add to the problem rather than helping to solve it.

The heart muscle cells respond to chronic stress by growing. The ventricles appear to become larger and thicker, allowing the heart to push harder against resistance in the pulmonary or systemic circuits. Ultimately, however, this cardiac hypertrophy causes the ventricles to become stiff so they don’t fill or contract normally.

Some chemicals that influence heart function can also help to compensate for short-term problems but add to long-term ones. Resistance in the system or injury to the heart causes the release of stress hormones, especially epinephrine and norepinephrine. These make the heart beat harder, which is appropriate for short-term situations but damaging for chronic ones. Shifts in hormones also cause the body to retain salt and water. Both of these features end up increasing blood pressure and adding to the workload of the overburdened heart.

Finally, the muscle simply wears out and functions so inefficiently that blood flow to the rest of the body is insufficient for the most basic kinds of activities: climbing a set of stairs, walking across a room, even getting out of bed. Left untreated, the heart muscle goes into fibrillations and the circulatory system collapses.

Most cases of heart failure are related to underlying cardiovascular disease. A history of atherosclerosis or heart attack with resulting scar tissue in the heart muscle increases the risk of developing heart failure. High blood pressure, untreated diabetes, smoking, and alcohol and drug abuse can all be contributing factors as well. An especially potent setup for heart failure is any combination of these risk factors: uncontrolled high blood pressure along with smoking, for instance.

A smaller number of heart failure patients do not have a history of cardiovascular disease but have sustained damage to the heart muscle for other reasons. Cardiomyopathy, valve diseases, infections of the valves or heart muscle, and congenital problems may all be factors in these cases.

Types of heart failure: systolic versus diastolic  Heart failure is sometimes classified by which cardiac function is most impaired.

• Systolic heart failure. The left ventricle has become enlarged and cannot contract efficiently. Consequently, it can’t push blood out into the aorta and the systemic circuit well enough to keep up with a person’s needs.

• Diastolic heart failure. The ventricles have become enlarged but stiff. They don’t relax and expand well to allow the inflow of blood from the atria. This also leads to inadequate pumping of blood through the pulmonary and systemic circuits.

Types of heart failure: left side versus right side  Heart failure is also classified according to which side of the heart is weaker.

• Left-sided heart failure. The left ventricle is impaired. A backup in the pulmonary circuit allows seepage of fluid back into the alveoli; this is pulmonary edema (Figure 5.16). Symptoms of left-sided heart failure include severe shortness of breath and stubborn coughing, perhaps with bloody sputum. Symptoms are worst when a person is active or lying down. One serious complication of this condition is the risk of pneumonia in the functionally impaired lungs.

• Right-sided heart failure. Also called cor pulmonale, it commonly results from pulmonary disease and high vascular resistance in the lungs—often as a complication of the pulmonary edema that accumulates with left-sided heart failure. Consequently, it becomes difficult to pump blood through the pulmonary circuit, and the backup is felt through the rest of the body. Symptoms include severe edema, especially in the legs.
(Figure 5.17). Someone with this type of heart failure has ankles that look like they’re spilling over the sides of the shoes. If the patient is bedridden, the edema may occur in the abdomen (ascites) or in the pelvis—wherever gravity is pulling most. Right-sided heart failure is also closely linked to enlarged liver (hepatomegaly) and renal failure. As blood flow to the kidneys is reduced, the kidneys begin to retain water, which systematically increases blood pressure and makes the heart work even harder to push blood through narrow tubes.

- **Biventricular heart failure.** This is left- and ride-sided failure simultaneously. It is the end stage of the disease, and if the patient doesn’t respond to medications, he or she may be a candidate for a heart transplant or other surgery.

**Signs and Symptoms**

Signs of heart failure depend on which side of the heart is dysfunctional, as already described. Along with shortness of breath (often exacerbated by lying down), low stamina, and edema, heart failure patients may also have chronic chest pain; indigestion; arrhythmia; visibly distended veins in the neck; cold, sweaty skin; and restlessness.
Symptoms typically develop over a long period. If they have a sudden onset, they present a medical emergency.

**Diagnosis**

Heart failure isn’t difficult to diagnose. It is often done through observation of systemic edema and auscultation, or listening to heart sounds and to the lungs for indications of fluid retention. Radiography may be indicated to look for cardiac enlargement, and an electrocardiogram may be conducted to analyze the efficiency of the heartbeat. An echocardiogram can show valve function if damage to these structures is suspected.

Heart failure may be rated on a scale of I to IV or from A to D. In either case, the mild end of the scale involves many risk factors but only minor symptoms or loss of function, while the extreme end of the scale indicates a life-threatening situation.

**Treatment**

The treatment options for heart failure depend on how severe it is and which side of the heart has been affected. Early interventions include rest, changes in diet, and modifications in phys-
ical activity so that the heart can be exercised without becoming overly stressed. Drugs for heart failure may include beta blockers, digitalis, diuretics, and vasodilators.

If a patient doesn’t respond well to these noninvasive treatment options, surgery may be considered. Surgery can range anywhere from repair to damaged valves, to wrapping a strong mesh bag around the heart, to a complete heart or heart and lung transplant.

**Massage?**

Heart failure means that the heart can’t function in a way that provides for essential needs. Most heart failure patients have other cardiovascular problems that contribute to their disease. Circulatory massage, which works with the intention of mechanically increasing blood flow through a basically healthy system, is inappropriate for clients whose blood vessels and heart cannot accommodate these changes in internal environment.

Energetic work that invites (rather than forces) the body to positive change may help to reduce blood pressure and perceived stress and so may be useful for heart failure patients, although it is not realistic to claim that massage can reverse the extensive damage seen with these clients.

### MODALITY RECOMMENDATIONS FOR HEART FAILURE

<table>
<thead>
<tr>
<th>Modality</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Deep Tissue Massage</strong></td>
<td>Systemically contraindicated.</td>
</tr>
<tr>
<td><strong>Lymphatic Drainage</strong></td>
<td>Systemically contraindicated.</td>
</tr>
<tr>
<td><strong>Polarity</strong></td>
<td>S: Indicated when working as part of health care team. R/D: Supportive within client comfort.</td>
</tr>
<tr>
<td><strong>PNF/MET/Stretching</strong></td>
<td>Supportive.</td>
</tr>
<tr>
<td><strong>Reflexology</strong></td>
<td>As part of health care team, work endocrine, heart, chest, lymphatic, solar plexus points.</td>
</tr>
<tr>
<td><strong>Shiatsu</strong></td>
<td>Indicated. Treat H, K meridians and extensions systemically.</td>
</tr>
<tr>
<td><strong>Swedish Massage</strong></td>
<td>Supportive with light work; stay within activity limitations and adjust for medications.</td>
</tr>
<tr>
<td><strong>Trigger Point Therapy</strong></td>
<td>Supportive with light work.</td>
</tr>
</tbody>
</table>

See Chapter 1 for a brief description of each modality, including definitions of abbreviations.

### CHAPTER REVIEW QUESTIONS: CIRCULATORY SYSTEM CONDITIONS

1. What is the causative agent for malaria?
2. Why are fatigue and low stamina symptoms of anemia?
3. Name two types of blood cancer.
4. How does sickle cell disease lead to severely reduced immunity?
5. Where are the blockages that lead to heart attacks?
6. What is a term for uncoordinated contractions of heart chambers?
7. Why is hypertension called the “silent killer”?
8. Describe the circular relationship between high blood pressure and kidney dysfunction.
9. Describe how a person may have any three of the following conditions at the same time: high blood pressure, chronic renal failure, edema, atherosclerosis, diabetes, aortic aneurysm, stroke.

10. A client has a deep ache in the lower leg. Distal to the knee the tissue is clammy and edematous. Pressing at the ankle leaves a dimple, which takes several minutes to disappear. What cautions must be exercised with this client? Why?

REFERENCES


