The Swollen Leg
Rocco G. Ciocca

Objectives

1. To describe the differential diagnosis of the swollen leg.
   • To discuss how to differentiate lymphedema from venous disease.
   • To discuss painful versus nonpainful swelling.

2. To describe the factors that lead to venous thrombosis and embolism.
   • To discuss the usual locations of thrombosis.
   • To discuss differing implications of deep and superficial venous thrombophlebitis.
   • To discuss the common invasive and noninvasive diagnostic tests for deep venous thrombosis (DVT).
   • To discuss methods for DVT prophylaxis and identify high-risk patients.
   • To discuss the risks, benefits, and available options for anticoagulation and thrombolysis.
   • To discuss the signs, symptoms, diagnostic evaluation, and treatment of pulmonary embolism.

3. To describe the diagnosis, workup, and management options for symptomatic varicose veins and venous ulcers.
   • To discuss the physical exam and tests for venous valvular competence.
   • To discuss the role of venography and ultrasound.
   • To discuss medical versus surgical management.
   • To discuss the role of stripping, sclerosis, and laser ablation.
Case

You are asked to see a 43-year-old woman with a “swollen leg.” She states that she has had a swollen left leg for several months and that her primary care physician wanted her to see a specialist for this condition. The left leg is somewhat larger on exam than the right leg, but, other than a sensation of “fullness,” the patient denies any discomfort.

History and Physical Examination

As in all things that pertain to patient care, the history and the physical exam are the cornerstones to getting at the etiology of the swollen leg. Giving the patient adequate time to explain the problem is critical and frequently can save valuable time and useless diagnostic studies. Of critical importance, however, is obtaining a sense of the immediacy of the problem. In other words, is this an acute or a chronic problem? Once the timing of the swelling is ascertained, then a relatively simple thought process can be followed. See Algorithm 29.1 for an algorithm of the management of the swollen leg.

The physical exam is critically important in the evaluation of the swollen leg, and, while not 100% accurate, it helps narrow the differential diagnosis of the problem. In the case presented above, the patient has had swelling for several months. The chronic nature of the situation may alter somewhat the aggressiveness of the workup. Things to focus on include any obvious trauma, evidence of infection, or bony abnormality. The presence of edema and the nature of the edema may be very telling. Ultimately, one must decide if the swelling is systemic in nature, due to a vascular (venous) abnormality, or secondary to lymphedema. The unilateral nature of the swelling described by the patient in the case presented leads one to think that the etiology of the swelling is not systemic in nature. Systemic conditions like obesity or congestive heart failure generally lead to bilateral lower extremity swelling.

All physical exams should include a thorough head-to-toe evaluation. Head and neck evaluation, with particular attention to the presence or absence of jugular venous distention, is important. Documentation of any masses may be telling when considering the etiology of venous or thromboembolic disease. The chest exam is important with regard to the presence or absence of rales or rhonchi. Decreased breath signs and dullness to percussion also are important to identify. A careful abdominal exam is critical. The presence of abdominal masses, which may be a source of venous or lymphatic obstruction, must be noted. Abdominal masses also may be indicative of an intraabdominal tumor and therefore a nidus for a hypercoaguable state. Checking the patient’s stool for occult blood also is important as an indicator of a possible neoplasm but also in planning therapy, particularly if anticoagulation is indicated. Obesity, a frequent cause of a “swollen” extremity, frequently is overlooked or disregarded as an etiology.
Unilateral swelling, as in the case patient, certainly could be due to an intrabdominal mass or deep venous thrombosis. It is critically important to examine both legs. Inspection often fails to identify any difference between the extremities. This implies that the swelling is bilateral in nature or that the “swelling” may be due to some other process. Remember the obesity discussion above.

The nature of the swelling, the presence or absence of edema, the nature of the edema, the evidence of trauma, cellulitis, the nature and texture of the skin, the presence of ulcerations, and the locations and nature of the ulcerations all are important to document. The presence of pain, the location of pain, and the presence or absence of varicosi-

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Algorithm 29.1. Algorithm for the management of swollen leg.
ties are important to document. While arterial insufficiency rarely presents as swelling, the presence of peripheral pulses is important to document.

**Acute versus Chronic**

When the history obtained from the patient indicates that the swelling has occurred acutely, the differential veers toward disease processes that need to be diagnosed quickly and treated aggressively. If the process is chronic, a more leisurely diagnostic workup may follow.

Deep venous thrombosis (DVT) is exceedingly prevalent. The annual incidence is estimated at 48 per 100,000.¹ Deep venous thrombosis is a component of a larger process, venous thromboembolism (VTE). The incidence of VTE is age dependent (Fig. 29.1). The diagnosis of DVT is always predicated on Virchow’s triad. Rudolf Virchow, a 19th century pathologist, surmised that three conditions tended toward thrombosis: intimal injury, stasis of blood flow, and a hypercoaguable state. These observations have stood the test of time and are as true today as they were in Virchow’s time. Most risk factors for DVT are related in some way to Virchow’s triad (Table 29.1). In the case pre-

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sented, the patient may have had a DVT for the past several months that simply had not been diagnosed. Therefore, the presence of a DVT needs to be ruled out.

Trauma is without question a risk factor for DVT (Table 29.2). Stasis frequently follows trauma or surgery and frequently is contributory to the development of DVT and pulmonary embolus (PE) (Table 29.3).

It can, at times, be difficult to differentiate an acute DVT from a chronic DVT. Obviously, the patient’s history is critical in helping to do this. If the patient had a normal extremity and suddenly developed a painful swollen extremity, the diagnosis is straightforward. Unfortunately, patients frequently present in a less than straightforward manner. In those cases, it is best to err on the side of caution and treat the patient as if he/she has an acute problem. A duplex scan, as discussed in the next section, may help in the diagnosis of an acute versus chronic DVT.

Diagnosis

As with many disease processes in medicine, the diagnosis of DVT has evolved. A thorough history and a thorough physical exam remain

Table 29.1. Risk factors for venous thromboembolism (VTE).

<table>
<thead>
<tr>
<th>History of VTE</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Major surgery</td>
</tr>
<tr>
<td>Malignancy</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Varicose veins/superficial thrombophlebitis</td>
</tr>
<tr>
<td>Cardiac disease</td>
</tr>
<tr>
<td>Hormones</td>
</tr>
<tr>
<td>Prolonged immobilization/paralysis</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Central venous catheterization</td>
</tr>
</tbody>
</table>

*Hypercoaguable states*


Table 29.2. Risk factors for venous thromboembolism in 349 trauma patients.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Odds ratio (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (each 1-year increment)</td>
<td>1.05 (1.03–1.06)</td>
</tr>
<tr>
<td>Blood transfusion</td>
<td>1.74 (1.03–2.93)</td>
</tr>
<tr>
<td>Surgery</td>
<td>2.30 (1.08–4.89)</td>
</tr>
<tr>
<td>Fracture of femur or tibia</td>
<td>4.82 (2.79–8.33)</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>8.59 (2.92–25.28)</td>
</tr>
</tbody>
</table>

* Determined by multivariate logistic regression.

the primary initial diagnostic modalities, but they suffer from a lack of sensitivity.

**Venography** had been considered the “gold standard” in the diagnosis of DVT. It currently is performed rarely. Venography or **phlebography** involves the cannulation of a peripheral hand or foot vein, application of a tourniquet to occlude the superficial venous system and to direct blood flow into the deep system, and injection of radiopaque contrast medium. Clot is outlined by the contrast material, thereby confirming the diagnosis. The procedure is painful for the patient, technically difficult to perform, and not always easy to interpret.

**Duplex ultrasonography** has surpassed venography as the diagnostic test of choice for DVT. It is a combination of B-mode ultrasonography and Doppler. The study allows determination of vein compressibility as well as flow characteristics. Veins generally are thin-walled and easily compressed. Veins that are incompressible with firm pressure applied by the ultrasound probe are considered thrombosed. Flow within the veins also can be assessed easily. Normal venous flow should be phasic, decreasing with inspiration. Flow can be increased by distal compression and decreased by increasing intraabdominal pressure. With experience, duplex ultrasonography can achieve greater than 90% sensitivity and greater than 95% specificity in the diagnosis of DVT. A venous duplex scan would be an appropriate diagnostic study for the patient in the case presented in this chapter.

Clinicians frequently ask the vascular lab to help differentiate between an acute and a chronic DVT. There are no strict criteria to achieve the differentiation; however, there are some “soft” signs that can be helpful. If the thrombus is acute, it generally is not echogenic on duplex and is relatively soft on compression. The veins distal to the thrombus frequently are dilated. The clot may appear free floating.

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and not completely adherent to the vein wall. Chronic DVTs are more commonly echogenic. There may be evidence of recanalization of the vein, and the veins distal to DVT are not as dilated as in an acute DVT. Comparison to a previous study, if available, can be very helpful.

A less specific but occasionally useful test for the diagnosis of DVT is the D-dimer assay. A new rapid assay currently is available to detect D-dimer, which is a specific derivative of cross-linked fibrin that is released when fibrin is lysed by plasmin. It is a useful screening test for patients suspected of DVT, with sensitivity approaching 93%. Its utility is questionable, however, if duplex ultrasonography is available readily.

**Pulmonary Embolus**

Embolization of DVT into the pulmonary artery is the most dreaded complication of DVT. **Pulmonary embolus (PE)** is estimated to be responsible for the deaths of 50,000 to 100,000 persons per year in U.S. hospitals who would otherwise not be expected to die of their underlying disease process.3

One should be suspicious of PE when a patient at risk develops signs and symptoms of dyspnea, tachypnea, chest pain, tachycardia, cyanosis, hemoptysis, hypotension, syncope, evidence of right-sided heart failure, and a pleural rub or rales.

The diagnosis of PE usually is not easy, but significant prospective data have been collected by the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED).4 There are multiple nonspecific tests that are helpful and usually are ordered when a patient presents with the above-mentioned symptoms. The tests include arterial blood gas, chest x-ray (CXR), and electrocardiogram (ECG). The arterial blood gas characteristically reveals a respiratory alkalosis, decreased CO₂, and decreased O₂, which results in an increased alveolar-arterial (A-a) gradient. Suggestive findings on CXR include prominent central pulmonary artery, decreased pulmonary vascularity (Westermark’s sign), and a pleural-based, wedge-shaped pulmonary density. These findings, while strongly suggestive of PE, rarely are seen. The most common ECG findings are nonspecific ST segment or T-wave changes. The importance of these nonspecific tests is to rule out other pathologic processes with similar signs and symptoms to PE.

**Pulmonary angiography (PA)** is the “gold standard” diagnostic study for PE. It is performed by placing a catheter into the pulmonary artery, usually via a femoral vein puncture, and injecting contrast into both lungs. The diagnosis of PE is confirmed by evidence of either com-

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plete obstruction of, or filling defects within, the pulmonary vessels. The study is invasive and has a significant list of complications, including cardiac arrhythmias, contrast reaction, and bleeding. It is both sensitive and specific, and a negative PA effectively rules out PE.

The noninvasive test of choice for the diagnosis of PE is a **ventilation/perfusion scan (V/Q)**. Perfusion scans involve the injection of radiolabeled colloid into a peripheral vein, followed by scanning of the lung in several positions. This is followed by inhalation of a radiolabeled aerosol for the ventilation portion of the study. The diagnosis of PE is confirmed by a perfusion defect without a corresponding ventilation defect. The scans are graded as normal, very low probability, low probability, intermediate probability, and high probability (Table 29.4).

When the V/Q scan is intermediate probability, many physicians also obtain a **lower extremity venous duplex scan**. If that is positive, then the patient should be anticoagulated, if there are no contraindications and no further testing is necessary. Additional diagnostic studies for PE currently being developed are **helical computed tomography (CT)** and **magnetic resonance angiography (MRA)**.

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**Table 29.4. PIOPED<sup>a</sup> central scan interpretation categories and criteria.**

<table>
<thead>
<tr>
<th>Category</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>High probability</td>
<td>≥2 large (≥75% of a segment) segmental perfusion defects without corresponding ventilation or roentgenographic abnormalities or substantially larger than either matching ventilation or chest roentgenogram abnormalities</td>
</tr>
<tr>
<td></td>
<td>≥2 moderate segmental (≥25% and ≤75% of a segment) perfusion defects without matching ventilation or chest roentgenogram abnormalities</td>
</tr>
<tr>
<td></td>
<td>≥4 moderate segmental perfusion defects without ventilation or chest roentgenogram abnormalities</td>
</tr>
<tr>
<td></td>
<td>Intermediate probability (indeterminate)</td>
</tr>
<tr>
<td></td>
<td>Not falling into normal, very low, low-, or high-probability categories</td>
</tr>
<tr>
<td></td>
<td>Borderline high or borderline low</td>
</tr>
<tr>
<td></td>
<td>Difficult to categorize as high or low</td>
</tr>
<tr>
<td>Low probability</td>
<td>Nonsegmental perfusion defects (e.g., very small effusion causing blunting of the costophrenic angle, cardiomegaly, enlarged aorta, hila and mediastinum, and elevated diaphragm)</td>
</tr>
<tr>
<td></td>
<td>Single moderate mismatched segmental perfusion defect with normal chest roentgenogram</td>
</tr>
<tr>
<td></td>
<td>Any perfusion defect with a substantially larger chest roentgenogram abnormality</td>
</tr>
<tr>
<td></td>
<td>Large or moderated segmental perfusion defects involving no more than 4 segments in 1 lung and no more than 3 segments in 1 lung region with matching ventilation defects either equal to or larger in size and chest roentgenogram either normal or with abnormalities substantially smaller than perfusion defects</td>
</tr>
<tr>
<td></td>
<td>&gt;3 small segmental perfusion defects (&lt;25% of a segment) with a normal chest roentgenogram</td>
</tr>
<tr>
<td></td>
<td>Very low probability</td>
</tr>
<tr>
<td></td>
<td>≤3 small segmental perfusion defects with a normal chest roentgenogram</td>
</tr>
<tr>
<td>Normal</td>
<td>No perfusion defects seen</td>
</tr>
<tr>
<td></td>
<td>Perfusion outlines exactly the shape of the lungs as seen on the chest roentgenogram (hilar and aortic impressions may be seen, chest roentgenogram and/or ventilation study may be abnormal)</td>
</tr>
</tbody>
</table>

<sup>a</sup> Prospective Investigation of Pulmonary Embolism Diagnosis.

*Source:* Reprinted from The PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism; results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). JAMA 1990;263: 2753–2759, with permission. Copyright © 1990 American Medical Association. All rights reserved.
Treatment

Once the diagnosis of a venous thromboembolic event has been confirmed and, occasionally, before it has been confirmed and, if the index of suspicion is high, the patient should be anticoagulated. In addition to conventional anticoagulation, a small subset of patients may benefit from thrombolytic therapy. There is a role for the use of thrombolytic therapy in the treatment of VTE in highly selected patients. Many patients have contraindications or relative contraindications to the use of thrombolysis that obviate their use.

Patients with VTE usually are treated with a bolus dose of unfractionated heparin (UFH), which functions as an anticoagulant via two mechanisms: (1) it binds to antithrombin III (ATIII) and amplifies the inhibition of thrombin and activated factor X by ATIII, and (2) it catalyzes the inhibition of thrombin by heparin cofactor II. The half-life of UFH is 90 minutes. The adequacy of anticoagulation is monitored by the activated prothrombin time (aPTT), which usually is monitored at 6-hour intervals until the level has achieved a steady state. One generally attempts to increase the aPTT to 1.5 to 2.5 times above control.

Evidence indicates that it is best to dose UFH according to a nomogram. Treatment with UFH generally is continued until the patient is fully anticoagulated with oral agents, namely warfarin. Complications of UFH use include bleeding and heparin-induced thrombocytopenia (HIT). HIT is estimated to occur in approximately 2% to 5% of patients receiving heparin. It is believed to be due to antibodies directed against platelet complexes with heparin. Due to the possibility of HIT, it is important that patients treated with UFH have a platelet count at some point after the initiation of therapy. Consequences of HIT include problems with both bleeding and thrombosis. All forms of heparin should be withheld once the diagnosis is considered.

Warfarin, the oral anticoagulant of choice, acts by interfering with the production of both the procoagulant (II, VII, IX, X) and anticoagulant (proteins C and S) vitamin K–dependent cofactors. Response to warfarin is variable depending on the patient’s liver function, diet, age, and concomitant medications. The levels of anticoagulation produced by warfarin are monitored by following the prothrombin time (PT). Secondary to a wide variability of thromboplastin reagents used, the international normalized ratio (INR) was created. An INR of 2.0 to 3.0 generally is accepted to be therapeutic in most patients with VTE.

It initially was thought that several days of UFH were necessary before initiating warfarin therapy. Multiple studies have shown that starting warfarin therapy in addition to heparin is safe and effective. Warfarin has a long half-life, variable depending on the patient, and must be withheld for several days prior to any significant intervention. It generally is felt that a patient with VTE should be continued on

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warfarin therapy for at least 3 to 6 months depending on their risk factors. Patients with a recurrence should be treated indefinitely.6,7

The most exciting new development in the treatment of VTE is the use of low molecular weight heparins (LMWHs) (Table 29.5). They have longer half-lives and better bioavailability than UFH and allow for once or twice daily subcutaneous dosing. They have a predictable anticoagulant effect based on body weight, so that laboratory monitoring is unnecessary. It is important to note that different LMWHs differ in their anti-Xa and anti-IIa activity and therefore do not perform in the same manner. Multiple randomized trials have been performed comparing various LMWHs to UFH in the initial treatment of VTE.8–10

Table 29.5. Randomized trials of LMWHa versus UFHb for treatment of deep venous thrombosis (DVT) (level I evidence).

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Recurrent DVT (%)</th>
<th>Major bleeding (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LMWH vs. UFH</td>
<td></td>
<td>LMWH vs. UFH</td>
</tr>
<tr>
<td>Fraxiparine6</td>
<td>&lt;55 kg 12,500 XaIUc</td>
<td>7 vs. 14 (p = ns)</td>
<td>1 vs. 4 (p = ns)</td>
</tr>
<tr>
<td></td>
<td>&gt;55 kg &lt;80 kg 15,000 XaIU (Q 12 h)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;80 kg 17,500 XaIU (Q 12 h)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dalteparine6</td>
<td>200 XaIU/kg (Q 24 h)</td>
<td>5 vs. 3 (p = ns)</td>
<td>None, either group</td>
</tr>
<tr>
<td>Enoxaparinf</td>
<td>100 XaIU/kg (Q 12 h)</td>
<td>1 vs. 10 (p &lt; .02)</td>
<td>None, either group</td>
</tr>
<tr>
<td>Logiparina8</td>
<td>175 XaIU/kg (Q 24 h)</td>
<td>3 vs. 7 (p = .07)</td>
<td>0.5 vs. 5 (p = 0.06)</td>
</tr>
</tbody>
</table>

- Low molecular weight heparin.
- Adjusted dose unfractionated heparin.
- Factor Xa inhibitory units.


The advantages of LMWH have allowed patients with VTE to be successfully treated as outpatients.\textsuperscript{11} The role for \textbf{surgical intervention} in the treatment of VTE is limited. The most commonly performed surgical intervention is the placement of an inferior cava filtration device. The indications for cava filter placement are the presence of VTE in the presence of an absolute contraindication to anticoagulation or the failure of anticoagulation. These are rare. Most commonly, cava filters are placed for relative contraindications to anticoagulation or, increasingly, for pulmonary embolus prophylaxis for patients who cannot be anticoagulated safely. While inferior cava filters are safe and effective, they do not treat VTE; rather, they prevent the most dreaded complication: pulmonary embolus.

Other more aggressive surgical interventions for VTE have been described, but they rarely are indicated. Simple procedures, such as high ligation of the greater saphenous vein at the saphenofemoral junction, are reasonable for superficial thrombosis of the greater saphenous vein. More significant operations, such as iliofemoral venous thrombectomy or surgical pulmonary embolectomy, have a role, but fortunately they only rarely need to be employed.

Referring back to the patient who presented with a swollen leg, one might surmise that she may have had a left lower extremity DVT that was missed. While the likelihood of this being the case is low in the absence of injury, stasis, or history of a hypercoagulable state, it would be reasonable to interrogate her venous anatomy with a venous duplex scan. If the duplex scan is positive for DVT, then the clinician may be left with a bit of a dilemma: Should the patient be treated with anticoagulants or not? One of the limitations of duplex ultrasonography is differentiating acute versus chronic DVTs. Particularly with the development of safe and effective outpatient therapies for DVT, a physician probably should treat a duplex proven DVT with anticoagulation.

\section*{Chronic Venous Insufficiency}

One of the most common causes of a swollen leg is \textbf{chronic venous insufficiency (CVI)}. It has been estimated that 27\% of the U.S. population has some form of detectable lower-extremity venous abnormality.\textsuperscript{12}

Patients with CVI frequently complain of leg fatigue, discomfort, and heaviness. Signs include venous telangiectasias, swelling, and varicose veins, as well as lipodermatosclerosis and venous ulceration. \textbf{Lipodermatosclerosis} represents a constellation of skin changes, including thickening of the skin, hemosiderin deposition of the skin, and a dry scaly dermatitis of the skin. These changes most commonly affect the

\begin{itemize}
\end{itemize}
medial aspect of the calf, the so-called gator region of the leg. Risk factors associated with varicose veins may include prolonged standing, heredity, female sex, parity, and history of phlebitis. Venous ulcerations particularly are problematic. In addition to the previously mentioned risk factors, patients with venous ulceration tend to be older, have a history of previous DVT, and have a history of lower extremity trauma; they tend to be male, and are obese.

The diagnosis of deep venous insufficiency generally is made clinically based on history and clinical exam. It can be confirmed by noninvasive studies, such as air plethysmography (APG) and, most commonly, via duplex ultrasound evaluation. The APG is performed by placing a polyvinyl chloride bladder on the lower leg. Various volumes of the leg are then calculated with the patient in several positions (Fig. 29.2). The information gathered from the APG may assess calf muscle pump function, venous reflux, and overall lower-extremity venous function. The duplex ultrasound, in addition to ruling out the presence of DVT, can be used to evaluate venous reflux in individual segments of the lower extremity. Particular attention currently is being paid to communicating veins, those that connect the deep and superficial venous systems. Incompetence of the perforating veins has been implicated in the development of venous stasis ulcers.

![Figure 29.2](image)

Treatment

Conservative, nonoperative, treatment for chronic venous insufficiency has been and remains the primary therapy. This form of therapy generally focuses on decreasing lower extremity venous hypertension. Bed rest with elevation is very effective in relieving symptoms of CVI, but it generally is not practical for more than a very short period.

Due to the limitation of bed rest and elevation, some form of compression is prescribed. The most common compression garment is a commercially made, graduated compression stocking that provides increased compression at the level of the ankle, but less compression as it ascends the leg. Patients with active venous ulceration can be treated with any of a number of layered compression dressings. The most common is the paste gauze dressing developed by the German dermatologist Paul Unna in 1896. The current Unna’s boot consists of dome paste dressing, containing calamine, zinc oxide, glycerin, sorbitol, and magnesium aluminum silicate. The dressing is applied with graduated compression from the foot to the knee. An additional layer of an elastic wrap is then added. The dressing is kept in place for several days and frequently for up to a week. The therapy effectively facilitates healing of venous stasis ulcers about 70% of the time. Once healed, the patient should convert to lifelong compression stocking use.

Multiple pharmacologic agents have been tried for the treatment of CVI and venous ulcerations. Most have little or no significant benefit. Diuretics, ironically, provide little benefit for patients with CVI, although they may benefit patients with systemic causes of lower extremity swelling, such as cardiac failure and renal insufficiency.

Surgical intervention rarely is indicated as a first line of therapy for patients with CVI. Commonly performed procedures include ligation and stripping of varicose veins, subfascial ligation of perforating veins, and, uncommonly, venous reconstruction. While vein ligation and stripping address only the superficial venous system, they frequently do provide significant symptomatic relief. Linton and others described procedures involving ligation of perforating veins for the treatment of CVI. Currently, a newer technique is available to accomplish this, subfascial endoscopic perforator vein surgery (SEPS). Retrospective analysis of the procedure demonstrates ulcer healing in 88% of cases treated with SEPS.

More aggressive venous reconstructions have been described for patients with advanced CVI. The operations have included venous valve repair, valve transplantation, and venous bypass procedures. The results of these procedures are encouraging, but the procedures should be reserved for extreme cases.

The patient presented at the beginning of this chapter may have chronic venous insufficiency. If she does not have chronic ulcerations,

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she most likely could be treated successfully with a compression stocking.

**Lymphedema**

**Lymphedema** represents another possible cause of a swollen lower extremity. The swelling of lymphedema is caused by an abnormality in the lymphatic drainage of the leg. It may be secondary to a congenital abnormality of the lymphatic system, leading to **primary lymphedema**, or it may be due to some acquired abnormality, leading to **secondary lymphedema**. Primary lymphedema has no demonstrable precipitating cause. Secondary lymphedema results from well-described causative mechanisms, such as recurrent infection or surgical or radiation therapy for tumor or trauma, all of which can lead to obliteration of the lymphatic vessels. Secondary lymphedema is the more common of the two forms. Tropical elephantiasis caused by infection with *Wuchereria bancrofti* is the most common form of secondary lymphedema internationally. Its incidence in the United States fortunately is low.

The diagnosis of lymphedema generally is based on **history and physical exam**. Careful history of trauma and chronic infections to the lower extremity should be elicited. A **history of surgery or radiation to the pelvis or extremity** obviously would lead one to think that the swelling was secondary to injury or obliteration of the lymphatic vessels.

The swelling from lymphedema generally starts at the level of the foot and ankle and progresses in a cephalad direction. The distribution of the edema produces a characteristic shape. In the lower extremity, the edema usually involves the forefoot and spares the metatarsophalangeal joint, so that, on lateral view, the foot and ankle resemble a buffalo hump. With more extensive edema, the limb assumes a tree trunk–link appearance. The edema usually does involve the digits, which rarely are involved when the edema is secondary to other causes. The edema may be pitting particularly early in the process, but it may lose the pitting with the onset of significant subcutaneous fibrosis. Unlike in venous insufficiency, the skin changes in lymphedema lack the dark pigment changes. Lymphedema certainly is part of the differential of the patient in the case presented here, particularly if the patient provides a history of previous surgery or infection.

A limited number of diagnostic tests may be helpful. It certainly is reasonable to obtain a **lower extremity duplex scan** to rule out DVT or CVI as etiologies of the swelling. Other studies, such as **CT scans or magnetic resonance imaging (MRI)**, may be helpful in assessing possible secondary etiologies of the lymphedema and in helping rule out lymphangiosarcoma. **Lymphangiography** rarely is indicated. **Lymphoscintigraphy** using radiolabeled albumin, gold colloid, and technetium colloid can be performed to assess lymphatic function and largely has replaced lymphangiography.
Lymphedema, whether it is primary or secondary, is a chronic condition and has no cure. Treatment, therefore, is palliative. The primary goal of therapy is to decrease limb volume in order to reduce discomfort, provide cosmesis, and avoid infection.

The noninterventional methods of treating lymphedema represent the first line of therapy, and, in fact, they are used to treat the vast majority of patients. The therapeutic interventions include adequate skin care, elevation and compression of the extremity, the use of pneumatic compression garments, manual lymph drainage and bandaging, the use of benzopyrones, and aggressive treatment of infections. Benzopyrones, theoretically, act by increasing protein lysis by macrophages in the interstitium. This action may decrease limb volume moderately and improve the softness of the skin. The other modalities mentioned above attempt to reduce limb volume via mechanical compression or manual massage.

The surgical forms of therapy, which generally are reserved for only the extreme cases, fall into one of two categories: physiologic or excisional. The physiologic procedures attempt to reestablish lymphatic drainage. Examples of physiologic procedures include lymphangioplasty, omental transposition, enteromesenteric bridge, lymphovenous anastomoses, and lymphohyphalastic anastomoses. It is important to note, however, that all of the above-mentioned procedures rarely are performed, and most vascular surgeons have seldom, if ever, performed any of them. Excisional procedures include total skin and subcutaneous excision, the Charles procedure, buried dermal flap, the Thompson procedure, and subcutaneous excision underneath flaps, the modified Homans procedure. These procedures are aggressive and have relatively high complication rates. Success rates are modest, in the range of 65%, and therefore these procedures should be reserved only for those patients who have not responded to measures that are more conservative.

Some of the important points to remember when dealing with lymphedema are that the condition is chronic, some form of compression garment is necessary, and any form of infection within the affected extremity should be treated aggressively. Patients need to be educated as to the signs and symptoms of infection and instructed to seek medical attention immediately if they develop signs of infection. Many physicians provide their patients suffering from lymphedema with a prescription for an appropriate antibiotic to avoid any delays in initiation of therapy.

If the patient in the case presented has lymphedema, she should be treated conservatively with compression of the affected extremity and education regarding the signs and symptoms of infection.
Summary

The presentation of a patient with a swollen leg is a rather common event. Most patients present with chronic symptoms and can be treated conservatively. The etiology generally is related to a systemic, venous, or lymphatic abnormality. Few patients present with potentially life-threatening problems (e.g., an acute DVT), and they warrant urgent and aggressive treatment. A thorough history and a thorough physical examination coupled with noninvasive testing lead to the appropriate diagnosis in the majority of cases. If a patient is found to have an acute DVT, aggressive therapy is indicated. Fortunately, with the advent of LMWHs, many of these patients can be treated safely as outpatients or with a relatively short hospital stay. Ironically, a vascular surgeon frequently is consulted when a patient presents with a swollen leg. The role of surgery is limited in the treatment of patients with swollen legs, but it may be useful in small subsets of patients. A reasonable understanding of the pathophysiology of the swollen leg as described in this chapter greatly assists a physician in making the correct therapeutic decisions regarding these sometimes difficult patients.

Selected Reading