

# CARDIOLOGY PATIENT PAGE

## Pulmonary Embolism and Deep Vein Thrombosis

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The heart pumps oxygenated blood through the aorta to smaller arteries. After the blood supplies nutrients to vital organs, it returns through veins for reoxygenation in the lungs (Figures 1 and 2). Blood clots called deep vein thrombi (DVT) often develop in the deep leg veins. Pulmonary embolism (PE) occurs when clots break off from vein walls and travel through the heart to the pulmonary arteries. The broader term venous thromboembolism (VTE) refers to DVT, PE, or to a combination of both.

### What Is the Epidemiology?

VTE poses a public health threat with an estimated incidence in the United States of 250 000 to 2 million cases per year. Predisposition to VTE arises from acquired conditions, inherited disorders, or both. Many of the acquired risk factors can be modified, thus lessening the likelihood of PE or DVT.

### What Are the Acquired Risk Factors?

Long-haul air travel is the most talked-about risk factor for PE. Other acquired risk factors include obesity, cigarette smoking, hypertension, immobilization, surgery, and trauma. Chronic medical illnesses such as congestive heart failure, chronic obstructive pulmonary disease, and cancer also predispose to PE. PE is also a prominent women's health issue. Risk factors include oral contraceptives, pregnancy, and hormone replacement therapy.

### What Are the Inherited Disorders?

Heredity plays an important role in a patient's susceptibility to PE. We are just beginning to develop genetic tests (such as factor V Leiden and the prothrombin gene mutation) that can identify those who are predisposed. The presence of these risk

factors is sometimes called a prothrombotic or thrombophilic state.

### How Is the Diagnosis Established?

DVT most often originates in the calf, with a persistent cramping or "charley horse" that intensifies over several days. Leg swelling and discoloration may accompany the increase in discomfort. Upper-extremity DVT may cause otherwise unexplained upper arm or neck swelling. The most frequently used diagnostic imaging test is the noninvasive venous ultrasound examination.

Many patients with PE have a vague sense that something is wrong but have difficulty defining or describing the problem. Consequently, they often delay seeking medical attention. At times, because symptoms are so vague and nonspecific, medical professionals will diagnose anxiety rather than PE. To establish the diagnosis of PE, the most frequently used noninvasive imaging test is the rapid-speed chest computed tomography (CT) scan.

### What Are the Warning Signals of PE?

- Unexplained shortness of breath (the most common symptom of PE)
- Chest discomfort, usually worse with a deep breath or coughing
- A general sense of anxiety or nervousness
- Lightheadedness or blacking out

### What Should I Expect at the Hospital?

#### Definitely Expect:

- Questions about symptoms of chest or leg discomfort, breathing difficulties, or lightheadedness
- Questions about whether you or your family members have suffered prior VTE
- A check of your blood pressure, pulse rate, breathing rate, heart, lungs, and legs
- An ECG and chest x-ray

#### Possibly Expect:

- A blood test (D-dimer) that screens for PE (If results are normal, PE is extremely unlikely.)

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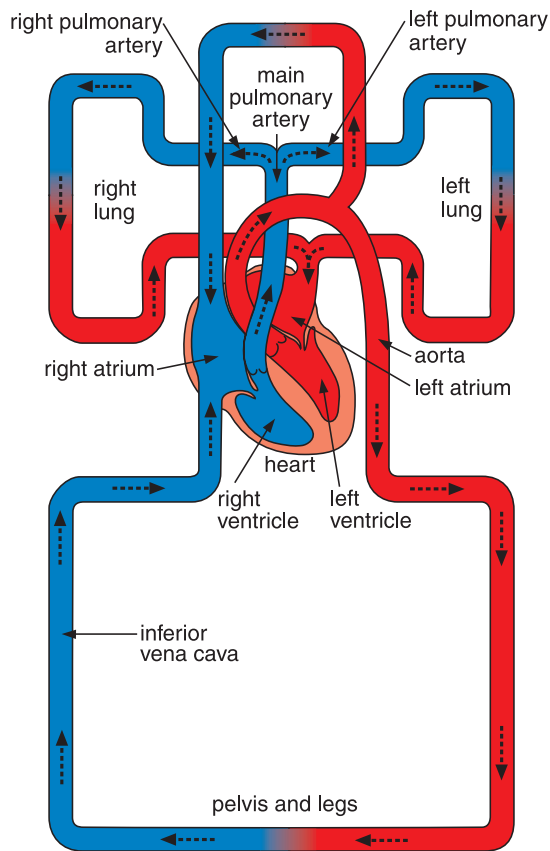
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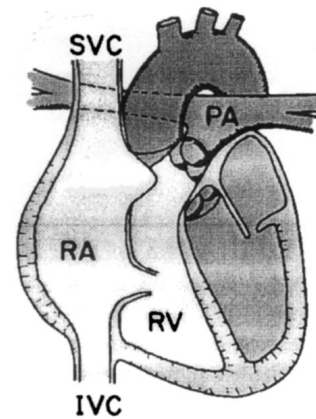
**Figure 1.** The arterial (red) and venous systems (blue) are depicted. The left ventricle (LV) ejects oxygenated blood into the aorta, which circulates blood to vital organs. Deoxygenated blood travels through the venous system to the right atrium, right ventricle, and pulmonary artery. Adapted with permission from MediClip, Clinical Cardiopulmonary Images 1997, CCP01026.TIF, Williams & Wilkins, Baltimore, Md.

- A chest CT scan, which directly images blood clots causing blockages in the pulmonary arteries
- A lung scan, which indirectly identifies areas of decreased blood flow in the lung tissue as a consequence of PE
- Blood tests to detect a prothrombotic state, especially in relatively young and otherwise healthy patients with PE or DVT

### What Treatment Will I Receive?

PE can range from mild to severe. Mild PE is managed with blood thinners (anticoagulation). Severe PE requires additional measures, such as clot busters (thrombolytic therapy) or embolectomy, a procedure in which the clot is removed with either a catheter or surgery.

Anticoagulation begins with a combination of 2 blood thinners: (1) heparin, administered intravenously or by injection, and (2) warfarin, an oral blood thinner. Heparin comes in 2 principal forms. The traditional unfractionated form ordinarily requires intravenous administration. There is no fixed dose for this type of heparin. Instead, the dose is titrated to a blood test called the partial thromboplastin



**Figure 2.** Deoxygenated blood returns to the heart and lungs for reoxygenation. SVC indicates superior vena cava; IVC, inferior vena cava; RA, right atrium; RV, right ventricle; and PA, pulmonary artery. Adapted with permission from MediClip, Clinical Cardiopulmonary Images 1997, CATHRHT.TIF, Williams & Wilkins, Baltimore, Md.

time. This blood test is usually performed several times daily for the first few days and then once daily thereafter. More recently, low-molecular-weight heparins have begun replacing unfractionated heparin. Low-molecular-weight heparins are ordinarily prescribed in proportion to the patient's weight, require no blood testing, and necessitate injection once or twice daily.

We overlap heparin treatment with warfarin until the oral blood thinner becomes effective, usually after 5 to 10 days of combined therapy. We determine the proper dose of warfarin by a blood test reported as the International Normalized Ratio (INR). The target INR range is usually between 2.0 and 3.0. Interactions with food, alcohol, and other drugs can dramatically alter the INR. Sometimes, major fluctuations in the INR occur for no apparent reason. Too high an INR may result in bleeding as a side effect. Too low an INR may result in recurrent clotting. Patients may need their INR checked every few weeks or months, according to the stability of the readings. It is important for patients to keep a record of their values over time.

PE is usually treated in the hospital with intravenous unfractionated heparin as a bridge to warfarin. In contrast, DVT can often be managed successfully on an outpatient basis with low-molecular-weight heparin injections as a bridge to oral anticoagulation with warfarin. The most controversial area in VTE therapy is the optimal duration of warfarin anticoagulation. The current recommendation is usually at least 6 months of anticoagulation, but it can sometimes be longer, according to individual patient circumstances.

In patients who cannot tolerate anticoagulation or those for whom anticoagulation fails, a permanent metal filter is inserted into the inferior vena cava, the largest vein below the heart, to prevent large blood clots from reaching the pulmonary arteries and causing PE. Unfortunately, the filter devices do not halt the clotting process. Their presence predisposes to future venous clots on or below the filter.

## Prevention

Maintaining ideal body weight with a healthy nutritional program and exercise regimen will generally reduce the likelihood of venous thrombosis. Specific other measures are shown below:

- To prevent immobility or inactivity: Walk, jog, bicycle, or swim.
- To prevent obesity: Limit caloric intake, exercise, and avoid saturated fats.
- To prevent VTE during air travel: Drink extra water, walk if feasible, wear vascular compression stockings, and avoid alcohol.
- To quit cigarette smoking: Use nicotine patch, gum, or spray, or consider the prescription drug bupropion.
- To control hypertension: Self-check blood pressure, and report elevated readings to primary care provider.
- To deal with a known genetic predisposition to VTE: Alert your healthcare provider about the family history and any abnormal blood tests related to a clotting tendency.
- To prevent VTE after trauma or surgery: Discuss with the treating physician the implementation of measures such as mechanical compression boots for the legs and/or blood thinners given either intravenously or as injections.

- To prevent VTE during a hospitalization precipitated by a medical condition: Discuss with the treating physician measures such as mechanical compression boots for the legs and/or blood thinners given either intravenously or as injections.
- To prevent VTE when planning birth control: Discuss VTE risks, and consider alternatives to oral contraceptives.
- To prevent VTE during pregnancy: Consider daily self-injected heparin if considered at high risk of VTE.
- To prevent VTE during hormone replacement therapy: Keep in mind that VTE risks with hormone replacement therapy are similar to those of oral contraceptives.

## Supplemental Reading

1. Goldhaber SZ. Pulmonary embolism. *N Engl J Med*. 1998;339:93–104.
2. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). *Lancet*. 1999;353:1386–1389.
3. Goldhaber SZ, ed. Frequently asked questions of the Pulmonary Embolism Support Group, Brigham and Women's Hospital. Available at: [http://icoper.cineca.com/faq\\_icoper.htm](http://icoper.cineca.com/faq_icoper.htm). Accessed August 23, 2002.
4. Geerts WH, Heit JA, Clagett GP, et al. Prevention of venous thromboembolism. *Chest*. 2001;119:132S–175S.
5. Goldhaber SZ, Ridker PM. *Thrombosis and Thromboembolism*. New York, NY: Marcel Dekker, Inc; 2002.

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