artery pressure and in pulmonary vascular resistance. When RV wall tension rises, RV dilation and dysfunction ensue, with release of the cardiac biomarker, brain natriuretic peptide. The interventricular septum bulges into and compresses an intrinsically normal left ventricle (LV). Diastolic LV dysfunction reduces LV distensibility and impairs LV filling. Increased RV wall tension also compresses the right coronary artery, limits myocardial oxygen supply, and precipitates right coronary artery ischemia and RV microinfarction, with release of cardiac biomarkers such as troponin. Underfilling of the LV may lead to a fall in LV cardiac output and systemic arterial pressure, with consequent circulatory collapse and death.

CLASSIFICATION OF PULMONARY EMBOLISM AND DEEP VENOUS THROMBOSIS

Pulmonary Embolism **Massive PE** accounts for 5–10% of cases, and is characterized by extensive thrombosis affecting at least half of the pulmonary vasculature. Dyspnea, syncope, hypotension, and cyanosis are hallmarks of massive PE. Patients with massive PE may present in cardiogenic shock and can die from multisystem organ failure. **Submassive PE** accounts for 20–25% of patients, and is characterized by RV dysfunction despite normal systemic arterial pressure. The combination of right heart failure and release of cardiac biomarkers indicates an increased likelihood of clinical deterioration. **Low-risk PE** constitutes about 70–75% of cases. These patients have an excellent prognosis.

Deep Venous Thrombosis **Lower extremity DVT** usually begins in the calf and propagates proximally to the popliteal vein, femoral vein, and iliac veins. Leg DVT is about 10 times more common than **upper extremity DVT,** which is often precipitated by placement of pacemakers, internal cardiac defibrillators, or indwelling central venous catheters. The likelihood of upper extremity DVT increases as the catheter diameter and number of lumens increase. **Superficial venous thrombosis** usually presents with erythema, tenderness, and a "palpable cord." Patients are at risk for extension of the thrombosis to the deep venous system.

DIAGNOSIS

Clinical Evaluation PE is known as "the Great Masquerader." Diagnosis is difficult because symptoms and signs are nonspecific. The most common symptom is unexplained breathlessness. When occult PE occurs concomitantly with overt congestive heart failure or pneumonia, clinical improvement often fails to occur despite standard medical treatment of the concomitant illness. This scenario presents a clinical clue to the possible coexistence of PE.

With DVT, the most common symptom is a cramp or "charley horse" in the lower calf that persists and intensifies over several days. Point score criteria help estimate the clinical likelihood of DVT and PE (Table 300-1). Patients with a low-to-moderate likelihood of DVT or PE should undergo initial diagnostic evaluation with D-dimer testing alone (see "Blood Tests") without obligatory imaging tests (Fig. 300-3). However, patients with a high clinical likelihood of VTE should skip D-dimer testing and undergo imaging as the next step in the diagnostic algorithm.

Clinical Pearls Not all leg pain is due to DVT, and not all dyspnea is due to PE (Table 300-2). Sudden, severe calf discomfort suggests a ruptured Baker's cyst. Fever and chills usually herald cellulitis rather than DVT. Physical findings, if present, may consist only of mild palpation discomfort in the lower calf. However, massive DVT often presents with marked thigh swelling, tenderness, and erythema. If the leg is diffusely edematous, DVT is unlikely. More probable is an acute exacerbation of venous insufficiency due to postthrombotic syndrome. Upper extremity venous thrombosis may present with asymmetry in the supraclavicular fossa or in the circumference of the upper arms.

Pulmonary infarction usually indicates a small PE. This condition is exquisitely painful because the thrombus lodges peripherally, near the innervation of pleural nerves. *Nonthrombotic PE* etiologies include fat embolism after pelvic or long bone fracture, tumor embolism, bone marrow, and air embolism. Cement embolism and bony fragment embolism can occur after total hip or knee replacement. Intravenous drug users may inject themselves with a wide array of substances that can embolize such as hair, talc, and cotton. *Amniotic fluid embolism* occurs when fetal membranes leak or tear at the placental margin.

TABLE 300-1 CLINICAL DECISION RULES

Low Clinical Likelihood of DVT if Point Score Is Zero or Less; Moderate Likelihood if Score Is 1 to 2; High Likelihood if Score Is 3 or Greater	
Clinical Variable	**DVT Score**
Active cancer	1
Paralysis, paresis, or recent cast	1
Bedridden for >3 days; major surgery <12 weeks	1
Tenderness along distribution of deep veins	1
Entire leg swelling	1
Unilateral calf swelling >3 cm	1
Pitting edema	1
Collateral superficial nonvaricose veins	1
Alternative diagnosis at least as likely as DVT	–2
High Clinical Likelihood of PE if Point Score Exceeds 4	
Clinical Variable	**PE Score**
Signs and symptoms of DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate >100/min	1.5
Immobilization >3 days; surgery within 4 weeks	1.5
Prior PE or DVT	1.5
Hemoptysis	1.0
Cancer	1.0

Nonimaging Diagnostic Modalities • ***BLOOD TESTS*** The quantitative *plasma D-dimer enzyme-linked immunosorbent assay (ELISA)* rises in the presence of DVT or PE because of the breakdown of fibrin by plasmin. Elevation of D-dimer indicates endogenous although often clinically ineffective thrombolysis. The sensitivity of the D-dimer is >80% for DVT (including isolated calf DVT) and >95% for PE. The D-dimer is less sensitive for DVT than for PE because the DVT thrombus size is smaller. A normal D-dimer is a useful "rule out" test. However, the D-dimer assay is not specific. Levels increase in patients with myocardial infarction, pneumonia, sepsis, cancer, and the postoperative state and those in the second or third trimester of pregnancy. Therefore, D-dimer rarely has a useful role among hospitalized patients, because levels are frequently elevated due to systemic illness.

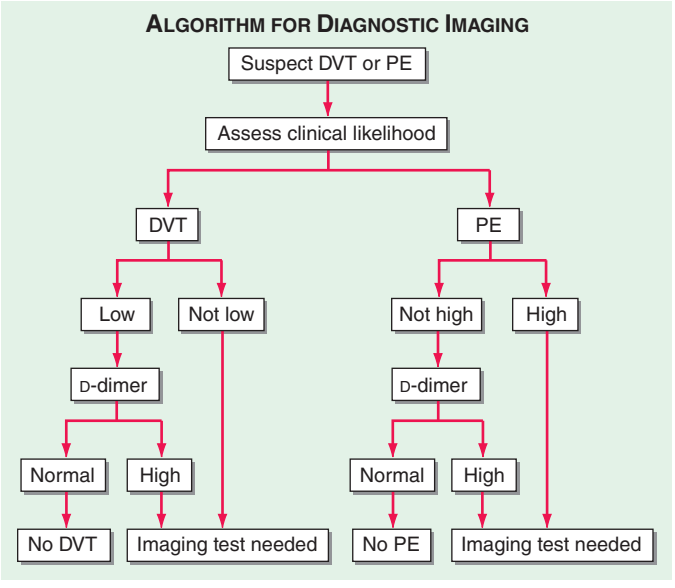

FIGURE 300-3 How to decide whether diagnostic imaging is needed. For assessment of clinical likelihood, see Table 300-1.