INTRODUCTION

Venous thromboembolism (VTE) is relatively common in clinical practice. Data suggest that the annual incidence of a first episode of deep venous thrombosis (DVT) ranges from 60 to 180 cases per 100,000 people, with more than 300,000 new cases occurring in the United States every year [1]. There is a male preponderance in the incidence of pulmonary embolism (PE). DVT and PE are part of the same disease process. Mortality and recurrence rates remain high.

The diagnosis of PE and DVT cannot be made on the basis of history and physical examination alone but must be conclusively proven or disproven with objective diagnostic testing. Autopsy findings show that the diagnosis is often missed antemortem. Successful management of VTE depends upon knowing the risk factors that increase likelihood of VTE, recognizing suggestive symptoms and signs of VTE, and proceeding with appropriate diagnostic testing and therapy as quickly as possible.

CASE STUDY

Initial Presentation

A 57-year-old obese woman presents to the emergency department of her local hospital with diaphoresis and dyspnea.

History

The patient’s symptoms began at approximately 9:00 pm after standing up from a chair. She did not attempt to call her physician given the later hour. Her symptoms were still present after 30 minutes of resting, so she had her husband drive her to the emergency department. The patient was discharged from the hospital 16 days ago after a right total knee replacement. She continued to have pain despite taking analgesics and, because of the pain, avoided ambulating as much as possible. Two days ago she experienced pain and swelling of the right leg but attributed this to the normal healing process. On presentation to the emergency department, the patient reports that the pain in her right leg persists but the swelling has improved slightly.

The patient takes a nonsteroidal anti-inflammatory drug for her rheumatoid arthritis and has been on postmenopausal hormone replacement therapy for 2½ years. The patient denies a history of fever, cough, wheezing, or lightheadedness. There is no history of smoking or VTE.

Physical Examination

On physical examination, the patient appears dyspneic and extremely anxious. She is tachycardic, with a heart rate of 115 bpm. Lungs are clear on auscultation. Her right calf and distal thigh are swollen.

• What are risk factors for VTE?

Risk Factors

This patient has several risk factors for developing VTE. Patients have a predisposition for DVT and PE up to 1 month postoperatively. Huber and colleagues [2] found that postoperative PE occurred a median of 18 days after surgery. In addition, this patient’s obesity, use of hormone replacement therapy, and decreased mobility place her at further risk for developing VTE. It is interesting that this patient has been taking hormones for 2½ years; it has been suggested that the risk of VTE is highest at the start of hormone therapy [3]. Other risk factors for VTE include malignancy, thrombophilia, high blood pressure, oral contraceptive use, and heavy cigarette smoking. The risk of PE increases by about threefold in smokers compared with nonsmokers.

Hypercoagulability with subsequent thromboembolic disease is a major cause of morbidity and mortality in cancer patients. In a few patients, venous thrombosis is the initial presentation of undiagnosed metastatic cancer. Studies have shown that the likelihood of cancer in patients with thrombosis but no identifiable risk factor for thrombosis is approximately 10% to 20% [4]. Some neoplastic cells actually
synthesize various procoagulants, which contribute to the increased predisposition to VTE.

Activated protein C resistance, antiphospholipid antibodies, and hyperhomocysteinemia also can lead to an increased risk of venous thrombosis. The factor V Leiden defect is the most common hereditary thrombophilic disorder and results in activated protein C resistance. It is found in 6% of whites of European descent and rarely in other ethnic groups. The risk of a thrombotic episode for homozygotes is 80 times greater than for normal individuals. Heterozygotes have a 7-times greater risk [5]. A more recently recognized genetic abnormality, the prothrombin G20210A defect, is being increasingly recognized.

The antiphospholipid syndrome was first described in patients with systemic lupus erythematosus. Antiphospholipid antibodies are a family of acquired circulating autoantibodies to negatively charged phospholipids and proteins important in coagulation. One to five percent of the population has antiphospholipid antibodies. The most common manifestations of this disorder are venous and arterial thrombotic disease.

Hyperhomocysteinemia is generally caused by mild deficiencies of folate and occasionally by inadequate intake of vitamin B12 or B6 [6]. About 20% of VTE in young patients may be associated with elevated levels of homocysteine in the blood. Hyperhomocysteinemia predisposes to thrombosis by damaging endothelium and causing platelet aggregation, and adhesion. This results in thrombosis formation by various mechanisms. Measuring fasting serum homocysteine levels can identify most patients with hyperhomocysteinemia. The most sensitive method for detecting hyperhomocysteinemia is measuring serum homocysteine levels before and after oral methionine loading. A recent study suggested that folic acid fortification has a substantial effect on plasma folate and homocysteine concentrations in a population-based sample of middle-aged and older adults [7].

**Table 1. Diagnoses That Can Resemble Pulmonary Embolism**

<table>
<thead>
<tr>
<th>Diagnosis</th>
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</thead>
<tbody>
<tr>
<td>Chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>exacerbation</td>
</tr>
<tr>
<td>Pneumonia</td>
</tr>
<tr>
<td>Anxiety</td>
</tr>
<tr>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
</tr>
<tr>
<td>Musculoskeletal pain</td>
</tr>
<tr>
<td>Myocardial infarction</td>
</tr>
</tbody>
</table>

*Pulmonary embolism may also occur concomitantly with some of these entities.*

**Clinical Findings in VTE**

Symptoms and signs of DVT include swelling, warmth, erythema, pain, or tenderness in the lower extremities. PE often manifests as nonspecific clinical symptoms. The sudden onset of dyspnea is a common symptom of PE. In some patients, the sudden onset of shortness of breath may be the only symptom. PE should always be considered in the setting of unexplained dyspnea and should be considered even in patients who have other possible explanations for their symptoms (e.g., pneumonia, chronic obstructive lung disease, or congestive heart failure). Patients with these conditions can develop PE superimposed over their underlying cardiopulmonary disorder. Other symptoms that may be associated with PE include cough, syncope, pleuritic chest pain, anxiety, general weakness, and hemoptysis. The physical examination may reveal suggestive findings such as unexplained tachycardia, but no physical findings are specific for PE.

**Presumptive Diagnosis in This Patient**

This patient presented to the emergency department with the acute onset of dyspnea, diaphoreses, and anxiety associated with tachycardia. These nonspecific symptoms raise suspicion for a number of problems, including an “anxiety attack.” Table 1 lists several other diagnoses that can resemble PE. However, the patient’s recent total knee replacement, particularly in the setting of obesity, clearly indicates the need for further evaluation for PE in light of her clinical presentation. In this particular patient, there are obvious risk factors for her presumed PE, and little would appear to be gained by testing for other thrombophilic disorders.
the chest radiograph is nondiagnostic for other cardiopulmonary disease, should prompt further evaluation for PE. It is not always clear, however, whether an altered PaO₂ level is the result of PE. The patient’s baseline PaO₂ level is often unknown, and associated cardiac and respiratory disease can also result in hypoxemia [8]. Although hypoxemia is common in acute PE, it is not present in all cases. Some patients, particularly young patients, may have PE and a normal PaO₂. Thus, the diagnosis of acute PE cannot be confirmed or excluded on the basis of a PaO₂.

The alveolar-arterial difference is usually abnormal in patients with PE. Patients with PE may breathe hard enough to decrease the PaCO₂ substantially, which may increase the PaO₂ level enough to keep it in the normal range. Thus, pulse oximetry may suggest adequate oxygenation and be misleading in the presence of acute PE. A normal alveolar-arterial difference cannot reliably exclude the diagnosis of PE, however. In a group of patients from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) [9], a small subset of patients with confirmed PE had a normal alveolar-arterial difference and a normal PaO₂.

### D-Dimer Testing

The D-dimer test is increasingly being used, particularly to exclude PE. When fibrin clots undergo fibrinolysis, D-dimer (a specific degradation product) is released into the circulation. Different assays of D-dimer have been analyzed to determine the utility of this test in detecting VTE. Perrier and associates [10] found that the negative predictive value of the quantitative ELISA plasma D-dimer in the emergency room setting was approximately 99%. However, other studies have shown that a normal latex agglutination D-dimer does not appear to be reliable in excluding PE.

In evaluating results, pretest probability should be considered. When clinical suspicion for VTE is very high, a negative D-dimer test result should not preclude further evaluation for PE. Different assays, including rapid bedside assays, are becoming increasingly available, and additional studies should further clarify the role of these tests. As shown by Ginsberg and colleagues [11], a normal D-dimer test result is useful in excluding PE in patients with a low pretest probability of PE or a nondiagnostic lung scan.

### Other Tests

Serum lactate dehydrogenase levels are often elevated in patients with PE. However, this is a nonspecific finding that is not useful in confirming or refuting the diagnosis.

### Chest Radiography

Most patients with PE have an abnormal chest radiograph, although the abnormalities are nearly always nonspecific. Chest radiograph abnormalities include elevation of a hemidiaphragm, atelectasis, pleural effusion, and pulmonary infiltrate(s). Greenspan and colleagues [12] demonstrated poor sensitivity (33%) and specificity (59%) in using the chest radiograph to predict PE. Decreased vascularity (Westermark’s sign) and suggestion of pulmonary infarction (Hampton’s hump) are infrequent findings. A normal chest radiograph in a patient with hypoxemia and dyspnea without an identifiable cause is suggestive of PE.

### Electrocardiography

Electrocardiography may be helpful in suggesting PE but is not diagnostic and does not provide sufficient information to exclude PE. While the S\_Q\_T\_ pattern has been suggested as useful, it is nondiagnostic. There are no electrocardiographic findings that are specific for PE.

### Leg Imaging

Commonly used diagnostic tools available to the clinician to evaluate the veins of the legs include compression ultrasonography, impedance plethysmography, magnetic resonance imaging (MRI), and contrast venography. Venography has been considered the most accurate (ie, the criterion standard) test for DVT. In the setting of clinically suspected DVT, a lower extremity ultrasound is considered both accurate and cost-effective as the initial diagnostic test [13]. However, caution is advised when ultrasonography is used as a screening tool in patients without symptoms or signs of DVT. Patients with lower extremity DVT often do not exhibit pain, swelling, warmth, or erythema. When patients are asymptomatic, a lower extremity ultrasound is less sensitive for the diagnosis. Venography or MRI should be performed when ultrasound is nondiagnostic or if patient factors (eg, plaster casts) render the use of ultrasound difficult or impossible.

As ultrasound findings may not return to normal after acute DVT has been diagnosed, it is important to use caution when attempting to diagnose recurrent DVT using ultrasonography. It is essential to distinguish between acute and chronic DVT because thrombi adhere to the wall of veins.

- Which imaging studies should be considered in a patient presenting with possible VTE?

The diagnostic test ordered depends upon whether the patient presents with suspected DVT or suspected PE. When the presentation suggests DVT, a leg study is ordered; if the clinical picture suggests PE, a lung imaging study is ordered.

- What is the value of radiographic and electrocardiographic studies in patients with suspected PE?
after several weeks and are not likely to embolize. Two prospective clinical investigations have shown that the rate of normalization of an abnormal ultrasound test after a first episode of acute DVT is only 44% to 52% after 6 months and 55% after 12 months [13,14]. Therefore, in the setting of previous DVT, ultrasound should be interpreted with caution. Since previous DVT is a risk factor for a subsequent episode, it has been suggested that a repeat ultrasound be performed between 3 and 6 months after anticoagulation to serve as a baseline should symptoms recur. Additionally, ultrasound techniques are less reliable in detecting DVT in the calf veins and iliac veins. Contrast venography or MRI is recommended if these areas are of concern.

The use of MRI for diagnosing both DVT and PE has been explored. Although there is little information on the value of MRI as a screening modality for DVT, early reports have shown that MRI is more than 90% sensitive and specific for acute symptomatic proximal DVT [15–17]. Advantages of MRI for diagnosing DVT include the lack of need for intravenous contrast and the ability to evaluate the pelvic veins and inferior vena cava [18]. However, MRI is expensive and currently is not an appropriate initial diagnostic modality for most patients with suspected DVT.

**Lung Imaging**

When the patient's presentation suggests acute PE rather than DVT, the focus is on lung imaging. The ventilation-perfusion (V/Q) scan has been the most commonly ordered diagnostic test for patients with suspected PE. A normal scan excludes PE. A high probability scan in a clinical setting consistent with acute PE is considered diagnostic. Most V/Q scans, however, are of low or intermediate probability and therefore inconclusive. The presence of other lung diseases such as chronic obstructive pulmonary disease or pneumonia are frequently present in the setting of PE and this complicates the diagnostic evaluation.

The PIOPED study [19] attempted to determine the sensitivity and specificity of the V/Q scan in patients with suspected acute PE (Table 2). This study showed that PE is often present in patients with nondiagnostic scans when there is a high clinical suspicion of PE. When clinical suspicion is high, a high probability lung scan is associated with proven PE in 96% of cases. However, even a low probability scan is associated with proven PE in 40% of cases when the clinical suspicion is high.

When the lung scan is nondiagnostic but the clinician feels that there is a high clinical probability of PE, pulmonary angiography should be considered as a safe and accurate diagnostic modality [20]. It appears that the procedure can generally be safely performed when the platelet count is at least 75,000/mm$^3$ and if coagulation studies are normal or minimally abnormal [21]. However, severely ill intensive care unit patients may not be stable for transport to angiography. An alternative approach would be to perform an ultrasound of the lower extremities to determine the need for anticoagulation. A low probability scan together with a negative lower extremity study (ie, ultrasound or impedance

### Table 2. Clinical Assessment and Ventilation-Perfusion (V/Q) Scan Probability in the PIOPED Study*

<table>
<thead>
<tr>
<th>V/Q Scan</th>
<th>High Likely (80–100%)</th>
<th>Uncertain (20–79%)</th>
<th>Unlikely (0–19%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High probability</td>
<td>28/29 (96%)</td>
<td>70/80 (88%)</td>
<td>5/9 (56%)</td>
</tr>
<tr>
<td>Intermediate probability</td>
<td>27/41 (66%)</td>
<td>66/236 (28%)</td>
<td>11/68 (16%)</td>
</tr>
<tr>
<td>Low probability</td>
<td>6/15 (40%)</td>
<td>30/191 (16%)</td>
<td>4/90 (4%)</td>
</tr>
<tr>
<td>Near normal/normal</td>
<td>0/5 (0%)</td>
<td>4/62 (6%)</td>
<td>1/61 (2%)</td>
</tr>
<tr>
<td>Total</td>
<td>61/90 (68%)</td>
<td>170/569 (30%)</td>
<td>21/228 (9%)</td>
</tr>
</tbody>
</table>

Adapted with permission from Value of the ventilation/perfusion scan in acute pulmonary embolism. Results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). The PIOPED Investigators. JAMA 1990;263:2753–9.

*Table shows number of patients with proven PE/number of patients with the specific scan result (%) for each of 3 levels of clinical probability.
plethysmography) is negative, additional diagnostic testing is recommended. Such testing could involve pulmonary angiography or serial lower extremity ultrasound testing.

Several clinical studies have suggested the utility of MRI for diagnosing PE [23]. In patients with suspected PE, the average specificity of CT was 89% compared with 90% for MRI in 1 study [24].

What is the role of spiral CT in suspected PE?

Spiral CT scanning has been utilized increasingly for suspected PE. This technique appears to have very good sensitivity for emboli in the main, lobar, or segmental pulmonary arteries and is less sensitive for subsegmental emboli. In the PIOPED, the interobserver agreement for subsegmental emboli even with angiography was only 66%. However, the importance of subsegmental emboli needs to be further defined. Studies evaluating the diagnostic ability of spiral CT for diagnosing PE have shown a sensitivity ranging from 53% to 100% and a specificity ranging from 78% to 97% [25–31]. However, study design has been variable. One study utilized spiral CT in patients with suspected PE after V/Q scan was found to be of intermediate probability and lower extremity ultrasound was normal. At 3-month follow-up, 6 of 112 patients (5.4%) with normal spiral CT had experienced PE (6 apparently false-negative CT scans) [30]. It is expected that advances in spiral CT technology will further increase its utility for diagnosing PE. One should keep in mind that spiral CT scanning requires contrast dye injection and caution should be used in patients with renal insufficiency. As with other techniques, reader expertise is required. Advantages and disadvantages of the spiral CT scan for diagnosing acute PE are listed in Table 3.

Which diagnostic approach to VTE is most cost-effective?

There are few good cost-effectiveness analyses available with regard to VTE. Van Erkel et al [32] performed a cost-effectiveness analysis on diagnostic algorithms for PE. Strategies that included spiral CT proved more cost-effective than pulmonary angiography strategies. The ultrasound followed by spiral CT strategy seemed most cost-effective. These results also suggested that the use of the d-dimer test does not, at present, result in optimally cost-effective strategies. A limitation of this analysis was that it was necessary to assume conditional independence of the diagnostic tests in order to simplify the decision model. The actual sensitivity and specificity of a test may actually depend on the results of the preceding test and are unlikely to remain constant. At this point, recommendations for cost-effective algorithms serve only as approximations. More data are needed.

Diagostic Testing

A V/Q scan and chest radiograph are ordered. The patient is noted to have minimal atelectasis in the right lower lung, but otherwise her chest radiograph is normal. The patient’s V/Q scan is read as high probability for PE, confirming the diagnosis.

Which therapies are available for the treatment of VTE?

Acute Treatment

DVT and PE represent 2 manifestations of the same disease and usually have the same treatment implications: Anticoagulation is usually indicated. Standard unfractionated heparin has been the primary initial therapy for acute VTE. It has been demonstrated that initial therapy with an oral anticoagulant and no heparin may intensify hypercoagulability and increase the frequency of recurrent venous thromboembolism.

Anticoagulation

It has been recommended that in the absence of overt contraindications (eg, active gastrointestinal hemorrhage), patients with a moderate or high clinical likelihood of PE

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Table 3. Potential Advantages and Limitations of Spiral CT Scanning for the Diagnosis of Pulmonary Embolism

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity (except for subsegmental emboli)*</td>
<td>Reader expertise</td>
</tr>
<tr>
<td>Specificity</td>
<td>Expense†</td>
</tr>
<tr>
<td>Availability</td>
<td>Not portable</td>
</tr>
<tr>
<td>Safety</td>
<td>Contraindications (renal insufficiency, contrast allergy)</td>
</tr>
<tr>
<td>Relative rapidity of procedure</td>
<td>Poor visualization of certain regions</td>
</tr>
<tr>
<td>Diagnosis of other disease entities</td>
<td></td>
</tr>
<tr>
<td>Retrospective reconstructions</td>
<td></td>
</tr>
<tr>
<td>Advancing technology</td>
<td></td>
</tr>
</tbody>
</table>

*In clinical trials to date, high sensitivity and specificity have been limited to emboli in the main, lobar, and segmental vessels.

†A number of issues impact upon cost-effectiveness, including the need for subsequent studies after a nondiagnostic initial study (eg, V/Q scan) and the potential cost-savings in patients with alternative diagnoses detected at CT scan.
should receive anticoagulation with heparin during the diagnostic evaluation [33]. It is no longer appropriate to consider a standard bolus of heparin at 5000 U per hour followed by a constant infusion of 1000 U per hour. Such a dosing regimen often delays achieving a therapeutic activated partial thromboplastin time (aPTT). It has been demonstrated that an inadequate aPTT at 24 hours after the initiation of therapy is associated with a higher VTE recurrence rate. Weight-based regimens are the standard of care. A bolus of unfractionated heparin of 80 U/kg followed by a continuous infusion (initiated at a dose of 18 U/kg per hour) can be given to achieve an aPTT of 60 to 80 seconds; with this regimen, the recurrence rate for acute VTE has been shown to be lower at 24 hours [34]. Warfarin can be started on day 1 once a therapeutic partial thromboplastin time has been achieved.

An alternative treatment for patients presenting with acute DVT is the use of low molecular weight heparin (LMWH) [33,35]. These smaller heparins (mean molecular weight, 5000 daltons) have substantial advantages over standard unfractionated heparin (mean molecular weight, 15,000 daltons) (Table 4). Advantages include excellent bioavailability with a more predictable dose-response, subcutaneous delivery, and lack of need for monitoring. There also appears to be significantly less heparin-induced thrombocytopenia with LMWHs. These drugs have relatively more anti-factor Xa effect than antithrombin effect and also result in tissue factor pathway inhibitor release.

In the United States, only one LMWH, enoxaparin, is FDA-approved for use as treatment in patients presenting with established DVT (with or without PE). Because of the ease with which these drugs can be used, outpatient therapy of carefully selected patients is becoming increasingly common and has been shown to be cost-effective. Clinical trials have demonstrated that outpatient therapy with enoxaparin at 1 mg/kg every 12 hours is at least as effective as standard heparin for the treatment of established DVT [36]. While nadroparin also appears efficacious in this setting, it is not approved for use in the United States [37]. Patients with a first episode of proximal DVT, with no risk factors for bleeding (eg, thrombocytopenia, liver diseases, renal insufficiency), and who demonstrate a good medical compliance history are candidates for outpatient therapy with LMWH.

For inpatients, enoxaparin can be administered as either 1.5 mg/kg once daily or 1 mg/kg every 12 hours. The heparin (standard or LMWH) is discontinued after approximately 5 days if adequate warfarin levels have been obtained (ie, 2 consecutive international normalized ratio [INR] values of 2.0 or greater, measured 24 hours apart). In general, a treatment period of 3 to 6 months is recommended.

For patients presenting with PE, standard unfractionated heparin is recommended as initial therapy. Preliminary data suggest that 1 or more LMWHs should be effective in this setting, and it is likely that additional clinical studies will confirm this. One large multicenter clinical trial performed in Europe suggests that another LMWH, tinzaparin, is effective as treatment in patients presenting with acute PE [38]. This drug is not, at present, approved for use in the United States. The current indications for various LMWH products are outlined in Table 5.

**Thrombolytic Therapy**

When a patient develops PE with extreme hypoxemia or particularly with hypotension, it suggests an extensive embolic burden and more aggressive therapy with thrombolytic therapy is warranted unless contraindications exist. Streptokinase, urokinase, and tissue-type plasminogen activator are approved for use for massive PE and are administered intravenously [39,40]. Careful attention must be paid to potential contraindications such as intracranial pathology, surgery within the previous 2 weeks, or other potential for significant bleeding. This form of therapy will not be discussed here in detail.

**Filters**

Occasionally, anticoagulation therapy is contraindicated in patients with DVT and PE, often because of the risk of bleeding. In this setting or when bleeding has already occurred, it is recommended that an inferior vena caval filter be placed [33]. Some individuals believe that a filter should also be considered in the setting of massive PE if it appears that additional emboli may be fatal. One study to assess the efficacy and safety of vena caval filters in the prevention of PE in patients with proximal DVT [41] found that in addition to heparin therapy, the use of a permanent filter initially reduced the occurrence of symptomatic or asymptomatic PE. However, no effect was seen on mortality. After 2 years, there was a significant increase in recurrent DVT, possibly related to thrombosis at the filter site.

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**Table 4. Potential Advantages of Low Molecular Weight Heparins Over Unfractionated Heparin**

- Similar or superior efficacy
- Similar or superior safety
- Superior bioavailability
- Once or twice daily dosing
- No laboratory monitoring*
- Less phlebotomy
- Subcutaneous administration*
- Earlier ambulation
- Home therapy in certain patient subsets

*For both prophylaxis and treatment.
Prophylaxis

Subcutaneous standard unfractionated heparin at 5000 U every 8 to 12 hours, LMWH, or warfarin are the most commonly utilized regimens for prevention of DVT, but the specific DVT prophylaxis regimen administered depends upon the clinical setting. There are currently 3 LMWHs approved by the FDA for the prophylaxis of VTE: enoxaparin, dalteparin, and ardeparin. LMWHs are administered in a fixed dose for prophylaxis against VTE. As seen in Table 5, enoxaparin 30 mg every 12 hours with the initial dose given 12 to 24 hours postoperatively and continued every 12 hours for 7 to 10 days can be administered to patients undergoing total hip or knee replacement surgery. The results of several studies suggest that a screening lower extremity ultrasound at discharge in high-risk orthopedic patients receiving enoxaparin or warfarin prophylaxis is unnecessary [42,43]. Specific prophylaxis regimens and doses are outlined in more detail in the 1998 American College of Chest Physicians guidelines [44].

Treatment

The patient is admitted to the hospital and treated with intravenous standard unfractionated heparin; she is switched to therapy with warfarin when her aPTT level reaches the therapeutic range; the INR is maintained between 2.0 and 3.0. The patient is discharged on the 4th hospital day on warfarin. The patient continues on warfarin therapy for 6 months, during which time she is closely monitored by her primary care physician. Initially, INR testing is done weekly. When a stable INR between 2.0 and 3.0 is achieved, monitoring frequency is reduced to every 2 weeks.

References


Table 5. Low Molecular Weight Heparin for Prophylaxis and Treatment of Established Venous Thromboembolism

<table>
<thead>
<tr>
<th>Drug</th>
<th>Prophylaxis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin</td>
<td>30 mg q12h or 40 mg qd</td>
<td>1 mg/kg bid*</td>
</tr>
<tr>
<td>Dalteparin</td>
<td>2500–5000 U qd</td>
<td>200 U/kg qd</td>
</tr>
<tr>
<td>Tinzaparin</td>
<td>75 U/kg qd</td>
<td>175 U/kg qd</td>
</tr>
<tr>
<td>Nadroparin</td>
<td>41–62 U/kg qd</td>
<td>&lt; 50 kg 4100 U bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50–75 kg 6150 U bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 70 kg 9200 U bid</td>
</tr>
<tr>
<td>Reviparin</td>
<td>4200 U qd</td>
<td>35–45 kg 3500 U bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>46–60 kg 4200 U bid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 60 kg 6300 U bid</td>
</tr>
<tr>
<td>Ardeparin</td>
<td>50 U/kg bid</td>
<td>Not evaluated</td>
</tr>
</tbody>
</table>

*FDA-approved for use for uncomplicated deep venous thrombosis in inpatients or outpatients.
†FDA-approved for use for uncomplicated deep venous thrombosis ± PE in inpatients.
ACUTE VENOUS THROMBOEMBOLISM

(continued from page 54)


The Unanticipated Response

E.R. on the horn
His amylase at midnight
1600 again!
The third time
in three months.
The seventh time
this year.
It was gin again
A full fifth
Beer chasers
and Oh, the remorse
The kindly white stubble
The kindly black face
The kindly eyes
I said, “Grandpa
don’t do it so slow
You are wasting the valuable time
and the talents of a team
of dedicated professionals.
Shoot yourself!”
So, the next day
he swore off
saying I’d talked to him
“like a son.”

John Brodsky, MD
Swarthmore, PA