Pulmonary Embolism in Orthopaedic Patients: Diagnosis and Management

Abstract

Orthopaedic patients are at particularly high risk for pulmonary embolism. There has been a trend recently toward overdiagnosis of pulmonary embolism; thus, evaluation of the nature of a clinically relevant pulmonary embolism is needed, as is assessment of the timing, risks, and outcomes of therapeutic anticoagulation in surgical patients. Recent literature shows the incidence of pulmonary embolism to be increasing without a corresponding increase in mortality, suggesting that not all emboli may be clinically relevant and that increasingly sensitive tests may be picking up small emboli. The size and location of a clot or clots may matter when deciding on management. A risk-benefit evaluation can assist in deciding treatment.

Orthopaedic patients are at high risk for both pulmonary embolism (PE) and deep vein thrombosis (DVT). These disorders have been well studied in the arthroplasty population; an American Academy of Orthopaedic Surgeons guideline is available for public access.1 However, this guideline does not apply to the nonarthroplasty orthopaedic population and addresses prophylaxis rather than management. Here, we discuss the diagnosis and management of PE in general orthopaedics, particularly trauma.

Symptomatic PE occurs in 2% to 10% of pelvic trauma patients, and fatal PE, in 0.5% to 2%.2 Deep vein thrombi appear to occur with greater incidence in proximal rather than distal lower extremity fractures, although the clinical significance of this finding is unclear.3 The most common signs of PE are tachycardia, low oxygen saturation, and shortness of breath; however, the clinical presentation of PE is notoriously unreliable, and many embolisms are silent. In a review of 695 patients, Kim et al4 found a 27.8% rate of positive CT results for PE in postoperative orthopaedic patients. In that study, a prior history of thromboembolic disease was the only significant predictor of a positive scan; a high body mass index was a marginal predictor. Thus, a combination of patient characteristics and symptoms may predict PE.

The magnitude of PE is a spectrum ranging from central large clots to tiny subsegmental clots. The clinical relevance of this spectrum of condition is also quite varied. It is well known that untreated emboli can lead to death; however, many have no clinical sequelae. How, then, should management such as aggressive anticoagulation, which has its own risks and costs, be chosen? As methods of embolus detection become more sensitive for smaller occlusions, the clinical significance of these small emboli is called into...
question. Is the management we are instituting rational, or are we over-treating some percentage of patients?

**Diagnostic Methods and Time Trends**

PE is diagnosed in several ways. Clinically, many patients do not present with the classic symptoms of pleuritic chest pain and shortness of breath. Often, unexplained tachycardia in a postoperative patient, combined with low oxygen saturation, is all that is needed to prompt a search for PE. D-dimer, a blood test that measures fibrin degradation, may be elevated in patients with PE, but it is also elevated in postoperative patients generally and is thus unreliable as a standalone diagnostic test. The Wells score has undergone numerous iterations and remains one of the most popular clinical prediction models for PE diagnosis. The Wells score assigns points based on patient characteristics and, when combined with the D-dimer level, can be a very useful predictive tool (Tables 1 and 2). However, this score has been developed primarily from a nonsurgical patient population and thus may not apply to orthopaedic injury patients.

With regard to imaging studies for the diagnosis of PE, pulmonary angiography is the benchmark, but it is expensive and invasive. The two major and most common imaging diagnostic modalities are CT pulmonary angiogram (CTPA) and ventilation perfusion (VQ) scanning. The advantages of CTPA are direct visualization of the clot, high sensitivity, identification of alternative pathology, and rapid testing time. The advantages of VQ scanning are low radiation exposure and low cost. A randomized trial comparing the two methods in 1,417 patients showed that CTPA had a higher positive predictive value than did VQ scanning but that the two methods were similar in ruling out PEs. The negative predictive value of CTPA appears to be adequate; a meta-analysis of 23 studies showed that the 3-month rate of recurrent, symptomatic PE in 4,657 patients with high clinical suspicion for PE and a negative CTPA was 1.4%. One study in patients with moderate to high probability for PE and/or elevated D-dimer levels gave the negative predictive value of CTPA as 99.5%. CTPA is now the most common diagnostic method for PE, but it is used as part of an overall approach that includes clinical prediction, ultrasonography, and other modalities.

General time trends from the past decade show that the incidence of PE is increasing. In a sample of more than 1 million patients from the Nationwide Inpatient Sample database, the number of PE diagnoses increased from 126,546 cases in 1998 to 229,637 in 2005, but fatal PE rates dropped from 12.3% to 8.2%. The decreases in mortality rate may suggest that management of PE be-

**Table 1**

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of Points Assigned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs and symptoms of DVT (ie, minimum leg swelling, pain on palpation of deep veins)</td>
<td>3</td>
</tr>
<tr>
<td>An alternative diagnosis is less likely than PE</td>
<td>3</td>
</tr>
<tr>
<td>Heart rate &gt;100 BPM</td>
<td>1.5</td>
</tr>
<tr>
<td>Immobilization or surgery in previous 4 weeks</td>
<td>1.5</td>
</tr>
<tr>
<td>Previous DVT/PE</td>
<td>1.5</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>1</td>
</tr>
<tr>
<td>Malignancy (on treatment, treated in last 6 months, or palliative)</td>
<td>1</td>
</tr>
</tbody>
</table>

DVT = deep vein thrombosis, PE = pulmonary embolism

* The number of points is added. The higher the score, the greater the likelihood of clinical probability. (See Table 2 for percentages.) >6 = high, 2 to 6 = moderate, <2 = low probability.


**Table 2**

<table>
<thead>
<tr>
<th>Wells Score</th>
<th>Normal D-dimer (95% CI)</th>
<th>Elevated D-dimer (95% CI)</th>
<th>Overall (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>2.7 (0.3–9)</td>
<td>0 (0–13.2)</td>
<td>2 (0.2–7.1)</td>
</tr>
<tr>
<td>2–6</td>
<td>2.9 (0.4–10)</td>
<td>37.3 (25–50.9)</td>
<td>18.8 (12.4–26.6)</td>
</tr>
<tr>
<td>&gt;6</td>
<td>20 (5.1–71.6)</td>
<td>60 (32.3–83.7)</td>
<td>50 (27.2–72.8)</td>
</tr>
</tbody>
</table>

CI = confidence interval

came more effective over time; however, better treatment modalities cannot also explain the increased PE incidence. The other explanation is that our PE tests are overly sensitive. This would result in increased diagnosis of small emboli, which may have fewer clinical sequelae. In other words, the diagnosis exists but may not be clinically significant. This trend has been termed overdiagnosis by Wiener et al.\(^1\)\(^0\)

Several studies have focused on overdiagnosis of PE. An analysis of 27 million patients over a 10-year period (1994 to 2004) showed a doubling of PE diagnoses without a change in mortality rate,\(^1\)\(^1\) which suggested a strong association of the increased use of CT to explain these changes (Figure 1). This trend leads to the issue of appropriate selection of patients for these imaging studies, which are not without adverse effects (eg, radiation exposure equivalent of 100 to 400 chest radiographs). Wiener et al\(^1\)\(^0\) looked at trends before and after the advent of CTPA and reported an 81% increased incidence of PE after CTPA with little change in mortality, as well as a 71% increase in complications from anticoagulation. This suggests that overdiagnosis occurs and is prevalent; in addition, the lack of change in mortality implies that previously missed small emboli picked up by CTPA may not be clinically important. Furthermore, overdiagnosis is potentially harmful to patients when they are anticoagulated for clinically insignificant emboli. In one study, a comparison of clinical indicators of PE compared with results of CTPA showed that 25% of positive CTPA findings were not associated with high clinical probability of PE; awareness of this fact could assist in patients’ not receiving unnecessary anticoagulation.\(^1\)\(^2\)

**Clinically Relevant Pulmonary Embolism**

Symptomatic PE can present in different ways. One study describes PE according to three syndromes that increase in severity, from pulmonary infarction syndrome (least severe) to isolated dyspnea to circulatory collapse (most severe). Patients with less severe syndromes are more likely to have a normal electrocardiogram and \(\text{PaO}_2\) >80 mm Hg but are less likely to have tachypnea, dyspnea, or a high-probability VQ scan.\(^1\)\(^3\) This suggests that PE can be stratified in terms of clinical severity. It follows that some PEs are completely asymptomatic and found incidentally. The prevalence of incidental PE is approximately 2.6% according to one meta-analysis of patients undergoing CT for reasons other than PE diagnosis (eg, evaluation of metastatic disease).\(^1\)\(^4\) Incidental PE also is more common in hospitalized patients and those with cancer. Incidental PE is more likely to occur in lobar and segmental arteries. The clinical history of asymptomatic PE has been evaluated by several studies; however, the studies had small patient sample sizes, limited clinical history of the patient populations, or limited follow-up time. It is clear that more evidence is needed to assess the potential clinical relevance of asymptomatic PE.

Several studies have investigated the size and location of the embolism (central versus segmental or subsegmental) (Figure 2) in relation to clinical significance. Although some authors have found no correlation between signs and symptoms and clot size or location,\(^1\)\(^5\) others agree that clot characteristics make a difference. In a 5-year study of postoperative cancer patients, Auer et al\(^1\)\(^6\) found a 7.8% average annual increased incidence in segmental and
subsegmental PE, no change in overall incidence of central PE, and no change in overall incidence of fatal PE. Central PE was more severe than peripheral and was associated with hypoxia, tachycardia, a higher Wells score, a greater number of symptoms, higher 30-day mortality (33.3%, central PE, versus 5%, peripheral PE), and a higher rate of admission to the intensive care unit. Le Gal et al also reported that subsegmental PE presented with less dyspnea and less chance of being characterized as having a high clinical probability of PE.

Small emboli are being detected with increased frequency, but this does not appear to influence PE outcome. Carrier et al found an increased detection rate of subsegmental PE in multidetector CTPA compared with single-detector CTPA. However, the 3-month risk of venous thromboembolism (VTE) in untreated patients with suspected PE and negative CTPA was similar be-
It is clear that anticoagulation is of paramount importance in treating clinically relevant PE to prevent death. In a landmark study, Barritt and Jordan\(^24\) conducted a randomized controlled trial of patients with PE. The group that received unfractionated heparin with vitamin K antagonist (n = 16) experienced no PE recurrences or fatalities, whereas the control group (n = 19) had 10 recurrences and 5 fatalities. Since then, multiple studies have shown benefits for anticoagulation and, based on their results, societies have released guidelines for anticoagulation therapy for PE.\(^{25,26}\) The American College of Chest Physicians provides the following grade 1 (strong) recommendations: a confirmed PE is treated with low-molecular-weight heparin (LMWH), monitored intravenous or subcutaneous unfractionated heparin, weight-based subcutaneous unfractionated heparin, or

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### Characteristics of Patients With Pulmonary Embolus of Different Sizes\(^a\)

<table>
<thead>
<tr>
<th>Factor</th>
<th>Subsegmental (n = 22)</th>
<th>Segmental (n = 67)</th>
<th>Central (n = 245)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidentally found</td>
<td>14 (63.6%)(^b)</td>
<td>36 (53.7%)</td>
<td>45 (18.4%)</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>4 (18.2%)</td>
<td>16 (23.9%)</td>
<td>117 (47.8%)</td>
</tr>
<tr>
<td>Surgery or trauma</td>
<td>14 (63.6%)</td>
<td>28 (41.8%)</td>
<td>44 (18.0%)</td>
</tr>
<tr>
<td>Coexisting DVT</td>
<td>9 (40.9%)</td>
<td>41 (61.2%)</td>
<td>177 (72.2%)</td>
</tr>
<tr>
<td>Received anticoagulation</td>
<td>15 (68.2%)</td>
<td>54 (80.6%)</td>
<td>229 (93.5%)</td>
</tr>
<tr>
<td>PE recurrence</td>
<td>None</td>
<td>None</td>
<td>6 (2.4%)</td>
</tr>
<tr>
<td>PE-related death</td>
<td>None</td>
<td>3 (4.5%)</td>
<td>7 (2.9%)</td>
</tr>
</tbody>
</table>

DVT = deep vein thrombosis, PE = pulmonary embolism

\(^a\) Note the higher incidence of recurrence and death in larger clot sizes, despite receiving anticoagulation at a higher rate than that of the smaller clots. (Where applicable, all P values are <0.05.)

\(^b\) Parentheses denote percentages of total patients in group.

of subcutaneous fondaparinux for 5 days, with concurrent initiation of vitamin K antagonists (eg, warfarin) until the international normalized ratio is ≥2. For PE in a patient with transient risk factors, 3 months of treatment is recommended; for PE in a patient without risk factors, 3 months plus evaluation for lifetime anticoagulation is recommended, with a target international normalized ratio of 2.525 (Table 5). The British Thoracic Society has similar guidelines, adding that thrombolytic therapy should be used only in massive PE and that oral anticoagulation should not be started unless PE is confirmed with imaging. These guidelines emphasize the risk of anticoagulation in certain patient subgroups, such as pregnant patients and those with cancer, but postoperative patients are not mentioned.

At least one response to the current anticoagulation guidelines questions whether they are appropriate for orthopaedic patients. Each of these medications has drawbacks. Warfarin requires close monitoring and is not predictable in its pharmacokinetics.

### Table 4

**Suggestions for Anticoagulation Therapy for Small Pulmonary Emboli**

<table>
<thead>
<tr>
<th>Cardiopulmonary Reserve</th>
<th>Coexisting DVT</th>
<th>Other Factor(s)</th>
<th>Anticoagulate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate</td>
<td>No</td>
<td>Symptomatic</td>
<td>No</td>
</tr>
<tr>
<td>Adequate</td>
<td>No</td>
<td>Asymptomatic/Incidental finding</td>
<td>No</td>
</tr>
<tr>
<td>Adequate</td>
<td>No</td>
<td>Anticoagulation contraindicated</td>
<td>No</td>
</tr>
<tr>
<td>Adequate</td>
<td>Yes</td>
<td>—</td>
<td>Yes</td>
</tr>
<tr>
<td>Adequate</td>
<td>—</td>
<td>Recurrent PE</td>
<td>Yes</td>
</tr>
<tr>
<td>Inadequate</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
</tr>
</tbody>
</table>

DVT = deep vein thrombosis, PE = pulmonary embolism

### Table 5

**American College of Chest Physicians Recommendations for Treatment of Pulmonary Embolism**

<table>
<thead>
<tr>
<th>Type of Pulmonary Embolism</th>
<th>Management</th>
<th>Goals of Management/Rationale</th>
<th>Grade of Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>LMWH, UFH, or fondaparinux × 5 d</td>
<td>LMWH: prevent formation of new thrombi&lt;sup&gt;27&lt;/sup&gt; UFH better for increased bleeding risk because rapidly reversed&lt;sup&gt;28&lt;/sup&gt;</td>
<td>1C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Initiation of VKA on day 1, discontinuation of heparin with INR ≥2 × 24 h</td>
<td>Prevent extension of thrombus and disease recurrence&lt;sup&gt;28&lt;/sup&gt;</td>
</tr>
<tr>
<td>With transient risk factors</td>
<td>VKA × 3 mo</td>
<td>—</td>
<td>Depends on clotting factors II and X depletion&lt;sup&gt;27&lt;/sup&gt;</td>
</tr>
<tr>
<td>Unprovoked/No risk factors</td>
<td>VKA × 3 mo</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Evaluation for lifetime VKA</td>
<td>Incidence of recurrence higher at 2 yr than provoked PE&lt;sup&gt;29&lt;/sup&gt;</td>
<td>—</td>
</tr>
<tr>
<td>First unprovoked PE, no bleeding risk, patient preference</td>
<td>Lifetime VKA, target INR = 2.5</td>
<td>—</td>
<td>1A</td>
</tr>
<tr>
<td>PE in patients with cancer</td>
<td>LMWH × 3 mo</td>
<td>LMWH more effective in preventing recurrence than VKA&lt;sup&gt;30&lt;/sup&gt;</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>LMWH or VKA as long as cancer remains active</td>
<td>LMWH more effective in preventing recurrence than VKA&lt;sup&gt;30&lt;/sup&gt;</td>
<td>—</td>
</tr>
</tbody>
</table>

INR = international normalized ratio, LMWH = low-molecular-weight heparin, PE = pulmonary embolism, UFH = unfractionated heparin, VKA = vitamin K antagonist

<sup>a</sup> A = high-quality evidence, C = low-quality evidence
ics. LMWH and fondaparinux require nonoral administration. The newly introduced direct thrombin inhibitor ximelagatran is an oral agent and requires no monitoring; however, it causes elevation of transaminase levels and an increased rate of coronary events of unclear significance.32

The timing of therapeutic anticoagulation for PE in postsurgical patients has not been thoroughly researched. In a study of spine surgery patients with PE, three patients were treated with inferior vena cava (IVC) filters because the PE presented within the first week of surgery, and three were treated with anticoagulation because the PE presented 1 week after surgery. Neither group of patients had severe complications from the treatment.33 The authors recommended a waiting period of at least 8 postoperative days before instituting a full dose of anticoagulation. Another study of neurosurgical patients cited a high mortality rate (15%) associated with anticoagulation therapy; the authors could not comment on safe timing.34

Several studies focus on the outcomes of anticoagulation therapy for PE (Table 6). In a study of 673 patients with PE (of whom only 10 were surgical) who were treated with anticoagulants for 3 months with complete follow-up, Nijkeuter et al15 found a 3% recurrence rate of thromboembolic events (20 patients) and a 2% recurrence rate of PE (14 patients), of which 79% were fatal, mostly in the first week. Immobilization for >3 days posed significant risk for both recurrent VTE and fatal recurrent PE (odds ratio, 2.79). The overall 3-month mortality risk factors in the entire PE cohort were age, immobilization, cancer, and inpatient status. Douketis et al16 researched a longer treatment period; in 2,052 patients (310 with PE, 292 with both DVT and PE) who were anticoagulated for 6 months, with an average 54-month follow-up, the risk of fatal PE after stopping therapy was 0.2 to 0.5 events per 100 person-years (case fatality rate, 4% to 9%). For an even longer treatment period, Palla et al17 looked at 497 patients with PE (33% surgical) anticoagulated for 1 year. Forty-eight patients (9.6%) had recurrent PE, which was fatal in 36 of 48 cases. Thirty-nine of 48 recurrences (81.2%) occurred within 10 days of diagnosis, and 2 patients had recurrent nonfatal PE between 6 and 12 months. Finally, Stein et al17 reviewed several studies of timing of heparin versus vitamin K antagonist in DVT patients. Most VTE (DVT or PE) recurrences took place after 5 days, even in those who were not treated with heparin; a therapeutic level of heparin in 24 hours resulted in fewer recurrent events. These studies suggest that the greatest risk of PE arises within the first 2 weeks and that these early recurrences have a high fatality rate. It follows that anticoagulation therapy is most important in the first days of diagnosis, precisely when it is most dangerous to a surgical patient. However, these studies did not delineate the nature of the embolus, such as size and location; this must be taken into account, given the earlier stated research on the effect of embolus size.

Other studies focus on the mortality of untreated PE, although there are, to our knowledge, no randomized controlled trials of treated versus untreated PE. One study reviewed data from the Prospective Investigation of Pulmonary Embolism Diagnosis of 20 patients who had PE but had not received anticoagulation therapy. These patients were more likely to have segmental perfusion defects than were the treated patients. One patient died, and one had recurrent PE. The authors concluded that “mild” PE has a low mortality risk.38 Nielsen et al39 presented a trial of 87 patients with DVT, 43 of whom had a silent, asymptomatic PE on lung scan. All 87 patients were randomized to receive or not receive anticoagulation.

### Table 6

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients</th>
<th>Treatment Duration (mo)</th>
<th>Follow-up (mo)</th>
<th>Recurrence Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nijkeuter et al35</td>
<td>673 (PE)</td>
<td>3</td>
<td>3</td>
<td>2%</td>
</tr>
<tr>
<td>Douketis et al36</td>
<td>2,052 (310 PE, 292 DVT + PE)</td>
<td>6</td>
<td>54</td>
<td>0.2–0.5/100 person-years&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Palla et al37</td>
<td>497 (PE)</td>
<td>12</td>
<td>≥12</td>
<td>9.6%</td>
</tr>
</tbody>
</table>

DVT = deep vein thrombosis, PE = pulmonary embolism

<sup>a</sup> Risk of fatal pulmonary embolism
Anticoagulation therapy did not influence PE resolution on lung scan at 3-month follow-up. The type of anticoagulation may have an effect on outcomes, as well. In a randomized trial, Hull et al \(^{40}\) looked at patients with both DVT and nonmassive PE who received either LMWH (97 patients) or unfractionated heparin (103 patients). Rates of recurrent VTE events were zero versus 7%, respectively, suggesting that LMWH is at least as good a therapy, if not better, than standard intravenous heparin.

Therapeutic anticoagulation involves several risks: bleeding, thrombocytopenia, and osteoporosis, as well as skin necrosis and, in the case of warfarin, teratogenicity. Of these, bleeding is most important and relevant for orthopaedic patients. Bleeding can lead to pain, prolonged rehabilitation, compartment syndrome, return to the operating room, anemia and transfusion, and wound infection. Bleeding is a strong predictor of mortality in hospitalized patients. \(^{41}\) Even prophylactic anticoagulation carries a bleeding risk at the surgical site: a 6.38 relative risk for LMWH compared with aspirin and a 4.88 relative risk for warfarin compared with aspirin in one study. \(^{42}\)

Much of the data on bleeding complications come from total joint literature. The rates quoted for bleeding range from minimal to as high as 50%. \(^{43-46}\) None of these studies is randomized; however, they provide useful data on risk. In a review of 112 arthroplasty patients who were treated for DVT or PE with intravenous heparin, Patterson et al \(^{43}\) found an overall bleeding rate of 30%, with decreasing risk over time (50% if anticoagulated within 5 days of surgery, 40% if within 7 days, and 15% if >1 week). In 41 patients (35%), heparin was discontinued because of complications; 38 of those patients received warfarin instead and did well. Twenty-three patients treated for mild PE with warfarin alone had no bleeding or PE-related complications. The authors reached several important conclusions: that a bolus heparin dose may be too aggressive; that a PE should be confirmed before instituting treatment; and, most importantly, that anticoagulation in the first postoperative week carries a very high risk and should be avoided. These findings are echoed in other studies, which show an increased bleeding rate in the first month of anticoagulation. \(^{44}\) Supratherapeutic levels of international normalized ratio or partial thromboplastin time are also associated with bleeding complications, as well as higher transfusion requirements and longer hospitalizations. \(^{44,45}\)

Anticoagulation therapy for even small pulmonary emboli carries risks. In one study of 43 false-negative CT scans that eventually were read as positive for PE, 21 patients did not receive therapeutic anticoagulation. Patients who received no anticoagulation had a significantly lower rate of hemorrhage, renal failure, and early death than did those who received anticoagulation therapy. \(^{47}\) Another study of 71 patients who received anticoagulation for subsegmental PE and 22 who did not showed 8 instances of hemorrhage, including 5 major events, all in the anticoagulated patients. Interestingly, no patient in either group died of PE, and there was one PE recurrence in the anticoagulated group. \(^{48}\) These findings emphasize the potential harm of anticoagulant therapy and warn against a “one size fits all” approach to PE treatment.

IVC filters are an alternative for patients with PE who have a high risk of bleeding (ie, recent surgery) or who cannot tolerate anticoagulants. They are also indicated for patients who experience a recurrent PE despite therapeutic anticoagulation. When placed, these filters block the passage of emboli from the lower extremities to the pulmonary circulation. They can be permanent or retrievable (for up to 1 year); removal carries a risk of iatrogenic IVC injury, which increases with the time since filter placement. Anticoagulation therapy is begun, either concurrently with the filter or just before removal, when the patient’s bleeding risk is acceptable. Additionally, in multiple-trauma patients, particularly those with long bone injury, spinal cord injury, or pelvic fractures, IVC filters are often placed in an attempt to prevent massive PE. One review of 9,348 orthopaedic patients found a 1% total rate of filter placement (90 patients), with 61% of 90 filters placed prophylactically. \(^{49}\) The ratio of prophylactic-based to treatment-based filters was 3.25 in fractures and 2.1 in joint arthroplasties. In that study, 10% of the retrievable filters were not able to be removed, and a further 11% had complications during removal. Despite these risks, filters are commonly used in orthopaedics and provide another weapon in the arsenal of PE prevention and treatment.

Summary

Orthopaedic patients present a dilemma for treating clinicians: they are at high risk for both PE and bleeding events. The orthopaedic community has extensively debated anticoagulation therapy for prophylaxis of PE, and the American Academy of Orthopaedic Surgeons has presented clinical practice guidelines to help guide care. However, data suggest that we may be overdiagnosing PE, that not all pulmonary emboli are the same, that small emboli are of questionable clinical relevance, and that the risks of anticoag-
ulation therapy are not minor.

We believe that the risks and benefits of management of the diagnosis of PE should be reviewed with the patient and that a spectrum of treatment should be based on the size and clinical presentation of the PE, as should the magnitude and timing of any orthopaedic surgical procedures. At this juncture, we are unable to make recommendations for treatment of small emboli. We feel that the subject merits additional clinical study and call for development of guidelines for anticoagulation in orthopaedic patients with PE, specifically including the size and location of the embolus and the potential risk of bleeding.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 6, 21, 24, 36, 37, and 39 are level I studies. References 3, 7, 12, 14, 17-20, 22, 27, 32, 33, 35, 44, 45, and 47-49 are level II studies. References 4, 15, 16, and 34 are level III studies. Reference 13 is a level IV study.

References printed in bold type indicate those published within the past 5 years.


