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Clinical Reviews

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EMERGENCY EVALUATION FOR PULMONARY EMBOLISM, PART 1: CLINICAL FACTORS THAT INCREASE RISK

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□ **Abstract—Background:** Pulmonary embolism (PE) can be fatal, but profligate testing for PE can harm patients. **Objectives:** With consideration of potential medicolegal implications, this two-part review provides current evidence about the care of patients with suspected and diagnosed PE in the emergency department (ED) setting. **Discussion:** In part 1, we review published evidence to describe the epidemiology, risk factors, and clinical presentation of PE in the ED setting. Older age, surgery requiring endotracheal intubation within the past 30 days, new use of oral contraceptives, and prior unprovoked venous thromboembolism in nonanticoagulated patients are clear risk factors for PE in ED patients. Recent history of unexplained dyspnea, pleuritic chest pain, and hemoptysis increase probability, but the effect of syncope is less clear. Treated and inactive cancer, smoking, obesity, and pregnancy have not been found to increase the probability of PE in symptomatic ED patients. Unexplained dyspnea, tachycardia, and a low pulse oximetry reading increase probability of PE. Finding of wheezing on lung auscultation reduces the probability of PE, and findings that suggest deep venous thrombosis increase the probability of PE. **Conclusions:** Understanding of risk factors, historical data, and physical findings that have been found to increase or decrease the probability of PE in symptomatic ED patients can help create rational guidelines for the diagnostic approach to PE.   © 2015 Elsevier Inc.

□ **Keywords—**pulmonary embolism; medicolegal; defensive medicine; decision making; venous thromboembolism; pregnancy; diagnosis

INTRODUCTION

In this first part of a two-part review, we analyze published evidence to determine which risk factors increase or decrease the probability of acute pulmonary embolism (PE) and the clinical presentation of acute PE in the emergency care setting. In this topical review, and its companion (part 2), the authors provide recommendations for the diagnostic approach to PE based upon contemporary, published evidence. In situations where published evidence is lacking, we draw from expert opinion. Recommendations herein suggest decisions and actions that a reasonably diligent and prudent emergency physician should render when caring for patients in a full-service emergency department (ED). If our recommended actions are not available at the initial site of care, this may require patient transfer to a hospital that can provide a higher level of care. However, our recommendations do not necessarily define the sole standard of care, nor do we describe all possible decisions and actions that could be considered reasonable and prudent.

The authors performed systematic free text query of www.pubmed.gov (October 2014) to locate contemporaneous references for all issues covered, but the decision to include or exclude specific topics, how to address, and which reference to use was at the discretion of the authors based upon their clinical and research experience.

DISCUSSION

Emergency Care Epidemiology of PE

Approximately 1 out of every 400 to 1500 adult ED patients will be diagnosed with PE in the United States (US) (1–5). The incidence of PE in the ED increases with patient age such that PE is diagnosed in about 1 in 10,000 ED patients in their third decade of life, and increases to about 1 in 200 patients in the ninth decade of life, after which the incidence of diagnosis levels off (reflected in Figure 1) (6). Thus, the age and comorbid illness of the population served by an ED will greatly determine the frequency of PE diagnosis (7,8).

The all-cause, 30-day mortality after diagnosed PE is about 8% (9–13). Autopsy studies suggest that PE is the second leading cause of sudden, unexpected, nontraumatic death in outpatients (14–16). In most cases, the PE was not diagnosed premortem. The mortality of untreated PE has been estimated to be 30% (17). These results may lead some to overemphasize the lethality of PE in the ED. Among patients diagnosed with PE, with systolic blood pressure >90 mm Hg, aged <50 years, the case fatality rate for death directly attributed to diagnosed PE in patients is below 1% (9,18). The PE-attributable mortality of small, subsegmental pulmonary emboli, which now account for about 25% of all PEs diagnosed in US EDs, is likely even lower (19–22). The majority of deaths occurring after PE are therefore secondary to other causes (e.g., cancer) (9–12,18). However, in the presence of circulatory shock, defined as a sustained arterial blood pressure <90 mm Hg, the mortality rate from PE may be 45% or higher even with anticoagulation and fibrinolytic treatment (12,23).

Risk Factors for PE

(Table 1) Population-based epidemiology studies have identified a large number of risk factors for venous thromboembolism (VTE). Although these studies are important from a public health perspective, they are not always useful to the emergency physician determining whether or not to work up a given patient in the ED for VTE. Many factors that increase risk in the general public do not translate to increased risk in ED patients with symptoms of PE. Several reasons account for this. First, longitudinal studies enroll volunteers who represent the US population as a whole, are usually not patients, and are asymptomatic at enrollment. Investigators then follow the cohort over long periods of time to assign statistical risk of an exposure or feature that some members of the cohort have, compared with those who not have the exposure or feature. In contrast, patients enrolled in diagnostic studies in the ED have selected themselves because they
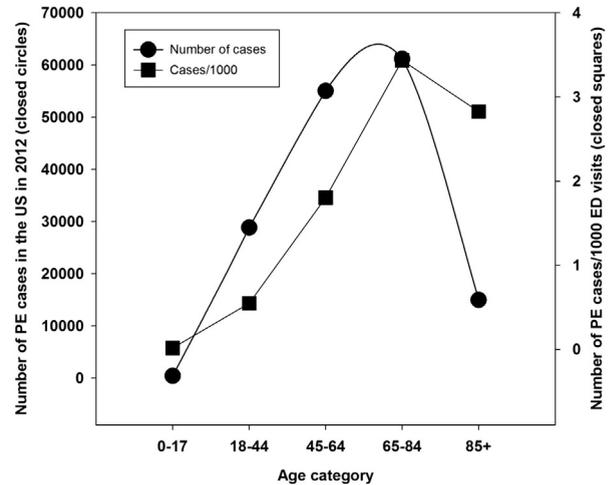

Figure 1. Number of patients diagnosed with pulmonary embolism (PE) (left axis, closed circles) and incidence of diagnosis of pulmonary embolism in emergency departments (ED) in the United States (US) for the calendar year 2012. Data from http://hcupnet.ahrq.gov/ HCUPnet, accessed November 15, 2014 (6).

have symptoms they think may be an emergency. Moreover, ED patients in PE diagnostic studies are then further selected by a clinician who believes the patient should have a diagnostic test for PE. Because emergency clinicians may tend to overweigh the importance of some factors that cause risk in the community, those factors may be diluted, and not increase probability of PE outcome in self-selected, then clinician-selected populations. Accordingly, many factors that increase risk of PE in longitudinal investigations do not appear in clinical probability scoring systems for PE (Tables 2–4).

Risk factors, signs, and symptoms of PE overlap other disease states. No single risk factor, sign, or symptom is sufficiently pathognomonic for VTE that a work-up must be undertaken when it is present. Although occasionally the decision to test a patient for VTE can be based on a single finding from the history and physical examination, applying this approach to all ED patients would result in enormous over-testing, over-diagnosis, and would ultimately harm more patients than it helps. Thus, although it is important for emergency clinicians to know population-based risk factors for developing VTE, it is even more important that they understand which factors are associated with an ED patient being diagnosed with VTE.

In deciding a patient's underlying risk for PE, emergency clinicians should consider most strongly age, prior VTE diagnosis (venous thromboembolism, referring to deep venous thrombosis [DVT] with or without PE) and the presence of provoking factors, as the primary risk factors for PE. Most guidelines and experts categorize VTE as either provoked (synonymous with secondary) or

Table 1. Risk Factors for Pulmonary Embolism in the Emergency Setting

Factor	Comment
Gender	No consistent increase in first-time PE risk, but males have a 2–3-fold increased risk in recurrent VTE (24–28).
Age	Risk becomes significant at 50 years and increases with each year of life until 80 years of age (see Figure 1).
Prior VTE	Highest risk of recurrence is for unprovoked VTE in men (OR 1.5–2), particularly if D-dimer remains elevated (OR 2.0–3.0) (29).
Solid cancers	Risk greatest with adenocarcinomas and metastatic disease (OR 2.0–3.5). A history of remote, inactive cancer probably does not increase risk of PE.
Hematologic cancers	Acute leukemias and myeloma confer the greatest risk, particularly when treated with L-asparaginase and the thalidomide derivatives, respectively (OR 3.0–6.5) (30).
Thrombophilias	Non-O blood type (OR 1.5–2.0) (31,32) lupus anticoagulant, shortened aPTT (OR 2.1–2.7) (33), Factor V Leiden or prothrombin variations (OR 2.0–3.0 heterozygous and OR 8–20 homozygous or compound heterozygous), and familial protein C, S, and antithrombin deficiency have the strongest risk (ORs 4.0–8.0).
Recent surgery or major trauma	Highest risk about 10 days after endotracheal intubation or epidural anesthesia and continues at least 4 weeks after exposure. Risk increases with more invasive surgery, neurosurgery, and cancer surgery (OR 3–5) (34–36).
Extremity immobility	Acute limb immobility of two contiguous joints or flaccid paralysis confers the highest risk.
Travel	Risk increases with >6 h continuous seated position. Effect of recent travel on probability of PE in the ED is less clear (37–39).
Bed rest	Becomes a risk factor at approximately 72 h (OR 2.0–3.0).
Indwelling catheters	Cause of most upper extremity deep venous thrombosis but not commonly associated with diagnosed PE.
Smoking	A population risk factor, but not a factor that increases probability of VTE in the ED setting. May increase risk of other factors such as obesity.
Congestive heart failure	Population risk factor for PE that may be less important in the ED. Related primarily to severity of systolic dysfunction; effect of diastolic dysfunction unknown (40,41).
Stroke	Risk greatest in first month after deficit, especially with severe stroke with flaccid paralysis, and higher for ischemic stroke than hemorrhagic stroke (42).
Estrogen	Highest risk period is in the first few months after starting therapy; all contraceptives containing estrogen increase risk of VTE, including transdermal and transvaginal preparations (OR 2.5–3.5) (43–47).
Obesity and metabolic syndrome	In the general population, VTE risk starts at BMI >35 kg/m^2 and increases with increasing BMI but studies are lacking to show that obesity is a significant risk factor for PE in symptomatic ED patients (30,48). Similarly, no evidence has shown metabolic syndrome confers increased probability of PE in symptomatic ED patients.
Pregnancy and postpartum state	70% of all peripartum PEs occur post partum; risk increases with trimester, but overall risk remains low throughout pregnancy (OR 0.4–0.8) (49,50).
Noninfectious inflammatory conditions	Examples are inflammatory bowel disease, lupus with lupus anticoagulant, and nephrotic syndrome. Risk of VTE increases roughly in proportion to severity of underlying disease.

PE = pulmonary embolism; VTE = venous thromboembolism; OR = odds ratio; aPTT = activated partial thromboplastin time; ED = emergency department; BMI = body mass index.

Table 2. Pulmonary Embolism Rule-out Criteria (PERC Rule)* (51,52)

1. Clinical low probability (<15% probability of PE based upon gestalt assessment)
2. Age <50 years
3. Pulse <100 beats/min during entire stay in ED
4. Pulse oximetry >94% at near sea level (>92% at altitudes near 5000 feet above sea level)
5. No hemoptysis
6. No prior VTE history
7. No surgery or trauma requiring endotracheal or epidural anesthesia within the last 4 weeks
8. No estrogen use†
9. No unilateral leg swelling‡

PE = pulmonary embolism; ED = emergency department; VTE = venous thromboembolism.
* All nine factors must be present to exclude PE.
† Oral, transvaginal, or transcutaneous.
‡ Defined as asymmetrical calves on visual inspection with patient's heels raised off the bed.

unprovoked (synonymous with primary or idiopathic) (53). Provoked VTE refers to clots associated with certain *acquired* conditions, generally including surgery requiring endotracheal intubation or epidural anesthesia within the previous 30 days, major trauma requiring

Table 3. Wells Score for Pulmonary Embolism (PE) (54)

Factor	Points*
Suspected deep venous thrombosis	3
Alternative diagnosis less likely than PE	3
Heart rate > 100 beats/min	1.5
Prior venous thromboembolism	1.5
Immobilization within prior 4 weeks	1.5
Active malignancy	1
Hemoptysis	1

* Risk score interpretation (probability of PE): ≤4 points, eligible for exclusion with D-dimer.

Table 4. Simplified, Revised Geneva Score for Pulmonary Embolism (55)

Clinical Variable	Points*
Age > 65 years	1.0
Previous venous thromboembolism	1.0
Surgery requiring anesthesia or fracture of lower limb in past month	1.0
Active malignancy	1.0
Unilateral leg pain	1.0
Hemoptysis	1.0
Pain on lower-limb deep venous palpation and unilateral edema	1.0
Heart rate	
75–94 beats/min	1.0
>95 beats/min	2.0

* Clinical probability: Low 0–1; Intermediate 2–4; High ≥ 5.

hospitalization, new immobility, pregnancy or post-partum condition, cancer, or new use of estrogen. Some consider travel as a provoking factor. Unprovoked or idiopathic PE refers to patients with none of the above provoking factors, though it is important to recognize that calling a PE idiopathic does not imply that a patient has no risk factors for PE.

Most patients diagnosed with PE in the emergency care setting have unprovoked PE (9,30). Unprovoked PE implies the excessive tendency to clot, which could be due to a genetic or acquired thrombophilia. Unprovoked PE increases the risk of VTE recurrence compared with unprovoked PE whether or not it is associated with a laboratory-proven thrombophilia (24–27,31,56). As a result, patients with unprovoked PE require longer duration of treatment than patients with provoked PE (28). However, when determining a patient's risk of having recurrent VTE, emergency physicians should not ruminate on the name of a patient's thrombophilia, but instead should simply distinguish whether the prior VTE was provoked or unprovoked.

Surgery. Any surgery that requires endotracheal intubation or epidural anesthesia increases risk of thrombosis and PE for about the next 30 days. That recent surgery appears as a factor in most validated clinical prediction rules and scoring systems for PE demonstrates the strong association between surgery and venous thrombosis (e.g., Tables 2–4, recognizing the existence of other rules and scoring systems). Over one-half of postoperative PEs occur after hospital discharge, with a peak incidence at around the 10th postoperative day (34–36). The highest-risk surgeries include abdominal surgery to remove cancer, joint replacement surgery, and surgery on the brain or spinal cord in the setting of neurologic deficits (35,57,58).

Immobility. Patients who are newly immobilized for >72 h, those with new limb immobility from neurological disease, and those with joint fixation by splints, casting, or external fixators have a two-to threefold increase in probability of PE compared with equally symptomatic and age-matched patients without immobility (37). Case-control data show that immobilization of the ankle alone confers at least an eightfold increase in risk of VTE, and the risk increases for patients with trauma and inherited thrombophilia (38). Prolonged travel within the previous 72 h increases risk of thrombosis in a dose-dependent fashion; the risk becomes significant at about 6 h of continuous seated position (39). However, the absolute risk of long-haul travelers in the ED is small. In one study, travel per se did not increase the risk of being diagnosed with PE in symptomatic patients with suspected PE in the ED (37). Travel is not part of any published clinical decision rule to assess probability of PE in ED patients.

Hormones. Administration of exogenous estrogen, whether for contraception or hormone replacement, and whether by oral, transvaginal, or transcutaneous delivery, increases a woman's risk for PE by two-to threefold in the general population and in the ED (30,43). The Pulmonary Embolism Rule-out Criteria (PERC rule, covered in more detail in part 2), does not allow exclusion of PE in patients taking estrogen (Table 2). The risk of estrogen use in men is unknown. The risk of VTE is greatest in the first few months after starting an estrogen regimen (43,44). The third-generation oral contraceptives containing desogestrel or gestodene as the progestin component confer significantly (1.5–3-fold) higher risk for VTE than preparations containing levonorgestrel (45). The risk of drospirenone remains controversial (46). Progestogen-only contraception, including certain subcutaneous implantable and intrauterine devices (Implanon® [etonogestrel; Merck, Kenilworth, NJ] and Mirena® [levonorgestrel; Bayer, Pittsburgh, PA]) and long-acting injections of progestins (Depo-Provera®; Pfizer, New York, NY) do not seem to increase risk (47).

Pregnancy. Pregnancy causes hypercoagulability, but a recent meta-analysis of over 25,000 patients found that pregnancy among ED patients evaluated for possible PE did not increase the risk of PE diagnosis compared with all other ED patients or women of childbearing age (49). The pooled relative risk (RR) of pregnancy for PE diagnosis was 0.60 (95% confidence interval [CI] 0.41–0.87) compared with all other patients. Patients in the third trimester had an RR of 0.85 (95% CI 0.40–1.77), and pregnant patients, compared to patients of childbearing age (≤45 years) had an RR of 0.56 (95% CI 0.34–0.93). whether these data suggest that pregnancy is not a high-risk state, or that US emergency physicians over-test pregnant patients for PE, or a

combination of these influences, remains unknown. The concept that pregnancy may not be such a high-risk condition was supported by a meta-analysis of 23 epidemiological studies in which Meng et al. found that PE occurs in only 3 of 10,000 pregnancies (50). In contrast, the postpartum condition (within 2 weeks of vaginal or caesarean section delivery) causes a high risk for VTE, such that 70% of all pregnancy-associated VTEs occur after childbirth (50).

Family history/genetics. Whether or not family history of VTE per se should be considered in assessing risk in the ED depends upon the quality and reliability of the information from the patient. This judgment requires acumen and experience that is beyond the scope of this review to describe or teach. Patients may confuse other conditions suffered by family such as stroke or myocardial infarction with VTE. Whereas conventional epidemiological cohort studies show that a family history increases longitudinal risk of VTE, no study has found that a patient report of a family history of VTE has a significant independent risk factor for VTE diagnosis in the ED setting (30,59).

Several inherited risk factors also increase the risk of first-time VTE. The most common genetic risk factors among patients with PE are factor V Leiden and the prothrombin gene mutation, and protein C and S and antithrombin deficiencies (25,60,61). Factor V Leiden causes resistance to activated protein C and is present in about 4–5% of the population at large (62,63). Heterozygous factor V Leiden triples the risk of an initial VTE, and homozygous factor V Leiden increases the risk 15- to 20-fold (61,63,64). The prothrombin G20210A variation leads to overproduction of prothrombin, and increases the risk of VTE two-to threefold for the heterozygous state and 5–10-fold for the homozygous state (61,63,64). Polymorphisms in numerous other genes, including methylenetetrahydrofolate reductase, endothelial nitric oxide synthase, plasminogen, α_2 antiplasmin, factors VIII, IX, and XI, and glycoprotein 6, also produce thrombophilia (31,65,66). Testing for genetic hypercoagulability is generally not indicated in the ED, even in patients diagnosed with idiopathic VTE, as it does not predict VTE recurrence nor determine the intensity or duration of anticoagulation (24,25,28).

Gender. Although gender does not increase the risk of first-time VTE, as shown in the OR data in Table 1, male gender strongly increases risk of recurrent VTE (24,26–29).

Cancer. Emergency physicians will often consider the need to test for VTE in patients with cancer. Patients with cancer that has been treated and is inactive do not have an increased risk for PE compared with age-matched patients, whereas patients with active cancer do have an increased risk (30,67). Cancer can be considered active if the patient is under treatment or the cancer is metastatic. Active cancer is included in the Wells and Geneva clinical probability rules (Tables 3 and 4). The thrombogenic potential of cancer varies the patient's age, tumor stage, and origin. In general, the more undifferentiated the cell type, and the larger the tumor burden, especially in the presence of distant metastasis, the higher the risk (68). Highest-risk cancers include adenocarcinomas (e.g., pancreatic, colon, ovary, stomach, and renal cell), glioblastoma, metastatic melanoma, lymphoma, and multiple myeloma (69,70). Approximately 15–25% of patients with these cancers are diagnosed with VTE during treatment. Similar rates are found in patients treated for acute lymphocytic leukemia treated with L-asparaginase, and acute promyelocytic leukemias treated with all-trans-retinoic acid (71). Approximately 10% of patients with advanced-stage breast cancer, or breast cancer patients undergoing chemotherapy, develop symptomatic VTE (72). Lower-risk cancers include localized breast, cervical, prostate, and nonmelanomatous, localized skin cancers such as squamous cell carcinoma and basal cell carcinoma not treated with chemotherapy. When treating cancer patients, clinicians should be especially vigilant for VTE during the induction phase of chemotherapy, as this is the most thrombogenic period. Especially high-risk chemotherapy agents include L-asparaginase and bolus fluorouracil treatment, lenalidomide and thalidomide, and any red cell growth factors (69,73). Indwelling central catheters, especially peripherally inserted central catheters (PICC lines), increase the risk of DVT in all patients, especially those with cancer, but have not been shown to increase risk of PE (74). The reason for this lack of association is not known, but could be related to a decreased embolic potential of upper-extremity catheter-based clots, that clinicians do not search assiduously for PE in these patients, or simply a lack of statistical power in the studies that have examined this issue.

Other factors. Although smoking, obesity, metabolic syndrome, and physical inactivity probably all increase the risk of VTE in the general population, none of these factors has yet been found to increase the likelihood of PE diagnosis among symptomatic ED patients selected for testing (30,75–78).

History of Present Illness

Disclosure of a factor known to provoke VTE indicates the most powerful information obtained from history that physicians should use to increase their gestalt probability of PE in symptomatic ED patients. Symptoms that have been positively associated with PE outcome include

new dyspnea that is not explained by a known medical problem (67,69). Most patients with PE accumulate an embolic burden over days or weeks prior to diagnosis, and this embolic accumulation is often painless and insidious, manifesting only as dyspnea on exertion in the review of systems (80,81). Morpurgo and Schmid referred to this phenomenon as the "heterochronic" nature of PE (80). Therefore, dyspnea that limits a patient's ability to walk should be considered tantamount to new-onset dyspnea (79). A history of dyspnea on exertion is sometimes more accurately obtained from family members, rather than the patient. Pleuritic chest pain (lateral or posterior thoracic pain between the costal margin and clavicles that increases with breathing) was found in several studies to significantly increase the probability of PE (30,79,82). Hogg et al. found that 5% of ED patients with pleuritic chest pain had PE (83). In contrast, in a secondary analysis of PIOPED II data, Stein et al. found pleuritic chest pain more commonly in patients without PE than patients with PE (79). Some clinicians believe the PE produces sudden onset of symptoms. Although Miniati et al. found "sudden onset of dyspnea" to significantly increase probability of PE, more generally, Courtney et al. found sudden onset of the chief complaint (dyspnea or chest pain) had a nonsignificant OR for PE diagnosis (0.88, 95% CI 0.75–1.07) (30,67,84). Although relatively uncommon, hemoptysis consistently shows a significant predictive value for PE (51,54). Most PEs originate as DVT, therefore, known DVT triples the odds of the patient having a PE (54,85). Factors that have either not been found to have significance or have not been evaluated to determine their influence on PE probability include orthopnea, dizziness, anxiety, and palpitations (79). The predictive value of syncope is less clear. For example, one US registry found that only 4% of ED patients with PE had syncope, whereas an Italian study found that 22% of patients with PE had syncope (9,82). In a systematic review, West et al. found syncope to be highly predictive of the diagnosis of PE, with a likelihood ratio positive of 2.6 (95% CI 1.5–3.8) (86). However, <1% of patients with presyncope and <3% of patients with syncope have the diagnosis of PE, explaining why syncope does not appear as a risk factor for PE in any validated clinical prediction rule for PE (Tables 2–4) (87,88). Patients with PE who present with syncope do tend to have larger and more dangerous PE (89,90). Despite this, syncope appears in only one of 13 published prognostic rules designed to predict the clinical course of patients with diagnosed PE (91). If a patient is taking a vitamin K antagonist, clinicians may be tempted to assume a lower risk if the patient's prothrombin time is in the therapeutic range (international normalized ratio [INR] of 2.0–3.0). However, clinicians must recognize that the quality of the pa-

tient's anticoagulation over the past several weeks, referred to as the time in therapeutic range, determines risk to a far greater extent than the INR value measured in the ED (92–94). Atrial fibrillation does not seem to increase risk of PE (95).

Physical Examination

On physical examination, findings that have demonstrated statistically significant associations with PE diagnosis include vital sign abnormalities, lung auscultation, and findings suggestive of deep vein thrombosis. Regarding the patient's general appearance, one small study has suggested that patients with significant cardiopulmonary disease, including PE, manifest decreased facial expression variability (96). Vital sign abnormalities that clearly increase probability of PE include an elevated heart rate (>100 beats/min) and a reduced pulse oximetry reading (<95% with the patient breathing room air near sea level) (Tables 2–4) (51,54,85,97). The normalization of vital signs with treatment or time does not change the likelihood that a patient will be diagnosed with PE (98).

Studies are inconsistent on the significance of an elevated respiratory rate, and the definition of tachypnea varies. Two studies, one using a definition of >20 breaths/min and the other >24 breaths/min, found tachypnea significantly associated with PE (30,79). Approximately 10% of patients with PE have an oral temperature of >38°C (100.4°F), though <2% of patients with PE have a temperature > 39.2°C (102.5°F), and one decision rule found high fever a negative predictor (67,99). Wheezing on lung auscultation has been found to reduce the probability of PE (100,101). Derivation and validation studies of several prediction rules have shown that findings of DVT increase probability of PE, including unilateral leg swelling (assessed by raising the patient's legs from the heels and observing for asymmetry of the calves), or tenderness along the deep venous system, which also includes calf tenderness (51,54,85,97). At least one-third of patients with DVT have concomitant PE, even when the patient lacks symptoms of PE (102). However, only about 40% of ambulatory ED patients with PE have concomitant DVT that can be found on standard compression ultrasonography (103,104).

CONCLUSION

This review critically evaluates clinical factors thought to affect the probability of PE in ED patients. The vital sign abnormalities that increase probability of PE are a pulse oximetry <95% and a heart rate over 100 beats/min. However, tachypnea has less predictive value. Fever over

39.2°C (102.5°F) decreases probability of PE. Patients that describe unexplained dyspnea and pleuritic chest pain have an increased likelihood of PE, whereas those with isolated substernal chest pain have a lower probability of PE. Syncope is a less certain predictor and does not appear as a risk factor for PE in any major pretest probability scoring system. The most pertinent risk factors for PE in the ED are age over 50 years, prior VTE, surgery within the past 30 days, limb immobilization, active cancer, and estrogen use. Current evidence does not indicate that obesity, smoking, travel, and family history increases probability of PE diagnosis in symptomatic ED patients. On physical examination, wheezing decreases probability of PE and leg swelling or pain increases the probability of PE. In part 2, we will discuss how these factors can be used to help decide which patients should have a formal evaluation for PE.

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ARTICLE SUMMARY

1. Why is this topic important?

Emergency clinicians have a duty to consider the possibility of pulmonary embolism (PE) in many patients on any given shift. Knowledge of which risk factors are valid in the emergency department (ED) is crucial to formulating the decision to initiate a formal work-up for PE.

2. What does this review attempt to show?

With awareness of medico-legal implications, this first part of a two-part review presents the clinical features that increase and decrease the probability of PE in the emergency care setting.

3. What are the key findings?

Some factors that increase risk for PE in epidemiological studies do not increase probability of PE patients with symptoms of PE in the ED setting. Findings from the medical record, or the patient, can increase or decrease clinical probability of PE, but the literature is conflicting on many features. On physical examination, a finding that points away from PE is wheezing on lung auscultation, whereas findings of deep venous thrombosis increase the probability of PE.

4. How is patient care impacted?

As will be presented in part 2, accurate recognition of risk factors for PE can help justify the decision as to whether or not to initiate a formal evaluation of PE.