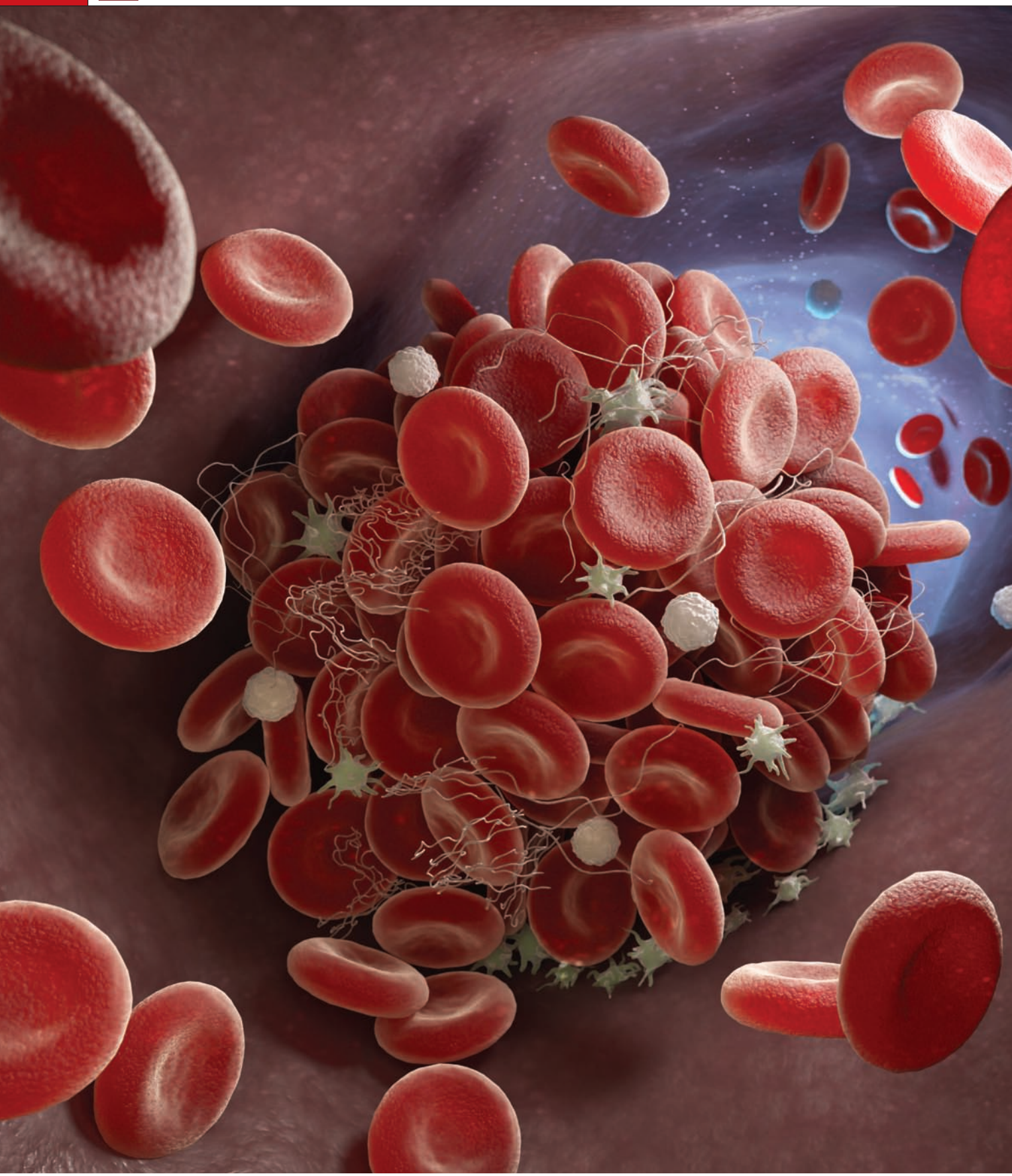
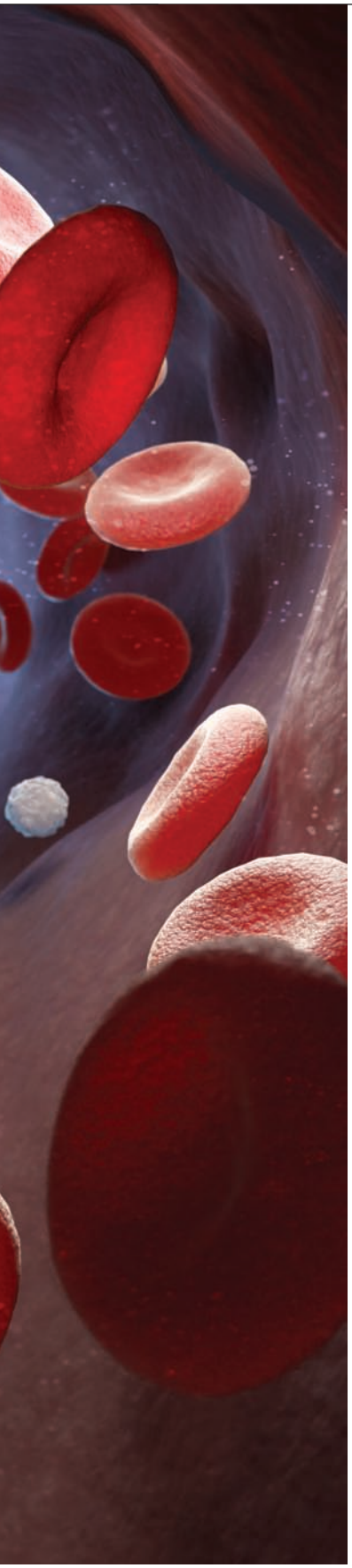




Tatiana Shepeleva

CHEST: Mnemonic approach to manage pulmonary embolism

Abstract: Acute pulmonary embolism is a challenging and potentially fatal disease that requires prompt assessment and precise management. Due to the lack of specific symptoms, NPs need to know how to identify a pulmonary embolism to manage it safely. This article discusses risk factors, initial approach, and diagnosis of acute pulmonary embolism using pretest probability and risk stratification tools. A mnemonic is proposed to guide medical management.

By Mohamed Toufic El Hussein, PhD, RN, NP and Jenna Habib, MN, NP

Acute pulmonary embolism (PE) is a serious and life-threatening condition that affects about 1 to 2 in every 1,000 adults annually in Canada and the US.[1] It is the third leading cause of cardiovascular death after acute coronary syndrome and stroke.[2] The ambiguity and overlap of its presenting symptoms, such as tachycardia, dyspnea, chest pain, and syncope, make the diagnosis of PE a challenge. The mnemonic approach and treatment algorithm presented in this article will allow NPs to better manage PE by initiating appropriate and timely treatment. Current management of PE no longer requires lengthy hospital stays, if any, as treatment modalities have evolved to benefit both the patient and the healthcare system.[1]

Introduction to identification of acute PE

Venous thromboembolism (VTE) includes deep vein thrombosis (DVT) and PE.[1] The incidence of acute PE and PE-related mortality increases exponentially with age.[3] Interestingly, acute PEs are the most preventable cause of death in hospitalized patients.[4] Acute PEs occur when a piece of a blood clot such as a lower extremity DVT (or, rarely, other material such as fat or tumor cells) dislodges and travels through the bloodstream to the pulmonary circulation, where it obstructs pulmonary arterial blood flow.[2]

The majority of acute PEs originate from the proximal deep veins of the legs.[1] Severe obstruction of blood flow through the lungs increases pulmonary arterial pressure, increasing the risk of elevated right ventricular (RV) pressure, and causing right heart strain and eventually right heart failure.[2,5]

Presence of certain factors is correlated with an increased risk of developing PE. These range from weak risk factors such as bedrest and diabetes, which have an odds ratio for risk of VTE of less than 2 to strong risk factors such as lower limb

Keywords: anticoagulation, pretest probability, PTP, pulmonary embolism

fracture and previous VTE which have an odds ratio of greater than 10 (see *Risk factors for venous thromboembolism*).[6] Failure to initiate treatment can lead to a cascade of physiologic events, including cardiac ischemia, manifested as tachycardia, hypoxia, systemic hypotension, and eventually cardiac collapse.[2]

Depending on the embolic burden, signs and symptoms of PE can be nonspecific and may include sudden-onset dyspnea, tachypnea, tachycardia, presyncope or syncope, pleuritic chest pain, cough, hypoxemia, increased supplemental oxygen requirements, palpitations, or hemoptysis.[2,6,7] Symptoms associated with DVT may also present as unilateral leg pain, swelling, and/or warmth.[2]

Due to the vague nature of these symptoms, the CHEST mnemonic, along with the effective use of validated predictive scores for initial identification and workup of acute PE, is proposed to help guide the NP's approach to a patient with a possible PE. The CHEST mnemonic provides an incremental, evidence-based approach starting with **C**linical suspicion, **H**emodynamic stability, **E**vidence-based diagnostic pathway, **S**everity index of pulmonary embolism, and **T**reatment with anticoagulation. The Wells score, Revised Geneva Score, Pulmonary Embolism Rule-out Criteria (PERC), and Pulmonary Embolism Severity Index (PESI) score were developed to assist providers in clinical decision-making.[2]

Risk factors for venous thromboembolism[1,6,10,15]

- Fracture of lower limb*
- Orthopedic surgery*
- Hip or knee replacement*
- Spinal cord injury*
- Major trauma*
- Myocardial infarction (within previous 3 months)*
- Previous DVT or PE*
- Intravascular catheter*
- Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months)*
- Cancer (highest risk in metastatic disease) & chemotherapy
- Arthroscopic knee surgery
- Bed rest >3 days
- Extended car or air travel
- Lower-extremity immobilizer or cast
- Blood transfusions
- Infections
- Pregnancy & postpartum period
- *In vitro* fertilization
- Minimally invasive surgery (for example, laparoscopic surgery)
- Antiphospholipid antibody syndrome
- Thrombophilia
- Inflammatory bowel disease
- Erythropoietin-stimulating agents
- Oral contraceptives
- Hormone replacement therapy
- Superficial vein thrombosis
- Varicose veins
- Diabetes mellitus
- Hypertension
- Autoimmune diseases
- Congestive heart or respiratory failure
- Obesity (body mass index ≥30 kg/m^2)
- Advanced age (particularly >75 years)

*Carries the highest risk for VTE development.
Note: Not a complete list of all possible risk factors.

Initial approach and diagnosis (CHEST mnemonic)

C–Clinical Suspicion

Due to the low specificity of PE signs and symptoms, PEs are among the most commonly missed diagnoses.[8]

PE is suspected because of pleuritic chest pain with or without dyspnea in 65% of patients.[7] Isolated dyspnea, either acute or progressive, without an obvious alternate cause points toward PE in 20% of patients.[7] PE is associated with significant morbidity and mortality; untreated PE is fatal in up to 30% of patients.[5,9] Approximately 20% of patients diagnosed with PE will die from hemodynamic instability within the first 30 days after diagnosis.[9] About 30% of patients with long-term VTE have a recurrence within 10 years.[9] Chronic thromboembolic pulmonary hypertension may also ensue secondary to PE in 0.1%-4% of cases.[7,9]

Virchow's triad of factors predisposing a patient to VTE includes hypercoagulability, venous stasis, and vascular wall dysfunction.[6,9] The mere presence of one variable of the triad increases the risk of PEs.[10] Interestingly, 40% of patients with acute PE have no predisposing risk factors.[6]

The first step in diagnosing suspected acute PE is the use of a clinical prediction rule, also called the pretest probability (PTP) or risk stratification rules.[5] These tests were developed to assess the probability that the patient has an acute PE and to support clinical reasoning for further lab testing and diagnostic imaging.[8] One of the most commonly used PTP rules in the diagnosis of PE is the Wells score, which stratifies patients into two categories based on the calculated scores: "PE unlikely" or "PE likely."[5] A pitfall of the Wells score is that it has been noted to be too subjective.[5] Another PTP rule is

the Revised Geneva score, which similarly predicts a patient's PE risk, but is thought to be more objective and less open to clinical judgment.[5] Rule-out tests can also be helpful, such as the PERC, which was deemed better suited for patients with a low-risk PTP than the two aforementioned scores thanks to its use of a set of clinical characteristics that indicate no further recommended testing when all eight are negative.[5]

Clinical suspicions may be supported by use of D-dimer, a quick and simple lab test. A serum D-dimer is used to rule out PE and is often a first-line test in patients with low-to-moderate PTP for PE.[5] D-dimer is a biomarker of fibrin formation and degradation that is measured in whole blood or plasma.[11] An elevated D-dimer is found in conditions associated with thrombosis but lacks specificity and can also be elevated due to other conditions such as advanced age, cancer, trauma, surgery, necrosis, pregnancy, infection, chronic inflammation, liver or renal disease, and thrombolytic therapy.[11] A positive D-dimer is a value that supersedes a threshold level of 500 mcg/L in patients below the age of 50 years. A value less than 500 mcg/L usually rules out acute PE.[1] Age-adjusted cut-offs for D-dimer values increase the specificity of D-dimer testing without sacrificing sensitivity.[1] Age adjusted D-dimer is specific for those over the age of 50 years, where the D-dimer is considered negative if it is less than the age of the patient multiplied by 10 mcg/L (that is, a 65-year-old person would have a negative D-dimer if the result was less than 650 mcg/L).[1,6]

Similarly, a modified D-dimer threshold which accounts for PTP also has proven validity.[12] The D-dimer threshold for ruling out PE can be increased in the presence of low PTP.[12] A combination of a Wells score of less than or equal to 4.0 (low probability of PE) and a modified D-dimer threshold of less than 1,000 mcg/L can substantiate the absence of a PE and indicate that no additional testing is needed to exclude a PE.[12] A D-dimer is not required when PTP for PE is high, as the clinical likelihood of PE remains excessively high even among those with negative D-dimer results.[1,13] Thus, a patient with a Wells score of 4.5 or greater should receive immediate imaging to establish a diagnosis.[12] Diagnostic imaging is recommended for patients with low or intermediate PTP for PE but an elevated D-dimer.[5,6]

An arterial blood gas (ABG) and chest X-ray may also be used to help with the diagnosis of acute PE. With PE, abnormal ABGs are common, and chest X-rays may be normal or abnormal; however, both of these tests are neither specific nor sensitive to provide a definitive diagnosis.[7] Chest X-rays may help identify alternative diagnoses such as pneumonia, heart failure, or pneumothorax.[7]

Twelve-lead ECG is an important tool to determine the degree of RV strain. The ECG of patients with PE may show T-wave inversions in leads V1-V4, a QR pattern in V1, an S1Q3T3 pattern, or a right bundle-branch block.[14] Atrial arrhythmias, such atrial fibrillation, may also result from acute PE and can be detected on the ECG.[14]

H–Hemodynamic stability

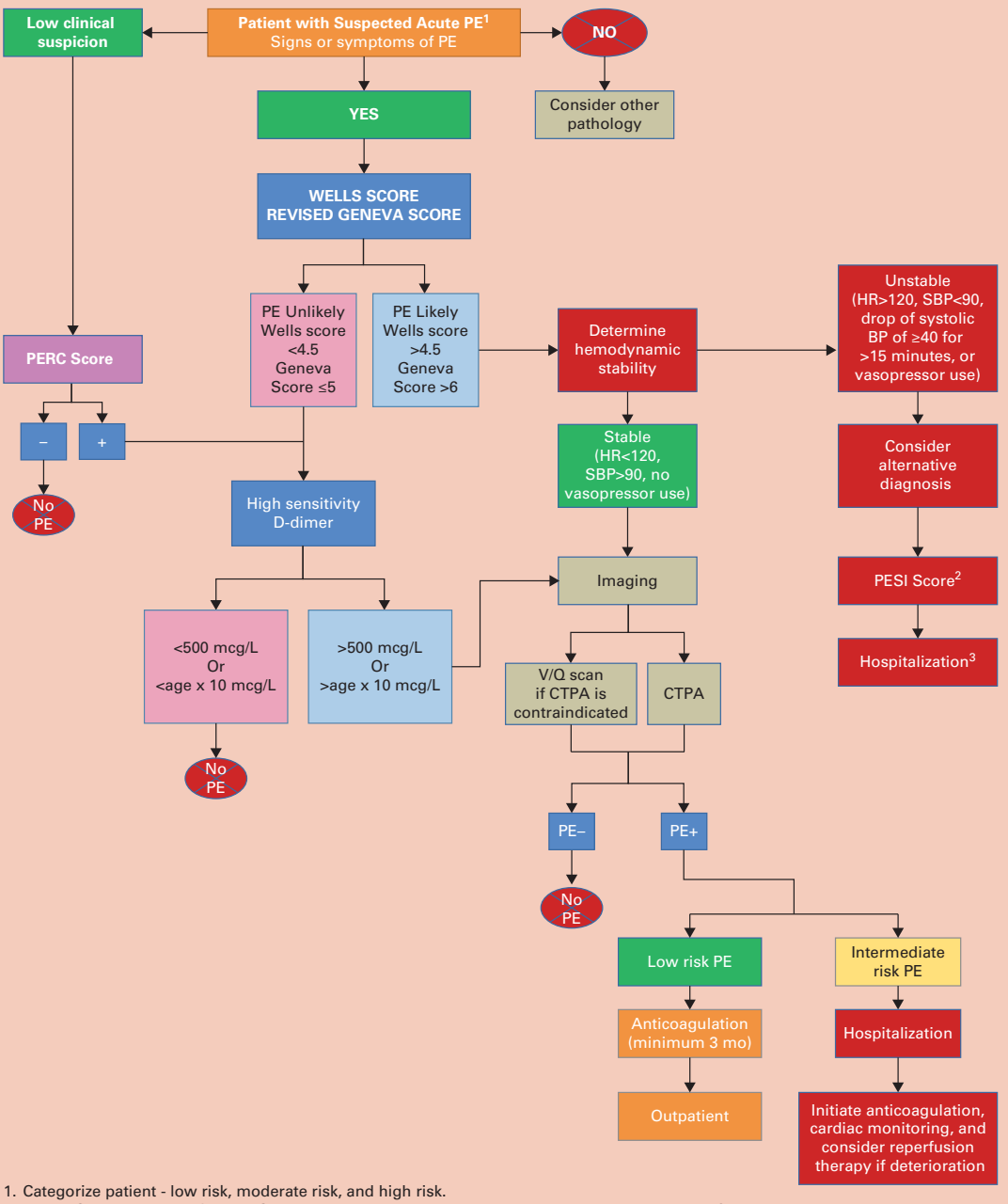
More than 95% of patients with acute PE are or appear to be hemodynamically stable at presentation and are therefore not at high risk for vascular collapse.[14] The clinical presentation of acute PE depends on the degree of physical obstruction in the pulmonary artery, which can be partial or complete, leading to circulatory failure and hypotension.[15] RV pressure overload increases RV wall stress, thus increasing its oxygen demand. The increased RV wall stress and pressure impedes myocardial perfusion, causing RV ischemia and dysfunction.[6,10] Acute PE also interferes with both circulation and gas exchange.[6] RV failure is caused by acute pressure overload and is the primary cause of death in severe PE.[6] Prolonged RV failure impedes left ventricular filling in early diastole, potentially leading to decreased cardiac output and contributing to systemic hypotension and hemodynamic instability.[6]

Acute right-sided heart failure is a primary cause of death that results from increased pulmonary vascular resistance and the associated acute increase in RV afterload.[16] This rapid rise in RV afterload coupled with systemic hypotension compromises coronary perfusion and results in myocardial ischemia.[16]

Hemodynamic instability is defined as sustained hypotension with a systolic BP below 90 mm Hg or a drop of greater than 40 mm Hg for at least 15 minutes along with supporting evidence of hemodynamic compromise.[15] Patients presenting with cardiogenic shock have between a 38% and 58% mortality.[15] Respiratory failure in acute PE is a result of hemodynamic disturbances, hypoxemia, and RV failure.[3] Echocardiography in patients with PE may show RV hypokinesis and dilation, interventricular septal flattening and paradoxical motion toward the left ventricle, and loss of inspiratory collapse of the inferior vena cava.[6] It is important to

note that because echocardiography has a negative predictive value of 40% to 50%, a negative result cannot exclude acute PE and is therefore not a mandatory part of routine diagnostic workup.[6] Right heart strain depicted by RV pressure overload and dysfunction can also be detected on echocardiogram.[6] RV dilation is found in 25% or more of patients with PE and is useful in risk stratification.[6] Echocardiography has a value in helping clinicians exclude other diagnoses that may present with similar symptoms such as cardiac

PE algorithm



1. Categorize patient - low risk, moderate risk, and high risk.
2. Use PESI score: High-risk via PESI, anticoagulation should be initiated while awaiting results of diagnostic testing. Intermediate- to low-risk PESI, treatment can safely be delayed if diagnostic testing will be completed within 4 hours or 24 hours, respectively.
3. Also consider: systemic thrombolytic therapy, catheter-directed therapy such as percutaneous suction thrombectomy, surgical thromboembolectomy, and/or extracorporeal membrane oxygenation.

Abbreviations: CTPA, computed tomography pulmonary angiogram; HR, heart rate; PE, pulmonary embolism; PERC, Pulmonary Embolism Rule-out Criteria; PESI, Pulmonary Embolism Severity Index; SBP, systolic BP; V/Q, ventilation/perfusion.

tamponade, acute coronary syndrome, or aortic dissection, if acute PE has been ruled out.[1,6,12]

E–Evidence-based diagnostic pathway

A computed tomography pulmonary angiogram (CTPA) is the gold standard for diagnosing acute PE.[8] CTPA is highly sensitive (83%), highly specific (96%), and has high predictive value for diagnosing PE as well as identifying alternative diagnoses.[12,15] CTPA is associated with radiation exposure, risk of CT contrast reactions, high cost, and risk of contrast-induced nephropathy; therefore, clinical assessment tools to calculate PTP and D-dimer testing should be used in ruling out PE.[12] It is recommended as a first-line test for patients with high PTP for PE and for patients with low or intermediate PTP who have a positive D-dimer.[6]

Due to the exposure to CT contrast and radiation, ventilation/perfusion (V/Q) scan may be the preferred diagnostic test in patients with renal insufficiency, contrast allergy, young patients with a normal chest X-ray, and in pregnant patients with a normal chest X-ray.[13] V/Q scanning can identify a perfusion defect when ventilation is normal and has 96%-97% sensitivity and 90% to 95% specificity (see *PE algorithm*).[6,17]

S–Severity index of pulmonary embolism

Once a PE is diagnosed, its severity can be stratified along a continuum of nonmassive, submassive, and massive.[4] The PESI aids in assigning a risk classification of I to V based on 11 criteria (see *PESI risk classification*).[4] Low-risk or nonmassive PE (PESI class I or II) do not demonstrate RV dysfunction or hemodynamic compromise.[4] Moderate-risk or submassive PE (PESI III or IV) demonstrates RV dysfunction, with an abnormal echocardiogram, computed tomography, or elevated cardiac biomarkers in an otherwise hemodynamically stable patient.[2,4] Patients with severe or massive PE are hemodynamically compromised with a systolic BP of less than 90 mm Hg or a drop of systolic BP by more than 40 mm Hg from baseline.[2] Therapeutic management of PE is dependent on the patient's hemodynamic stability. Risk stratification (PESI) aids in identifying whether in-hospital treatment is required or outpatient management is sufficient.[1]

T–Treatment

It is important to assess temporary and permanent risk factors that can contribute to the possibility of PE recurrence. Risk factors will help NPs in the process of decision-making regarding the course of anticoagulation treatment.[6]

Historically, patients diagnosed with acute PE, regardless of PESI class, were admitted to the hospital for a minimum of 5 days and treated with unfractionated heparin (UFH) and oral vitamin K antagonist (VKA) therapy.[18] As treatment regimens have evolved, direct oral anticoagulants (DOACs) have become more appealing due to their safety profile, efficacy, and convenience.[18] DOACs have simplified initial treatment, prophylaxis, and long-term management of PE without any required lab coagulation monitoring and have been found to reduce the risk of major bleeding by 39% compared with VKA therapy.[9,19] However, low-molecular-weight heparin (LMWH)/fondaparinux and UFH still continue to play a vital role in the initial treatment of acute PE.[19] Although there is no evidence that UFH, LMWH/fondaparinux, or DOACs have a direct effect on existing thrombi, these anticoagulation methods are used to inhibit the coagulation cascade, thereby allowing endogenous fibrinolysis to dissolve the existing thrombus.[19] The goal of initial management of anticoagulation therapy is to reduce mortality by preventing subsequent thrombin-mediated platelet aggregation, embolization, and further thrombus formation.[20] Anticoagulant therapy is the mainstay

PESI risk classification[2]

Factors	Points
Age	1 point/year
Male sex	10
Chronic heart failure	10
Chronic lung disease	10
O_2 saturation <90% on room air	20
Heart rate ≥110 beats per minute	20
Respiratory rate >30 breaths per minute	20
Temperature <36° C (96.8° F)	20
Malignancy	30
Systolic BP <100 mm Hg	30
Altered mental status	60
Score	**Risk Classification**
≤65	I
66-85	II
86-105	III
106-125	IV
>125	V

treatment for PE and is divided into acute phase (first 5 to 10 days after PE diagnosis), maintenance phase (3 to 6 months after diagnosis), and extended phase (greater than 6 months).[7] The American Society of Hematology (ASH) guidelines indicate that the minimal length of time for treatment of initial VTE is 3 to 6 months.[6,13] Treatment regimens and corresponding dosing can be found on the Thrombosis Canada website.[1] (See *Classification and initial management of PE.*)

Advanced treatment and support for patients with intermediate- and high-risk PE

Reperfusion therapy is the primary goal for high-risk, massive PE with hemodynamic instability.[3] For patients categorized as high-risk via PESI, anticoagulation should be initiated while awaiting results of diagnostic testing.[1,6] For those categorized as intermediate- to low-risk via PESI, treatment can safely be delayed if diagnostic testing will be completed within 4 or 24 hours, respectively.[1,6]

In most high-risk cases, systemic thrombolytic therapy is indicated, with goals of hemodynamic improvement, reversal of RV dilation, and prevention of further hemodynamic decompensation.[21] Dependent on degree of cardiovascular compromise, percutaneous catheter-directed treatment, such as catheter-directed thrombolysis or aspiration thrombectomy, surgical embolectomy, or extracorporeal membrane oxygenation, may be indicated.[2] Other hemodynamic supportive measures might include supplemental oxygen, parenteral analgesics, I.V. fluid resuscitation, and/or vasopressors.[1]

Whether or not parenteral UFH is used first, the 2019 European Society of Cardiology PE guidelines indicate the preferred use of DOAC over VKA when oral anticoagulation is initiated.[3,6] Oral anticoagulants are not administered concurrently with thrombolysis, but administered subsequently.[15] Providers should be cognizant of contraindications to DOACs, such as severe renal impairment, pregnancy, breastfeeding, and antiphospholipid antibody syndrome.[1] In these situations, a LMWH bridge to VKA regimen is an alternative.[3]

Outpatient treatment options

The ASH guidelines for management of VTE suggest offering home treatment over hospital treatment for patients with PE who have a low risk of complications, unless the patient has other conditions that would require hospitalization.[13] Select low-risk patients whose home circumstances are adequate can safely receive outpatient treatment without hospitalization.[6] Outpatient treatment provides improved quality of life, increased social function, and improved physical activity.[18] Patients who are classified as intermediate-risk and hemodynamically stable are recommended for inpatient management; however, thrombolysis is not routinely recommended for these patients, as risks usually outweigh the benefits.[1,6,19] In such patients, anticoagulant therapy with UFH, LMWH/fondaparinux, or DOACs is recommended for the initial treatment of PE.[19] DOACs are preferred over VKA as first-line oral anticoagulation therapy for low-risk patients as well as for intermediate- and high-risk patients once they are hemodynamically

Classification and initial management of PE[1,21]

For **low-risk (nonmassive) PE** (no RV dysfunction or hemodynamic compromise, biomarkers within normal limits) the NP should initiate anticoagulation, such as DOACs, and prepare for outpatient management or early hospital discharge.	For **intermediate-risk (submassive) PE** with RV dysfunction/strain on echocardiography or CT, elevated troponin, BNP, and/or NT-proBNP and for **high-risk (massive) PE** with hemodynamic instability*, thrombus in transit, syncope, or cardiac arrest, the NP should initiate anticoagulation. UFH is the recommended anticoagulant for patients who will receive catheter-directed therapy, as well as for those at high risk for bleeding, with dosing modification. Use of UFH may be indicated in those with CrCl <30 mL/min. Finally, UFH should be considered in patients who may undergo surgical embolectomy or receive mechanical support for PE-induced cardiogenic shock.

BNP, B-type natriuretic peptide; CrCl, creatinine clearance; CT, computed tomography; DOACs, direct oral anticoagulants; NT-proBNP, N-terminal pro B-type natriuretic peptide; PE, pulmonary embolism; RV, right ventricular; UFH, unfractionated heparin.

*Defined as systolic BP <90 mm Hg for ≥15 min, drop in systolic BP of ≥40 mm Hg, or vasopressor use.

stable, unless contraindicated.[21] Considering the pharmacokinetics of DOACs, an equally rapid anticoagulation effect can be achieved with a DOAC compared with a UFH or LMWH/fondaparinux.[22] Upon hemodynamic stabilization and reassessment of RV size, function, intracardiac thrombi determined by echocardiogram or CTPA, and fulfilling early discharge criteria, treatment may be continued on an outpatient basis.[3]

All patients with PE should be treated with anticoagulants for a minimum of 3 months.[6] Individual risk assessment should be performed and treatment should be tailored based on a benefit versus risk assessment of continuing anticoagulation treatment after the first 3 months.[3] Patients with a PE caused by a major transient risk factor usually shouldn't receive anticoagulation past 3 months, whereas PE in those with persistent risk factors such as cancer, antiphospholipid antibody syndrome, history of recurrent VTE not related to a major transient risk factor, or PE with no identifiable risk factor may benefit from continuation of anticoagulation.[1,6] Providers should refer to relevant guidelines for more information.

The main barrier to outpatient treatment is provider uncertainty in identification of patients at low risk for adverse outcomes.[18] However, as criteria, reliability, and predictive capability have improved, practitioners are gaining more comfort with outpatient treatment.[18]

Finally, while use of mnemonics in medical education has proven effective as a strategy to boost memory and improve recall of facts, the goal of mnemonics is not to enhance comprehension.[23-25] Furthermore, the clinical practice of NPs who depend on mnemonics may fall short when generalizing disease-specific details to the presentations of patients with complex needs. Therefore, NPs should understand the concepts behind the mnemonic letters before employing them.[25]

Conclusion

Knowledge of the presentation and management of acute PE are pertinent to the role of the NP as this condition carries a high risk of mortality (of up to 34%) without timely diagnosis.[26] As signs and symptoms are nonspecific, the NP plays a pivotal role in the safety and management of patients with PEs by conducting patient risk assessments, managing medical therapy, and providing patient education.[27] PE management initiatives involving NPs can be highly effective and provide significant clinical and economic benefits.[27] The need for NPs to be knowledgeable and aware of current practice guidelines enables appropriate and timely PE assessment, diagnosis, and treatment.[28] A pillar of clinical competency is implementing evidence-based practice thereby providing ethical, safe, and current care resulting in improved patient outcomes.[28]

Clinical awareness of disease burden, practice guidelines, assessment tools, and risk stratification enables practitioners to provide comprehensive, effective, and patient-cared care. The ability to determine appropriate management is strongly dependent on the practitioner's ability to identify risk factors associated with acute PE. PTP scores and risk stratification criteria guide practitioners to initiate appropriate testing, diagnostic imaging, and prompt treatment. Clinical interventions are dependent on patient symptoms, hemodynamic stability, and PESI. PTP scores and treatment algorithms are easy to use, cost-effective, and reliable methods for practitioners to implement into their practice. NP

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The authors and planners have disclosed no conflicts of interest, financial or otherwise.

DOI-10.1097/01.NPR.0000841924.43458.25