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Summer 2018

Pulmonary Embolism

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Pulmonary Embolism

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Introduction

Deep vein thrombosis (DVT) and Pulmonary Embolism (PE) are often under diagnosed, but are serious medical conditions that can be prevented. Specifically, "a pulmonary embolism is a blockage in the pulmonary artery, which supplies the blood to the lungs. It is one of the most common cardiovascular diseases in the United States"(Crosta, 2018). Due to this very reason, the pathophysiological topic of acute pulmonary embolism was chosen to be researched so that a more thorough understanding of the topic could be reached.

Acute pulmonary embolism is a life-threatening diagnosis that can present with a multitude of nonspecific symptoms and can quickly become a life-threatening diagnosis. Clinicians must approach suspected cases of PE with a heightened awareness and follow evidence-based recommendations for the diagnosis and treatment of acute PE (Morici, 2014). Pulmonary embolism is among the most treatable and preventable causes of death in the United States (Minges et al., 2015).

There are many risk factors such as genetics, obesity, birth control, pregnancy, smoking, recent surgeries, prolonged bed rest and more that will increase an individuals chances of developing a DVT/PE. When a patient presents with any signs and symptoms of a possible PE, such as shortness of breath, chest discomfort, hemoptysis, leg swelling/discomfort, etc. it is the clinicians responsibility to take the proper steps in diagnosing and treating the possible pulmonary embolism.

There are many aspects of a patients care that must be considered by the clinician when taking care of a patient with a PE. The articles used throughout this presentation will help to provide additional resources and education to those individuals looking to increase their knowledge base in regards to caring for a patient with a pulmonary embolism.

Pathophysiological Process

Acute PE caused by thrombo-emboli may be spontaneous and often originates in the deep venous system of the lower extremities, upper extremities, right side of the heart, or pelvis (Morici, 2014). There are several factors that can increase a patient's susceptibility to thrombus formation. Virchow triad (stasis, vascular wall injury, and hypercoagulability) can be used to assess the patient's risk of developing thrombi (Morici, 2014). Stasis is often considered the most prominent factor, which, in conjunction with either vessel damage or hypercoagulability, can lead to clot formation; stasis can present in a variety of settings, including immobilization, chronic venous insufficiency, paresis secondary to stroke or other causes, and varicose veins (Morici, 2014).

Once deep vein thrombosis develops, clots may dislodge and travel through the venous system and the right side of the heart to lodge in the pulmonary arteries, where they partially or completely occlude one or more vessels (Tapson, 2018). The outcome depends on the size and number of emboli, the underlying condition of the lungs, how well the right ventricle is functioning, and the ability of the patients body to dissolve the clot(s).

Small emboli may have no acute physiologic effects and may begin to lyse immediately and resolve within hours or days. Larger emboli can cause a reflex increase in ventilation (tachypnea), hypoxemia due to ventilation/perfusion (V/Q) mismatch, and low mixed venous oxygen content as a result of low cardiac output, atelectasis due to alveolar hypocapnia and abnormalities in surfactant, and an increase in pulmonary vascular resistance caused by mechanical obstruction and vasoconstriction (Tapson, 2018). When large emboli occlude major pulmonary arteries, or when many small emboli occlude > 50% of the more distal vessels, RV pressure increases, which may lead to acute RV failure, shock, or sudden death. The risk of death depends on the degree and rate of rise of right-sided pressures and on the patient's underlying cardiopulmonary status (Tapson, 2018).

Signs and Symptoms

- Shortness of breath (dyspnea)
- Chest discomfort
- Coughing up blood (hemoptysis)
- Palpitations
- Dizziness and fainting
- Leg swelling and discomfort
- Increased respiratory rate (tachypnea)
- Shortness of breath when lying flat (orthopnea)
- Increased heart rate (tachycardia)
- Decreased breath sounds
- Jugular Venous Distention (JVD)

Risk Factors

- Genetic and acquired tendencies
- Pregnancy; use of birth control pills or hormone therapy
- Obesity
- Smoking
- Cancer
- Medical illnesses including hear disease, lung disease, and kidney disease
- Older age
- Recent surgery, trauma, hospitalization, or prolonged bed rest
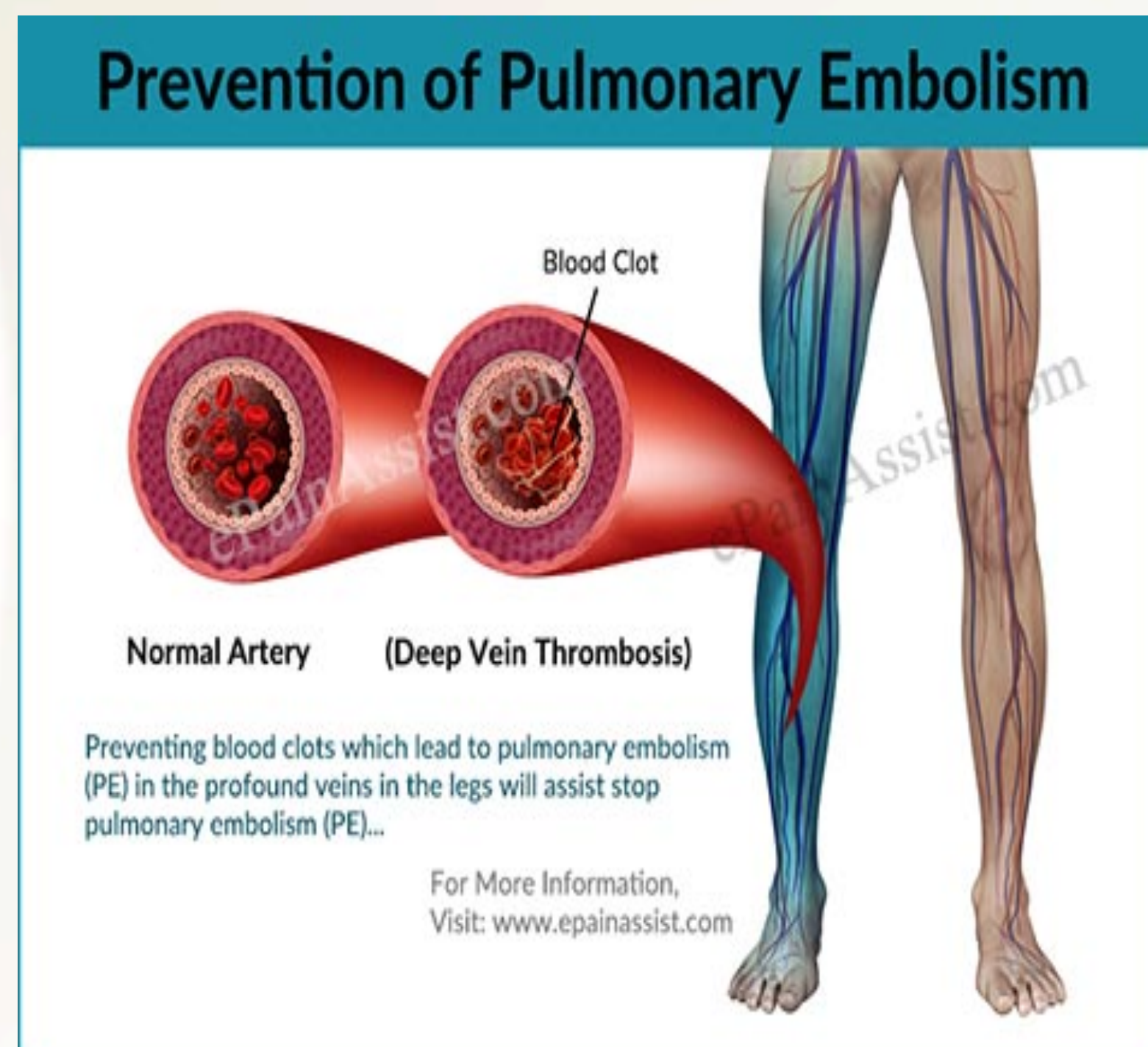
(Kerkar, 2018) Prevention of Pulmonary Embolism

Case Study

- **Presentation**

Harry is an 81 year-old man who presents to your ED with a 3 day history of breathlessness. He reports coughing up fresh blood and a sharp pain on the left side of his chest on taking a deep breath.

- **Question**

You believe Harry has symptoms of a suspected PE. What would you do next?

- **Answer:**

Carry out an assessment of his general medical history, a physical examination and a chest x-ray to exclude other causes. On examination his vitals are RR 20 breaths per minute, BP 145/90 mmHg, HR 72 beats per min, Temp 37.5c, SpO2 92% on RA. Upon auscultation of his lungs you hear crackles at the left base. Chest x-ray shows a small left-sided pleural effusion. Upon further questioning you find out that Harry has been in bed for the part 4 days because he felt unwell.

- **Question**

You still suspect pulmonary embolism. What would you do next?

- **Answer**

Offer Harry a D-dimer test.

- **Question**

Harry's D-dimer is positive. What would you do next?

- **Answer**

Offer an immediate CPTA. On this occasion CTPA is available for Harry immediately. The consultant in charge assesses Harry and deems him stable for transfer for CTPA. The CT department agree to accept him for CTPA and he is transferred with medical escort.

- **Question**

Harry's CTPA is positive for PE and he remains hemodynamically unstable. What would you do next?

- **Answer**

Diagnose PE and treat as soon as possible. Because of Harry's hemodynamic instability start unfractionated heparin and consider offering systematic thrombolytic therapy. In considering systematic thrombolytic therapy, expert clinical opinion advises provision of adequate monitoring facilities. Harry agrees to being treated with systemic thrombolytic therapy and appropriate facilities are made immediately available to safely administer and monitor systematic thrombolytic therapy. Following the acute management phase Harry is stabilized and continues on UFH until the international normalized ratio (INR) (adjusted by a VKA) is 2 or above for at least 24 hours.

("Venous Thromboembolic Diseases," 2012)
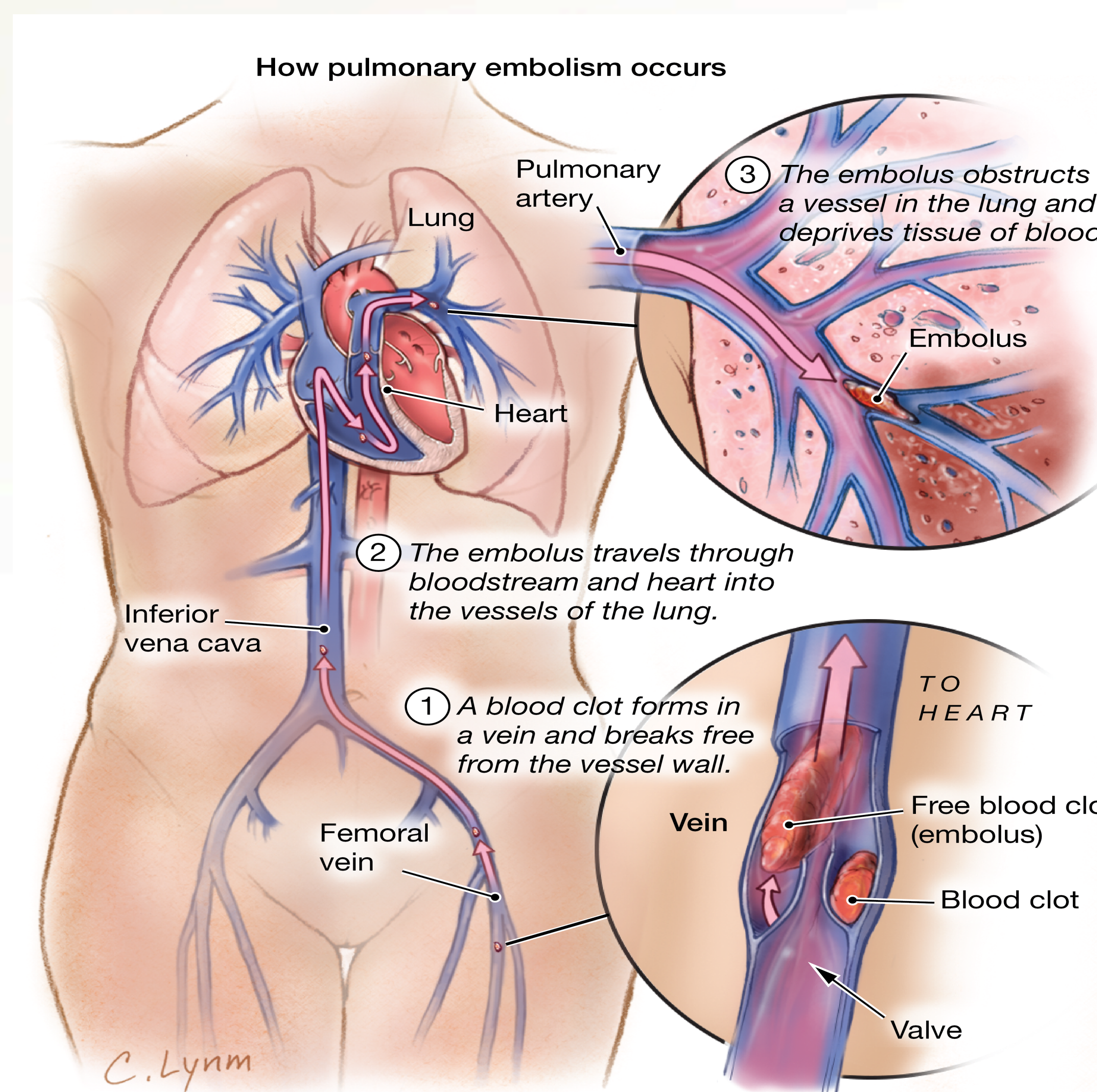
(Merrigan, Piazza, & Lynm, 2013) How Pulmonary Embolism occurs

Nursing Implications

As an advanced practice nurse, it is important to know the signs and symptoms of pulmonary embolism (PE). Due to the fact that the signs and symptoms associated with PE can be nonspecific, the condition can be a diagnostic challenge. Often a patient may present atypically or have none of the preceding symptoms; because of the risk of sudden death from acute PE, clinicians should have a heightened awareness of PE when assessing patients (Morici, 2014).

Being proactive and preventing a DVT/PE from ever developing is a key responsibility of clinicians. Bedbound patients and patients undergoing surgical, especially orthopedic, procedures can be identified as "high risk" before a thrombus ever forms. Preventive measures include low-dose unfractionated heparin, low molecular weight heparin, warfarin, fondaparinux, oral anticoagulants (rivaroxaban, apixaban, dabigatran), compression devices, and elastic compression stockings (Tapson, 2018). Choice of drug or device depends on various factors, including the patient population, the perceived risk, contraindications, relative costs, and ease of use.

If a PE develops in the acute setting, the initial treatment should be focused on stabilizing the patient. Provide supplemental oxygen to ensure adequate oxygenation, and monitor pulse oximetry continually, with a reasonable goal of 92%. The mainstay therapy for PE is anticoagulation. Treatment options include unfractionated heparin, fondaparinux, warfarin, and rivaroxaban. The American College of Chest Physicians (ACCP) suggests LMWH as the initial anticoagulant; however, treatment should be individualized based on the patient's characteristics and comorbidities (Morici, 2014).
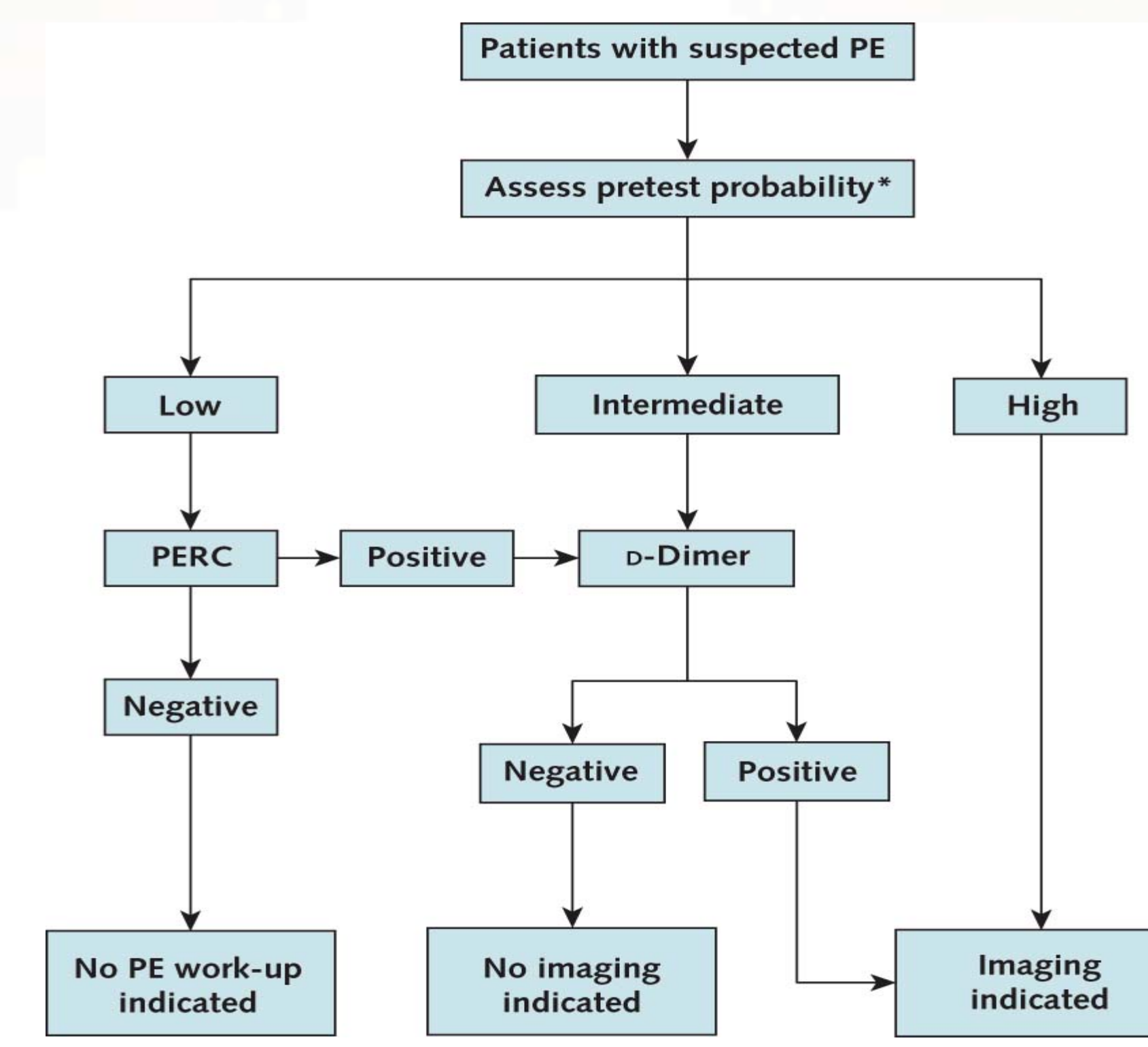
(Raja et al., 2015) Pathway for the evaluation of patients with suspected PE

Conclusion

- Acute PE is a common and potentially devastating medical condition.
- In an acute or ambulatory care setting, providers will likely be faced with patients who present with signs and symptoms indicative of PE.
- Clinical suspicion and confirmatory diagnosis are essential because in most patients who die from acute PE, PE is not even suspected.
- Due to the poor specificity of these presenting cases, clinicians must be vigilant about suspecting PE in patients at an increased risk.
- Prompt triage, imaging, and treatment can improve morbidity and mortality significantly.
- Discovering the underlying pathology can assist providers in determining the length of therapy and need for continued treatment and prevention.
- Because anticoagulation improves survival, patients with PE should be considered for thrombolytic therapy or embolectomy.
- Recent pharmaceutical and technological advances have added to the treatments available for PE and will continue to evolve as scientific research continues, with a goal of further improving identification, diagnosis, and treatment of acute PE.

Facts/Statistics

- The risk of PE increases with age
- The risk of PE is high for individuals who have had a blood clot in the leg/arm
- In rare cases, a PE can be caused by amniotic fluid
- In about 25% of people who experience a PE, the first "symptom" is sudden death
- 10% to 30% of people affected by DVT/PE will die within one month of diagnosis
- One-third (about 33%) of people with DVT/PE will die within one month annually
- Acute PE's affect 300,000 to 600,000 people in the United States annually

("Quick Facts," n.d.) National Blood Clot Alliance

(Zagaria, 2009) Factors Increasing Risk for Embolism

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