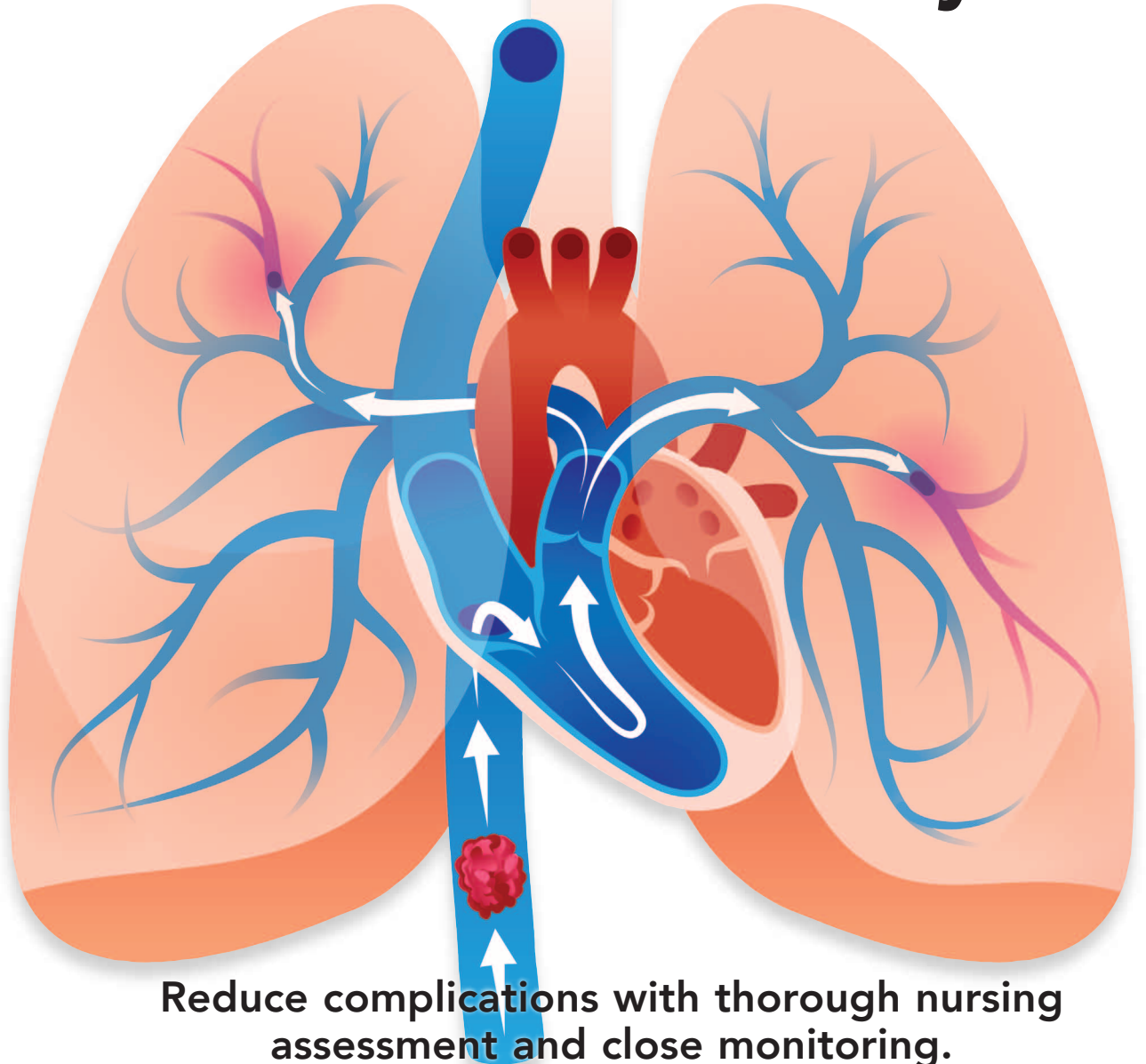


Pulmonary embolism: Surveillance is key



Reduce complications with thorough nursing assessment and close monitoring.

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LEARNING OBJECTIVES

1. Describe risk factors for and how patients might present with pulmonary embolism (PE).
2. Discuss nursing implications to consider when caring for patients with suspected or confirmed PE.
3. Identify medication management options and associated safety concerns for patients with PE.

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Expiration: 3/1/25



PE by the numbers

According to the European Society of Cardiology, pulmonary embolism (PE) is the third most common cause of acute cardiovascular death (after myocardial infarction and stroke). A high mortality rate persists despite advances made in treatment.

- The exact incidence of PE is unknown, but according to Turetz and colleagues, an estimated 600,000 cases occur in the United States each year, resulting in approximately 100,000 deaths annually.
- PE incidence is rising over time in developed countries, likely due—at least in part—to increasingly sedentary lifestyles.
- PE disproportionately affects the elderly population. Individuals over age 70 years are six times more likely to develop PE than those less than 44 years.
- If left untreated, PE mortality is significant and can be as high as 30%, with 10% of patients with acute PE succumbing to sudden death.

PULMONARY EMBOLISM (PE) occurs when a clot (thrombus) that formed somewhere in the body dislodges, travels through the right side of the heart to the lungs and blocks one of the arteries that supplies blood flow to the lungs. The vascular obstruction can cause sudden death, ventilation-perfusion (V/Q) mismatching, pulmonary hypertension, pulmonary infarction, hemodynamic compromise, right-sided heart failure, and multiple organ failure. Some patients with PE exhibit clinical manifestations such as hypoxemia and chest pain; however, some are asymptomatic, emphasizing the need for a high index of suspicion and close surveillance of patients at high risk. (See *PE by the numbers*.)

Etiology

Most PE results from deep vein thrombosis (DVT), which occurs when a blood clot that develops in one of the deep veins of the body, usually the leg, breaks free from the vessel wall and travels through the right side of the heart into the lungs. Keep in mind that PE also can consist of fat, air, bacteria, amniotic fluid, or other substances. Clinical presentation, diagnostic approaches, and physiologic alterations are essentially the same, but treatment varies based on embolus composition.

PEs can occur in multiple conditions, including healthy states such as pregnancy and inactivity as well as pathologic states that involve blood vessel injury and surgery. They can be acute or chronic, and treatment and complications may vary in duration and intensity.

Risk factors

Several factors increase an individual's risk of developing PE. Virchow's triad (mechanisms underlying the risk factors) include venous stasis, hypercoagulability, and injury to the thin inner lining (tunica intima) of the vessel. Identifying known risk factors and educating patients about lifestyle adjustments can help decrease individual risk. (See *PE risk factors*.)

Pathophysiology

Sisson and colleagues describe PE as a partial or complete occlusion of the pulmonary artery or one or more of its branches by an embolus or thrombus. The pulmonary vascular occlusion results in impaired gas exchange and circulation. When the embolus lodges in the pulmonary vasculature, several neurohormonal

PE risk factors

Pulmonary embolism (PE) risk factors are stratified as high, moderate, and low. Patients with high-risk factors require close nursing assessment and monitoring and a standardized screening protocol.

High risk

- Age >60 years
- Atrial fibrillation/flutter
- Cancer
- Erythropoiesis-stimulating agents
- Fracture of the leg or hip
- Genetics
- History of heart attack or stroke
- Hypercoagulable states/thrombophilia
- Major surgery
- Major trauma
- Severe COVID-19 symptoms

Moderate risk

- Blood transfusion
- Family history of embolism
- Infection
- Long trips
- Sedentary lifestyle
- Spinal cord injury

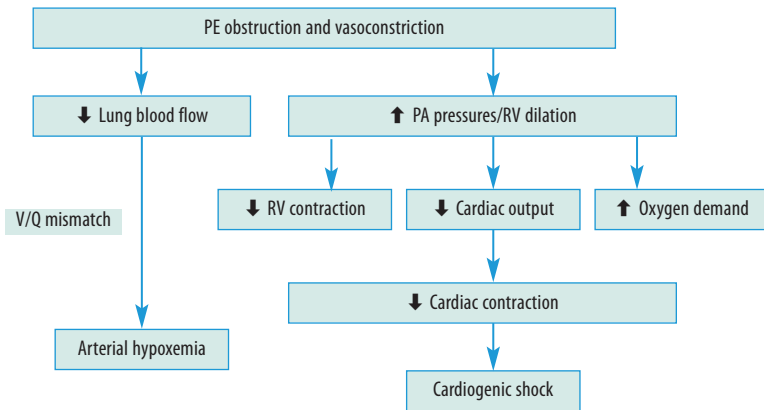
Low risk

- Estrogen or testosterone therapy
- Obesity
- Post-partum period
- Pregnancy
- Smoking

and inflammatory mediators—including serotonin, catecholamines, histamine, leukotrienes, angiotensin II, thromboxane, and toxic oxygen free radicals—are released. These substances cause widespread vasoconstriction, which impedes blood flow to the lungs and increases pulmonary arterial pressure (pulmonary hyperten-

PE pathophysiology

Pulmonary embolism (PE) reduces oxygenation and cardiac output. The more extensive the embolus in size or number, the greater and more rapid the physiologic deterioration.



PA = pulmonary artery, RV = right ventricle, V/Q = ventilation to perfusion ratio

sion). The vasoconstriction results in right ventricular heart failure because the right ventricle has to pump against higher-than-normal pulmonary vascular resistance, which dilates the right ventricle and decreases contractility and cardiac output. Subsequent imbalance between oxygen demand and supply further decreases cardiac contractility, which may lead to hemodynamic instability and cardiogenic shock. (See *PE pathophysiology*.)

The reduction or cessation of blood flow to lung tissue creates a V/Q mismatch. The blocked vessel can't carry blood to areas of alveoli that are ventilated, so less oxygenated blood is brought to the left side of the heart, leading to hypoxemia and tissue hypoxia. Although the mechanisms aren't fully understood, hypoxemia is enhanced by impaired production of surfactant in the alveoli deprived of blood supply, leading to atelectasis.

Clinical manifestations and evaluation

In most cases, PE clinical presentation is nonspecific and depends on the number and size of emboli and the number and size of blood vessels occluded. Patients may be asymptomatic or experience critical hemodynamic instability. The most common symptoms include dyspnea and pleuritic or substernal chest pain, but patients also may experience tachycardia, cough, hemoptysis, cyanosis, and syncope. (See *Clinical manifestations*.)

Diagnosis requires evaluation of risk factors, patient history, and predisposing factors. Diagnosing non-thrombotic PE can be chal-

lenging because micro-emboli may not be detectable with computed tomography (CT). Patients with high clinical suspicion of PE should be evaluated with a CT pulmonary angiogram, which allows visualization of a thrombus or blocked blood flow distal to the thrombus. Patients who lack access to an angiogram or for whom this test is contraindicated should be evaluated with a V/Q scan, which permits assessment of pulmonary arterial blood flow and alveolar ventilation distribution. Although lower extremity doppler ultrasound can't diagnose PE, Thompson and colleagues recommend it for patients with contraindications for both angiogram and V/Q scan. Ultrasound detects reduced blood flow, but doesn't provide evidence of the cause.

A chest X-ray can help rule out pleural effusion, pneumonia, and other pulmonary disorders. Keep in mind that chest X-rays usually are unremarkable for the first 24 hours before atelectasis develops. A 12-lead ECG may show right heart strain, tachycardia, and nonspecific ST segment and T wave changes. Arterial blood gas analysis may reveal hypoxemia with respiratory alkalosis secondary to tachypnea to compensate for the hypoxemia; however, hypoxemia and metabolic acidosis are present with a massive PE that significantly reduces perfusion. Results from a basic metabolic panel can be used to evaluate renal function for the contrast dye used for a pulmonary angiogram and for later selection of pharmacologic agents, and a complete blood count may reveal leukocytosis from the inflammatory response to PE. Serum D-dimer may show the products of thrombus degradation (according to Thompson and colleagues, levels over 500 ng/dL increase the likelihood of PE). (See *Diagnostic tests*.)

Complications

PE complications can be life-threatening and include pulmonary hypertension, pulmonary infarction, right-sided heart failure, multi-organ dysfunction, and, if the clot is large enough to obstruct a pulmonary artery, sudden death. Lack of oxygen to the lung results in lung tissue death (pulmonary infarction). Because the right ventricle normally pumps blood against low pressure in the pulmonary vasculature, the workload of pumping against high pulmonary arterial resistance (pulmonary hypertension) causes the right ventricle to fail, leading to potentially catastrophic cardiac decompensation

Clinical manifestations

Pulmonary embolism clinical manifestations vary based on the size and number of emboli. For example, a massive pulmonary embolus that occludes the pulmonary artery will result in loss of consciousness and cardiac arrest, whereas a small embolus may manifest as tachypnea and tachycardia.

- Altered level of consciousness/mentation
- Anxiety
- Compensatory respiratory alkalosis, followed by respiratory acidosis if embolus persists
- Cough
- Crackles on chest auscultation
- Cyanosis
- Dyspnea
- Fever
- Hemoptysis
- Hypotension
- Hypoxemia
- Metabolic acidosis for hemodynamic compromise
- Pleural friction rub
- Pleuritic chest pain
- Syncope
- Tachycardia
- Tachypnea

as reduced cardiac output impairs blood flow to vital organs.

If a clot persists and the patient continues to have significant respiratory distress that results in hypoxemia and hypercapnia, acute respiratory failure may occur. Pulmonary hypertension caused by a persistent clot may result in scarring and chronic thromboembolic pulmonary hypertension. Increased right ventricular workload, from acute or chronic PE, may cause arrhythmias.

Pharmacologic treatment

Pharmacologic PE treatment includes anticoagulants, thrombolytics, and antiplatelet therapy.

Anticoagulants, administered either intravenously or subcutaneously, are first-line PE treatment. Rather than breaking up a clot, anticoagulants prevent them from enlarging. Bleeding is the main complication.

A *thrombolytic* may be administered if the PE is large and life-threatening and the existing clot must be dissolved to avoid hemodynamic and respiratory compromise. Although thrombolytics increase bleeding risk, they can be lifesaving if a patient is hemodynamically compromised. Thrombolytics are administered locally near the clot via a long catheter or systemically via I.V.

Antiplatelet therapy may be initiated for pa-

tients who are at high risk for developing a PE during a planned surgical procedure, particularly if the procedure is of long duration. Antiplatelet medications prevent platelet aggregation in the blood and subsequent clot formation. Evidence shows that these medications significantly reduce the risk of fatal and non-fatal PEs. (See *Pharmacologic treatment options*.)

Nonpharmacologic treatment

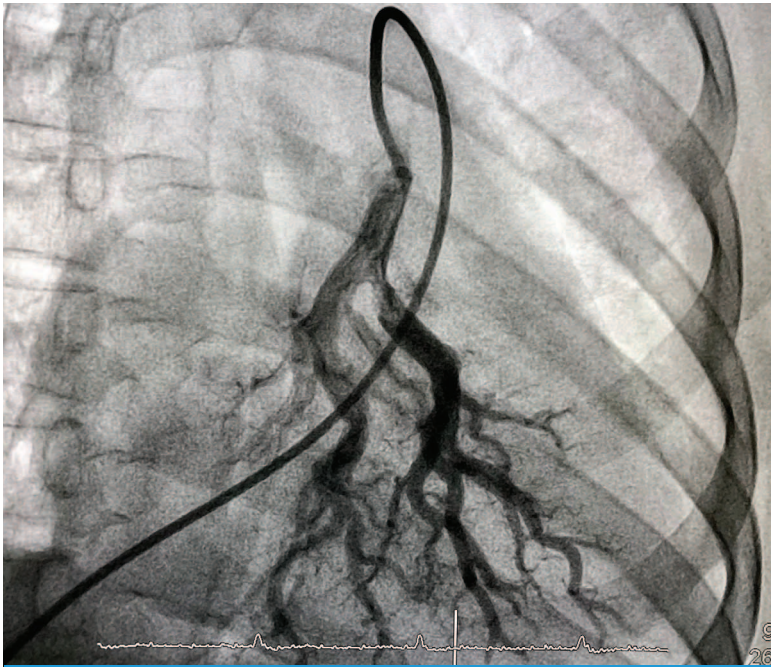
Patients who have contraindications or are unresponsive to pharmacologic therapy may require clot removal. The least invasive option is suction thrombectomy, in which a catheter is threaded through a major vessel to the clot and the clot is removed. If this treatment isn't an option, surgical embolectomy may be required.

Placement of an inferior vena cava filter, preferably a retrievable one, is reserved for patients with recurrent PE despite appropriate management with medication, those who are unable to take anticoagulants and don't require surgical intervention, and patients with active bleeding. Jaber and colleagues reported improved outcomes with these filters, including reductions in 3-month mortality rate and recurrent PE. When used for patients who subsequently develop active bleeding from concurrent anticoagulation, the European Society of Cardiology guidelines recommend removing the filter and re-initiating anticoagulation when the bleeding stops and the patient is stable.

Providers can use oxygen therapy to treat hypoxemia from the V/Q mismatch of perfusion to under-ventilated lung regions that results from redistribution of pulmonary blood flow. Some organizations have success using extracorporeal membrane oxygenation (ECMO) as a bridge for diagnosis and treatment of life-threatening, massive PE. ECMO pumps oxygenated blood to the body, bypassing the failing heart and lungs. Keeling and colleagues recommend that PE centers have ECMO readily available for treatment. (To learn more about ECMO, visit myamericannurse.com/?p=315947.)

Post-treatment evaluation and prevention

Patients receiving acute thrombolytic therapy require close observation to monitor for clinical manifestations of allergic and anaphylactic reactions. Chronic thromboprophylaxis may be used for months or years to prevent subsequent PE. Bleeding risk varies by drug, dosage, and individual patient factors. The provider



Diagnostic tests

Diagnostic tests for pulmonary embolism are selected based on the severity of the clinical manifestations and the risk factor level.

- Arterial blood gases
- Basic metabolic panel
- Chest X-ray
- Complete blood count
- Computed tomography pulmonary angiogram (shown in photo)
- D-dimer, coagulation studies
- History and physical examination
- 12-lead electrocardiogram
- Ventilation/perfusion scan

may use a bleeding risk score (such as the Pulmonary Embolism Severity Index) to determine the duration, type, and dose of chronic anticoagulation. Successful treatment requires periodic bleeding risk reassessment, hepatic and renal function tests, and patient adherence with and tolerance of the prescribed regimen.

For immobilized patients, mechanical prophylaxis with intermittent pneumatic compression can help prevent recurrent PE, but low patient adherence has been reported by many authors, including Dunn and Ramos. The devices may disrupt sleep and discourage mobility in bed. According to Dunn and Ramos, the devices are misapplied easily and present risks for skin breakdown.

Anti-embolism stockings (thromboembolic deterrent hose) increase venous blood flow velocity, decreasing venous stasis and the risk of developing DVTs. Appropriate pressure distribution to prevent blood pooling in the lower

extremities requires proper sizing of the stockings. The elastane in the fabric loses its compressibility and effectiveness over time, so stockings should be replaced monthly or more frequently as needed to maintain their preventive effect.

Nursing implications

Sudden onset and high mortality of PE make continuous surveillance of at-risk patients imperative. Nurses should know the risk profile for each patient and plan surveillance accordingly. For many patients who die from PE, the diagnosis is made post-mortem, so the condition should be at the top of the differential diagnosis list, along with myocardial infarction, when a patient has acute chest pain, dyspnea, or a cardiopulmonary arrest.

According to Rivera-Lebron and colleagues, because PE treatment options will vary based on provider experience and skill, medical specialty, organization, region, and available resources, protocols may be different within and across clinical settings. To account for these variations, individualize nursing surveillance, assessment, and patient education.

Patients with PE who have hemodynamic compromise may require fluid resuscitation. Monitor them closely for fluid overload effects on a poorly functioning right ventricle. Maintain good communication with the prescribing provider about the patient's blood pressure, heart rate, respiratory rate, jugular vein distention, and clinical manifestations of perfusion so that fluid resuscitation can be balanced to enhance cardiac output vs. cardiogenic shock.

Manage pain in patients who are symptomatic and take steps to assuage fear and anxiety. Provide close surveillance (face-to-face or electronic), ensure the call bell is within reach, and offer patient and family education.

Education about acute or chronic anticoagulation should be appropriate to the setting and include discussion of the risk of PE recurrence, the importance of following the therapeutic regimen, and the consequences of missing medication doses. Provide information about monitoring for bleeding and clinical manifestations of thrombus formation; foods, drugs, and herbs to avoid as appropriate for the prescribed anticoagulant; and lifestyle modifications to prevent or minimize medication adverse effects and PE recurrence. Although most pulmonary pathology will resolve after about 3 months,

Pharmacologic treatment options

Understanding the mechanisms of action and the parameters used to monitor pulmonary embolism (PE) treatment aids safe patient care and prevents treatment complication. Prophylactic and therapeutic ranges vary, with the latter being higher. Nursing considerations common to all PE pharmacologic treatment include monitoring for bleeding from over-anticoagulation and clotting from under-anticoagulation.

Pharmacologic treatment	Mechanism of action	Monitoring parameter	Prophylactic (P) and therapeutic (T) ranges	Nursing considerations
Anticoagulants				
Unfractionated heparin (UFH)	Binds to AT-III to inhibit thrombin conversion	aPTT	25-37 s (P) 50-70 s (T)	<ul style="list-style-type: none"> I.V. injection or continuous infusion Observe for HIT Use caution with NSAIDs (they increase bleeding risk)
Low-molecular-weight heparin (LMWH) (e.g., bemiparin, certoparin, dalteparin, enoxaparin, fondaparinux*)	Binds to AT-III to inhibit thrombin conversion	Anti-Xa	0.2-0.4 U/mL (P) 0.5-1 U/mL (T)	<ul style="list-style-type: none"> Subcutaneous injection Observe for HIT Use caution with NSAIDs (they increase bleeding risk) Hold two doses prior to surgery Closely monitor for variable effects in frail and obese patients and those with renal failure
Vitamin K antagonist (e.g., warfarin, phenindione, dicoumarol)	Interferes with the interaction between vitamin K and coagulation factors II, VII, IX, and X	INR	<ul style="list-style-type: none"> 2.0-3.0 (P and T) Patients at high risk: 2.5-3.5 (P and T) 	<ul style="list-style-type: none"> Ensure access to reversal agent Given with UFH or LMWH for 2 days until target INR is reached Target INR level based on recurrent PE risk Teach patient and family self-monitoring
Non-Vitamin K antagonist oral anticoagulant (e.g., apixaban, dabigatran, edoxaban, rivaroxaban)	Inhibits specific clotting factors: <ul style="list-style-type: none"> thrombin for dabigatran Factor Xa for apixaban, edoxaban, and rivaroxaban 	N/A	N/A	<ul style="list-style-type: none"> Ensure access to reversal agent Check contraindications in renal and hepatic failure Routine laboratory monitoring not needed
Thrombolytics				
Direct thrombin inhibitor (e.g., bivalirudin)	Inhibits circulating and clot-bound thrombin	INR	2.0-3.0 (P and T)	<ul style="list-style-type: none"> Preferred replacement for heparin in patients with HIT
Thrombolytic (e.g., rt-PA, urokinase, streptokinase, alteplase)	Dissolves existing clots	Blood pressure and neurologic status	Similar to baseline	<ul style="list-style-type: none"> I.V. injection
Antiplatelet therapy				
<ul style="list-style-type: none"> Acetylsalicylic acid Clopidogrel Prasugrel Ticagrelor 	Prevents platelets from sticking together and forming a clot	N/A	N/A	<ul style="list-style-type: none"> Easily accessible, some without prescriptions Important to know if patient is receiving antiplatelet and additional anticoagulant therapy

*Fondaparinux is not an LMWH; however, its half-life and selective Factor Xa inhibition are similar to LMWH.

aPTT = activated partial thromboplastin time, AT-III = antithrombin-III, HIT = heparin-induced thrombocytopenia, INR = international normalized ratio, NSAIDs = nonsteroidal anti-inflammatory drugs, rt-PA = recombinant tissue plasminogen activator, s=seconds

advise patients and their families to report new onset of dyspnea or stamina deterioration to their healthcare provider.

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Access references at myamericannurse.com/?p=319625.

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Please mark the correct answer online.

1. A pulmonary embolism (PE) can be composed of which of the following?

- a. Fat
- b. Air
- c. Amniotic fluid
- d. Blood clot
- e. All of the above

2. A ventilation/perfusion (V/Q) scan

- a. helps visualize a thrombus and blocked blood flow.
- b. helps visualize distribution of blood flow and distribution of alveolar ventilation.
- c. helps rule out pneumonia.
- d. may reveal right heart strain and nonspecific T wave changes.

3. First-line pharmacologic treatment for pulmonary embolism includes which of the following?

- a. Thrombolytics
- b. Antiplatelets
- c. Anticoagulants

4. Thrombolytics are administered to treat PE in which of the following scenarios?

- a. For planned surgery in patients with high risk for PE
- b. If the PE is large and life threatening
- c. To prevent clots from enlarging
- d. To prevent platelet aggregation in the blood

5. Extracorporeal membrane oxygenation may be considered for patients with PE to

- a. provide intermittent pneumatic compressions.
- b. remove a clot in the pulmonary artery.
- c. bridge oxygenated blood between diagnosis and treatment for massive PE.
- d. rule out pleural effusion, pneumonia, and other pulmonary disorders.

6. Which of the following is correct about the incidence of PE? (select all that apply)

- a. PE incidence is rising over time in developed countries.
- b. PE disproportionately affects those younger than 44 years.
- c. If left untreated, PE mortality is low (less than 5%).
- d. PE is the third most common cause of acute cardiovascular death.

7. Which of the following is considered a high-risk factor for PE (select all that apply)?

- a. Blood transfusion
- b. Cancer
- c. Leg fracture
- d. Estrogen therapy
- e. Testosterone therapy
- f. Major surgery
- g. History of stroke
- h. Pregnancy

8. Anticoagulant mechanism of action includes which of the following (select all that apply)?

- a. Inhibits clotting factors such as thrombin
- b. Interferes with the interaction between vitamin K and coagulation factors
- c. Prevents platelets from sticking together
- d. Dissolves existing clots
- e. Binds to AT-III to inhibit thrombin conversion

9. Pulmonary embolism clinical manifestations vary based on the size and number of emboli. Describe clinical manifestations of patients with PE, and include examples specific to patients with massive PE and those with a small PE.

10. Patients with PE who have hemodynamic compromise may require fluid resuscitation. Describe nursing care to enhance cardiac output and prevent cardiogenic shock in patients requiring fluid resuscitation.

11. Which of the following is correct about anti-embolism stockings (select all that apply)?

- a. They should be replaced every 6 months.
- b. They decrease venous stasis.
- c. They prevent blood pooling in the lower extremities.
- d. Proper sizing is required.
- e. They constrict and decrease venous blood flow.

12. Which of the following is correct about diagnostic tests for PE (select all that apply)?

- a. Chest X-ray can help rule out pleural effusion, pneumonia, and other pulmonary disorders.
- b. Chest X-ray can detect atelectasis early.
- c. A 12-lead ECG may show left heart strain.
- d. Complete blood count may reveal leukocytosis.

13. Arterial blood gases ordered for patients with a PE would include which of the following (select all that apply)?

- a. Respiratory acidosis
- b. Respiratory alkalosis
- c. Metabolic Acidosis
- d. Metabolic alkalosis