

## **Caring for Patients with Acute Pulmonary Embolism**

### **NYSNA Continuing Education**

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## How to Take This Course

Please take a look at the steps below; these will help you to progress through the course material, complete the course examination and receive your certificate of completion.

### 1. REVIEW THE OBJECTIVES

The objectives provide an overview of the entire course and identify what information will be focused on. Objectives are stated in terms of what you, the learner, will know or be able to do upon successful completion of the course. They let you know what you should expect to learn by taking a particular course and can help focus your study.

### 2. STUDY EACH SECTION IN ORDER

Keep your learning "programmed" by reviewing the materials in order. This will help you understand the sections that follow.

### 3. COMPLETE THE COURSE EXAM

After studying the course, click on the "Course Exam" option located on the course navigation toolbar. Answer each question by clicking on the button corresponding to the correct answer. All questions must be answered before the test can be graded; there is only one correct answer per question. You may refer back to the course material by minimizing the course exam window.

### 4. GRADE THE TEST

Next, click on "Submit Test." You will know immediately whether you passed or failed. If you do not successfully complete the exam on the first attempt, you may take the exam again. If you do not pass the exam on your second attempt, you will need to purchase the course again.

### 5. FILL OUT THE EVALUATION FORM

Upon passing the course exam you will be prompted to complete a course evaluation. You will have access to the certificate of completion **after you complete the evaluation**. At this point, you should print the certificate and keep it for your records.

## **Objectives**

At the completion of this learning activity the learner will:

- Recognize patients at increased risk of pulmonary embolism.
- Identify signs and symptoms of acute pulmonary embolism.
- Choose, implement and evaluate appropriate nursing measures for patients with or at risk for pulmonary embolism.
- Discuss underlying causes, predisposing factors, diagnostic tools and treatment used in pulmonary embolism.

## **Introduction**

Of all the emergencies a hospital nurse may encounter, pulmonary embolism (PE) ranks among the most serious. Approximately 10% of patients who exhibit signs and symptoms of PE will die within one hour of the onset of symptoms (Feied & Handler, 2006; Rumsey & Seaberg, 2004; Sharma, 2006).

Without prompt treatment even nonfatal PE carries a dire prognosis; about a third of patients who survive a first untreated PE will eventually succumb to a thrombosis (Sharma, 2006).

Pulmonary embolism, occurring about 600,000 to 650,000 times annually, is the third most common cause of death in the United States, and is the second or third leading cause of unexpected death across all age groups (Fedullo & Tapson, 2003; Feied et al., 2006; "National Quality Forum," 2006). Autopsy studies show that approximately 60% of patients who have died in hospitals have had a PE, so the chances are very good that most nurses will care for patients with PE at some time in their nursing career (Feied et al., 2006).

The good news is that with timely intervention and proper treatment, recurrence and mortality are rare (Fedullo et al., 2003). The bedside nurse is in an ideal position to pick up on the first symptoms of PE and initiate care that may literally make the difference between life and death (Meyer & Lavin, 2005). In order to recognize the onset of PE, make key decisions, and take effective measures, bedside nurses should be familiar with the clinical aspects of this life-threatening condition.

This course is suitable for:

- Novice nurses

## **About the Author(s)**

**Ellen Falin, MA, RN**, has a twenty-seven year nursing career with extensive experience in Med-Surg, OB, ICU, and ER. Ms. Falin has worked as a nurse manager at a 200-bed nursing home, conducted utilization review and discharge planning, and was also a regional director for the New York State Alzheimer's Association. For two years she served on the Otsego County Legislature and sat on committees overseeing public health and nursing home administration. Currently, Ms. Falin also reviews medical negligence cases to keep up-to-date with best practice and advances in medical and nursing technology.

**Nancy Jacobson, BS, RN**, is currently a staff nurse on an acute care and medical unit at Mary Imogene Bassett Hospital. Ms. Jacobson has experience with managing the care of patients with pulmonary embolism in various stages, including active and post-active. She has background experience in Med-Surg and ICU.

## **Pathophysiology**

The word “thrombus” is from the Greek word “thrombos” meaning lump or clump (“Definition of Thrombus,” n.d.)

By definition, “pulmonary embolism is the process by which a lesion derived from the downstream venous circulation migrates to the pulmonary vasculature” (Dickson, 2004, p.168). Pulmonary embolism is the blockage of one or more pulmonary arteries by a thrombus which has obstructed the vessel(s). The thrombus itself can be composed of fat, air, amniotic fluid, tumor, or a foreign body (CCRN Review, n.d.). By far the most common type of thrombus in PE is a blood clot from the deep veins of the legs (CCRN Review, n.d.; Garg, 2005), usually the popliteal and saphenous veins (Garg, 2005; Netter, 2004).

However, deadly PE can begin in the pelvic veins, or the subclavian or axillary veins of the shoulder and arm. Patients with central venous catheters are at high risk for fatal PE (Feied et al., 2006).

The thrombus detaches from its site of origin and travels through the body’s venous circulatory system to the heart. When the thrombus begins to move through the circulatory system, it is referred to as an embolus. From the heart, the embolus travels with unoxygenated blood to the lungs via the pulmonary arteries (Netter, 2004). The severity of the PE is determined by the size of the embolus and the respiratory and cardiac status of the patient (Garg, 2005).

Emboli are classified by size as minor, submassive, and massive (Shaughnessy, 2007) or small, medium and large. Microscopic emboli can block the lungs’ arterioles, causing multiple areas of lung tissue infarction (Brunner & Suddarth, 2006). Massive “saddle” emboli span the bifurcation of the main pulmonary artery and are often fatal (Wells, Kapoor & Whelan, 2007).

When an embolus blocks pulmonary blood flow, there is no circulation to the alveoli beyond the blockage. This creates a condition known as “dead space ventilation” in which the affected section of the lung is being ventilated by respiration but not perfused by circulation (Shaughnessy, 2007).

The sequelae of this condition are: rising pressure in the blood vessels upstream of the embolus causing pressure to build in the right ventricle of the heart with pulmonary hypertension, right-sided heart failure, and decreased cardiac output. Downstream from the embolus, lung tissue becomes ischemic (Garg, 2005; Shaughnessy, 2007). Atelectasis often occurs due to loss of lung surfactant (Feied et al., 2006).

If the occlusions are small enough, the event may be unnoticed or misdiagnosed; unfortunately, this is too often the case. The diagnosis is commonly missed and the majority of PE victims who are diagnosed at autopsy have never had any workup or treatment for PE (Feied et al., 2006).

Particularly significant is the fact that the diagnosis of PE is made correctly in only 10% of people over the age of 70, making it the most frequently missed diagnosis leading to death in the institutionalized elderly (Feied et al., 2006). Pulmonary embolism is the leading cause of maternal death following live birth (Shaughnessy, 2007) while the death rate in patients with hemodynamic compromise is approximately 20% to 30% (Fedullo et al., 2003).

## Risk Factor Assessment

PE has extremely high morbidity and mortality rates, and it is often misdiagnosed or overlooked completely (Sharma, 2006). The most important nursing intervention for PE prevention and/or management is the initial identification of patients at high risk for PE (Charlebois, 2005).

A thrombus may be composed of a variety of material such as fat or amniotic fluid. However, approximately 90% of all PEs are caused by deep vein thrombosis (DVT) in the legs (Garg, 2005). Additionally, PE may be present in 60% to 80% of patients who have DVT but may be asymptomatic (Sharma, 2006).

In the early 1800s, Rudolf Virchow developed a theory that accurately describes three physiologic precursors to DVT formation that include: alterations in blood flow, vascular endothelial injury, and alterations in the constitution of the blood (Dickson, 2004; Sharma, 2006). Virchow's Triad is useful when assessing for PE risk factors since it is generally applicable to all patients. However, patients with widely differing illnesses also will fit into the Triad.

Aside from the universal preconditions of Virchow's Triad, there are specific conditions or disease processes that should alert you to the possibility of PE in your patient.

Review the following case scenario.

### Case Study #1

George Johnson is a 54-year-old male admitted for chest pain and shortness of breath. His past medical history includes: atrial fibrillation, a myocardial infarction in 2005, and smoking from the age of 16 until five years ago. Further history includes left lower lobe pneumonia in 2002, a left total knee replacement 2 weeks ago, multiple sinus infections, and a sedentary lifestyle with limited exercise prior to his knee surgery.

Mr. Johnson arrived on the medical unit at 1500. Vital signs revealed a B/P 148/78 P 88 RR 20. Diagnostic testing in the emergency department revealed a normal chest x-ray and telemetry demonstrated normal sinus rhythm at a rate of 80 beats per minute with occasional PVCs. Arterial blood gases and a complete blood count were all found to be within normal limits. The provider did not order telemetry monitoring.

The patient was alert and oriented to person, place and time, and was asking many questions about how long he would be in the hospital and what testing might be ordered. His skin was warm, dry, and pale. Mr. Johnson denied chest pain or SOB upon admission, lung sounds were diminished without adventitious breath sounds, and no cough was noted.

Medications upon admission:

- Lasix 40 mg po qd;
- Potassium 20 po qd;
- propranolol 40 mg tid;
- hydralazine 10 mg qd;
- Ventolin Inhaler 2 puffs PRN
- Coumadin had been discontinued approximately one month prior, per his cardiac physician.

Although he currently had no chest pain, Mr. Johnson revealed that he had experienced on and off episodes of chest pain for about one week, and he denied SOB before being admitted. He took extra Lasix prior to coming into the ED because he felt bloated. The patient reported that he hadn't voided much at all on the day he was admitted.

After about an hour, Mr. Johnson called for assistance several times and appeared very anxious. He stated that he felt like "it's all over for me." He complained several times about the inability to urinate. The physician was notified of his increased anxiety and inability to void and ordered Ambien 10 mg for sleep and that he be straight catheterized.

When he was catheterized and no urine drained, a catheter was ordered to straight drainage. Mr. Johnson called for a nurse, was visibly upset, anxious and confused to time and place, stating: "I can't breathe, I can't breathe!"

At that point he began gasping for air and had a harsh productive cough with thick phlegm, which was red-tinged. The physician ordered oxygen to be started, ABGs, and an ultrasound of both legs. A VQ scan was ordered stat. The results of the chest x-ray revealed consolidation and small pleural effusions in the left upper lobe. The ultrasound was positive for a blood clot in the great saphenous vein in the left calf. Heparin was initiated via a central line and a loading dose was given (5000 Units IV). The patient was then transferred to ICU.

### **Test Yourself! Case Study Question #1**

**What factors placed Mr. Johnson at high risk for PE?**

(Answer can be found on page 21.)

Specific risk factors for a pulmonary embolus include (Feied et al., 2006; Kaufman, 2007):

- Prolonged bed rest or inactivity (including long trips in planes, cars, or trains)
- AIDs
- Blood type A
- Indwelling venous catheters
- Venous pacemakers
- Oral contraceptive use/hormone replacement therapy
- Surgery (especially pelvic surgery)
- Pregnancy and childbirth
- Massive trauma
- Burns
- Cancer, chemotherapy
- Stroke
- Heart attack
- Heart surgery
- Lupus
- Fractures of the hips or femur
- Lower extremity varicosities
- Venous stasis
- Obesity
- Old age
- Drug abuse
- Inflammatory bowel disease
- History of prior PE
- Blood clotting abnormalities/hypercoagulability
- Diseases affecting blood clotting



A thorough assessment of risk factors can help you anticipate and prepare for the possibility of PE. Meyer et al. (2005) describe this process as surveillance diagnosis where the nurse recognizes the patient risks for PE and remains ready to act in the event of an actual occurrence.

These same authors conclude that “vigilance is...the sustained attention, the perpetual scanning, that must always be present as nurses practice. Vigilance is not the action of taking the vital signs, dressing the wound, or starting the IV. It is the “watch-ful-ness” that is always a part of the nurse’s thinking process as activities such as these are completed” (Meyer et al., 2005, p. 2).

The patient’s health history is of prime importance, and the presence of any risk factor noted above should alert the nurse to the possibility of current or future PE. A complete physical assessment must be based not only on the admitting diagnosis, but should include an analysis of risk factors in view of existing signs and symptoms.

## Signs and Symptoms

Feied et al. (2006) state: "PE is so common and so deadly that the diagnosis should be sought actively in every patient with any chest symptoms that cannot be proven to have another cause" (p. 1).

The clinical signs of PE are nonspecific and may vary in quality depending on the extent of the occlusion and the patient's baseline cardiopulmonary status. However, the most common physical signs of PE are sudden onset dyspnea, tachypnea, chest pain, rales, and tachycardia (Fedullo et al., 2003; "Medical Emergency," 2003; Rumsey et al., 2004; Sharma, 2006).

Acute dyspnea is episodic, intense shortness of breath. Sixty percent of patients who have died from a massive PE had exhibited dyspnea, while 96% of patients with massive PE have tachypnea, 58% have rales, 44% have tachycardia, and 43% have a fever (Feied et al., 2006).

Chest pain in PE is commonly pleuritic or chest wall pain as opposed to cardiac pain. Patients may also have hemoptysis (Feied et al., 2006; Fedullo, et al.; Sharma, 2006).

Let's look at another case study.

### Case Study #2

John Rasmussen is a 65-year-old gentleman who was admitted for a total hip replacement. On the day of his planned discharge, he was ambulating in the hallway with physical therapy on his 4th postoperative day. He had an uneventful recovery from the surgery and had been compliant with his rehabilitation. He ambulated about 200 feet from his bedside when he suddenly complained of shortness of breath and dizziness. He stopped to rest at the nursing station. While talking to the nurse he dropped to the floor right in the middle of a sentence.

He was assisted back to bed where he complained of shortness of breath and slight chest pain. Vital signs revealed a heart rate of 110 and a respiratory rate of 30. Oxygen was immediately applied via a non-rebreather mask. Five minutes later, as he engaged in conversation with his nurse, he suffered a respiratory arrest and subsequent cardiac arrest. Attempts to resuscitate him were unsuccessful, and after 30 minutes he was pronounced. An autopsy revealed a PE. Mr. Rasmussen clearly illustrates the acute nature of the PE and the symptoms that may be mistaken for other conditions.

### Test Yourself! Case Study Question #2

**What medications could this patient have been prescribed that may have prevented a PE from developing?**

(Answer can be found on page 21.)

PE is difficult to diagnose on the basis of symptoms alone. Below are some common diseases and conditions that share similar symptoms (Fedullo et al., 2003; Feied et al., 2006):

- Acute Coronary Syndrome
- Acute Respiratory Syndrome
- Asthma
- Congestive Heart Failure
- Myocarditis
- Pericarditis/Cardiac Tamponade
- Pneumonia

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- Pneumothorax
- Lung carcinoma
- Pancreatitis

Armed with a detailed picture of the patient's health history and risk factors and familiarity with the pathophysiology of PE, the nurse can contribute more valuable input, possibly, than any other healthcare professional toward a quick and accurate diagnosis (Meyer et al., 2005).

### Diagnostic Testing

Because the symptoms of pulmonary embolism mimic so many other diseases and conditions there is no single specific diagnostic test that's adequate to diagnose PE in every patient (Fedullo et al., 2003; Kearon, 2003). The following diagnostic methodologies are used to rule in or rule out PE.

**Chest x-ray** – Chest x-rays are not considered sufficient to confirm the diagnosis. Early in the development of PE, x-rays may be normal (Feied et al., 2006; Sharma, 2006). As damage from the embolism progresses, the x-ray may show atelectasis, which is indistinguishable from pneumonia (Feied et al., 2006).

**EKG** – An EKG may show a sinus tachycardia or changes associated with increased right-sided cardiac pressure. An EKG is not a specific test for PE, but it may help clarify the diagnosis (Rumsey et al., 2004).

**D-dimer** testing is very sensitive to thrombus; however, it is not specific to DVT or PE. A positive D-dimer may not indicate a PE, but a negative D-dimer can help rule out a suspected PE in cases where the probability of PE is small to begin with (Feied et al., 2006).

**Arterial Blood Gases (ABGs)** – Blood gases may be normal, or when patients are tachypnic the patient may show mild respiratory alkalosis with a pH of greater than 7.44. ABGs are indicative of a patient's general respiratory status and can only contribute limited data toward a diagnosis (Feied et al., 2006).

**Pulse Oximetry** – As a strong warning to nurses who may rely too heavily on bedside technology, Feied et al. (2006) state: "In particular, pulse oximetry is extremely insensitive, is normal in the majority of patients with PE, and should not be used to direct a diagnostic workup" (p. 11).

**Echocardiography** – Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) are not generally accepted as definitive tests for PE (Kearon, 2003; Sharma, 2006). TEE in particular is unattractive because of its invasive nature. However, it is capable of finding central embolism in 70% of patients with right ventricular dysfunction (Kearon, 2003).

**VQ scans (lung perfusion scans)** – Also an invasive test, the perfusion test uses radioisotopes to track pulmonary airflow and blood flow. But VQ scans are frequently inconclusive, difficult to interpret, and, while still widely used, they are gradually being replaced with less invasive and more accurate diagnostics such as MRI or CT (Feied et al., 2006).

**Pulmonary angiography** – Angiography has long been considered the gold standard for PE diagnosis. But it is slow and highly invasive, requiring a catheter to be threaded through the heart to the pulmonary arteries. It requires a high level of technical skill and carries the possibility of serious risks. Like VQ scans, it is being replaced by speedier, less invasive and equally accurate scanning technology (Feied et al., 2006).

**Computerized Tomography (CT) scan** – CT scans are becoming more widely used because of their accuracy and less invasive nature. While there are some technical drawbacks, CTs are less

costly than angiography and can provide other helpful diagnostic information (Garg, 2005; Kearon, 2003).

**Spiral (helical) computerized tomography (CT) scan** – A helical CT scan is invasive because it uses dye contrast. The helical CT is an attractive diagnostic option because images of the target area can be taken in as little as 20 seconds. There are questions about its accuracy, which varies widely from 5% to 36% sensitivity and ranging up to 100% sensitivity for non-central PE (Garg, 2005).

**Multidetector computed tomographic angiography (MDCT)** – Newer imaging technology, MDCT has been used with good success for coronary artery imaging (Fratt, 2007). Although there are problems with how to manage the extraneous information generated by such scanning, MDCT is being used more frequently to diagnose PE (Fratt, 2007; Garg, 2005).

**Magnetic Resonance Imaging (MRI)** – MRI does not expose the patient to radiation or intravenous contrast, and can have a sensitivity of 85% and specificity of 96% for PE in certain areas of the lung (Sharma, 2006). Because it is a more expensive technique, it is often reserved for pregnant patients or those who may suffer kidney damage from the dyes used in other types of scanning.

**Doppler Studies for DVT** – Doppler studies are positive in approximately 50% of patients with known PE; however, this test can be misleading because of false positive results and because PEs occasionally originate in other areas of the venous circulation (Fedullo et al., 2003).

## Treatment

**ABCs** – As with any emergency situation, immediate treatment focuses on the ABC's (airway, breathing and circulation) when your patient develops a PE. With massive embolism, or when multiple small emboli block 50% of the lung's arterial system, the patient's condition can rapidly deteriorate into hypotension, right-sided heart failure and shock.

**Oxygen** – Dyspnea and tachypnea, are common signs of acute PE. Both are signals of hypoxia. Make sure any patient with these symptoms has an open airway (Rumsey et al., 2004). Particularly in the case of a massive PE, emergency measures must be taken to stabilize the patient's breathing and circulation. Initial treatment should include oxygen administration. Nasal O<sub>2</sub> can be given immediately to combat hypoxia and respiratory distress (Brunner et al., 2006).

**Fluid support** – Intravenous lines should be established, with normal saline (0.9%) or per order (Brunner et al., 2006).

**Diagnostics and Monitoring** – Diagnostic testing should be initiated as soon as possible. The patient's overall condition will dictate further treatment, which may include admission to intensive care. The patient's respiratory and cardiac status must be closely monitored (Brunner et al., 2006; "Pulmonary Embolism," 2005).

**Thrombolytic Therapy** – In severe cases when the patient is hemodynamically unstable, thrombolytic therapy may be warranted. The medications used are the same as those used for other major clotting emergencies (Kaufman, 2007; Sharma, 2006; "Thrombolytic Agents," 2001). Thrombolytics are classified as blood modifiers which activate the patient's own fibrinolytic mechanism to break up clots, thus restoring pulmonary blood flow (Kaufman, 2007; "Thrombolytic Agents," 2001). The following drugs have been approved by the Food and Drug Administration (FDA) for use in PE:

- Streptokinase
- Urokinase
- Alteplase (Recombinant tPA)

These drugs must be used with great caution. Your facility should have protocols in place for their use ("National Quality Forum," 2006). Check warnings and side effects specific to individual drugs. For example, streptokinase is linked to serious allergic and febrile reactions, and urokinase, which is derived from human neonatal kidney cells, is recommended by the FDA for use in only the most critical of situations.

Because of the potential for hemorrhage with these drugs, there are some absolute contraindications for their use ("Pulmonary Embolism," 2005). These include:

- Prior history of hemorrhagic stroke
- Any active bleeding
- Intracranial trauma or surgery within the past 2 months
- Recent large vein or artery puncture
- Recent CPR

There are also many related contraindications. These include ("Thrombolytic Agents," 2001):

- Pregnancy up to 10 days postpartum
- Severe hypertension (>180/110)
- Recent major surgery
- Severe liver impairment
- Recent trauma
- Active cavitating tuberculosis

- Organ biopsy within past 10 days

The decision to use thrombolytics is not made lightly. With a thorough and accurate physical assessment and health history, the bedside nurse can provide information critical to making this choice.

**Anticoagulant Therapy** should be given to any patient with DVT or suspected PE (Sharma, 2006). Unlike thrombolytics, anticoagulants interfere with the body's clotting cascade, in effect preventing an existing clot from enlarging and embolizing, while allowing more rapid resolution of the clot (Kaufman, 2007).

As with thrombolytic therapy, there are some general absolute contraindications to anticoagulant therapy ("Thrombosis Risk Factor Assessment," 2007). These include:

- Active hemorrhage of any kind
- Heparin-induced thrombocytopenia
- Coumadin use in pregnancy
- Severe trauma with substantial risk of hemorrhage

Be sure to document any of these conditions in the patient's record and alert healthcare providers appropriately.

**Heparin** is the drug of choice for treatment of both acute DVT and PE. It is given as soon as a clot is diagnosed or is clinically probable ("Heparin Sodium Injection, USP," 2006). Either the unfractionated or low molecular weight (LMWH) forms may be given (Feied et al., 2006; Quinlan, McQuillan, & Eikelboom, 2004).

Heparin sodium may be derived from beef or pork tissue, so sensitivity to either beef or pork products is a specific contraindication for heparin therapy ("Heparin Sodium Injection, USP," 2006; "Thrombosis Risk Factor Assessment," 2007).

**Unfractionated heparin (UFH)** is usually given as a bolus and then as a continuous infusion. The administration rate will be determined by the patient's clotting tests, aiming for a partial prothromboplastin time (PTT) of 1.5 to 2.5 times greater than normal ("Pulmonary Embolism," 2005).

Patients may later be converted from UFH to an oral anticoagulant such as warfarin (Coumadin) ("Heparin Sodium Injection, USP," 2006). The goal of long term anticoagulation therapy is the prevention of DVT. The duration of anticoagulant therapy is determined by reversible and irreversible risk factors and the number of thromboembolic events in the patient's history. Generally the therapy will continue for 3 to 6 months for the first occurrence and up to a year for repeated episodes (Schreiber, 2005; Sharma, 2006).

**Low molecular weight heparin (LMWH)** is a group of drugs chemically similar to unfractionated heparin but somewhat easier to manage (Feied et al., 2006; Schreiber, 2005). They can be given subcutaneously (SC) once or twice daily. The dosage doesn't have to be adjusted to clotting times. Contraindications include bleeding risks, and the major adverse reaction is hemorrhaging (Sharma, 2006).

Anticoagulants may also have interactions with many other medications, so before administering any anticoagulant, you should be familiar with your patient's medication regime (Sharma, 2006). The following is a partial list of LMWH medications available in the United States:

- Enoxaparin (Lovenox)
- Fondaparinux (Arixtra)
- Dalteparin (Fragmin)
- Andeparin (Normiflo)

**Warfarin (Coumadin)** is an oral medication that is given for long term anticoagulation for DVT or PE. It interferes with Vitamin K-dependent clotting mechanisms. It does not act on existing clots (Schreiber, 2005). Initial doses of Coumadin are overlapped with heparin until INR (international normalized ratio) values are maintained at 2-3.5 (Feied et al., 2006; Schreiber, 2005). Dosages are continuously adjusted according to clotting times. These must be checked daily until optimal anticoagulation is reached, then weekly and monthly. If the patient's circumstances prevent close and regular monitoring, Coumadin should not be used (Schreiber, 2005).

**Antiplatelet agents** such as aspirin, clopidogrel (Plavix), and dipyridamole/aspirin (Aggrenox) are not commonly used for the treatment or prevention of DVT or PE. These drugs have not been proven as effective as anticoagulants in the treatment or prevention of DVT or PE and current guidelines advise against the use of aspirin as the drug of choice for DVT prevention (Kehl-Pruitt, 2006; Tang, 2006).

**Surgery** involves removal of the embolism from the pulmonary circulatory system, or embolectomy, is done under extremely emergent conditions when the patient cannot maintain adequate cardiac output and is not a candidate for thrombolysis (Shaughnessy, 2007). Pulmonary embolectomy requires a thoracotomy and cardiopulmonary bypass. Catheters can be used to remove the clot, although the situation sometimes requires that the pulmonary artery be incised to reach and manually remove the clot (Brunner et al., 2006; Shaughnessy, 2007). The survival rate for this type of surgery only approaches 10% (Kaufman, 2007).

For recurrent PE, or when the patient cannot tolerate or is not a candidate for anticoagulation, surgery may be done to place a filter into the inferior vena cava. The filter, which resembles a tiny umbrella, is inserted via catheter through the common femoral vein or internal jugular vein and opened to block emboli and prevent them from entering pulmonary circulation (Brunner et al., 2006, Schreiber, 2005; Tang, 2006).

## Prevention/Prophylaxis

Most PEs are caused by clots originating in the deep veins of the legs (Kaufman, 2007). Pulmonary embolism is one of the leading causes of preventable in-hospital deaths (Schreiber, 2005). Data suggest that 40 to 60% of patients with total hip replacement will get a DVT (“DVT/PE Prophylaxis,” n.d.). Yet studies show that hospital or nursing home patients are at high risk for DVT because they may not receive any preventive care. Surgical patients are far more likely to receive prophylaxis than high-risk medical patients (Evans, 2004).

In 2006, the National Quality Forum published a set of consensus standards addressing institutional management of venous thromboembolism (VTE) including DVT and PE. These standards emphasize evidence-based risk assessment, diagnosis, treatment and clinically appropriate prophylaxis, and advise that “every health care facility shall have a written policy” incorporating these principles (“National Quality Forum,” 2006). Clearly the best treatment for PE is prevention (Kehl-Pruitt, 2006; Pathophysiology: Pulmonary Embolism,” n.d.).

A word about evidence-based assessment—While the phrase may sound daunting, it is less challenging if you keep in mind that “evidence-based” assessment is just that. It uses assessment criteria and risk factors based on evidence compiled from scientific research, as opposed to the traditional guidelines of expert opinion, local standards of care, or the “we’ve always done it that way” principle (Borgstrom, 2007).

Preventive care aims to correct the components of Virchow’s Triad, which include alterations in blood flow, vascular endothelial injury, and alterations in the constitution of the blood. Although there is ongoing debate in the community about the efficacy of one treatment over another, and the safety versus effectiveness of anticoagulant therapy, preventive care generally consists of these simple basics (Dosch, 2003; Kehl-Pruitt, 2006; Lotke & Lonner, 2005; Shaughnessy, 2007):

- Elevation of the legs, intermittent compression devices for bedridden patients, graduated compression stockings, (GCS, TEDs)
- Range of motion and exercises for the lower legs
- Ambulation whenever possible, as early as possible
- Adequate hydration
- Anticoagulation

If your patient develops any one or a combination of any of the following symptoms, he or she should be immediately evaluated for DVT (Dosch, 2003; Shaughnessy, 2007):

- Tenderness of the calf or inner thigh
- Leg, ankle or foot edema
- Temperature elevation without other obvious cause
- Redness/discoloration and warmth of skin on the leg
- Positive Homan’s sign (calf pain from dorsiflexion of the foot)
- Discrepancy in size of legs

Since long-term IV access lines are also a potential source of PE, check patency frequently and flush the lines as ordered—lines that are not in use should be flushed with heparin on a regular basis according to facility policy.



## **Nursing Care for PE**

Prevention is the primary goal of nursing care for patients at risk for developing PE. Once you have identified your patient's risk factors, document them in the patient records and communicate them to the patient's healthcare providers. Preventive care should be incorporated into every care plan for high risk patients.

Many preventive interventions are nursing measures such as monitoring respiratory status, assisting with ambulation, monitoring temperature and vital signs, and maintaining adequate hydration. Once a preventive plan is in place, evaluate the patient's response to your interventions and adjust them as necessary. It is appropriate to teach your patient to do leg and ankle exercises, including dorsiflexion and plantarflexion of the feet every two to three hours, but patients who are not able to do this independently will need help with range of motion to prevent pooling of venous blood (Kehl-Pruitt, 2006).

Make sure compression devices are applied and functioning correctly and encourage your patient to comply with their proper use (Kehl-Pruitt, 2006). Be particularly careful when fitting the patient with antiembolism stockings. This requires meticulous measurement of the patient's legs. If they aren't fitted and put on properly they are of little benefit and may actually interfere with circulation (Sharma, 2006). A sock-aide device may be used to help the patient get the stockings on; rubber gloves can help grip the slippery material. The patient's skin should be dry before any device is applied (Samala, 2006) and the stockings should be removed for 30 minutes every 8 hours and the underlying skin assessed for breakdown (Kehl-Pruitt, 2006; Samala, 2006). Graduated compression stockings are available for pregnant women.

Patients on anticoagulant therapy should be assessed on a continuous basis for bleeding. This can be any type of abnormal bleeding including bleeding gums, bruising, hematuria, and GI bleeding. In addition to the methodologies discussed above, nursing care for the patient with an acute PE should center on the following concerns.

**Monitoring therapies** – Treatment for acute PE includes oxygen, IV fluids, thrombolytics and anticoagulants.

Oxygen administration must be performed and documented meticulously. Airway and breathing assessments should be done frequently to evaluate the patient for response to therapy and for change in respiratory status (Brunner et al., 2006). Any unexplained restlessness, tachypnea or tachycardia may signal a worsening condition (Shaughnessy, 2007). Deep breathing and incentive spirometry are used to limit or prevent atelectasis and improve ventilation. More aggressive respiratory treatments may be appropriate if secretions are problematic (Brunner et al., 2006).

IV fluid administration should also be monitored carefully, and oral fluid intake encouraged when possible. A decrease of 2% body weight in fluid deficit is considered mild dehydration; 5% is moderate and 8% is severe. Dehydration can increase coagulability, or tendency to clot, by decreasing the blood's fluid component. This may be reflected in a higher-than-normal hematocrit. Conversely, fluid overload can place more stress on the heart and worsen any heart failure that may exist (Nagendran, 2001).

Thrombolytics and anticoagulants are commonly used in treatment of acute PE. Thrombolytics should be given in an intensive care setting with protocols as guidelines (Marchigiano, Reindeau, & Morse, 2006). Patients receiving therapy must be monitored very closely for allergic reactions and uncontrolled bleeding. If this occurs the infusion is stopped immediately. Urine and stool should be checked for occult or frank blood. Patients are kept on strict bedrest, with vital signs and neurovascular checks every hour. Invasive procedures are contraindicated. When blood is drawn, pressure must be applied to the site for 30 minutes (Brunner et al., 2006; Marchigiano et al., 2006).

Since dosages of anticoagulants such as heparin and Coumadin are closely tied to blood clotting values, verify that testing is done as ordered and the healthcare provider notified of the results. Patients should be monitored for response to treatment. Abnormal bleeding is the major danger with these medications.

**Managing discomfort** – Your patient may experience significant chest wall or pleuritic pain with acute PE (Marchigiano et al., 2006; Sharma, 2006). Assess for pain at least every 4 hours, and after medicating as ordered and providing comfort measures such as positioning in bed, reassess the effectiveness of your pain management interventions (Brunner et al., 2006; Marchigiano et al., 2006).

Anxiety is also common in patients with acute PE. A calm and comforting approach helps decrease the patient's distress. Allow the patient to verbalize fears. Answer questions and explain treatments in a simple and direct manner, and reassure the patient that this condition can be successfully treated. (Brunner et al., 2006; Marchigiano et al., 2006; Shaughnessy, 2007).

**Teaching** – You may never have a better opportunity to use your teaching skills than when working with a patient with PE. Supplying the information your patient needs at a level adapted to individual learning capabilities is one of the most important aspects of the care you provide (Shaughnessy, 2007).

Topics for discussion will include the nature of the condition, causes and treatments. The patient will need explanations of whatever diagnostic tests are chosen. Medication regimens should be discussed in detail with the patient, their family or caregiver, with clear instructions as to side effects, precautions, contraindications, and interactions with other medications. Patients on anticoagulant therapy should be encouraged to wear an emergency identification bracelet or carry their health information in a purse or wallet.

Anticoagulants interact with a number of other drugs, and many common foods containing vitamin K. Your patient will need information about drug interactions and what to discuss with her medical provider, such as the use of over-the-counter medications (Sharma, 2006).

Dietary restrictions should be reinforced, particularly when patients are taking Coumadin, with its many food interactions. Dietary counseling should teach avoidance of such foods as spinach, green tea, peas, cauliflower, soybeans, apricots, and lettuce. Your patient will need to understand the importance of having regular blood work done and the need to take the medication just as it's ordered (Brunner et al., 2006).

The patient should know the signs of abnormal bleeding and how to recognize and prevent it. For example, you can teach the patient to look for bruising after minor trauma, to use an electric shaver instead of razors, and to use soft-bristled toothbrushes to protect the gums from injury (Brunner et al., 2006).

A home fall prevention plan may be appropriate (Marchigiano et al., 2006). Patients should be reminded to stretch their legs frequently when in confined situations such as long distance travel (Marchigiano et al., 2006; Shaughnessy, 2007). Remind your patient that walking and keeping physically active can lower the risk of recurrent PE.

If the patient is expected to be on LMWH therapy, assess his or her ability to self-administer the drug, and tailor your teaching accordingly. Make sure you know what the discharge plan is, so therapy can continue without interruption.

Some patients may need additional counseling and support; for example, obesity in women is thought to be a factor in PE formation (Poirier et al., 2006). The fact that obesity is a reversible risk factor is good news, but these women may need special counseling and encouragement to successfully cope with the challenge of being overweight.

Patients with cancer who are receiving chemotherapy should be counseled to be particularly vigilant for signs of DVT or PE (Hamilton, n.d.). Having cancer increases the risk of hypercoagulability.

Once any patient has had a PE, there's always a risk that it will recur. Be sure your recovering patient knows about this risk and understands that it is very important to follow through with medical and non-medical prevention strategies.

## **Conclusion**

Acute pulmonary embolism is one of the most serious situations you may face as a nurse. Being aware of the risk factors, the mechanisms, signs and symptoms, and emergency care for PE can help you save your patient's life. Knowledge of treatment and prevention modalities will allow you to work with and assist your patient to achieve recovery, avoid injury, comply effectively with therapies and reduce the risk of recurrence after discharge.

## Answers to Case Study Questions

1. Atrial fibrillation, myocardial infarction, smoking, knee replacement, sedentary lifestyle, and hypercoagulability
2. Lovenox SC, warfarin (Coumadin) PO, heparin IV or SC, streptokinase, or urokinase. *Even with the use of these medications some PEs cannot be prevented.*

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## Caring for Patients with Acute Pulmonary Embolism

### Course Exam

After studying the downloaded course and completing the course exam, you need to enter your answers online. **Answers cannot be graded from this downloadable version of the course.** To enter your answers online, go to e-leaRN's Web site, [www.elearnonline.net](http://www.elearnonline.net) and click on the Login/My Account button. As a returning student, login using the username and password you created, click on the "Go to Course" link, and proceed to the course exam.

Please read the following scenario and choose the best answer to each question.

*As a nurse on the day shift, you'll be caring for Mrs. Walters, a 70-year-old woman. Mrs. Walters recently fell at home and fractured her right ankle, requiring extensive surgical repair. Mrs. Walters is now 48 hours post-op. Although she should be preparing for discharge home, she is having difficulty moving in bed and is reluctant to get out of bed due to fear of pain and falling.*

*The offgoing nurse reports that Mrs. Walters used her patient controlled analgesia system to self-medicate at 12 AM and slept soundly through the night. This morning she refused to transfer to a bedside commode because she was afraid she would fall. She has been afebrile. Respirations at 12 AM were 16, clear and unlabored, pulse 68 and regular, blood pressure 130/80. Her post-op checks of peripheral circulation have revealed no problems, and she is alert and oriented.*

*Mrs. Walters' admission assessment indicates that she is obese, has a history of venous stasis ulcers and has had a mild stroke which resolved without therapy.*

*As you walk toward Mrs. Walters' room, her call light comes on. You find her sitting upright in bed sweating profusely and saying that suddenly she has begun to feel short of breath. She denies having chest pain but feels as if she's "gasping for air." She appears extremely anxious.*

*You do a quick assessment: her skin is pale and diaphoretic, and her respirations are 22 and labored with rales throughout the right side. Her pulse is 110, blood pressure 150/90. Her pulse oximetry reading is 98% on room air.*

1. You know that Mrs. Walters has several risk factors for pulmonary embolism (PE). Other risk factors include:
  - A. Blood type A and varicose veins
  - B. Blood type B and a history of pneumonia
  - C. A history of recent motor vehicle accident
  - D. A and C
2. As you finish your assessment, Mrs. Walters says clutches your hand and says, "I can't breathe! What's wrong with me?" You suspect she may have developed a PE. You should:
  - A. Reassure her that nothing's wrong since her pulse oximetry is normal
  - B. Reassure her that she'll be getting help very quickly, then prepare to administer O<sub>2</sub> via nasal cannula
  - C. Check to see what her clotting times are
  - D. Encourage her to get out of bed as soon as possible
3. You notify Mrs. Walters' medical care provider of her change in respiratory status and continue her emergency care. You should:
  - A. Prepare to start a second IV line
  - B. Measure her calves for a discrepancy in size
  - C. Tell her she may need to go home on anticoagulant medications
  - D. Have her do active range of motion

4. 90% of all pulmonary embolisms arise from:
  - A. Pelvic surgery
  - B. Central catheters
  - C. Deep vein thrombosis (DVT)
  - D. Fat emboli
  
5. After Mrs. Walters has received emergency care, you continue your assessment. This will include:
  - A. Rechecking her breathing and circulatory status
  - B. Checking for calf tenderness, swelling, redness and warmth
  - C. Checking for abnormal bleeding at IV insertion sites
  - D. A and B
  
6. Patients with acute PE may experience:
  - A. Abdominal pain and distension
  - B. Pleuritic pain
  - C. Signs of right sided heart failure
  - D. B and C
  
7. A bedside Doppler ultrasound test has been arranged for Mrs. Walters. When she asks you about the test, you tell her:
  - A. It is designed to find clots in the legs
  - B. It is nearly 100% accurate
  - C. A negative test result doesn't rule out a PE
  - D. A and C
  
8. Mrs. Walters tells you her medical provider has ordered a "VQ scan" but she's not sure what the scan does. You can tell her that the VQ scan:
  - A. Examines lung circulation by means of a catheter threaded through the heart
  - B. Will not expose her to radiation
  - C. Shows blood flow and airflow in the lungs
  - D. Shows heart rhythm abnormalities related to the clot in her lung
  
9. Mrs. Walters' medical provider is considering the use of thrombolytic therapy. An absolute contraindication for this therapy is:
  - A. Recent pregnancy/delivery
  - B. Kidney failure
  - C. Recent cardiopulmonary resuscitation (CPR)
  - D. Organ biopsy within 10 days of therapy
  
10. Mrs. Walters is started on heparin therapy. Prior to administering the therapy, you should ask:
  - A. If she has recently had chemotherapy
  - B. If she is allergic to pork or beef products
  - C. If she is allergic to shellfish
  - D. If she has a history of pancreatitis

11. As you update Mrs. Walters' care plan, you'll include:
- A. Incentive spirometry
  - B. Strict bed rest
  - C. Range of motion exercises
  - D. A and C
12. The following is a true statement about anticoagulant therapy:
- A. Aspirin is highly recommended as a prophylactic treatment for DVT and PE
  - B. Heparin is the drug of choice for acute DVT and PE
  - C. Heparin should never be given until PE has been clearly diagnosed
  - D. Heparin is contraindicated for cancer patients
13. Mrs. Walters can be switched from heparin to Coumadin when her blood clotting times (PTTs) have reached a therapeutic level. This would be:
- A. At normal levels
  - B. 2-3 times the normal level
  - C. 1.5-2.5 times greater than normal
  - D. Slightly below normal levels
14. Mrs. Walters prepares to go home on Coumadin therapy. Her discharge plan should include:
- A. Arranging to have her clotting times checked every 6 months
  - B. Teaching her to include several servings of green leafy vegetables in her daily diet
  - C. Reassuring her that it's okay to skip a dose of her anticoagulant
  - D. Arranging home nursing visits
15. Mrs. Walters should be reminded to:
- A. Report any unusual bruising to her medical care provider
  - B. Use a soft-bristled toothbrush
  - C. Flex her feet every 2-3 hours throughout the day
  - D. All of the above