To the Faculty of Washington State University:

The members of the committee appointed to examine the Intercollegiate College of Nursing Research requirements and manuscript of TANDA M. FERGUSON find it satisfactory and recommend that it be accepted.

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Abstract

Pulmonary embolus is known as a Great Masquerader for the non-specificity of symptoms and inconclusiveness of diagnostic tests. The annual incident in the United States alone is about 600,000 with 50,000 to 200,000 of those people dying. With rapid recognition and treatment, mortality and morbidity rates can be significantly improved. Most pulmonary emboli are diagnosed at autopsy, making this a subject needful of careful consideration during evaluation of any patient. Knowing how to interpret diagnostic tests and which are the most cost effective with the greatest specificity enhances the practitioner’s ability to rapidly diagnosis or rescue patients.
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Introduction

Patient's down between the wall and toilet, escape rhythm, no respirations, call a code! Crack the chest. Why aren't fluids running? The left ventricle is empty! Thus begins my own introduction to pulmonary emboli.

Various people from diverse walks of life develop life threatening pulmonary emboli. Most will live, thanks to rapid diagnosis and state of the art treatment. This article explains how practitioners, at all stages of health care delivery, learn to recognize patients at risk, to diagnose pulmonary emboli and then the life sustaining or rescuing treatments available today. Using the theoretical framework of the Health Belief Model (HBM), we will explore who is affected and why, what are the individual’s and the practitioner’s barriers to rapid, correct diagnosis, and the self-efficacy of correct, efficient treatment.

The HBM was developed in the 1950’s to understand the lack of public participation in the tuberculosis screening projects. In order to grapple with why the public was not flocking to government projects to help eradicate a public menace, the Public Health Department developed 5 stages, with 2 sub stages, which are found in table 1. In 1977, the 6th stage, also in table 1, was added to become the model used today. Pulmonary embolism appears to randomly choose the victims, yet using the HBM we can begin to recognize patterns that put people at risk, either from themselves or their naive practitioners. Then the HBM would be of help in the development of messages that are most likely to persuade individuals and practitioners to make health-aware decisions (NIH, 2001).
Epidemiography: Who is affected? What is the prevalence?

Pulmonary embolism is difficult to diagnose, related to the non-specificity of symptoms, and the inconclusiveness of diagnostic tests. With disturbing frequency clinicians and researchers alike report that pulmonary emboli is a necroptic diagnosis, discovered only on autopsy. Pulmonary embolism is called the “Great Masquerader in medicine” (Wells & Salyer, 2001). The estimated annual incidence of pulmonary embolism in the United States alone is about 600,000, with 50,000 to 200,000 deaths per year. Pineo (2001), states that pulmonary embolism is the direct cause or contributor in the deaths of approximately 200,000 U.S. citizens. Pulmonary emboli represent about 5 to 10% of all hospital deaths with massive pulmonary embolus, class 4, (Table 3) having a mortality rate of 70%, often in the first 1 to 2 hours. Called “the silent killer of the elderly” pulmonary embolism is one of the most under diagnosed disease processes of the elderly (Webster & Marquardt, 1974). Similar statistics estimate that 30% of pulmonary embolism patients will experience a fatal pulmonary embolus, with 80% of those dying 1 to 2 hours from onset of symptoms (Schumann, 2000). With the estimate of mortality at 30%, and the diagnosis being made at autopsy in 2/3 of those patients, in comparison with a mortality rate of only 2 to 8% in timely and appropriately treated patients, clinicians need always to maintain a high level of suspicion, could this be a pulmonary embolus (Wells & Salyer, 2001).
Pathophysiology

Virchow first described pulmonary embolism in the 1800’s, discovering the three physiologic factors that predispose someone to inappropriate thrombogenesis. Virchow's Triad, postulated more than 150 years ago still stands true today, consisting of abnormalities of blood flow or venous stasis, hypercoagulability, and intimal injury to the vessel walls (Schumann, 2000). Thrombolic events are the most common form of pulmonary embolism, but other considerations could be fat, amniotic fluid, air, tumor, foreign material such as bullets, sutures, catheter tips, and precipitated crystals from medications or total parenteral nutrition (Schumann, 2000; Reedy et al., 1999). Another postulated abnormality is the autosomal dominant disorder of the coagulation system known as Activated Protein C resistance (APC-R). APC-R is a vitamin K-dependent zymogen to a serine protease that acts as an anticoagulant on the surface of the endothelial cells by a complex of thrombin and thrombomodulin. Those with the mutated gene carry a seven-fold risk of venous thrombosis. In order to determine the incidence of the APC-R gene medical examiners need to autopsy victims of pulmonary embolus (Rulon, Cho, Guerra, Bux, & Gulley, 1999).

The Bexar County Forensic Science Center in San Antonio, Texas studied 66 cases of sudden death from pulmonary embolism with a median age of 46 years and range from 14 to 93. Of the 66 cases, 56% were males and the other 44% were female, 77% were under the age of 60 and 18% were under the age of 30. Nationality ranges were 53% non-Hispanic Caucasian, 24% African American, and 23% Hispanic (Rulon, et al., 1999). Risk factors were obesity,
immobility, oral contraception, malignancy, prior thrombotic events, spontaneous vaginal delivery, pelvic obstruction from benign ovarian masses and a persistent vegetative state. Three of the 66 cases (4.5%) were heterozygous for factor V 506Q mutation (APC-R) with a 95% confidence interval. The American population reported to carry the APC-R gene is 3 to 5%, similar to that of the Bexar study results (Ruion, et al., 1999). Those with the mutated genome are at no greater risk of pulmonary embolus than the general population, despite the increased risk of deep vein thrombosis. This study should be replicated in multiple centers to confirm that APR-C does not increase the risk of death from pulmonary embolism. Further study should be done on those who survive their experience with pulmonary embolism to determine the influence, if any, that APR-C might have in their risk and resolution of pulmonary embolus. Having a definite marker for pulmonary embolism would have significant implication in diagnosis and prevention.

Clinical Presentation

Kra (2000), a cardiologist, giving his personal experience with multiple pulmonary emboli, indicated that his first symptoms were on the tennis court, becoming sloppy, exhausted and increasingly short of breath. Universally, every article, textbook and algorithm (Figure 1) will list as the primary, yet non-specific symptom as becoming increasingly short of breath. Often a progressive dyspnea will be rationalized, first by the victim, thinking “I’m just out of shape” or other denial self-talk. Once they do present for treatment with the complaint of exertional dyspnea or shortness of breath, other diagnoses are considered, often
without any consideration given to pulmonary embolism (Wells & Salyer, 2001).
Patients will most often present with acute symptoms including, but not limited to, (Figure 1) dyspnea, pleuritic chest pain, mild to severe and sudden. Patients describe pain as a “thrust of a knife” rather than as an “elephant sitting on my chest”. The clinician can be expected to observe a cough, with or without wheeze, tachypnea, hypotension, fever, tachycardia and possibly hemoptysis (Figure 1) (Schumann, 2000, Farg & Costello, 2001; Kra, 2000; Wells & Salyer, 2000). An elderly patient’s only presentation might be syncope (Farg & Costello, 2001).

When a patient presents with relatively generic symptoms that are associated with pulmonary embolism, a provider should consider other potential causative sources (Table 2) and rule each one out, while proceeding towards a diagnosis. Often, the patient’s presentation will depend on the extent of the pulmonary embolus and segmental versus central location.

Diagnostics Testing
Diagnosis of pulmonary embolism will consist of three tiers. First, is clinical evaluation, or “the story and the assessment” which uses the clinician’s skills, then non-imaging, or laboratory testing and electrocardiogram. Finally, imaging tests such as x-ray, computed tomography (CT) or nuclear medicine studies should be done to confirm the diagnosis, (see algorithm in Figure 1).

When a patient presents with shortness of breath and chest pain, baseline oximetry should be done, then oxygen delivery should be titrated according to the result. EKG patches should be placed on the chest while vital signs are being
obtained. A twelve lead EKG should be done to look for right heart enlargement or strain, possible pulmonary hypertension and elevated or depressed T-wave morphology, which would be indicative of cardiac ischemia or injury. A change in T-wave morphology is presumptive for cardiac involvement rather than pulmonary embolism. However, EKG changes of S_{1}Q_{3} or S_{1}Q_{3}T_{3} pattern, a rightward shifting of the QRS axis, an incomplete or complete transient right bundle branch block and T-wave inversion in the right precordial leads will alert the practitioner to consider pulmonary embolus (Chou, 1986). As soon as intravenous access is obtained, and blood samples available, venous labs such as complete blood count (CBC), complete metabolic profile (CMP), Troponin-I and a D-dimer ELISA should be done, see the algorithm in Figure 1). Arterial blood is needed for pCO_{2}, pH, pO_{2} and bicarbonate, to establish baseline criteria along with determining when and if the need for intubation & mechanical ventilation exists.

A D-dimer, which is a marker for fibrinolytic activity, may be normal if drawn too early in the course of events. If normal, it has a 90% negative predictive value for pulmonary embolism. Specificity of the D-dimer can be influenced by other causes such as heritage, which includes a family history of deep vein thrombosis, or pulmonary embolus. As is noted in the 1992 Epidemiologic Studies of the Elderly, (Duke University), those with a black heritage were 40% more likely to have elevated D-dimers than caucasians, even when controlled in the multivariable analysis (Pieper, Rao, Currie, Harris & Cohen, 2000). Other influences on D-dimer elevations are hypertension,
pneumonias, cancer, sepsis, myocardial infarction and surgery (Goldhaber, 2000; Pieper, Rao, Currie, Harris & Cohen, 2000). The Troponin I will direct the practitioner in most cases towards a cardiac diagnosis; whereas a D-dimer ELISA, will provide an implication that there is clot present within the body. Troponin I levels can be elevated in pulmonary embolism related to right heart strain releasing the enzyme into the blood stream (Meyer et al., 2000). Troponin I can also be normal during the onset time of ischemic angina, because measurable levels from striated muscle damage take approximately twenty minutes to occur.

Chest films provide an indication of cardiac silhouette size, pulmonary congestion, pleural effusions and pneumonias. Physical assessment findings may be limited or absent, if a pulmonary embolus is present. Any assessment abnormalities might mislead the provider to assume that this is a cardiac event, including but not limited to diaphoresis, shortness of breath, chest pain and tachycardia.

Occasionally, a patient might present with a S4 or an accentuated P2. The landmark study, Prospective Investigation of Pulmonary Embolism Diagnosis (PRIOPED, 1990) listed the most common signs and symptoms of pulmonary embolus presenting as a person "without pre-existing cardiac disease, dyspnea, tachypnea, pleuritic chest pain, crackles, cough, tachycardia, S4, accentuated P2, and hemoptysis," as seen in Table 3 (Wells & Salyer, 2001). Syncope can be the only presenting symptom in the elderly (Masotti et al., 2000).
Physical symptoms can assist in separation of the two diagnoses. They can be pleuritic chest pain, or increased pain by cough and or deep breaths, which would indicate an involvement of lung, pleura or chest wall versus a deep pain and pressure that is unaffected by coughing or breathing which would be more indicative of cardiac ischemia.

While rest and or oxygen often relieve cardiac pain, pain from pulmonary emboli will not be affected by the same intervention. Visible cyanosis can be present with the inhibition of gas exchange, but rarely in a cardiac event. With community-acquired pneumonia, the white blood count is usually elevated. Pneumonia is also associated with an increased production of sputum, cough and fever. Asthma might acutely present with cyanosis, but is accompanied by wheezes, stridor and a significant decrease in air exchange. Pulmonary embolus impedes the blood flow, not airflow, but the patient might have wheezes associated with histamine release secondary to clot lyses, (see Table 2 for a differential diagnosis listing).

EKG changes, with the exception of those listed above; with or without a positive Troponin I, indicate acute cardiac problems. Compromised arterial blood gas exchange with the physical symptoms of wheeze and negative Troponin I and D-dimer, may indicate asthma. As stated above, community acquired pneumonia is often accompanied by elevated WBC’s, chest film changes and above or below clinically normal temperature, usually indicative of bacterial or viral origin.
An Echocardiogram may be done to determine cardiac size, cardiac and pulmonary pressures, and the ejection fraction of the right and left ventricle, valvular dysfunction or even a newly opened foramen Ovale. Observation of movement of the interventricular septal wall towards the left ventricle is present in 31% of those with positive pulmonary emboli (Masotti et al., 2000). On echocardiogram an ischemic heart presents with increased left-sided filling, poor wall motion and pulmonary congestion and hypotension. A right-sided infarct potentially enlarges the right ventricle, increases preload and peripheral edema, usually without pulmonary congestion. Associated with pulmonary emboli on echocardiogram, the right ventricle will be enlarged with a left shift or deviation of the septum. Pulmonary embolism can be accompanied by pulmonary hypertension and or congestion with small left heart chambers. The primary diagnosis should proceed to pulmonary embolus until ruled out, if this is the clinical presentation.

Dependent on the capabilities of the facilities, and the availability of diagnostic teams there are three methods of determining occlusion of the pulmonary vessels. The gold standard of diagnosis for pulmonary embolism has been pulmonary angiography, but it is costly, invasive and not without risk (Fang & Costello, 2001; Velmahos et al., 2001). Angiography involves the threading of a femoral catheter up towards the pulmonary artery in order to inject dye. The dye imaging provides a visual picture of the pulmonary vasculature, demonstrating the presence or absence of occlusions. Angiography is expensive, and requires a team of people who are usually not on site, outside of
normal business hours. The greatest “expense” is actually in the risk to the already compromised patient. If there is a positive diagnosis of pulmonary embolism, using thrombolytics might be inhibited related to disruption of the integrity of the femoral vessel, putting the patient at an increased probability of significant bleeding. While statistically low in incidence, the risk of further catheter induced emboli, bleeding, dye induced renal failure and other complications of any invasive procedure do occur.

The most commonly used test, both in the United States and internationally, is that of Ventilation/Perfusion (VA/Q) Scan. V/Q scans use an isotope to determine, if there is impedance to the transfer of gases across the alveolar membrane. The results are graded into very low, low, intermediate and high probability of embolus. If the patient falls into either end of the spectrum then the diagnosis is either ruled out, very low probability for PE, or the patient is diagnosed with pulmonary embolism. However, most often you are faced with an indeterminate reading, about 50% of the time, and must use clinical judgment to proceed.

Increasingly, the choice of scanning tests is becoming the computed tomography (CT) of the chest. The CT scan is helpful for several reasons. With the use of dye, you can diagnose 100% of life threatening central emboli. The CT scan is generally considered inaccurate in segmental pulmonary arteries occlusions; (see Table 3 for classifications of pulmonary embolisms) (Goldhaber, 2000; Gotway, Edinburgh, Feldstein, Lehman, Reddy, & Webb, 1999; Velmahos et al., 2001). Another advantage that CT has over all other tests is that it is
diagnostic for malignancies, pneumonias, dissections, tamponades and fractures. CT also takes less time than nuclear medicine scanning or angiography. However, like angiography, since a dye is used, there is the risk of anaphylaxis or renal failure.

Magnetic Resonance Imaging (MRI) is still in the investigational stages of diagnosing PE. MRI has excellent imaging resolution and may soon be added to the clinician’s arsenal of diagnostic tools.

Treatment

Treatment of pulmonary embolism is dependent to a certain extent on the severity and the acute or chronic nature of the emboli. Table 3 presents classifications or levels of pulmonary embolism. The provider, when looking for something else, such as a tumor, might discover class or level one. Treatment of Class 1 and 2 pulmonary emboli should be with anticoagulant therapy using intravenous Heparin for seven to ten days, while maintaining activated partial thromboplastin time (aPTT) 1.5 to 2.5 times over the standard control value along with supplemental oxygen. Warfarin, (Coumadin) therapy should be started while on Heparin until a therapeutic International Normalized Ratio (INR) of 1.5 to 2.0 times greater than the control is achieved.

Class or level 3 and 4 (table 3) by their nature, will require more aggressive management. Rapid thrombolytic therapy should be instituted, as soon as diagnosis is determined. Streptokinase (Streptase), Urokinase (Abbokinase) and Tissue Plasminogen Activator (Alteplase) (TPA) are the current choices with differing onset and half-life. TPA has the most rapid onset
and effective half-life of four minutes. Intravenous Heparin therapy should be started simultaneously. Again, Warfarin (Coumadin) therapy should be started and therapeutic levels achieved prior to stopping Heparin infusion. Contraindications to thrombolytic therapy will be gastrointestinal bleeding, uncontrolled hypertension, surgical procedures and cerebrovascular or intracranial bleeding. Class 3 and 4 patients often need hemodynamic lines, such as a pulmonary artery catheter to manage fluids and inotropic medicines. While Class 3 patients might get by with a 100% non-rebreather mask or Bi-pap, Class 4 patients will need to be intubated and mechanically ventilated.

In some cases, if available, a venous umbrella or intraluminal filter device (VCF) will be placed percutaneously to filter out clots 95% of the time. These devices can be inserted into the inferior vena cava, in some cases safely at the ICU bedside, but usually in the catheterization lab or surgery. Clots will not be ensnared in the filter when migration is from the brachial or subclavian veins. The devices will not be able strain any clots that originate in the right atrium or ventricle. The devices carry a low morbidity and mortality rate. In rare cases, PE has occurred from, as early as 8 days to 5.5 years after first VCF insertion (David, Gross, Colaiuta, Gonda et al., 1999).

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Often patients will need oxygen therapy at time of discharge, especially if Class 4. All pulmonary emboli patients will need to be on Warfarin (Coumadin) therapy. The duration is controversial, but standard usage is considered to be six months (Chesnutt, Prendergast & Stauffer, 1999; Webb et al., 1999).

Case One

MC, is a 54-year-old female presenting with a long history of depression and obesity. She has recently retired from floor nursing. She has added a walking and mild exercising program to her new life. Several days after instituting the new physical activity, she is seen in the local ER for shortness of breath and chest pain.

MC has a heart rate of 100, blood pressure of 125/80 and a respiratory rate of 20. Physical assessment reveals no jugular venous distention, a few bibasilar lung crackles, with no expiratory wheezes, no dullness to percussion, no focal chest wall pain. Her cardiac assessment was regular heart tones, normal S₁S₂, and without any murmur, rubs or clicks. Her abdomen is soft, non-tender with active bowel tones, no masses or organomegaly.

Her lab work is noted in Table 5 and is non-contributory to her sorting out disease process. ABG’s and D-dimer were not ordered at the time, as the
prevailing diagnosis was cardiac ischemia and her oxygen saturation was 96% on 4 liters per nasal cannula. MC had a year old EKG available in the system for comparison. Her current EKG indicated possibly a two-week-old myocardial infarction by story and changes. There was evidence of left ventricle dysfunction leading to the supposition of early CHF. In the review of old records, it was determined that there was a previous Dobutamine Echo that was normal with no evidence of a prior infarct. Furthermore there was no inducible ischemia. An Echo was ordered to determine the need of immediate cardiac catheterization or elective catheterization during the normal business hours. The left ventricular ejection fraction was 45%, with mild hypokinesis of the anterior septum, anterior wall and inferior wall. The left ventricle was small with a normal wall thickness, but the right ventricle was enlarged and there was mild tricuspid regurgitation. Pulmonary artery systole was 45 mm Hg. None of these changes were present in the previous years study. Neither the nuclear medicine team nor the catheterization teams were in house, but CT was immediately available for scanning.

By use of the CT (figure 4, 5 & 6), MC was diagnosed with extensive bilateral pulmonary emboli, which most likely started two weeks prior and presented acutely. Since the clots had been in all probability accumulating over a prolonged period of time immediate lyses was not attempted but heparin was started and her oxygenation was supported with Bi-pap. After 10 days of hospitalization, MC was discharged to home with the Echo & EKG both returned
to baseline. The patient was discharged on Warfarin (Coumadin), aspirin and oxygen.

Case Two

MM, 52 years old, had untreated diabetes, obesity and a significant smoking history. She complained to her house partners of becoming increasingly short of breath over several days but refuses to seek treatment until she was blue, respiratory rate in the 60’s and is unable to deny 911 being called. Living close, she arrived at the ER on 100% oxygen, normal saline wide open to maintain blood pressure and an oxygen saturation of 77%. She was immediately intubated and mechanically ventilated.

Primary assessment reveal cyanosis from the nipple line up, non-palpable pulses, clear breath sounds, and normal S1, S2 heart tones. The EKG monitor showed a narrow complex rhythm with a variable rate of 125 to 225. She was given adenosine per normal protocol without response. She was then cardioverted anteriorly-posteriorly with 75 bi-phasic joules to a consistent sinus tachycardia rate of 125. Her portable chest film showed clear lung fields with a generous right heart border. Her D-dimer was mildly positive, at 0.4 to 0.8 %. A subsequent CT-scan was positive for massive pulmonary embolism and she is started on Alteplase (TPA) while in the CT-scan room, and then moved to ICCU. While in the ER she had been hydrated related to her blood glucose greater than eight hundred and started on an insulin drip. While establishing an IV line blood was drawn for blood cultures, CBC, CMP, Troponin I and D-dimer. Her lab values
were essentially normal, including a negative Troponin I, potassium greater than 4, a white blood cell count of 15,500.

Her course over the next several hours was quite rocky, needing one-on-one nursing care, cardiac resuscitation several times and inotropic support. Subsequent cardiac echocardiogram demonstrated an open foramen ovale, Medications consisted of dose appropriate Heparin, Amiodarone (Cordarone), narcotics for sedation and comfort, H2 blocker and empirical antibiotics. Dopamine (Intropin), fluids and insulin were continued.

For the first several days’ survival was definitely in question. However, she slowly responded, her cardiac echocardiogram returned to normal, as did her EKG. She was discharged from the hospital on oxygen and Coumadin, along with diabetic and antihypertensive medications. At her two-week office visit she presented on room air having “forgotten” her portable oxygen at home. She was encouraged to use oxygen on a pro re nata (PRN) basis. As an outpatient, her primary care needs of hypertension and diabetes took the forefront in her care.

Summary

Using the theoretical framework of the Health Belief Model (HBM), which is oriented to the prevention of disease, the practitioner can look at what might make some people more susceptible to a disease and therefore interventions or possibly prevention. We can further look to the HBM to determine the deficiency or barriers to rapid diagnosis of pulmonary embolism.

At first look, Kra (2000) did not fit Virchow’s triangle of venous stasis, hypercoagulability or intimal injury. He was athletic, didn’t smoke, his “lipids and
weight were just right", he took a daily baby aspirin and had an "enjoyable, balanced lifestyle". His perceived susceptibility for disease was very low. He knew that he was doing everything right. Yet, shortly after returning to the United States, by plane, from a vacation in Europe, he had trouble breathing and was fatigued while playing his normal tennis game. Mentally, he perceived no threat, therefore was not motivated to take any action until he was so short of breath and wheezing, that he could no longer see any of his patients several days later. Still, his barrier to seeking appropriate care in the emergency room was the fact that he was certain that he should not be susceptible to serious disease because of his life-style. He, instead, went to a colleague to “check my lungs.”

Throughout his hospitalization, he was unable to see himself as a patient, despite a near respiratory and cardiac collapse. Being a respected cardiologist and professor, in his own unit, surrounded by fellow staff members and seeing his own patients became his greatest barrier to self-efficacy. Writing his story was his demonstration of the perception of benefits and self-efficacy by alerting other health professionals concerning the power of denial.

MC & MM started at different points. Both had a history of hypertension, obesity and diabetes. The difference was in perception of threat. MC perceived her susceptibility to potential health problems, sought treatment for the hypertension and diabetes, while starting an exercise regime. When she became short of breath, she knew that it was not normal for her and perceiving a threat of significant disease she sought the benefits of rapid treatment in the emergency room. Having just retired, but on COBRA insurance, there was no
perception of barriers, therefore quick action on her part decreased the morbidity of her significant and potentially life-threatening disease.

MM was in denial of any health risks. She knew that she had a diagnosis of diabetes and hypertension, yet told her house-partners that she was controlling it with diet. She perceived no susceptibility to disease from the barriers of education level, finances and the opinion that she was doing just fine without medications. She continued to smoke heavily and had a persistent gain in her weight demonstrating her decreased motivation to take appropriate action. Even after her presentation in extremis with Nonketotic hyperglycemic hyperosmolar diabetes, massive central pulmonary emboli her partners maintained that she was strong, she could overcome anything. They enabled her inability to maintain health behaviors, before she became severely ill and even after her hospitalization. Despite being witness to her time in critical care, the fact that she survived reinforced their perception that she could survive anything. Their attitude potentially becomes a barrier to her self-efficacy and motivation of healthy behaviors, by reinforcing MM previous perceptions of susceptibility.

Health professionals often do not perceive the risk of threat. Today's media and research money go into breast cancer awareness, HIV and cardiac disease. Despite a significant number of deaths each year, pulmonary embolism are essentially silent, lulling professionals into a decreased sense of patient susceptibility and a lack of understanding of the severity of the problem. Since pulmonary embolism masquerades its presentation, often as other disease
processes, we are not cued into action, because we don’t recognize it. That is why it is “discovered” at autopsy, when we are trying to understand what we missed, or why our patient died.

My own personal self-efficacy began in just this manner at the beginning of my nursing carrier, why did my patient die? I was fortunate in that there was an autopsy done to answer that question, but it took years and many more patients before I learned enough to become proactive. Now I propose the consideration of pulmonary embolism in puzzling patients to others, and look for it when seeing my own patients.
Table 1: Health Belief Model

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<tr>
<th>Concept</th>
<th>Definition</th>
<th>Application</th>
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<tr>
<td>Perceived Susceptibility</td>
<td>One's opinion of chances of getting a condition</td>
<td>Define population(s) at risk, risk levels; personalize risk based on a person's features or behavior; heighten perceived susceptibility if too low.</td>
</tr>
<tr>
<td>Perceived Severity</td>
<td>One's opinion of how serious a condition and its sequelae are</td>
<td>Specify consequences of the risk and the condition</td>
</tr>
<tr>
<td>Perceived Benefits</td>
<td>One's opinion of the efficacy of the advised action to reduce risk or seriousness of impact</td>
<td>Define action to take; how, where, when; clarify the positive effects to be expected.</td>
</tr>
<tr>
<td>Perceived Barriers</td>
<td>One's opinion of the tangible and psychological costs of the advised action</td>
<td>Identify and reduce barriers through reassurance, incentives, and assistance.</td>
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<tr>
<td>Cues to Action</td>
<td>Strategies to activate &quot;readiness&quot;</td>
<td>Provide how-to information, promote awareness, reminders.</td>
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<tr>
<td>Self-Efficacy</td>
<td>Confidence in one's ability to take action</td>
<td>Provide training, guidance in performing action.</td>
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(National Institute of Health, 2001)
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<th>Table 2: Differential Diagnosis: PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Aortic dissection</td>
</tr>
<tr>
<td>• Pericardial Tamponade</td>
</tr>
<tr>
<td>• Myocardial infarction</td>
</tr>
<tr>
<td>• COPD</td>
</tr>
<tr>
<td>• Malignancy</td>
</tr>
<tr>
<td>• Pleural disease</td>
</tr>
<tr>
<td>• Pulmonary edema</td>
</tr>
<tr>
<td>• Pulmonary Hypertension</td>
</tr>
<tr>
<td>• Rib Fracture</td>
</tr>
<tr>
<td>• Pneumonia</td>
</tr>
<tr>
<td>• Pneumothorax</td>
</tr>
<tr>
<td>• Congestive Heart Failure</td>
</tr>
<tr>
<td>• Pericarditis</td>
</tr>
<tr>
<td>• Asthma</td>
</tr>
</tbody>
</table>
Table 3: Levels in treatment of PE; level 1-3

<table>
<thead>
<tr>
<th>Presentation Levels</th>
<th>Symptoms</th>
<th>Potential ABG's &amp; Respiratory Rate</th>
<th>Pulmonary Vascular Occlusion</th>
<th>Pulmonary sequelae</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1</strong></td>
<td>None</td>
<td>PaO2 &gt;80 mmHg RR &lt;20</td>
<td>&lt; 20%</td>
<td>Normal</td>
<td>IV Heparin 7-10 days</td>
</tr>
<tr>
<td>Minor Segmental PE</td>
<td>Chest pain, anxiety &amp; cough</td>
<td>PaO2 80 mmHg RR &lt;/= 20 PaCO2 &lt;35 mmHg</td>
<td>20 – 30 %</td>
<td>Tachycardia</td>
<td>Warfarin, (Coumadin) or daily Aspirin</td>
</tr>
<tr>
<td><strong>3</strong></td>
<td>Dyspnea Orthostasis</td>
<td>PaO2 &lt;65 mmHg PaCO2 &lt;30 mmHg RR &gt; 20</td>
<td>30 – 50 %</td>
<td>Va/Q mismatch, alveolar hypocapnia causing pneumoconstriction, loss of surfactant, atelectasis &amp; mild pulmonary hypertension (PHT)</td>
<td>Consider Thrombolysis IV Heparin 7-10 days Oxygen PRN Coumadin</td>
</tr>
</tbody>
</table>
Table 3: Levels in treatment of PE; level 4 & 5

<table>
<thead>
<tr>
<th></th>
<th>Dyspnea</th>
<th>PaO2 &lt;50 mm Hg</th>
<th>&gt; 50 %</th>
<th>Previous DVT or PE history</th>
<th>Thrombolysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Massive Shock &amp; Death</td>
<td>PaCO2 &lt;30 mm Hg</td>
<td>RR &gt; 20</td>
<td>Oxygen PRN, Coumadin</td>
<td>IV Heparin 7-10 days</td>
</tr>
<tr>
<td>5</td>
<td>Chronic Syncope, dyspnea</td>
<td>PaO2 &lt;50 mm Hg</td>
<td>&gt;50 %</td>
<td>Coumadin</td>
<td>Intraluminal filters</td>
</tr>
</tbody>
</table>

Table 4: Symptoms of PE

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>73%</td>
</tr>
<tr>
<td>Tachypnea</td>
<td>70%</td>
</tr>
<tr>
<td>Pleuritic chest pain</td>
<td>66%</td>
</tr>
<tr>
<td>Crackles</td>
<td>51%</td>
</tr>
<tr>
<td>Cough</td>
<td>37%</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>30%</td>
</tr>
<tr>
<td>S4</td>
<td>24%</td>
</tr>
<tr>
<td>Accentuated P2</td>
<td>21%</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>13%</td>
</tr>
<tr>
<td>Syncope in elderly</td>
<td>13%</td>
</tr>
</tbody>
</table>
Table 5: MC’s Lab values

<table>
<thead>
<tr>
<th>Troponin I</th>
<th>White blood cells</th>
<th>Hematocrit</th>
<th>Platelets</th>
<th>Comprehensive metabolic panel</th>
<th>Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.8</td>
<td>5,900</td>
<td>40%</td>
<td>229,000</td>
<td>Normal limits</td>
<td>162</td>
</tr>
</tbody>
</table>
Figure 1: Algorithm for Pulmonary Emboli

R/O Pulmonary Emboli

Presentation
- Shortness of breath
- Tachypnea

Laboratory Tests
- Arterial Blood Gasses
  $146.99
- Venous Tests
  CBC, CMP,
  $41.00, $71.70
  TROPONIN
  $76.18

Imaging Tests
- Chest Film
  R/O pneumonia,
  pulmonary congestion
  pleural effusions
  $290.00

If none of the above, proceed
- Venous Doppler study
  $470.00
- Ventilation/Perfusion Study
  $1355.00
- Computed Tomography
  $1675.00
- Pulmonary Angiography
  $2600.00

- Vital Signs
  Oxygen Saturation
- Pleuritic:
  Chest Pain
- Tachycardia
  S₄, P₂
- Crakles
  Cough
  Hemoptysis
- Crackles:
  S₄, P₂
- Suspected PE
  Check labs
  Move to imaging
- EKG
  $133.00
- Echo
  $500.00

- Right Heart Enlargement,
  Pulmonary Hypertension
- If positive move to imaging
- If positive move to imaging
- If none of the above, proceed
- Venous Doppler study
  $470.00
- Ventilation/Perfusion Study
  $1355.00
- Computed Tomography
  $1675.00
- Pulmonary Angiography
  $2600.00
Figure 2: MC Chest Films

Radiology Report: MC (Figure 2) (Ortiz, 2001)

Impression: No acute appearing process in the chest in comparison with 1/31/00: Mild pectus excavatum configuration is noted which accentuates the right hilar markings. Transverse cardiac diameter is mildly prominent as well. Lungs and pleural surfaces are clear.
Possible Myocardial Infarction, considered being about two weeks old by symptoms. Currently has Left Ventricular dysfunction with respiratory symptoms indicative of congestive heart failure.
Figure 7: MC's discharge EKG

(Ortiz, 2001)
Figure 4: MC Cut 1; Deviated Septum

(Ortiz, 2001)
Figure 5: MC Cut 2; first evidence of clot

(Ortiz, 2001)
CT: MC (Figure 4,5 & 6) (Ortiz, 2001)

The heart demonstrates an enlarged right ventricle, a small left ventricle, and a septum displaced to the left. Noted on the second picture is a clot in the right vascular bed of the right lung. Bilateral clots are noted centrally by the final cuts of the CT angiogram.
References


Velmahos GC; Vassiliu P; Wilcox A; Hanks SE; Salim A; Harrel D; Palmer S; Demetriades D. (2001). Spiral computed tomography for the diagnosis of pulmonary embolism in critically ill surgical patients: a comparison with pulmonary angiography. *Archives of Surgery.* 136:5. 505-11

