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Pulmonary embolism (PE) is an illness that frequently presents with nonspecific symptoms, that affects people of all ages and stages of life, and that is difficult to diagnose with available tests. Unfortunately, it also is the third most common cause of death in the United States,[1] and many patients with PE present to emergency departments (EDs) across the country each day.

The diagnosis of PE rarely is made in a straightforward manner, and when the diagnosis is clear, the patient usually is extremely ill. In diagnosing PE, emergency physicians must consider a variety of factors, all of which must be weighed carefully to determine whether the patient has PE, a potentially fatal illness for which effective treatments are available.

A number of advances in both the treatment and diagnosis of thromboembolic disease have been made during the past decade. Several diagnostic tests commonly are used today that were not available even five years ago. The first article in this two-part series will cover the epidemiology of PE, the factors that increase a patient's risk for the disease, and the pathophysiology and clinical features of PE. In addition, complicated issues regarding the diagnosis of PE and the controversies involved will be addressed. The second part of the series will examine the differential diagnosis of PE, its treatment, and prevention of the disease.

—The Editor

Pulmonary Embolism: Recent Advances in Diagnosis and Treatment Modalities

Part I — Epidemiology, Risk Factors, and Pathophysiology

Author: **Marianne Haughey, MD,** Assistant Professor of Emergency Medicine, Jacobi Hospital, Albert Einstein College of Medicine, Bronx, NY.

Peer Reviewers: **Brian K. Snyder, MD,** Assistant Clinical Professor of Medicine, Department of Emergency Medicine, University of California, San Diego; and **Albert C. Weihl, MD, FACEP,** Assistant Professor of Surgery-Emergency Medicine, Department of Surgery, Yale University School of Medicine, New Haven, CT.

Epidemiology

PE is a relatively common and potentially life-threatening disease. The diagnosis of PE is missed more than 400,000 times per year, and 100,000 of those patients may have lived if they had received the appropriate treatment.[1] PE is the third most common cause of death in the United States, and at least 650,000 cases of the disease occur each year.[2-5]

Ten percent of patients with PE die within the first hour.[4] One-third of the patients who survive that first hour eventually will be diagnosed and treated, and two-thirds will remain undiagnosed.[2] One-third of the undiagnosed group will die, usually due to recurrent embolism and right heart failure.[4] Identification of patients with

PE is the essential first step in improving the survival statistics. In one study, 45% of patients who died of PE had the correct diagnosis suspected premortem.[6] The correct clinical diagnosis was made even less often in those with a diagnosis of myocardial infarction (MI), coronary artery disease (CAD), or chronic obstructive pulmonary disease (COPD). The same study found that only 47% of the patients with a suspected diagnosis of PE were anticoagulated with proper dosages.[6] Of the group that is diagnosed and treated, only one in 12 will die of massive PE or PE complications.[4,7]

Venothromboembolic disease, which includes deep vein thrombosis (DVT) as well as PE, accounts for 250,000 hospitalizations per year and about 50,000 deaths from recognized events.[8-11] DVT and PE are two points on the continuum of thromboembolic disease. In fact, there is much more crossover than previously thought. Asymptomatic PE occurs in 50% or more of patients with DVT, and DVT is found in 80% of patients with PE at autopsy.[12-14]

One study found a positive trend in the prevention and treatment of thromboembolic disease. The age-adjusted mortality rates for PE in Minneapolis/St. Paul decreased 50% for adults ages 30-84 during the period of 1980-1995.[15] This trend was paralleled by declining hospital discharge rates for phlebitis and thrombophlebitis. It also is possible that this may reflect a changing natural history of the disease.

Pathophysiology

The basic pathophysiology of PE involves material that becomes lodged in the pulmonary vasculature. This material, most commonly blood thrombus, does not permit normal pulmonary blood flow into an area of the lung. This causes the ventilated area of the lung to become essentially ventilatory dead space.[2] A blockage of one part of the pulmonary vasculature results in an increase in pulmonary vascular resistance, which increases pulmonary arterial pressures and right ventricular pressures.[2] Pulmonary hypertension occurs, and the patient develops a ventilation perfusion mismatch. If the obstruction is large enough, the patient also can become hypoxic.[16] The hemodynamic effects of embolism depend on the amount of pulmonary vasculature that is occluded.

The pO_2 and pCO_2 may vary during different stages of PE severity. Increased ventilation rate actually can increase the PO_2 and decrease the PCO_2 in smaller pulmonary emboli. Larger emboli can cause enough of a shunt that a significant amount of blood is unable to exchange oxygen or carbon dioxide, and the PO_2 can decrease while the PCO_2 increases.[2]

PE does not always present in a uniform manner. Patients with a large clot load may present in shock with hemodynamic collapse. On the other end of the spectrum, some patients may present more subtly with a smaller clot load and normal vital signs. Between these two extremes of presentation is a group of patients that presents with normal blood pressure, but if an echocardiogram is done, it can show the right ventricle under strain.[17] If 50% of the pulmonary vasculature is blocked, significant pulmonary hypertension and acute cor pulmonale can occur, which, if not detected, can cause long-term morbidity or death.[2]

Thrombus is the most common cause of pulmonary emboli, and the most common sources of these emboli are clots in other parts of the deep venous system. Often, the main concern for most patients is not that the clot already in the pulmonary circulation will be fatal, but rather that the possible next clot could pose a danger.

Clots most commonly originate in the deep venous system of the legs, as evidenced by the fact that 70% of patients with PE are found by venography to have clot in the lower extremity.[2] Clots can embolize from the iliac, common femoral, and popliteal veins. The significant risk of clot embolizing from the veins in the calf often is discounted; however, about one-third of those with pulmonary emboli have a calf thrombus as the source for the emboli.[2,18] In addition, thrombus in the calf is likely to

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Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
Marketing Manager: Schandale Kornegay
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grow and extend, therefore becoming a larger thrombus that may be more dangerous to the patient if it embolizes.

Upper extremity DVT can embolize to become a PE. This type of DVT forms more often in those with malignancy or with central venous lines.[19] Pelvic veins also can be a source for thrombus, and may be missed in a search for a lower extremity DVT.

While thrombus is the most common material to embolize, air embolisms, amniotic fluid embolisms, and fat embolisms also have occurred. Other material, such as bullets and tumors, embolizes even more rarely. These nonthrombotic emboli require the usual supportive and interventional care, but they do not benefit from the anticoagulation treatment given to patients with thrombotic emboli.[2]

Risk Factors

In 1858, Virchow established that there were three main reasons for the pathogenesis of thrombus: venous stasis, intimal injury, and hypercoagulability.[2] The risk factors for thrombus formation, and hence PE, still may be categorized neatly within the umbrella of Virchow's triad. These risk factors may occur alone or in combination. *(See Table 1.)*

Venous stasis is the reason for the increase in thromboembolic disease among patients with prolonged immobility as a result of medical illness, burns, trauma, or surgery.[20] Patients who have experienced strokes or paralysis also are at high risk. Congestive heart failure (CHF) is another significant risk factor. Venous stasis may be the reason for the increased thromboembolic events in people who travel long distances, especially on long airline flights.[21,22] The hyperviscosity syndromes, varicose veins, and pelvic tumors may cause increased venous stasis and are risk factors for thromboembolism as well.[20,22] Obesity has been found to be an independent risk factor for VTE.[23-25] History of prior DVT increases venous stasis, and patients with proximal DVT who receive inadequate anticoagulation treatment have a 20-50% risk of recurrent VTE.[20]

Intimal injury can occur as a result of trauma or as an iatrogenic by-product of care. Central lines or intravenous drug abuse can contribute to a thromboembolic event. Injury to the vessel may result from orthopedic surgery, especially hip or knee surgery, or vascular surgery. Chemotherapeutic agents also have been shown to cause intimal damage.[20,22]

Hypercoagulability results from alteration of the clotting mechanisms. Malignancy, trauma, burns, and hormone replacement therapy are acquired causes of hypercoagulability.[20,26] The duration of pregnancy and the postpartum period are times of increased risk of thrombosis, and this is thought to be due to a state of hypercoagulability.[22,27,28]

In addition to these acquired states of hypercoagulability, there are inherited hypercoaguable states. These are rare causes for VTE but, if present, they are concerning as the patient is at risk for multiple recurrences. The more common of these rare, inherited states include protein C deficiency, protein S deficiency, antithrombin III deficiency, Factor V Leiden mutation, antiphospholipid antibodies, hyperhomocysteinemia, and high factor VII.

The risk of VTE also increases with age.[20] The reasons in this heterogeneous group are probably multiple, including a higher incidence of co-morbid conditions. Morbidity and mortality also increase with age.[20] The higher percentage of patients with underlying cardiopulmonary disease makes it more difficult to exclude those illnesses from the differential diagnosis list.[20]

Finally, the most significant risk factor for PE is the presence of a current DVT.

Table 1. Risk Factors for PE

PROLONGED IMMOBILITY	HYPERCOAGULABILITY	INTIMAL DAMAGE
• Paralysis	• Malignancy	• Trauma
• Long trips	• Previous PE or DVT	• Vascular surgery
• Bed rest	• High estrogen states	• Orthopedic surgery
• Pelvic tumors	• Inherited hypercoagulability disorders	• Central lines
		• IV drug use/abuse

Key: PE = pulmonary embolism; DVT = deep venous thrombosis IV = intravenous

Clinical Manifestations

The history and physical exam in a patient with PE do not confirm the diagnosis; they merely raise the suspicion of such a diagnosis, triggering further investigation. A patient with PE may present as severely ill or in cardiac arrest;[29] those who present in this manner may be among the 10% of patients with PE who will die within the first hour.[2] The other 90% of patients may present more subtly, with nonspecific symptoms.

The history may help elucidate whether the patient has any of the risk factors listed above, which may be the strongest indicator that the clinician should proceed with additional examinations. Other findings in the history may include dyspnea, pleuritic chest pain, cough, leg swelling, hemoptysis, wheezing, palpitations, or anxiety.[2,14,20] *(See Table 2.)*

Dyspnea is the most common symptom and is found in 70-90% of those found to have PE.[14] Although dyspnea is a sensitive sign for PE, it clearly is not specific. Sudden onset dyspnea may be more specific for PE.[30,31]

Chest pain is the second most common symptom of PE and is present in 49-85% of patients, with pleuritic pain being more common.[31-33] Pulmonary infarction near the innervation of the pleural nerves is thought to be the reason for pleuritic chest pain in a patient with PE.[14] Massive emboli and centrally lodged emboli are less likely to cause pleuritic chest pain.[14]

Cough, hemoptysis, sweating, and syncope are less common than the above symptoms. Hemoptysis occurs when a small embolus travels to the periphery of the pulmonary vasculature, resulting in infarction and hemorrhage. Syncope can result when a very large embolus increases the pulmonary resistance and, therefore, the afterload in the right ventricle, thus decreasing the blood flow return to the left ventricle. The right ventricle also increases in size somewhat, impinging on the left ventricle. Cardiac output decreases, and the result is syncope. Syncope occurs more commonly in massive PE, from 17% to 35% of the time.[32-34]

Signs of PE commonly include tachycardia, which occurs up to 71% of the time, and tachypnea, which occurs 92% of the

Table 2. Symptoms of PE

Dyspnea	70-90%
Chest pain	49-85%
Cough	3-55%
Leg swelling	17-35%
Hemoptysis	3-40%
Sweating	26-41%

Adapted from: Lee LC, Shah K. Clinical manifestation of pulmonary embolism. *Emerg Med Clin North Am* 2001;19:925-942.

time.[14] Rales occur in about 50% of patients, and a low-grade fever can be found in 30-54% of patients.[14] Because many patients with PE have a DVT at autopsy, the signs of leg swelling also may be found in those with PE. (In fact, only 17-35% of patients with non-massive PE are found to have leg swelling.)[14] Other signs demonstrate the right heart strain that occurs in large or massive PEs. These signs include jugular venous distension, right ventricle heave, circulatory collapse, and an increase in the intensity of the pulmonic component of the second heart sound. *(See Table 3.)*

Diagnosis

The diagnosis of PE frequently remains elusive despite the advances in testing that have been developed during the past decade. Emergency physicians have stated that this is one of the diagnoses for which they would most like to see a decision rule created.[35] There is no single test with adequate sensitivity and specificity that can be provided for the patient in a minimally invasive manner and that is available to the ED physician 24 hours per day.

A multitude of exams has been studied and used in the diagnosis of PE. Some of these exams have been used to evaluate other diagnoses on the differential diagnosis list. Others are an attempt to screen for PE. Still others are used to confirm a highly suspected PE. These tests include electrocardiograms (ECGs), arterial blood gases, chest radiographs, computed tomography (CT) scans, ventilation/perfusion scans, lower extremity or upper extremity ultrasound, pulmonary angiograms, and D-dimer products. Pretest clinical suspicion also has been accorded a significant weight by many authors in their studies. Unfortunately, the fact that there are a vast number of exams in use demonstrates that each has a significant weakness. There is no one test with adequate sensitivities or specificities for absolute inclusion or exclusion from treatment. Yet, each also has strengths in certain clinical situations. Multiple authors now have suggested that these tests may be used in combination, with a more powerful result than may be achieved by each individual exam.[36-40] In combination, the tests can be used very constructively to establish who should be treated and who may have alternate diagnoses.

Exams for PE may be divided into screening tests and confirmatory tests, as well as tests that are helpful in either excluding or confirming alternate diagnoses. Screening tests ideally have a high rate of sensitivity, and confirmatory tests ideally have a high rate of specificity.

Table 3. Clinical Signs of PE

Tachypnea—rate greater than 25	48-59%
- rate greater than 16	66-92%
Tachycardia—heart rate greater than 100	24-71%
- among those with a massive PE	48-71%
- among those with non-massive PE	24-44%
Rales/crackles	50%
Fever greater than 37° C	30-54%
- greater than 38° C	7-20%
Leg swelling	17-35%
Jugular venous distension	12-31%
Diaphoresis	10-41%
Circulatory collapse	3-24%
Circulatory collapse among those with massive PE	34%

Adapted from: Lee LC, Shah K. Clinical manifestation of pulmonary embolism. *Emerg Med Clin North Am* 2001;19:925-942.

Clinical Evaluation

Multiple authors now suggest that the pursuit of a diagnosis of PE utilize a decision tree system. The first step in each decision tree is the clinician's pretest evaluation of the probability of PE as the diagnosis.[41] PE is not a clinical diagnosis, yet the degree of suspicion held by the clinician has been shown to be significant clinically. The majority of authors, however, have not specified what criteria clinicians should use to come up with their pretest probability. Wells and Wicki each have developed separate systems for objectively evaluating the clinical probability of PE.[42,43] *(See Table 4 for Wells' criteria.)*

Each of the scoring systems places the patient into a low, intermediate, or high probability group. Both sets of criteria include risks such as recent surgery, previous thromboembolic event, and tachycardia. Wicki additionally considers age, hypocapnia, hypoxemia, and chest x-ray findings. Wells scores factors such as signs of DVT, immobilization, hemoptysis, malignancy, and whether PE is considered as likely or more likely than an alternate diagnosis by the treating physician. Wicki's score found that 10.3% of those considered to be low probability had PE. Of those found to be at intermediate risk, 38% had PE, and of those scored to be high probability, 81% had PE. A total of 49% of the patients were in the low probability group, thus permitting a large portion of those suspected to proceed through a less intensive arm of a diagnostic decision tree.[43]

D-Dimer. D-dimer is a fibrin degradation product that circulates in the bloodstream of patients with a dissolving fibrin clot.[44-46] D-dimer usually begins to circulate within an hour of thrombus formation and can be found to circulate for up to about a week after thrombus formation, as the continued fibrinolysis of clot continues to elevate the D-dimer level.[47-51] D-dimer has a circulating half-life of 4-6 hours, so the levels decrease over time.[45] After seven days, false-negative test results are more likely as the D-dimer normalizes.[50,52,53]

D-dimer, when used well, is a wonderful tool in helping the physician determine whether to treat a patient suspected of having a thromboembolic event. As with all of the other exams available, it is essential to understand the strengths and limitations of the exam. The initial point to understand is that the best use of any D-dimer is to exclude the disease in low probability patients. There is not adequate evidence to use a negative result to exclude the disease in patients whose clinical probability is intermediate or high.[36] In addition, a positive test has low specificity, with the most specific test only having a 20-50% specificity.[46,54,55]

A significant limitation of the D-dimer exam is that there are a heterogeneous collection of exams with varied sensitivities and specificities. It is essential for emergency physicians to understand which exam is available at their institution. There now are multiple suggested protocols for diagnosing PE via a decision tree.[36-39] They almost universally use a negative D-dimer test as a method to screen out patients who clinically are considered to have a low probability of PE. The D-dimer test used in these studies is the enzyme-linked immunoabsorbent assay (ELISA) technique. ELISA tests that show a normal D-dimer level have excellent negative predictive values of 91-98%.[51,56-58] A D-dimer level less than 500 ng/mL on an ELISA test has been found to have a 93% sensitivity in predicting a normal angiogram result.[51] Unfortunately, the ELISA test is not the more commonly available exam, and in fact it has other limitations that make it less attractive for rapid diagnosis in the ED. The drawbacks to the ELISA D-dimer test are that it is technician-dependent, expensive, run in batches, and inefficient for clinical use.[45,59]

The semiquantitative latex agglutination (LA) technique is a more commonly found and rapid technique. The LA assays are more widely available but they are not as sensitive as the ELISA assays. A normal LA D-dimer level alone or with V/Q scan results is not recommended to preclude the treatment of possible PE.[44,45] The LA exam has been found to have a sensitivity of 70%.[41] The SimpliRED exam is another commonly studied D-dimer exam. It is a whole blood agglutination assay and is easier and faster to perform than the ELISA-based tests. It is a bedside exam that can be performed in two minutes.[44] The sensitivity of SimpliRED was 68-85%, and the negative predictive value was 81% for VTE.[41,60,61] But the SimpliRED test may have more of a role in those with a low pretest probability of PE or a nondiagnostic V/Q scan. The sensitivity of this exam in low probability PE patients is 99-100%.[36,44,60]

There is an interesting problem in introducing a highly sensitive yet poorly specific screening exam into the diagnostic decision tree. When a rapid D-dimer (SimpliRED) test was introduced to a study ward, the number of V/Q scans done increased because the number of patients screened for PE increased. There also was an increase in those who were diagnosed as having thromboembolic disease.[46] While more VTE was diagnosed, there was no change in any three-month outcomes.[46]

Several second-generation rapid D-dimer tests are available. They work via an erythrocyte agglutination assay, a turbidimetric assay, the rapid ELISA test, and an immunofiltration assay. It is incumbent upon emergency physicians to determine which exam with which level of sensitivity is available at their institution to frame the results of the exam in the proper context. COPD does not affect the accuracy of the D-dimer test, although many other illnesses lead to false positives.[62]

Table 4. Wells' Criteria

VARIABLE	POINTS
Clinical signs and symptoms of DVT (objectively measured leg swelling and pain with palpation in the deep vein region)	3.0
Heart rate >100	1.5
Immobilization (bedrest, except for use of the bathroom for 3 or more days) or surgery (in the previous 4 weeks)	1.5
Previous objectively diagnosed DVT or PE	1.5
Hemoptysis	1.0
Malignancy (patients with cancer who are receiving treatment, those in whom treatment has been stopped in the last 6 months, or those who are receiving palliative care)	1.0
Pulmonary embolism as likely as or more likely than an alternate diagnosis	3.0

Low pretest probability	< 2points
Moderate pretest probability	2-6 points
High pretest probability	> 6 points

Arterial Blood Gases. Arterial blood gases (ABG) and the A-a gradient have been used in the past in the attempt to diagnose pulmonary emboli. Unfortunately, the sensitivity and specificity of the exam are both too low to make it a useful test for diagnosis.[44,63] Many patients who are suspected of having a PE have other pulmonary pathology, which, therefore, decreases the specificity of the exam. The sensitivity of the exam also is poor, as it takes only one or two extra breaths per minute to make the PaO_2 normal even when the pulmonary gas exchange is significantly impaired.[2] In a population of classically symptomatic patients with angiographically proven PE, at least 17% will have a normal PO_2, and one in 20 will have a PaO_2 greater than 100 mmhg on room air.[64,65] A completely normal ABG may be found in up to 23% of patients with known PE. There is no role for pulse oximetry in the diagnosis of PE.[2]

Certainly, an ABG may provide useful information about a patient who is critically ill the need for additional oxygenation, intubation, or choices of treatment. A large PE may cause a significant A-a gradient; however, A-a gradient is nonspecific and insensitive.

Electrocardiogram. A massive PE that causes severe pulmonary hypertension can produce specific ECG abnormalities. Sub-massive PE manifests a wide spectrum of nonspecific changes on surface ECG, ranging from no abnormality to multi-

Table 5. ECG Scoring System for Pulmonary Hypertension Caused by PE

FINDING	POINTS
Tachycardia	2
Incomplete RBBB	2
Complete RBBB	3
t wave inversion in all leads V1-V4	4
t wave inversion in lead V1	0-2
t wave inversion in lead V2	1-3
t wave inversion in lead V3	1-3
s wave in lead 1	0
q wave in lead III	1
Inverted t wave in lead III	1
If all s1, qIII, tIII is present	2

Adapted from: Daniel KR, Courtney DM, Kline JA. Assessment of cardiac stress from massive pulmonary embolism with 12-lead ECG. *Chest* 2001;120:474-481.

Table 6. Using ECG to Predict PE

Sreeram found three or more of the following predicted PE with 82% sensitivity:

- Incomplete or complete RBBB associated with ST-segment elevation and positive t-wave in V1
- S waves in lead 1 and AVL of > 1.5 mm
- A shift in transition zone in the precordial leads to V5
- Q waves in leads III and AVF but not in II
- Right axis deviation with a frontal QRS axis of 90° or an indeterminate axis
- Low voltage QRS complex of < 5 mm in the limb leads
- T wave inversions in III and AVF or V1-V4

Adapted from: Sreeram N, Cheriex EC, Smeets JL, et al. Value of the 12 lead ECG at hospital admission in the diagnosis of pulmonary embolism. *Am J Cardiol* 1994;73:298-303.

ple disturbances of rate, rhythm, and conduction pattern.[66,67] Common ECG findings in those with PE include tachycardia, nonspecific ST-T wave abnormalities, atrial fibrillation, right axis deviation, left axis deviation, S1-S2-S3, S1-QIII-TIII, and p-pulmonale.[2] ECG frequently is relegated to screening for other cardiac abnormalities in patients with PE.[68] One study found that inverted t waves in leads V_1-V_4 was the most common finding on ECG, and was found in 68% of patients with angiographically confirmed PE. The study also found 9% of those with PE had normal ECGs.[69] Another study looked at multiple studies and found that normal ECGs were noted in 10-46% of patients with angiographically proven PE.[70] New-onset atrial fibrillation must encourage the physician to consider PE as the cause.

Right ventricular strain on ECG may have clinical importance in determining the severity of the strain and, hence, the severity of pulmonary hypertension. In a patient who has PE, the severity of strain may correspond with the presence of the right ventricular dysfunction. One study produced a scoring system to grade the severity of pulmonary hypertension from PE.[71] *(See Table 5.)* A score of 10 or greater was found to be highly suggestive of severe pulmonary hypertension from PE.

Sreeram et al examined ECGs of patients with and without PE using the criteria in Table 6. The authors suspected PE if three or more of the listed findings were present. The sensitivity of their study was 82%.[72] The specificity of ECG is poor for PE, and its sensitivity certainly is not adequate. The purpose of an ECG is to evaluate for other causes of the patient's symptoms, especially myocardial infarction ischemia and pericarditis.

Chest Radiographs. Chest radiographs are important to obtain in the patient thought to have PE. Chest x-rays are essential in making the diagnosis of other respiratory illnesses that may masquerade as PE.

There are nonspecific findings that are associated with PE on chest x-ray, including an elevated hemidiaphragm, focal infiltrates, pleural effusions, or atelectasis. In addition, there are some eponymous signs for PE that can be found on chest x-ray, including Hampton's hump and Westermark's sign. Hampton's hump is a triangular pleural-based infiltrate with the apex toward the hilum, commonly adjacent to the diaphragm. Westermark's sign shows up as a dilation of the pulmonary vessels proximal to the embolism along with the collapse of more distal vessels. This also is called oligemia or regional pulmonary paucity. Unfortunately, all of these signs are nonspecific and insensitive.[73] In addition, 30% of those with PE have a normal chest x-ray.[74] *(See Figure 1.)*

Another important issue regarding the chest x-ray is that it is helpful in interpreting the V/Q scan.[63,75] In subjects investigated for PE, an abnormal chest x-ray increases the prevalence of nondiagnostic lung scans. A normal pre-test chest x-ray more often is associated with a definitive (normal or high) probability lung scan result. The chest radiograph may be useful in deciding the optimum sequence of investigations.[75]

Echocardiography. Transesophageal echocardiography (TEE) is a bedside test that has been used to evaluate right heart strain and to attempt to identify clots in those suspected of having PE. TEE has been found to be more sensitive than transthoracic echocardography for the diagnosis of PE.

TEE fails to identify some 50% of patients with angiographically proven PE.[76] Although echo findings of right ventricular strain, paired with a high clinical suspicion, support a diagnosis of PE, TEE has to have a better sensitivity to be used as a screening tool to rule out PE.

The role of echocardiography in PE may be more appropriately directed to evaluating the patient with known PE. Patients can present in hemodynamic embarrassment or with an apparently stable exam. There seems to be a group of patients who present with stable vital signs, yet who already have increased right ventricular strain and may be on the verge of decompensation. An echo may be used to identify this deceptively relatively well-appearing group before their clinical decompensation occurs.

DVT Evaluation. Because about 80% of PEs originate in the lower extremities, an evaluation for DVT can be an extremely useful test in the diagnostic workup for PE.[63,77] It is significant to realize that thromboembolism is a continuum of illness, and that the important issue first may be to diagnose the presence of a clot

and then its location. This becomes even more important as the outpatient treatment of DVT with low molecular weight heparins (LMWHs) becomes more common. It must be recognized that only 42% of patients with PE and DVT have any symptoms of DVT.[77]

Venous ultrasound (US) has a sensitivity of about 95% and a specificity of about 96% in patients with an initial episode of DVT. In isolated calf vein DVT, the sensitivity is about 73%. About one-fourth of isolated distal DVTs will extend proximally, so repeating the US after about one week is indicated after a single normal proximal venous US study. The risk of subsequent venous thromboembolism is very low (< 2% during six months of follow-up), provided that US of the proximal veins remains normal for one week in a patient with suspected DVT or for two weeks in someone with suspected PE (in a patient who also has a nondiagnostic lung scan).[78]

A negative single bilateral lower extremity venous ultrasound exam is not a reliable method of excluding PE in outpatients with nondiagnostic V/Q scans.[79]

One study has examined the use of serial impedance plethysmography in those who have nondiagnostic lung scans and adequate cardiopulmonary reserve to diagnose PE. The leg study was done on presentation and, if negative, was repeated on days 2, 3, and 5 or 7, 10, and 14. The study found this to be a safe manner of evaluating for PE in stable patients.[38]

CT Scans. In 1992, the first study was published comparing CT angiogram (CTa) to a pulmonary angiogram.[80] The use of helical CT has risen dramatically as more EDs have gained easier access to this diagnostic tool than to angiograms. Helical CT scans are fast, with the ability to scan from the aortic knob to the diaphragm in 20-30 seconds.[81,82] They have the potential to show images of the pulmonary vasculature from the right and left main pulmonary arteries to at least the segmental and possibly subsegmental arteries.[83] Using 2- to 3-mm sections is superior to using 5-mm sections. The study requires 120 mL of ionic or nonionic contrast.[44] An intraluminal filling defect or vascular occlusion characterizes a positive result.[44] False positives are possible with tortuous or oblique segmental arteries and kinetic effects from breathing.[83,84] Specificity ranges from 89-100% and sensitivity is 64-93%.[85,86] The CTa can be viewed as having an overall test accuracy that is equal to or slightly superior to that of the V/Q scan, but a CTa scan that is read as negative for PE is not as sensitive as a V/Q scan read as normal.[44]

Subsegmental vessels are difficult to identify and evaluate for PE.[44] This consistently has been presented as a significant disadvantage of CTa. It has been suggested that it may not be necessary to diagnose PE with the certainty of a pulmonary angiogram. Isolated subsegmental PEs appear to be uncommon, representing only 6% of all PEs.[52]

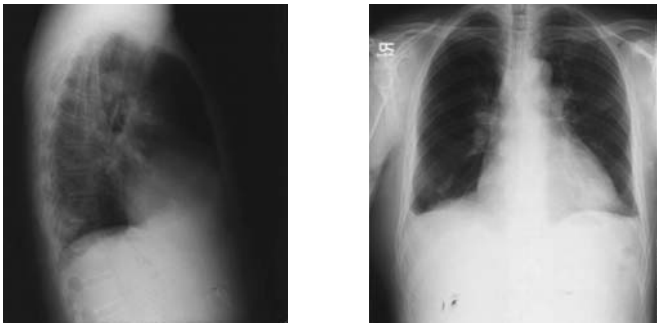
CTa is recommended for those who have a positive screening test or as an initial test in those who have conditions that cause nondiagnostic V/Qs, such as heavy smoking, chronic pulmonary disease, or pulmonary infiltrate.[87] It also can be useful in those with a high pretest clinical probability but indeterminate V/Q scans.[88]

The sensitivity of CTa for PE is too low to use it to rule out PE without other tests. One study looked at the rates of PE in those patients who had a negative CTa after a non-diagnostic V/Q and negative leg ultrasounds. The researchers found an overall false-negative rate for PE of 5% in the three months following their exams.[81] The false-negative rate included patients who had syndromes meriting a high clinical suspicion but negative CTa, but who had an angiography that was diagnostic for PE at initial presentation. Also included in the false negative rate were patients who only had a low or moderate clinical suspicion, and therefore no initial angiography, but who were diagnosed with PE in the three months following the initial presentation.[81] Other studies have found a 2-5% follow-up thrombosis rate.[89-91] The interobserver agreement on the CTa readings is high,[86] and is much higher than the interobserver agreement found in the Prospective Investigation of Pulmonary Embolus Diagnosis (PIOPED) study in reading V/Qs.[92] It has been suggested that CTa might replace pulmonary angiogram in the combined strategies that use US and V/Q scanning.[86,93] CTa can be found to confirm an alternate diagnosis in 11-67% of patients.[86] A meta-analysis of 277 patients showed only 4% with inconclusive results from CTa.[36] Others show up to a 10% indeterminate reading on the CTa.[2] This percentage still is far less than the 60-75% of indeterminate readings obtained with a V/Q scan.[92] Thrombi in the main pulmonary arteries mostly were true positives, while 15% of those at the lobar and 38% of those at the segmental level were false positives.[86] There also is the possibility, not yet widely practiced, of using the CT to evaluate the pelvic and thigh veins as well as the pulmonary vasculature.[94] The true clinical utility of this approach has yet to be evaluated.

The advantages of CTa are that it is fast, frequently available, relatively specific, and can be helpful in demonstrating other lung pathology. Disadvantages include the fact that the patient may have to leave the department for the test, there is a significant dye load, and the test is not sensitive enough to use alone. CTa is a powerful tool in the diagnosis of PE, but only when used with an understanding of its limitations.

V/Q. Ventilation-perfusion (V/Q) studies are among the most commonly ordered exams used to diagnose PE. A V/Q test requires injection of a gamma radioactive isotope into the patient. The patient must leave the ED for the test, which may be impossible for a critically ill patient. V/Q examines only the lungs and usually is not helpful in evaluating other diagnoses.

Figure 1. Hampton's Hump



V/Q scans in PIOPED (in which they were compared against the findings of pulmonary angiograms) had variable rates of sensitivity and specificity, depending on their reading. The scans were read as normal, very low probability, low probability, intermediate probability, and high probability. The sensitivity of a high probability scan was only 47%, but the specificity was 97%. The negative predictive value of a near normal/normal scan is 91%, and that of a low probability scan is 84%.[92] Other authors have recommended that all other scan readings be considered indeterminate.[75] An abnormal chest x-ray increases the prevalence of non-diagnostic scans. A normal pretest chest radiograph more often is associated with a normal or high probability scan (definitive scan).[75]

Lung scanning is diagnostic in only 25-40% of patients with a reading of normal/near normal or of high probability.[38,39,92,95] This is a serious limitation of the exam. It is imperative to remember to pursue other diagnostic routes when a non-diagnostic scan is obtained. Another drawback is that V/Q scans may be difficult if not impossible to obtain in some institutions during off-hours.

It also has been shown that when the readings are done, they are most clear and accurate when a standardized conclusion is included. Examples included high probability, no conclusion, or diagnosis excluded.[22] Given the difficulty of diagnosing PE, physicians should make it as clear as possible when results are obtained.

Angiography. Pulmonary angiography is considered to be the definitive gold standard in the evaluation for PE. Dye is injected into the pulmonary vascular tree (which has 26 branchings of the pulmonary vessels). If a defect is seen intraluminally, or if the dye abruptly is cut off, the presumption is that PE is occluding the vessel. This is not an infallible exam. Objects other than thrombus, including extraluminal tumor, can cause the vessel to appear occluded. Even a well-performed pulmonary angiogram cannot detect material in vessels below the third branch point. This corresponds with lobular arteries. As we have discussed, small emboli may herald the imminent arrival of the larger, life-threatening emboli.

Angiography is a highly accurate tool in the diagnosis of PE. Interobserver agreement for positive angiograms is 89%, and it is 83% for negative studies. Agreement was found to be more concordant as the size of the vessels involved increased.[96] In fact, the agreement between readers for the main or lobar arteries is the best, with 98% agreement. The agreement for readers of clots involving the segmental arteries (and not larger vessels) was 90%, and the agreement level was 68% in those clots that involved only the subsegmental arteries.[97]

In 1990, the PIOPED study used the pulmonary angiogram as the gold standard in its classic comparison of V/Q scans to angiograms, yet even a patient with a normal pulmonary angiogram can expect a 2.2% chance of a venous thromboembolic event within a year.[96] An angiogram also is invasive and frequently difficult to arrange in some institutions on a 24-hour basis. Some studies cannot be completed for technical reasons. In addition, if lytic therapy is used, the angiogram may be normal within 90 minutes of therapy.

Risks for the procedure include allergic reaction to the contrast, vascular injury, cardiac dysrythmias, endocardial injury, or perforation. Clearly, some of these injuries can lead to significant morbidity or death. Even given the multiple manners in which the risk expresses itself, the PIOPED data demonstrate a mortality rate of 0.5%.[2]

Combination Strategies

It is apparent that any individual test other than a pulmonary angiogram lacks either the sensitivity or specificity to use as a single instrument in determining whether to treat a patient for PE. It also is important to note that treatment for PE is not benign, and that the risk of bleeding from treatment with unfractionated heparin, LMWH, or thrombolysis is significant and cannot be dismissed. Likewise, the surgical placement of a filter has its own inherent risks.

To diagnose PE more accurately, multiple authors have begun to suggest using a decision tree approach rather than a single exam. The initial exams used are those that have a high sensitivity, while the later exams test a smaller group with more specific tests. Pulmonary angiogram is performed on fewer people, but is available if the rest of the analysis does not provide an adequate answer.

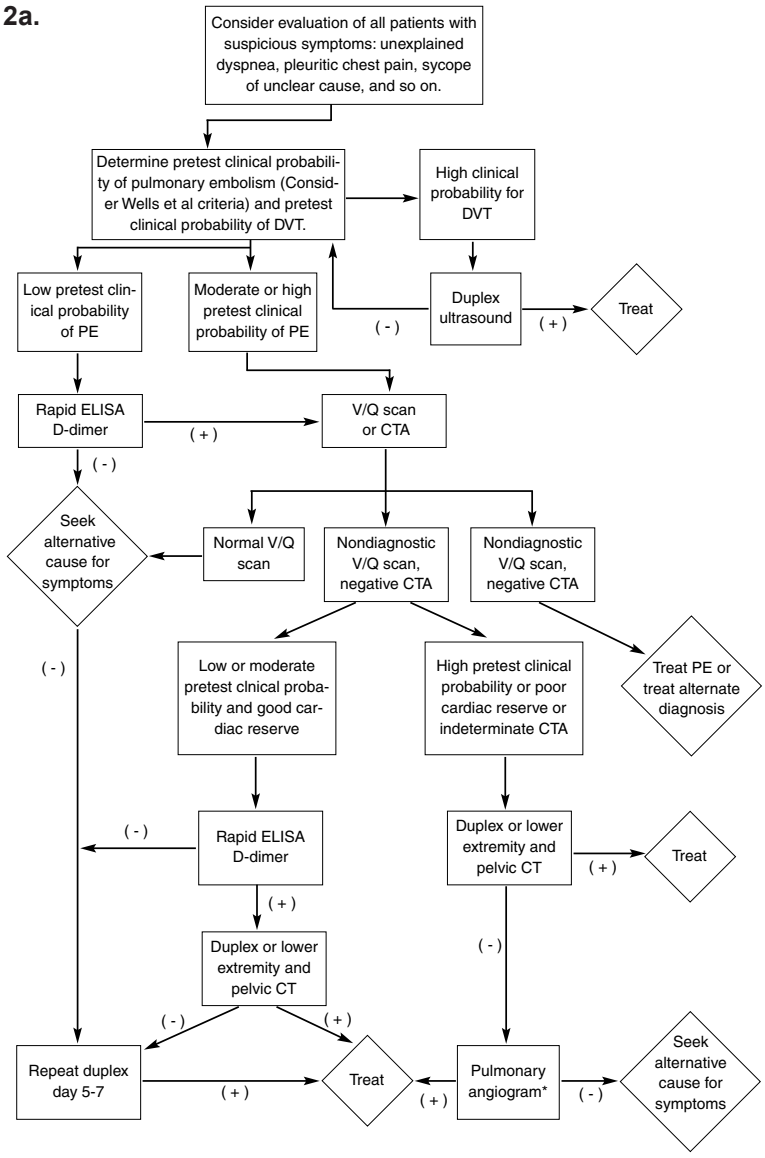
Some authors begin their decision trees with an interpretation of the clinician's pretest clinical probability. *(See Figures 2a and 2b for two strategies.)* In those patients considered to have a low pretest clinical probability of PE, a D-dimer (an ELISA D-dimer) is used and, if negative, precludes further testing for PE. If positive, or if the pretest clinical probability is moderate or high, further testing is pursued.

In one strategy for evaluating PE, lower extremity ultrasound is the next level of testing which, depending on the results, might be followed by a V/Q scan, then a spiral CT angiography with contrast (not requiring catheterization), and then, if needed, angiography.[36] Another approach includes duplex ultrasound if there is high clinical probability for DVT, but otherwise uses a V/Q or spiral CT as the next testing level for either moderate or higher clinical probability of PE or a positive D-dimer (an ELISA D-dimer test) in a patient considered to have a low pretest clinical probability.[37] Further testing may require a determination of the patient's cardiac reserve in evaluating treatment or follow-up. It has been found that in patients who are clinically suspicious for PE yet have nondiagnostic workups, including negative DVT studies of the legs, who also have good cardiac reserve, it is safe to follow them with outpatient duplexes of the legs during the next 14 days.[38] This is not to say that they do not have PE, because as many as 18% of patients with a high clinical suspicion of PE but a low probability lung scan and negative compression ultrasonography of the lower extremities will be found to have PE.[40] Wolfe uses further outpatient testing in those determined to be stable with a nondiagnostic V/Q scan, a positive D-dimer, and a negative duplex at the time of presentation.

Each of these algorithms attempts to determine which patients need treatment as early in the strategy as possible while minimiz-

Figures 2a and 2b. Algorithms for Pretest Clinical Probability of PE

2a.



2a. Proposed diagnostic algorithm for the evaluation of suspected PE.

Key: US = ultrasound; **CTA** = computed tomography angiography.
* If initial pulmonary imaging study was V/Q scanning, consider CTA before pulmonary angiography.

2b. Pretest clinical probability

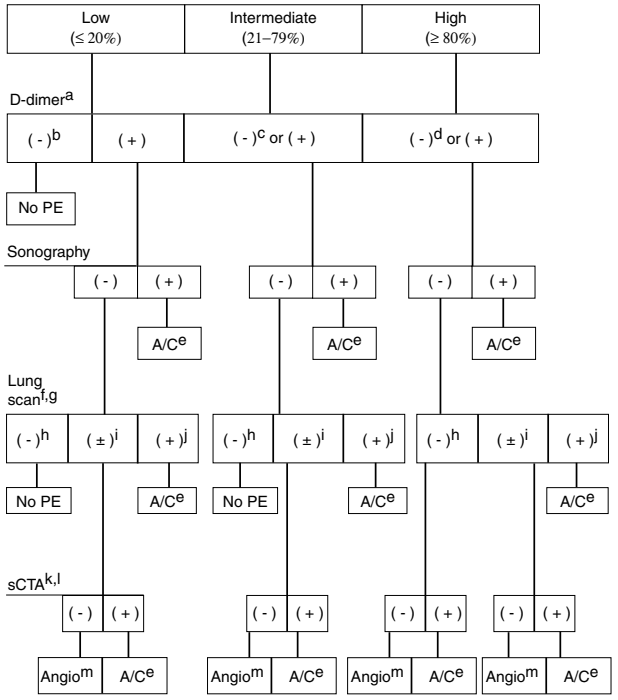


Fig. 2b: Suggested serial diagnostic testing strategy for patients presenting to the ED with varying clinical pretest probabilities of PE.

Key: a = conservative assessment of evidence supports use of VIDAS DD D-dimer assay; **b** = negative VIDAS DD D-dimer assay in low clinical pretest probability patients essentially excludes PE (< 1% posttest probability); **c** = evidence supporting use of negative VIDAS DD D-dimer result in intermediate clinical pretest probability patients for exclusion of PE appears valid; however, because the precision of the LR cannot be estimated, this test cannot be recommended at this time as unequivocally safe among the intermediate clinical probability patients; **d** = negative VIDAS DD D-dimer result does not exclude PE among high clinical pretest probability patients; **e** = anticoagulation; **f** = patients with underlying cardiopulmonary disease, especially chronic lung disease, may bypass radionuclide lung scanning and proceed directly to sCTA; **g** = ventilation scanning after a perfusion study, when indicated; **h** = negative lung scan finding indicates a reading of normal or near-normal; **i** = ± finding on lung scan indicates a clinically indeterminate reading (i.e., neither normal/near-normal nor high probability [very low, low, intermediate, and so forth]); **j** = positive (+) lung scan indicates a high probability reading; **k** = sCTA is spiral (helical) computed tomography angiography with contrast, not requiring catheterization; **l** = a plausible alternative diagnosis seen on sCTA is currently the only evidence-based means of excluding PE with this test; **m** = conventional pulmonary angioplasty, requiring catheterization; **PE** = pulmonary embolism.

Figures used with permission from: (2a) Wolfe TR, Hartsell SC. Pulmonary embolism: Making sense of the diagnostic evaluation. *Ann Emerg Med* 2001; 37:509; and (2b) Gallagher EJ. Clots in the lung. *Ann Emerg Med* 2000;35:182.

ing the need for pulmonary angiograms. They are complete and attempt to address all possible presentations of PE.

Another study looked at those patients with nondiagnostic lung scans and negative leg DVT exams on the day of presentation and found that they may be followed safely as outpatients with serial impedance plethysmography studies on days 2, 3, 5, 7, and 10. Anticoagulation was withheld in those whose exams remained negative. The author found this to be a safe method of evaluation, but clearly it is limited in the ED setting if the patient lacks access to ongoing care.[38]

Summary

PE is a common, potentially lethal disease that is under-diagnosed and under-treated. The second part of this two-part series

will address treatment methods and the differential diagnosis that should be considered.

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Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

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Physician CME Questions

101. What percent of patients with PE die in the first hour?
    A. 5%
    B. 10%
    C. 20%
    D. 30%

102. What is the most common pre-existing medical condition that leads to PE?
    A. Antithrombin deficiency
    B. Pregnancy
    C. DVT
    D. Factor V Leiden

103. PE is the:
    A. second most common cause of death in the United States.
    B. third most common cause of death in the United States.
    C. fourth most common cause of death in the United States.
    D. fifth most common cause of death in the United States.

104. Symptoms of PE include all the following *except*:
    A. hemoptysis.
    B. cough.
    C. sweating.
    D. sneezing.
    E. dyspnea.

105. Which of the following is *not* a clinical sign of PE?
    A. JVD
    B. Wheezes and rhonchi
    C. Tachypnea
    D. Rales and crackles

106. Regarding the physical examination for PE:
    A. A well-done physical exam can confirm the diagnosis of PE.
    B. Ninety percent of patients may present with subtle, non-specific symptoms.
    C. PE is not a consideration in cardiac arrest.
    D. All of the above are true of PE.

107. Venous ultasound has a poor sensitivity in the diagnosis of DVT.
    A. True
    B. False

108. The most common presenting symptom of PE is:
    A. cough.
    B. sweating.
    C. dyspnea.
    D. chest pain.

109. Fever is never associated with the diagnosis of PE.
    A. True
    B. False

110. Wells' criteria include which of the following?
    A. Fever
    B. Sweating
    C. Hemoptysis
    D. D-dimer

Answer Key:

101. B	105. B	109. B
102. C	106. B	110. C
103. B	107. B	
104. D	108. C	

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

In Future Issues: **PE, Part II**

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Pulmonary Embolism, Part I

Risk Factors for PE

PROLONGED IMMOBILITY	HYPERCOAGULABILITY	INTIMAL DAMAGE
• Paralysis	• Malignancy	• Trauma
• Long trips	• Previous PE or DVT	• Vascular surgery
• Bed rest	• High estrogen states	• Orthopedic surgery
• Pelvic tumors	• Inherited hypercoagulability disorders	• Central lines
		• IV drug use/abuse

Key: PE = pulmonary embolism; DVT = deep venous thrombosis IV = intravenous

Symptoms of PE

Dyspnea	70-90%
Chest pain	49-85%
Cough	3-55%
Leg swelling	17-35%
Hemoptysis	3-40%
Sweating	26-41%

Adapted from: Lee LC, Shah K. Clinical manifestation of pulmonary embolism. *Emerg Med Clin North Am* 2001;19:925-942.

Clinical Signs of PE

Tachypnea—rate greater than 25	48-59%
- rate greater than 16	66-92%
Tachycardia—heart rate greater than 100	24-71%
- among those with a massive PE	48-71%
- among those with non-massive PE	24-44%
Rales/crackles	50%
Fever greater than 37° C	30-54%
- greater than 38° C	7-20%
Leg swelling	17-35%
Jugular venous distension	12-31%
Diaphoresis	10-41%
Circulatory collapse	3-24%
Circulatory collapse among those with massive PE	34%

Adapted from: Lee LC, Shah K. Clinical manifestation of pulmonary embolism. *Emerg Med Clin North Am* 2001;19:925-942.

Wells' Criteria

VARIABLE	POINTS
Clinical signs and symptoms of DVT (objectively measured leg swelling and pain with palpation in the deep vein region)	3.0
Heart rate >100	1.5
Immobilization (bedrest, except for use of the bathroom for 3 or more days) or surgery (in the previous 4 weeks)	1.5
Previous objectively diagnosed DVT or PE	1.5
Hemoptysis	1.0
Malignancy (patients with cancer who are receiving treatment, those in whom treatment has been stopped in the last 6 months, or those who are receiving palliative care)	1.0
Pulmonary embolism as likely as or more likely than an alternate diagnosis	3.0

Low pretest probability	< 2points
Moderate pretest probability	2-6 points
High pretest probability	> 6 points

ECG Scoring System for Pulmonary Hypertension Caused by PE

FINDING	POINTS
Tachycardia	2
Incomplete RBBB	2
Complete RBBB	3
t wave inversion in all leads V1-V4	4
t wave inversion in lead V1	0-2
t wave inversion in lead V2	1-3
t wave inversion in lead V3	1-3
s wave in lead 1	0
q wave in lead III	1
Inverted t wave in lead III	1
If all s1, qIII, tIII is present	2

Adapted from: Daniel KR, Courtney DM, Kline JA. Assessment of cardiac stress from massive pulmonary embolism with 12-lead ECG. *Chest* 2001;120:474-481.

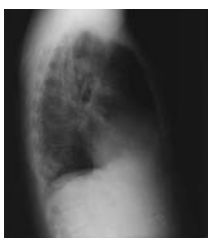
Using ECG to Predict PE

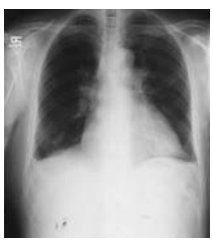
Sreeram found three or more of the following predicted PE with 82% sensitivity:

- Incomplete or complete RBBB associated with ST-segment elevation and positive t-wave in V1
- S waves in lead 1 and AVL of > 1.5 mm
- A shift in transition zone in the precordial leads to V5
- Q waves in leads III and AVF but not in II
- Right axis deviation with a frontal QRS axis of 90° or an indeterminate axis
- Low voltage QRS complex of < 5 mm in the limb leads
- T wave inversions in III and AVF or V1-V4

Adapted from: Sreeram N, Cheriex EC, Smeets JL, et al. Value of the 12 lead ECG at hospital admission in the diagnosis of pulmonary embolism. *Am J Cardiol* 1994;73:298-303.

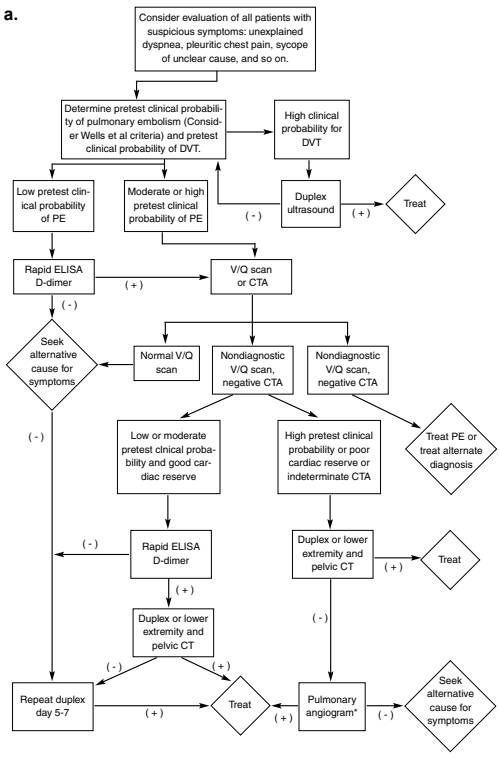
Hampton's Hump





Algorithms for Pretest Clinical Probability of PE

a.



a. Proposed diagnostic algorithm for the evaluation of suspected PE.

Key: US = ultrasound; **CTA** = computed tomography angiography.

* If initial pulmonary imaging study was V/Q scanning, consider CTA before pulmonary angiography.

b. **Pretest clinical probability**

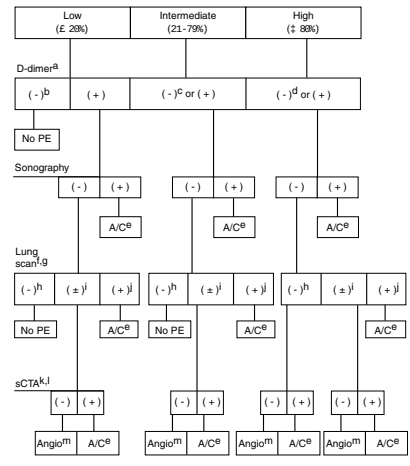


Fig. b: Suggested serial diagnostic testing strategy for patients presenting to the ED with varying clinical pretest probabilities of PE.

Key: a = conservative assessment of evidence supports use of VIDAS DD D-dimer assay; **b** = negative VIDAS DD D-dimer assay in low clinical pretest probability patients essentially excludes PE (< 1% posttest probability); **c** = evidence supporting use of negative VIDAS DD D-dimer result in intermediate clinical pretest probability patients for exclusion of PE appears valid; however, because the precision of the LR cannot be estimated, this test cannot be recommended at this time as unequivocally safe among the intermediate clinical probability patients; **d** = negative VIDAS DD D-dimer result does not exclude PE among high clinical pretest probability patients; **e** = anticoagulation; **f** = patients with underlying cardiopulmonary disease, especially chronic lung disease, may bypass radionuclide lung scanning and proceed directly to sCTA; **g** = ventilation scanning after a perfusion study, when indicated; **h** = negative lung scan finding indicates a reading of normal or near-normal; **i** = ± finding on lung scan indicates a clinically indeterminate reading (i.e., neither normal/near-normal nor high probability [very low, low, intermediate, and so forth]); **j** = positive (+) lung scan indicates a high probability reading; **k** = sCTA is spiral (helical) computed tomography angiography with contrast, not requiring catheterization; **l** = a plausible alternative diagnosis seen on sCTA is currently the only evidence-based means of excluding PE with this test; **m** = conventional pulmonary angioplasty, requiring catheterization; **PE** = pulmonary embolism.

Figures used with permission from: (a) Wolfe TR, Hartsell SC. Pulmonary embolism: Making sense of the diagnostic evaluation. *Ann Emerg Med* 2001; 37:509; and (b) Gallagher EJ. Clots in the lung. *Ann Emerg Med* 2000;35:182.

Supplement to *Emergency Medicine Reports*, May 17, 2004. "**Pulmonary Embolism: Recent Advances in Diagnosis and Treatment Modalities. Part I—Epidemiology, Risk Factors, and Pathophysiology."** *Author:* **Marianne Haughey, MD,** Assistant Professor of Emergency Medicine, Jacobi Hospital, Albert Einstein College of Medicine, Bronx, NY.

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Pediatric head injuries are common occurrences with the potential for serious morbidity or mortality. Fortunately, the incidence of traumatic brain injury (TBI) has been declining, mainly because of the development of effective prevention strategies (e.g., car seats and bicycle helmets). Although it is difficult to determine the exact incidence of head trauma in children due to variations in definitions and classifications, the majority of head injuries in children are minor and result in no long-term morbidity or mortality. However, early identification of a potentially serious injury and aggressive management of a child with a head injury facilitates the optimal possible outcome. The topic of pediatric TBI is extensive, and the majority of information is very familiar to the practicing emergency department (ED) physician. The author discusses two areas of controversy — patient selection for imaging and an update on management strategies for children with TBI. Selecting patients who require imaging following head trauma is easy if the child has an abnormal mental status or a Glasgow Coma Scale (GCS) score less than 15; he or she needs a head CT scan. The challenge is identifying high-risk patients with a GCS score of 15. The author reviews the available literature and presents currently available guidelines. Since TBI is the leading cause of death and disability, aggressive management of a child with a TBI is critical. The author reviews available therapies and their current application to pediatric patients.

—The Editor

Pediatric Controversies: Diagnosis and Management of Traumatic Brain Injuries

Author: **Kirsten Bechtel, MD**, Assistant Professor of Pediatrics,Yale University School of Medicine; Attending Physician-Pediatric Emergency Department, Yale New Haven Children's Hospital, New Haven, CT.

Reviewer: **Mary Jo A. Bowman, MD,** Associate Professor of Clinical Pediatrics, Ohio State University College of Medicine; Attending Physician, Columbus Children's Hospital, Columbus, OH.

Introduction

Trauma is the leading cause of childhood death,[1] and TBI is the leading cause of death and disability for children who sustain trauma.[2] Each year, more than 400,000 children younger than 14 years have emergent evaluations for head trauma.[3,4] Children younger than age 4 have considerable morbidity from head trauma. This age group has a prevalence of TBI that is more than twice the rate of the general population and nearly twice the rate for older children.[4] In addition, recent research indicates that even "minor" trauma may have the potential to result in life-long sequelae.[5,6] Thus, when evaluating children with head trauma, the practitioner must determine which patients are at risk, based on their history and physical exam, for significant injury requiring diagnostic imaging, careful monitoring, and aggressive intervention.

Evaluation of Children with Accidental Head Trauma

Injury patterns vary by the age of the child, with older children most likely sustaining an injury while participating in sports or when involved in motor vehicle collisions. However, children younger than 4 years most commonly sustain TBIs as a result of falls, motor vehicle collisions, or abuse. In the younger child, contact head injuries, such as linear skull fractures, hematomas, and cerebral contusions, can occur as the result of short, vertical falls.[7,8]

One study found that children who fell from a greater height were more likely to have injuries, but a number of patients had skull fractures or brain injury following falls from heights of less than three feet.[9] When there is a contact injury to the head, the point of impact causes the inner table of the skull to bend inward, which may injure blood vessels within the epidural or subdural space, as well as the parenchyma of the brain itself.[10] At the same time, there is also simultaneous outbending of the skull around the site of impact.[10] This puts the outer table of the skull under tension, and a fracture may result, either proximate to, or remote from, the site of impact. Children who sustain isolated skull fractures typically do not present with significant alterations in mental status, unless there is underlying brain injury with mass effect.[10]

Children Younger than 2 Years

Children younger than 2 years have been thought to be at high risk for significant brain injury after accidental head trauma.[11] Earlier studies often did not have enough data in the youngest age groups to recommend anything except a very cautious approach to evaluating head trauma in children younger than 2 years.[12,13] It has been estimated that the overall rate of brain injury after trauma in children younger than 2 years is approximately 5%, but infants younger than 2 months may have the highest prevalence of brain injury.[5]

Two studies in 1999 both evaluated infants younger than 1 year who presented to the ED with accidental head trauma.[5,9] The prevalence of brain injury was 12% in the 0-2 month age group, 6% in the 3-11 month age group, and 2% in infants older than 12 months.

Controversy exists in the literature regarding the ability of the physician to use clinical signs and symptoms to identify children at risk for brain injuries following blunt trauma. Obtaining an accurate history and a complete neurologic exam may be challenging, especially in younger children. Children younger than 2 years have been particularly identified as having subtle clinical presentations.[5] In addition, a computerized tomography (CT) scan of the head has disadvantages, including exposure to radiation, transport of the patient out of the ED, and a frequent requirement of sedation.[14-16]

Scalp Hematomas. Greenes and Schultzman sought objective markers of the presence of TBI and identified significant scalp hematomas as strongly associated with a skull fracture and brain injury in children younger than 2 years.[5] Another study also found the presence of a scalp hematoma to be the most important predictor variable for TBI identified on CT scan (e.g., intracranial hemorrhage, hematoma, or cerebral edema), in children 2 years and younger.[17] Finally, Greenes and Schutzman (2001) evaluated children younger than 2 years who sustained accidental head trauma, but had no neurological signs or symptoms.[18] The size and location of the scalp hematoma (e.g., parietal and temporal), and age younger than 3 months were each associated with skull fractures. This study also found that a skull fracture, large hematoma, and parietal location were associated with brain injury.[18] Children without a history of neurological symptoms and with a normal scalp exam were identified as a low-risk group.[9]

Abnormal Mental Status. Other series have examined the ability of an abnormal mental status to predict an abnormality on CT. Palchak et al found that of 194 children age 2 years and younger, all 15 children with a TBI on CT were predicted by the presence of a scalp hematoma and an abnormal mental status (sensitivity 100%; 95% CI 81.9—100%).[17] Of the 60 chil-

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This publication does not receive commercial support.

dren in this series age 2 years and younger who underwent CT and had a normal mental status examination and no scalp hematoma, none had a TBI identified on CT scan (negative predictive value 100%; 95% CI 95.1—100%). Lethargy, irritability, full or bulging fontanel, and vital signs suggestive of increased intracranial pressure (ICP) also have been associated with brain injury, while vomiting and loss of consciousness, at least in this age group, were not.[5]

Skull Fractures. Palchak et al found that of the 194 children age 2 years and younger who underwent CT scan, 15 (7.7%) had skull fractures on CT, and 46.7% had an associated TBI identified on CT.[17] Another study reported on 102 infants younger than 13 months with skull fractures. Fifteen of the 102 patients were found to have a brain injury. The authors found that patients with lethargy prior to presentation or in the ED and patients with parietal fractures were more likely to have sustained a brain injury.[19]

Guidelines. A multidisciplinary panel of nine experts in pediatric head trauma was convened.[20] All evidence gathered from a Medline search was reviewed, and using a modified Delphi technique, a set of guidelines for the evaluation of children younger than 2 years with minor head trauma was developed. Among the guiding principles the panel recommended were the following: One should have a lower threshold for diagnostic imaging in young children, with children younger than 12 months being at higher risk and children younger 3 months being at the highest risk for intracranial injury after head trauma; the greater the number and severity of signs and symptoms, the stronger the consideration should be for obtaining a CT. The greater the forces involved, the more pronounced the physical findings (e.g., scalp swelling), and the younger the age, the greater the risk for intracranial injury.

Specifically, the panel stratified the patients into risk categories based upon clinical features (e.g., history and physical examination), mechanism of injury, and absence/presence of a skull fracture.

High-risk patients had any of the following characteristics: depressed mental status, focal neurologic findings, signs of depressed or basilar skull fracture, seizure, irritability, acute skull fracture, bulging fontanel, vomiting greater than five episodes or for more than six hours, and loss of consciousness greater than one minute. All high-risk patients required a cranial CT scan.

Intermediate-risk patients had any of the following characteristics: vomiting three to four times; loss of consciousness less than one minute; history of lethargy or irritability, now resolved; caretakers concerned about current behavior; higher force mechanism; hematoma (especially large or nonfrontal in location); unwitnessed trauma; fall onto a hard surface; vague or no history of trauma with evidence of trauma; and nonacute skull fracture older than 24-48 hours. Patients in this category could be managed in one of two ways: a period of observation (4-6 hours recommended) and reevaluation, or a head CT scan.

Low-risk patients were defined as having low-energy mechanism (e.g., fall less than 3 feet), no signs or symptoms, and more than two hours since the injury; also, the panel found that as the patient's age increases, the risk decreases. These patients may be observed in the ED or at home with reliable caretakers.[20]

Apart from these findings and the panel recommendations, evidence exists suggesting that the youngest age group is more likely to have brain injury with no neurological findings.[6,21,22]

Children Older than 2 Years

For older children, it is easier to obtain historical information and an accurate physical examination. Many series have sought to determine historical factors and clinical features that are predictive of an intracranial injury. A recent prospective study found that in 2043 children younger than18 years with head trauma, an abnormal mental status, clinical signs of skull fracture or scalp hematoma (in patients younger than 2 years), history of headache and vomiting were predictive of intracranial injury.[17] The most important variable in this series was clinical findings of a skull fracture.

These five clinical findings identified 97 (99%; 95% CI 94.4—100%) of the 98 children with TBI on CT scan and all 105 children with TBIs that required acute intervention. Of the 304 (24%) children with CT scans who didn't have any of the five predictors, only one had a TBI on CT scan (0.3%; 95% CI 0—1.8%). Of the 825 patients who had none of the five predictors, no one had a TBI requiring acute intervention (negative predictive value 100%; 95% CI 99.6%—100%). Use of this rule would have decreased CT scan utilization by approximately 25%.[17] Similarly, another study found that children older than 2 years with closed head trauma who were neurologically normal and had no clinical signs of skull fracture could be managed safely without cranial CT.[23]

In 1999, the American Academy of Pediatrics published guidelines for the management of closed head trauma in previously healthy children 2-20 years of age.[24] This consensus statement used the historical features of loss of consciousness and the presence of symptoms as an indication for obtaining a CT scan of the head. For those children without a loss of consciousness, a thorough history and physical examination should be performed, and a competent caregiver should observe the patient for any deterioration in mental status. For those who have a history of a brief loss of consciousness, along with amnesia, headache or vomiting at the time of evaluation, the prevalence of intracranial injury may be as high as 7%.[25-27] Though many of these brain injuries may have little clinical consequence, a minority of these children may require neurosurgical intervention.[26-28] Therefore, in this group of symptomatic children with a brief loss of consciousness, CT scanning of the head may be useful. However, with a brief loss of consciousness alone in an otherwise asymptomatic patient, observation of the patient for neurological deterioration may be an acceptable alternative to obtaining a CT scan of the head.[24]

While CT scanning is usually a safe procedure, some children may require sedation to obtain the study. Therefore, one must consider the risks of sedation against the benefits of obtaining a CT scan in this group of asymptomatic patients.

Once a TBI has been detected, the type of facility where the child will be evaluated and treated is important to the recovery. Several studies have examined the impact of pediatric trauma centers on the initial management of pediatric trauma. One study evaluated the morbidity and mortality rates among pediatric trauma victims in Pennsylvania and found that morbidity and mortality from TBI was reduced significantly in patients who were treated at pediatric trauma centers.[29] More neurosurgical procedures were performed in pediatric trauma centers, and there was concomitant lower mortality from TBI.[29] Another study found that the mortality rate was significantly higher for children with TBIs who were first transported to non-pediatric hospitals and subsequently transferred to pediatric trauma centers.[30] Thus, it is important that children with brain injuries be transferred to the nearest pediatric trauma facility as soon as it is feasible.

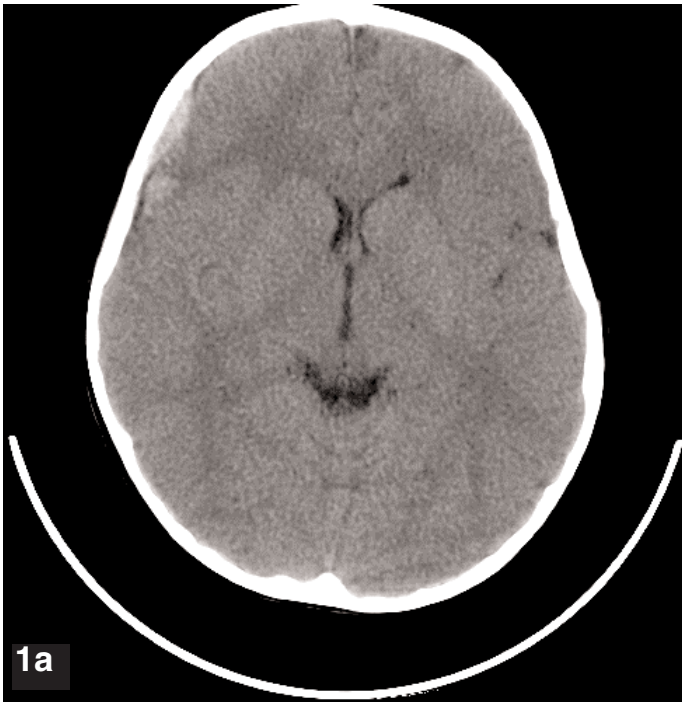
The guidelines for the acute management of severe TBI in infants, children, and adolescents made transfer to a pediatric trauma center a guideline based upon class II data (prospective and retrospective observation, cohort, and case control) and strong class III data (retrospective), and, as an option, an adult trauma center with qualifications for pediatric treatment.

Management of Intracranial Injuries

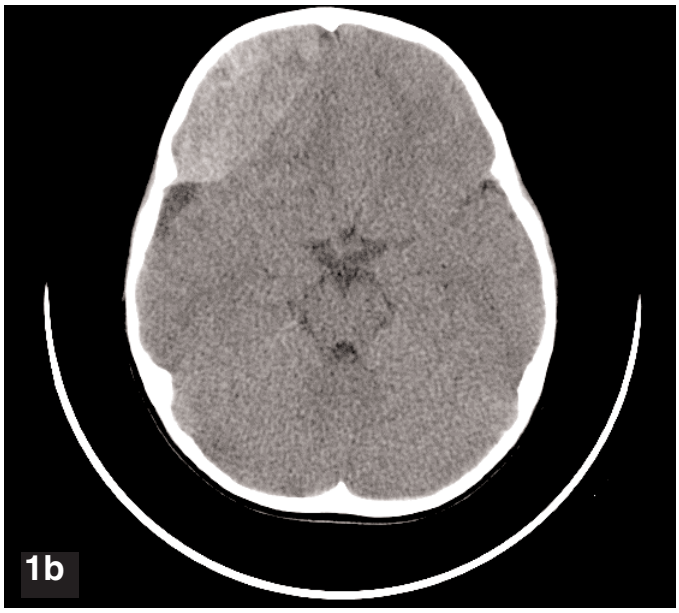
Group 1: Asymptomatic Intracranial Injuries. The optimal management and outcome of children who have intracranial injury as detected by CT scan, but who are otherwise asymptomatic, is controversial. Typically, such children are admitted to the hospital for close neurological assessment and monitoring. Many pediatric neurosurgeons have adopted an approach of expectant management for small intracranial and extradural hematomas, taking into consideration the size of the hematoma, its propensity to increase in size, shift of midline intracranial structures and surrounding cerebral edema.[24] (*See Figure 1.*) In some cases, children with subdural hemorrhage from minor trauma may do quite well with expectant management. Four patients were reported with unilateral subdural hemorrhage, of which three occurred from minor trauma and one from a fall out of a window. In all four cases, the subdural hemorrhage resolved spontaneously within 48 hours of injury.[31]

Critical to the management of children with an acute TBI is the initial assessment of the child's neurologic status and ongoing monitoring. Standardized assessment scores are the most accurate for detecting subtle changes in a patient. The GCS is useful for repeated neurological assessments in children with TBI. (See *Table 1.*) In one study, the most important prognostic indicators for pediatric TBI were demonstrated: the presence of associated trauma, admission GCS scores, traumatic mass lesions with ICP, and the presence of diffuse axonal injury.[32] There are modifications to the GCS to accommodate children who are preverbal or who are unable to verbally communicate due to sedation or endotracheal intubation. Such modifications include the Children's Coma Scale and the Infant Face Scale.[33,34] (*See Table 2.*)

Figure 1a and 1b. Rapidly Expanding Epidural Hematoma



1a. A head CT of a child performed two hours after a fall. The child had progressive emesis and lethargy. **1b.** Same patient's head CT five hours after the head trauma done secondary to increasing lethargy. Note the rapidly expanding epidural hematoma.



Group 2: Symptomatic Intracranial Injuries. The primary injury is the injury that occurs to the brain as a direct result of the trauma. Once an intracranial injury has occurred, management is directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to progressive brain injury. The major, avoidable secondary insults include hypoxia and hypotension, which may

Table 1. Glasgow Coma Scale

EYE OPENING	
Spontaneous	4
Verbal stimulation	3
Painful stimulation	2
None	1
MOTOR	
Obeys commands	6
Localizes	5
Withdraws	4
Flexion	3
Extension	2
None	1
VERBAL	
Oriented	5
Confused	4
Inappropriate	3
Incoherent	2
None	1

Table 2. Glasgow Coma Scale — Modifications for Children

CHILDREN'S COMA SCALE (HAHN ET AL 1988) BEST SCORE = 15	
• Modification to best verbal response	
Smiles, orients to sound, follows objects, interacts	5
Consolable	4
Inconsistently consolable	3
Inconsolable	2
No response	1
INFANT FACE SCALE (DURHAM ET AL 2000) BEST SCORE = 15	
• Modification to best motor response	
Spontaneous normal movements	6
Hypoactive movements	5
Nonspecific movement to deep pain	5
Abnormal, rhythmic, spontaneous movements	3
Extension, either spontaneous or to pain	2
Flaccid	1
• Modification to best verbal response	
Cries spontaneously to handling or pain, alternating with quiet wakefulness	5
Cries spontaneously to handling or minor pain, alternating with sleep	4
Cries to deep pain only	3
Grimaces only to pain	2
No facial expression to pain	1

occur in the patient with multiple trauma; and intracranial hypertension, which may occur after the primary brain injury. Secondary brain injury causes a loss of cerebrovascular autoregulation and may result in cerebral edema, thereby reducing cerebral blood flow. Secondary brain injury also may be due to release of excitatory neurotransmitters, which can alter intracellular ion concentrations; and to the formation of inflammatory mediators, which can disrupt the blood-brain barrier and exacerbate neuronal damage.[35-37] Therefore, the goals of treatment of children with significant brain injury are to lower ICP and maximize cerebral perfusion pressure and oxygen delivery to the brain.

Monitoring of the ICP is appropriate in patients who have GCS score of 8 or less; have an abnormal initial CT scan of the head that demonstrates hematomas, contusions, or cerebral edema; or in whom serial neurological examinations are not possible due to other injuries, sedation, or neuromuscular blockade. There have been several studies in children that demonstrate an association between intracranial hypertension and poor neurological status at hospital discharge.[38,39]

ICP Monitoring. Recently published guidelines for the management of severe TBI in children recommend that a ventricular catheter connected to an external strain gauge is the most accurate and reliable manner in which to monitor ICP. [41] Such a device also allows for therapeutic diversion and analysis of cerebrospinal fluid.[40] These guidelines also recommend that the ventricular ICP be used as the reference standard in comparing the accuracy of ICP monitors placed in other cranial compartments.[41] Intracranial hypertension is defined as an ICP greater than 20 mmHg. The guidelines recommend that therapy be instituted when the ICP is consistently between 20-25 mmHg.[41] Other authors have suggested that the treatment of elevated ICP should be age dependent. In the young infant, treatment should begin when the ICP is greater than 15 mmHg; for children younger than 8 years, when the ICP is greater than 18 mmHg; and for older children and adolescents, when the ICP is greater than 20 mmHg.[35]

ICP Reduction.There are several methods to reduce ICP. Hyperventilation to reduce the pCO_2 below 35 mmHg may be useful in the setting of an acute rise in ICP or when signs of impending herniation are present. While hyperventilation may temporarily reduce intracranial hypertension, it also increases the volume of hypoperfused tissue in the injured brain; thus long periods of hypocarbia should be avoided.[41] The child's head should be maintained in a neutral position, and the head of the bed elevated to 30°. These maneuvers may decrease ICP without significantly changing cerebral perfusion pressure.[35] Jugular venous obstruction, which can elevate ICP, should be avoided by ensuring that cervical collars and endotracheal tube ties are not constrictive around the neck.[35]

Cerebral perfusion pressure (CPP) is defined as the difference between the mean arterial pressure and the ICP. The CPP is the gradient that promotes cerebral blood flow and substrate delivery to the brain. A CPP of 40-65 mmHg represents a spectrum to guide the efficacy of therapeutic interventions. Children with a CPP of 40-50 mmHg tend to have better survival after TBI.[42-45] Some authors have recommended that in young children, the CPP be maintained above 40-45 mmHg and above 50 mmHg in older children and adolescents.[35]

Therapeutic Interventions

Airway Management. *Hypoxia.* Patients should be well oxygenated throughout their ED course. Sedation and neuromuscular blockade may be useful to reduce the untoward effects of painful and noxious stimuli in patients with TBIs. Such stimuli include endotracheal intubation and mechanical ventilation, endotracheal suctioning, placement and maintenance of intravascular or intracranial catheters and monitoring devices, and transport for diagnostic procedures. Painful or stressful stimuli may increase the brain's oxygen consumption and increase sympathetic tone, leading to systemic hypertension and bleeding from operative sites.[46-48] There has been no systematic study of the efficacy of sedative and paralytic agents in children with TBI, and thus, there is no consensus as to what constitutes the ideal agents for sedation and neuromuscular blockade in this group of patients. There are case reports of the systematic, but limited, use of benzodiazepines, barbiturates, propofol, and non-depolarizing paralytic agents in children with TBI.[48] Prolonged use of propofol should be avoided in children because of reports of metabolic acidosis associated with its use. When using such agents, one must be aware of potential age-related differences in the response to pain and stress and in the level of sedation that patients may have.

Hypotension. Hypotension, which may occur in a pediatric multi-trauma patient, should be managed aggressively. Patients should be monitored carefully for the early signs of shock, including tachycardia, prolonged capillary refill, and loss of peripheral pulses. All volume deficits should be corrected and transfusions, when indicated, should not be delayed, to maintain hemoglobin and hematocrit at 10 mg/dL and 30%, respectively.[49]

Osmolar Agents. Osmolar agents, such as hypertonic saline and mannitol, have long been used in the treatment of children with TBI. Hypertonic saline works by increasing serum sodium concentration and serum osmolarity, creating an osmotic gradient by which water is pulled from the intracellular and interstitial compartments into the intravascular compartment. This increases intravascular volume and cerebral perfusion pressure, and reduces cerebral edema and ICP. One study reported results of a double-blind, crossover study comparing 3% saline and 0.9% saline boluses in 18 children with TBI.[50] During the initial trial boluses with hypertonic saline, the ICP decreased and there were reduced requirements for additional interventions. The guidelines for the acute management of severe traumatic brain injury in infants, children, and adolescents lists hypertonic saline as an option. The guidelines point out that hypertonic saline has evidentiary support, but mannitol has clinical acceptance and safety. Though mannitol works in a similar fashion, the blood brain barrier is able to exclude sodium chloride from the intracranial compartment, making it less likely to accumulate in the interstitial space.[51] Hypertonic saline also causes a reduction in vascular resistance by decreasing edema in the vascular endothelium of injured tissues.[52] Hypertonic saline also may normalize resting membrane potentials and cell volumes by restoring normal intracellular electrolyte balance in injured brain cells.[53] Rapid lowering of the serum sodium concentration should be avoided. Rebound cerebral edema can occur due to intracellular fluid shifts when the serum sodium concentration falls rapidly in the face of a residual hyperosmolar intracellular environment.[52]

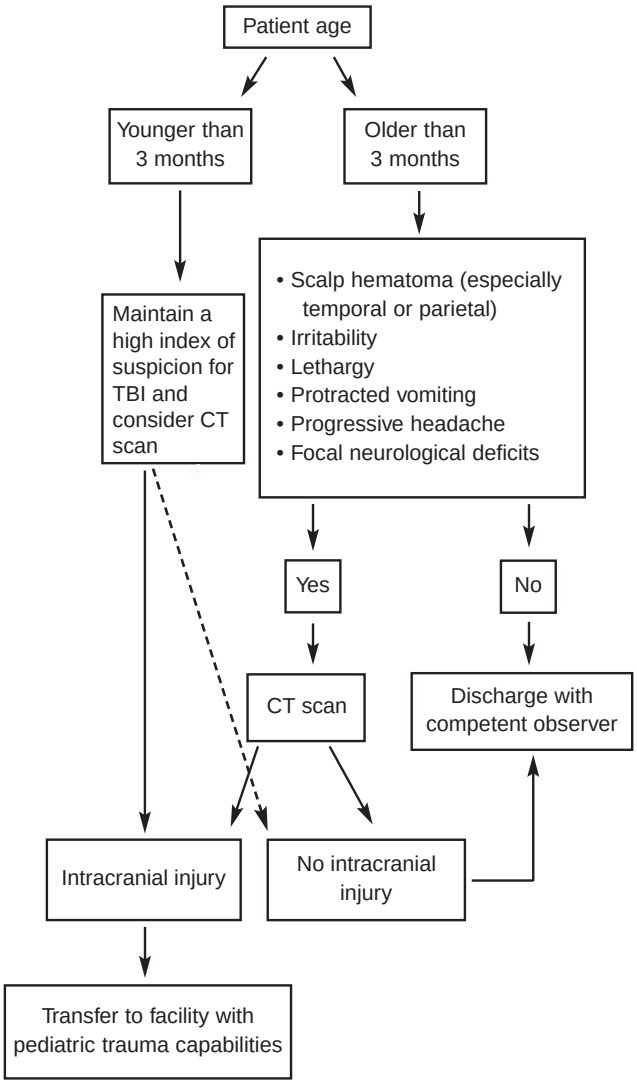
Mannitol works in a similar fashion by decreasing blood viscosity and, thereby the diameter of cerebral blood vessels. Cerebral blood flow is maintained by reflex vasoconstriction of the cerebral vasculature, but cerebral blood volume and ICP are reduced.[54] This mechanism relies on intact autoregulation of cerebral blood flow by the brain. Mannitol also reduces ICP by changing the osmotic gradient within the cerebral vasculature, causing water to move from injured tissues into cerebral blood vessels.[54] Mannitol should be administered as intermittent bolus doses. Prolonged administration of mannitol can result in its accumulation within injured tissues, reversing the osmotic gradient with the cerebral vasculature and worsening cerebral edema.[55]

Cerebral Metabolism Reduction. Reducing cerebral metabolism may be helpful in reducing ICP. Early initiation of barbiturate coma may reduce the risk of secondary brain injury. Barbiturates can lower ICP by reducing cerebral metabolism, altering cerebrovascular tone and reducing neuronal, free-radical injury.[35] Lower doses of pentobarbital initially may be given to prevent myocardial depression and systemic hypotension. It may not be necessary to use higher doses of pentobarbital to obtain burst suppression on the electroencephalogram (EEG), as lower doses still may have significant neuroprotective effects.[35]

Seizure Control. Seizures can cause a rise in ICP by increasing the brain's metabolic demands, releasing excitatory neurotransmitters, and raising systemic blood pressure. Antiepileptic drugs (e.g., phenytoin, fosphenytoin, or phenobarbital) may be helpful to prevent seizures within the first week after severe TBI, but their effectiveness in preventing late onset (i.e., longer than one week) seizures has not been demonstrated.[56] Some authors have recommended antiepileptic prophylaxis if there is significant parenchymal injury in children with severe TBI.[35] Children younger than 2 years of age are at high risk of post-traumatic seizures, with 44-70% of those with severe brain injuries having post-traumatic seizures.[35,57]

Hypothermia. The role of hypothermia in the treatment of children with TBI is unclear. While initial studies in adults demonstrated benefit in adults with TBI and intracranial hypertension, a recent randomized prospective study showed that hypothermia did not reduce morbidity and mortality in adults with severe TBI.[58-60] A similar degree of hypothermia has been shown to be efficacious in children with uncontrolled intracranial hypertension after TBI.[61] While intracranial hypertension was ameliorated after 48 hours of induced hypothermia when compared with the normothermic group, functional outcomes of survivors were similar between the two groups. A larger randomized trial is needed to definitively determine if induced hypothermia improves survival in children with TBI. Currently, the Guidelines for Acute Management of Severe Traumatic Brain Injury in Infants, Children and Adolescents recommend as an option, to avoid hyperthermia (i.e., temperature is higher than 38.5°C), and consider hypothermia (i.e., temperature is

Figure 2. Children Younger than 2 Years with a Head Injury



32-33°C) if refractory intracranial hypertension occurs.

Operative Intervention. Finally, operative intervention may be a necessary adjunct to medical therapy for severe TBI. Significantly depressed skull fractures should be elevated and intracranial and intraparenchymal mass lesions should be evacuated or debrided when ICP and CPP cannot be optimally managed by medical measures.[35] Some studies have demonstrated that decompressive craniectomy may be useful for pediatric patients with severe head injuries with uncontrolled intracranial hypertension.[62,63]

Predictors of Outcome

There has been a significant decline in the morbidity and mortality of pediatric TBI in the United States during the past two decades.[64] The overall mortality of children with TBI in the United States has been reported to be 6%, and those children with severe head injury requiring mechanical ventilation have a mortality of approximately 18%.[65,66]

There may be several reasons for such a decline in morbidity and mortality. One study analyzed consecutive admissions of children with TBIs to three different pediatric intensive care units. He found that while there was significant variation among centers with respect to the use of neuromuscular blockade, induced hypothermia and ICP monitoring, none of these modalities had an effect on mortality. Only the use of antiepileptic agents significantly reduced mortality in this study.[67] Another study found that in children with severe traumatic brain injuries, survival was associated significantly with the maintenance of supranormal systolic blood pressure (i.e., greater than 135 mmHg).[68] Mannitol was associated with a prolonged length of stay in the pediatric intensive care unit, but had no effect on survival. Similarly, Pigula found that children with severe head injuries and systemic hypotension had a much greater mortality rate.[69] Further study is needed to determine which interventions have an impact on morbidity and mortality in children with TBIs.

Several investigators have evaluated which factors may predict both survival and functional outcomes of children with TBI. In severe TBI, the GCS score and Pediatric Risk of Mortality Score (PRISM) may be predictive of survival.[66] In a retrospective study, children with GCS scores less than or equal to 5, but with lower PRISM scores, were more likely to survive and be discharged from the hospital. At hospital discharge, 40% of these patients were functioning independently; and at two years after the injury, nearly 66% were functioning independently. However, independent functioning in childhood may not persist into adulthood. In another study, 39 adults who had sustained TBI during the preschool years were evaluated.[70] While 59% of these patients attended a regular school after recovering from their TBI, only 29% eventually had full time employment as adults.[70] Most of these patients had sustained their TBI nearly 30 years ago, and it can be argued that recent advances in resuscitation of brain-injured children eventually may improve functional outcomes that persist into adulthood. Finally, serum levels of protein S-100 beta, a calcium-binding, dimeric protein found in astroglial and Schwann cells, when obtained and measured at the initial time of injury, may have predictive value in determining functional outcome in children and adults with mild to severe TBI.[71,72]

School-age children who survive TBI are at risk for having neuropsychological deficits and developing psychiatric syndromes. Children who survive severe TBI are at risk of having deficits in verbal reasoning, learning and recall, attention, executive functions, and constructional skills within 12 months of hospital discharge. Even when evaluated as long as four years after the injury, there may be little long-term recovery of such skills.[73] Children who recover from both mild and severe TBI are more likely than those who recover from orthopedic injuries to have psychiatric disturbances, such as organic personality disorder, attention deficit-hyperactivity disorder, major depression, and anxiety disorders.[74] Siblings and parents of children who survive severe TBI may also experience psychological distress during the patient's recovery and rehabilitation periods.[75,76]

Summary

TBI can cause considerable morbidity in young children. Children younger than 1 year, and particularly those younger than 3 months, are at higher risk of sustaining a TBI after head trauma than are older children. Scalp hematomas, especially those over the parietal region, altered mental status, and focal neurological signs, are the best clinical indicators of TBI in children.

Children with TBI are best managed at trauma centers, and transfer to such facilities should be expedited when TBIs are diagnosed in children. Once a primary brain injury, or trauma that results directly from impact, has occurred, the goals of management are directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to secondary brain injury. Maximizing CPP and reducing ICP are the goals of management of children with TBIs. Sedation, neuromuscular blockade, hyperosmolar therapy, barbiturate therapy, and antiepileptic prophylaxis are management options in children with TBIs.

Finally, children and their families will require considerable support during the rehabilitation phase after a TBI. Psychological and psychiatric sequelae are common in children after a TBI, and significant family stress can occur during the patient's recovery and rehabilitation period.

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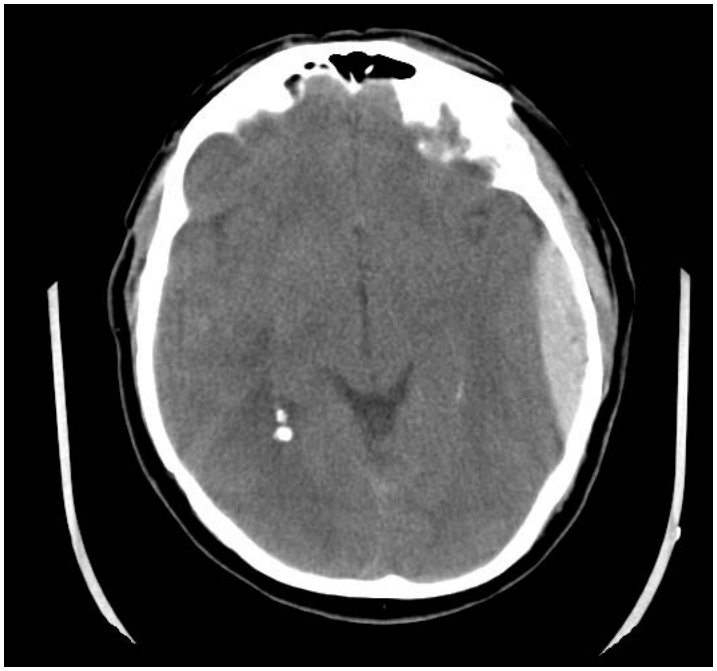
CE/CME Questions

1. Which of the following is true regarding a child younger than 2 years who sustains a head injury?
   A. The younger the child, the higher the risk for traumatic brain injury.
   B. The incidence of brain injury in a child younger than 2 years is about 5%.
   C. CT scans do have certain disadvantages, including exposure to ionizing radiation.
   D. All of the above

2. A 3-month-old male presents after his mother dropped him when she tripped. He fell approximately five feet. He is irritable, but consoles and has a large parietal hematoma. The most appropriate next test is:
   A. MRI.
   B. CT scan of the head.
   C. skull films.
   D. skeletal survey.

3. A 7-year-old male was involved in a fight at school four hours ago. He did not lose consciousness, remembers the entire event, and has had no vomiting. His neurologic examination is normal. On physical examination, he has a hematoma on his forehead. The next best test is:
   A. an MRI.
   B. a CT scan of the head.
   C. skull films.
   D. None of the above

4. Which of the following has/have been associated with an intracranial injury in a child younger than 2 years?
   A. Skull fracture
   B. Parietal scalp hematoma
   C. Large scalp hematoma
   D. All of the above



5. What is shown in the image above?
   A. Epidural hematoma
   B. Subdural hematoma
   C. Intraparenchymal hematoma
   D. None of the above

6. Which of the following is *not* considered to be high-risk criteria for TBI in a child younger than 2 years?
   A. Depressed mental status
   B. Signs of depressed or basilar skull fracture
   C. Two episodes of emesis
   D. Acute skull fracture

7. Which of the following children does *not* require a cranial CT following a fall?
   A. A 3-year-old with an occipital hematoma, no other symptoms, and a normal exam
   B. A 4-month-old who has a large scalp hematoma and is irritable
   C. A 1-year-old who has a GCS score of 13
   D. A 6-year-old with hemotypanum

8. Which of the following are critical in the initial stabilization of a child with a head injury?

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) Recognize or increase index of suspicion for pediatric head injury;
b.) Identify how to correctly and quickly stabilize and manage pediatric head trauma;
c.) Employ appropriate diagnostic modalities for pediatric head trauma; and
d.) Recognize indications and potential risks with therapeutic options for children with head trauma.

A. Avoiding hypoxia
B. Avoiding hypotension
C. Maintaining an adequate cerebral perfusion pressure
D. All of the above

9. In which of the following scenarios is ICP monitoring *not* an appropriate consideration?
A. A child with a GCS score less than 8
B. A child with a GCS score of 12 five minutes after a seizure
C. A child who was intubated at the scene, is unresponsive and has cerebral edema on CT scan
D. A child who is intubated for a multi-system trauma and must be paralyzed and sedated

10. Which of the following may be used in the management of a child with a head injury and a GCS score of 8?
A. Early intubation
B. ICP monitoring
C. Correction of hypotension
D. All of the above

Answer Key:

1. D	**6. C**
2. B	**7. A**
3. D	**8. D**
4. D	**9. B**
5. A	**10. D**

In Future Issues:

Knee Injuries