Pulmonary Embolism

Massive pulmonary embolism (MPE) results whenever the combination of embolism size and underlying cardiopulmonary status interact to produce hemodynamic instability; the presence of shock adds a threefold to sevenfold increase in mortality, with a majority of deaths occurring within 1 h of presentation; thrombolytic therapy is acknowledged as the treatment of choice, with embolectomy reserved for those in whom thrombolysis is contraindicated.

ANESTHETIC CONSIDERATIONS:

- Potential life threatening emergency causing hypoxemia, RV failure, obstructive shock, & PEA arrest
- Timely diagnosis with simultaneous aggressive supportive management & early definitive thrombolysis or embolectomy is essential
- Risk of CV collapse with induction of anesthesia and positive pressure ventilation
- Co-morbidities: malignancies, prolonged immobilization, elderly, obese, CHF, Trauma

ANESTHETIC GOALS:

- Simultaneous diagnosis & management = aggressive resuscitation
- Minimize PVR, promote contractility and maintain SVR for vital organ perfusion
- Minimize anesthetic to prevent any further myocardial depression
- Be prepared for CV collapse

The scenarios the anesthesiologist may face with respect to PE are:

- The preoperative patient at high risk for PE or PE recurrence
- The elusive diagnosis of perioperative PE, followed by treatment
- Intraoperative massive PE and resuscitation
- Rarely – the patient presenting for embolectomy for massive PE or chronic thromboembolic pulmonary hypertension

- Difficult clinical diagnosis b/c signs, symptoms & basic lab tests are neither specific nor sensitive, thus, must have clinical suspicion based on risk factors, history, physical exam & investigations

HISTORY

TABLE 9–17 -- Signs and Symptoms of Pulmonary Embolism

Sign/Symptom	Incidence (%)
Acute dyspnea	75
Tachypnea (>20 breaths per minute)	70
Pleuritic chest pain	65
Rales	50
Nonproductive cough	40
Tachycardia (>100 bpm)	30
Accentuation of pulmonic component of second heart sound	25
Hemoptysis	15
Fever (38°–39°C)	10
Homans' sign (calf or popliteal pain with abrupt dorsiflexion of foot at the ankle with knee flexed to 90 degrees)	5

*"Classic symptoms of **pulmonary embolism** include distended neck veins, hypoxemia, tachycardia, tachypnea, and chest pain" Miller 7th ed. Ch 73*

Under GETA:

- Can be non-specific & often transient but can see:
  - Sudden decrease in SpO_2, $ETCO_2$, & BP
  - Combined with increased HR, CVP, PAP, +/- AWPs (bronchoconstriction)
  - Or sudden cardiac arrest (PEA > asystole)

TEE is KEY!!

TABLE 9–16 -- Differential Diagnosis of Pulmonary Embolism

Myocardial infarction

Pericarditis

Congestive heart failure

Chronic obstructive pulmonary disease

Pneumonia

Pneumothorax

Pleuritis

Thoracic herpes zoster

Anxiety/hyperventilation syndrome

Thoracic aorta dissection

Rib fractures

PHYSICAL

- **VITALS** - ↓ BP, ↓ SpO_2, ↑ HR, ↑ RR
- **CVS** – RV failure (↑ JVP, RV heave, hypotension, tachycardia, + HJR)
- **RESP** – tachypnea
- **MSK** – evidence of DVT (leg edema, inflammation, palpable cord), fat embolism (petechiae)
- **Pretest Clinical Probability Assessment**: Wells Criteria:

Wells criteria and modified Wells criteria: clinical assessment for pulmonary embolism

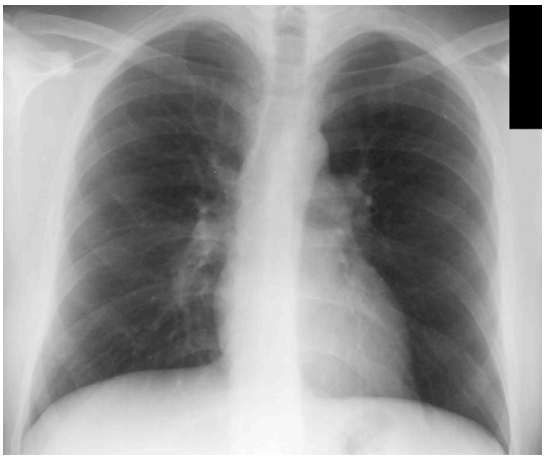
Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)*	
PE likely	>4.0
PE unlikely	≤4.0

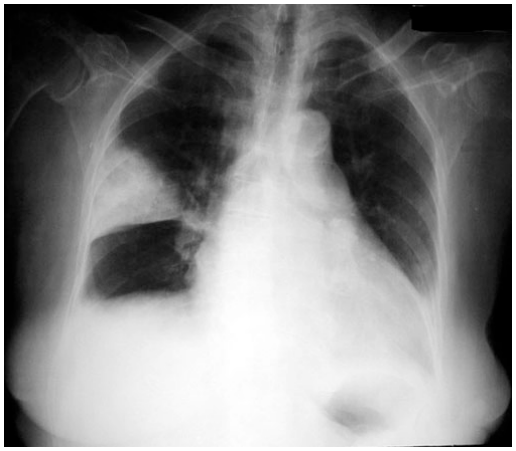
Data from van Belle, A, et al. JAMA 2006; 295:172.

INVESTIGATIONS

- **Labs**
  - CBC, Lytes, Cr, PT, PTT
  - D-dimer: good sensitivity & NPV, but poor specificity & PPV
    - +ve test means PE is possible. –ve D-dimer strongly suggests PE absent (NPV > 99%)
    - BUT keep in mind D-dimer positive in postoperative surgical patients!
  - ABGs: hypoxemia, hypocapnia, ↑ A-a gradient (PaO_2 > 80 & normal A-a gradient in about 15%)
  - Troponin: may be elevated due to myocyte damage due to acute RV strain
  - BNP: typically greater in patients with PE but may be normal (not specific or sensitive)

- **Imaging**
  - ECG: non-specific ST-T changes, RBBB, $S_1Q_3T_3$ (prominent S in I with Q & inverted T in III), T-wave inversion in V1-V4 (most specific) but most useful to r/o MI instead of rule in PE
    Peaked P waves, AFib & new RBBB if PE large enough to cause acute cor pulmonale
  - CXR: dilated PA, Westermark's sign (segmental oligemia), Hampton's hump (pleural-based cone infarct), pleural effusion can be seen

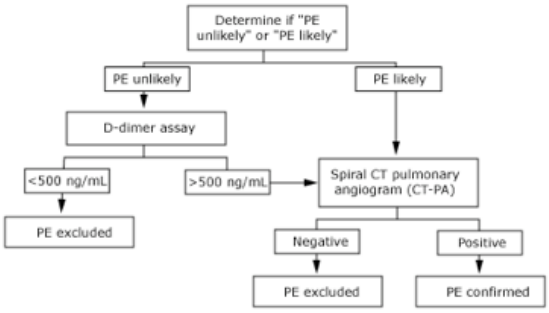




  - Venous U/S of legs: can have false positive (3% in one study) & false negative results (29% in another study). Quality of exam is operator dependent
  - V/Q scan: drawback is high number of intermediate probability scans (neither sensitive nor specific) (PIOPED study)
  - ECHO = best b/c it is transportable, and is capable of differentiating shock states and recognizing the characteristic features of PE:

        - Unexplained RV dysfunction has 50% sensitivity & 90% specificity for PE
        - Normal ECHO eliminates PE as the cause of a shock state
        - RV dysfunction, septal shift, visible thromboemboli, ASD / VSD / PFO, increased PAP / TR, decreased LV filling
        - Several features suggest chronic failure rather than acute PE: hypertrophied RV, preservation of the normal inspiratory collapse of IVC, minimal septal shift
    - Pulmonary Angiography - gold standard but not used much anymore
    - **Spiral CT** (CTA) - has replaced V/Q as imaging of choice with the exception of PE in pregnancy (discouraged d/t radiation exposure to fetus)
        - most useful for detecting clot in main, lobar & segmental arteries
        - less sensitive for detecting emboli in smaller blood vessels.

CT-based diagnostic strategy used in patients with suspected pulmonary embolism



Adapted from van Belle, A, et al. JAMA 2006; 295:172.

- Algorithm based on presence / absence of shock since this is major predictor of survival

Management Goals in Major PE

- Rapid diagnosis & institution of supportive & definitive therapy
- Aggressive resuscitation with goals to minimize PVR, promote contractility and maintain SVR for vital organ perfusion

Setup (usual setup & CAS monitors)

- Invasive HD monitors (a-line preinduction if possible, CVP, ± PAC); TEE for diagnosis and management
- Vasopressors and inotropes in syringes and infusions
- Pulmonary vasodilators (Inhaled NO or prostacyclin)

- Shock = major predictor of outcome so it is a useful clinical indicator for initial management
- ABCs w/ use of ACLS guidelines (PEA arrest)
- Consider CVT, respirology, & interventional radiology consults
- If PE suspected, anticoagulation with IV heparin is indicated while awaiting diagnostic workup
- Fluid resuscitation is controversial - increased fluid can help w/ RV dysfunction but can worsen LV dysfunction & pulmonary function - most use vasopressors 1st now
- Vasopressors:
    - Norepinephrine = 1st line since it improves RV function through alpha-mediated arterial & venous constriction, enhances RV CPP, & decreases RV ischemia
    - Phenylephrine increases CPP d/t increased SVR but also increases PVR
    - Milrinone = potentially another good choice (inodilator = beneficial ↓ PVR but need vasopressor to maintain systemic perfusion)
    - Dobutamine = useful if moderate (not severe) hypotension, but can cause hTN
    - Dopamine may also be useful, but in large doses may increase PVR
    - Vasopressin may be useful
- Pulmonary vasodilators
    - iNO will decrease PVR, offload RV (and thus improve LV function), increase CO and may improve V/Q matching
    - inhaled prostacyclin may also considered
- Heparin is given on spec for suspected PE
- Thrombolytics:
    - Treatment of choice for HD instability w/ decreased mortality & recurrence, improved reperfusion, greater improvement RV function compared to heparin
    - Probably safe in PEA arrest requiring CPR (some feel it is contraindicated in this setting but based on case series' and case reports, probably safe w/o ↑ incidence fatal hemorrhage)
    - Options:

        - rt-PA: 100 mg q30min x 2 doses (AMI dose), 0.6 mg/kg in 15min
        - Reteplase: 10 mg q30min x 2 doses
        - Streptokinase is slower and can produce hTN & anaphylaxis
- Embolectomy:
    - Indications include contraindication to or failed thrombolysis, refractory hTN, intermittent cardiac arrest, presence right heart thrombi
    - Can be done via catheter or sternotomy
    - Usually done with CPB but can be done w/ venous inflow occlusion and normothermic arrest
    - PEEP may help during actual embolectomy
    - Potential complications are hypoxemic respiratory failure from reperfusion pulmonary edema, RV dysfunction and persistent shock, pulmonary HTN, massive pulmonary hemorrhage

OPTIMIZATION

- Prevention:
    - TEDS (low efficacy in high risk)
    - Pneumatic compression device
    - Warfarin
    - Heparin (LMWH > unfractionated SC)
    - IVC filter (if DVT present)
- Elective surgery should be postponed in the 1st month after venous or arterial thromboembolism
- If postponement is not possible, patient should receive preoperative heparin while the INR is below 2.0
- Ideally, 3 months of anticoagulation is recommended before elective surgery
- Withholding warfarin for 5 days will allow the PT/INR to fall to normal if the INR is chronically maintained between 2.0 and 3.0 (the usual therapeutic targets)

ANESTHETIC OPTIONS

- GETA

ANESTHETIC SETUP

- **Drugs**
    - Resuscitation medications including norepinephrine, phenylephrine +/- dobutamine, milrinone, dopamine, vasopressin
- **Equipment**
    - Standard CAS & 5-lead ECG
    - Pre-induction art-line, +/- CVP / PAC
    - Intraoperative TEE: recommended for assessment of RV dysfunction while weaning from bypass & visualization of mobile emboli in right heart or PA (determines whether exploration necessary)
    - Perfusionist present & CPB / ECLS set-up for crash
    - Patient prepped and draped, surgeon ready
    - Consider secondary anesthesiologist

MANAGEMENT OF ANESTHESIA

- **Induction**
    - CV collapse on induction is common & HD instability occurs d/t:
        - Vasodilatation in the presence of a fixed CO & blunting of catecholamine drive
        - Mechanical ventilation decreases venous return & increases PVR
    - Etomidate is agent of choice - use caution with all other agents including ketamine (increased PAP)
- **Maintenance**
    - Avoid PEEP (decreased VR) and N_2O (increases PVR)
    - Maintain goals, likely need inotropic / vasopressor support along with minimal anesthetic
    - Maintain adequate preload which is essential for RV function
- **Emergence**
    - Usually keep intubated

DISPOSITION & MONITORING

- All patients should go to ICU postoperatively

IMPLICATIONS IN PREGNANCY

- PTE occurs in approximately 0.01% to 0.05% of all pregnancies
- ~15-24% of pregnant women with untreated DVT experience PTE with assoc mortality of 12-15%
- ~10% of all patients with PTE die within the first hour
- Pregnancy results in 2-5x ↑ relative risk of thromboembolism due to:
    - ↑ venous stasis
    - hypercoagulable state of pregnancy
    - vascular injury assoc with vaginal or C/S delivery
- Risks of DVT & PTE are as much as 8x higher after C/S delivery than after vaginal delivery
- Obstetric conditions & coincidental disease also plays a role to ↑ risk:
    - Pre-eclampsia, multiple gestation, heart disease, smoking, obesity
    - Antiphospholipid Ab syndrome, protein S & C deficiencies, AT III deficiency
    - Hyperhomocysteinemia, prothrombin gene or Factor V Leiden mutation
- Goals are to diagnose PTE early & minimize radiation exposure for fetus & mother
- Spiral CT involves less radiation exposure to fetus than V/Q scan during all trimesters
- Can minimize radiation by using half-dose radionucleotide protocols for V/Q scans & special shielding for CT
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- Treatment:
    - Unfractionated Heparin bolus 80-150 U/kg then infusion 15-25 U/kg/hr to keep PTT 2x normal

- Consider IVC filter or caval interruption in pts who can't be anticoag or suffer recurrent emboli
- Thrombolytic in massive PTE – Urokinase, Streptokinase, rt-PA have been used safely
  - Urokinase is less antigenic with fewer side effects
- Surgical embolectomy in extreme cases of rapidly deteriorating patient

- Follow ASRA Guidelines for neuraxial anesthesia in the anticoagulated patient

PATHOPHYSIOLOGY

- **Fatal pulmonary embolism** occurs in 0.1% to 0.8% of patients undergoing elective general surgery, 2% to 3% of patients having elective hip replacement, and 4% to 7% of patients undergoing repair of a fractured hip who do not receive prophylaxis (Miller 7th ed)
- **MPE** = a PE that results in hemodynamic instability = 3x - 7x increase in mortality
- ⅔ of MPE die within 1 hr of presentation:
  - 50% d/t massive embolus & 50% d/t recurrent emboli
  - If shock = 1st presentation = 30% mortality
  - If cardiac arrest = 1st presentation = 70% mortality
- **Causes**:
  - 95% come from proximal DVT
  - 5% come from fat, air, cement, AFE, or tumor
- **Risk factors** = remember Virchow's Triad:
  - Venous stasis, endothelial injury, hypercoagulable state
- **Outcome** is a function of the size of the embolus & the patient's underlying cardiopulmonary function
- HD instability d/t RV failure as a function of embolus size & cardiopulmonary dysfunction is more accurate indicator of severity than degree of angiographic obstruction (which measures PE size only)
- **Pathophysiology** = vicious circling the drain with multiple interactions
  - Increased RV impedance occurs d/t mechanical obstruction & pulmonary vasoconstriction (neural reflexes, humoral factors, & HPV)
  - Hypoxemia results from increased dead space, L-to-R intrapulmonary shunt, V/Q mismatching, & venous admixture (it can actually worsen after treatment d/t atelectasis)
  - Clinically results in RV pressure overload, then RV dysfunction from ischemia results in decreased RV CO & increased RV volume
  - This leads to septal shift & pericardial restriction & decreased LV preload resulting in systemic hypotension & worsened coronary perfusion and O_2 supply demand ratio

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