Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Pulmonary Embolism

Objectives :

1. Epidemiology
2. Pathophysiology
3. Diagnosis
4. Massive PE
5. Treatment

Done by :

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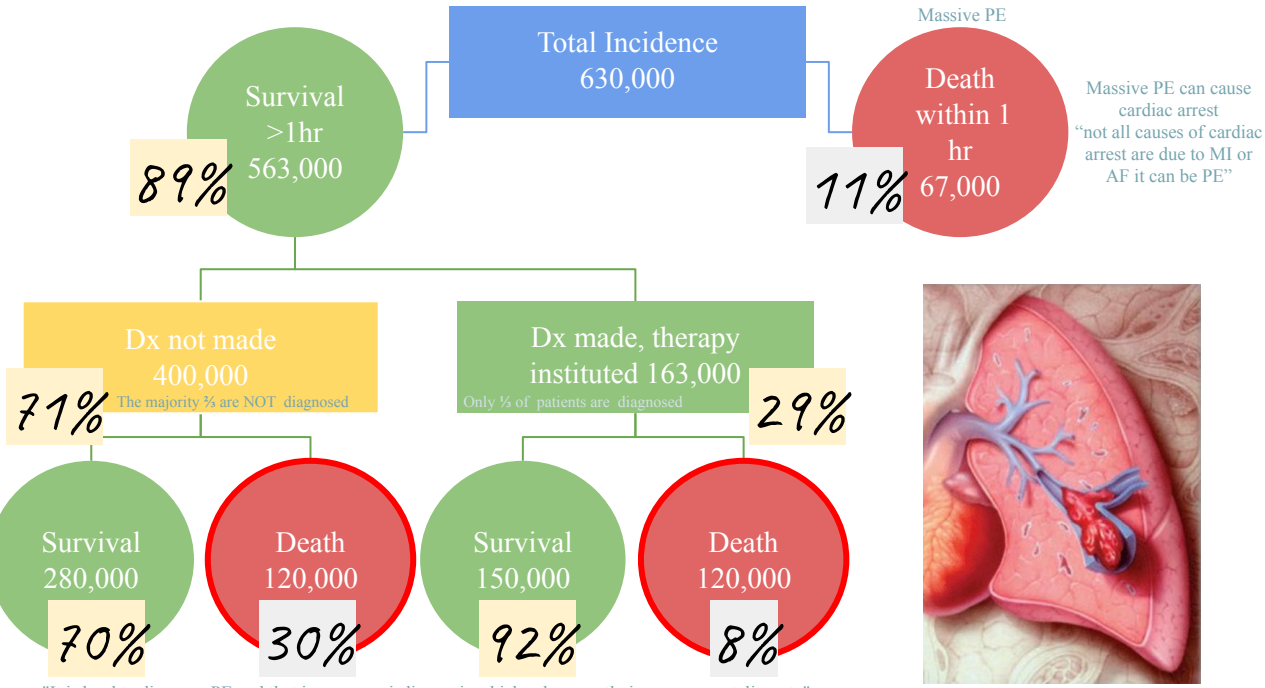
Resources :

- Dr. Ahmed Bahammam slides & notes
- Team 436 (Davidson & Kumar)

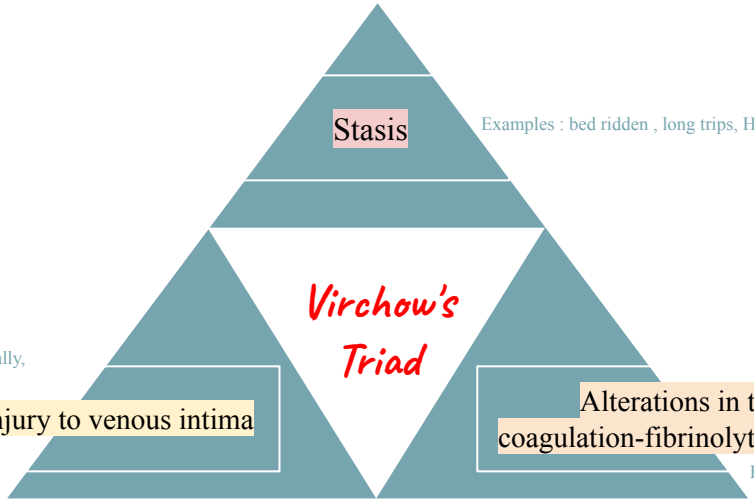
Epidemiology

- 50,000 individuals die from PE each year in USA
- The incidence of PE in USA is 500,000 per year
- Over 317,000 deaths were related to VTE Venous thromboembolism in six countries of the European Union (with a total population of 454.4 million) in 2004
  - **Of these cases,**
    - ➔ 34% presented with sudden fatal PE
    - ➔ 59% were deaths resulting from PE that remained undiagnosed
    - ➔ Only 7% of the patients who died early were correctly diagnosed with PE before death.



"It is hard to diagnose PE and that increases misdiagnosis which subsequently increases mortality rate"

Risk factors for venous thrombosis



1-It can happen physiologically, as in pregnancy the risk of thrombosis increases.
2- Protein C deficiency

Risk factor for venous thrombosis

Source of emboli

- Deep venous thrombosis (>95%)
- Other veins:
  - Renal
  - Uterine
  - Right cardiac chambers

This is important

Risk factors for DVT

1. General anesthesia
2. Lower limb or pelvic injury or surgery
3. Congestive heart failure
4. **Prolonged immobility**
5. Pregnancy
6. Postpartum
7. Oral contraceptive pills
8. Malignancy
9. Obesity
10. Advanced age
11. Coagulation problems (protein C and S deficiency, Antithrombin III deficiency , factor V leiden)

Clinical features

These are non-specific for PE but when you have them + Risk factors then you should suspect PE

1. **Sudden onset dyspnea**
2. Pleuritic chest pain
3. Hemoptysis

Only with infraction

- Clinical clues cannot make the diagnosis of PE; their main value lies in suggesting the diagnosis.

Signs or symptoms observed in patients with thromboembolism

Keep in mind:absence of DVT does NOT exclude PE! . sometimes the whole clot (instead of parts of it) travels to the lungs. In this case, **the legs won't show any sign of thrombosis when examined.**

		Study	
		Stein et al., % (n= 117)	Anderson et al., % (n= 131)
Deep vein thrombosis	Swelling	28	88*
	Pain	26	56
	Tenderness	—	55
	Warmth	—	42
	Redness	—	34
	Homan's sign	4	13
	Palpable cord	—	6

This study is for your interest

Table 3 Clinical characteristics of patients with suspected PE in the emergency department (adapted from Pollack *et al.* (2011)).[82]

Feature	PE confirmed (*n* = 1880)	PE not confirmed (*n* = 528)
Dyspnoea	50%	51%
Pleuritic chest pain	39%	28%
Cough	23%	23%
Substernal chest pain	15%	17%
Fever	10%	10%
Haemoptysis	8%	4%
Syncope	6%	6%
Unilateral leg pain	6%	5%
Signs of DVT (unilateral extremity swelling)	24%	18%

DVT = deep vein thrombosis.

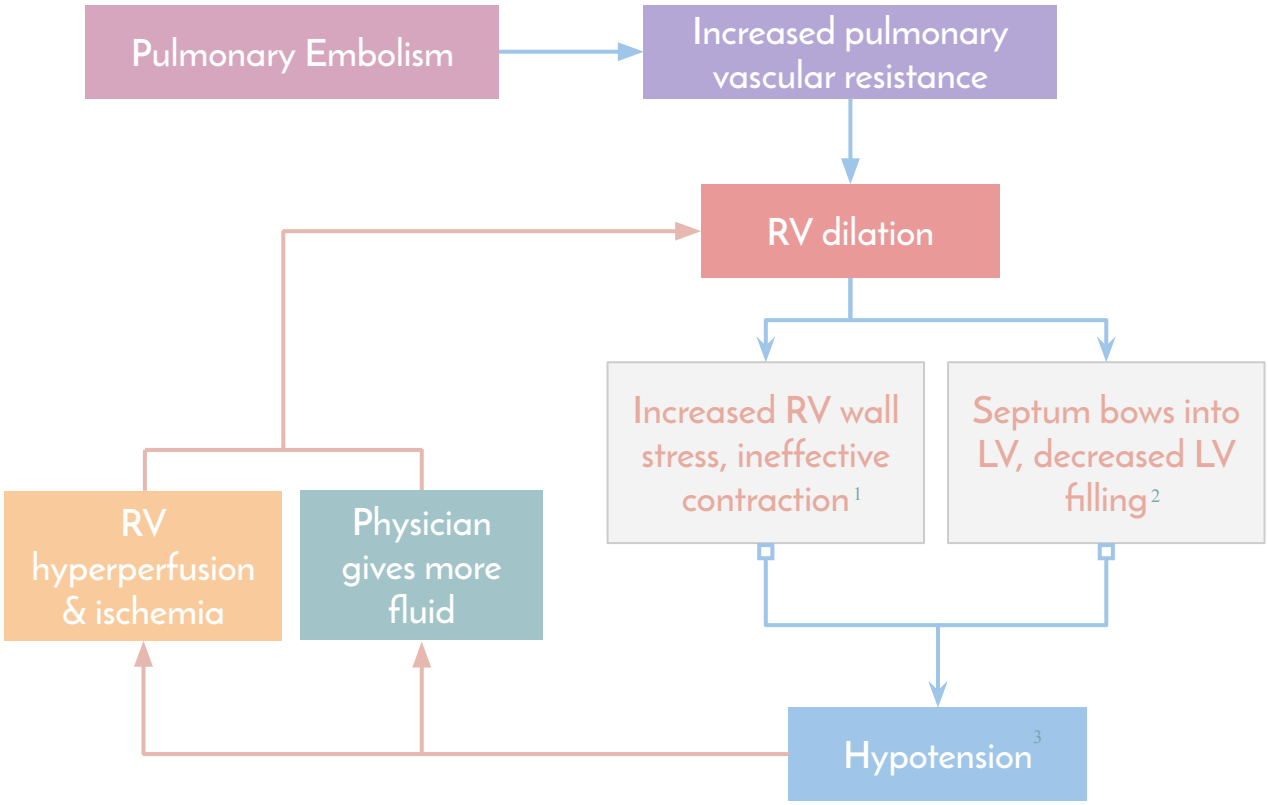
The point of this table is to tell you that you can't rule in/out PE based on clinical features

Massive Pulmonary Embolism:

Massive means major hemodynamic effect (shock & hypotension), not reserved to the size

- It is a catastrophic entity which often results in acute right ventricular failure and death.
- Frequently undiscovered until autopsy.
- Fatal PE typically leads to death within one to two hours of the event.

Pulmonary Embolism Death Spiral:

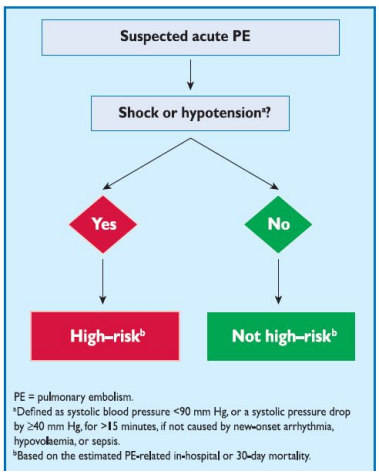


1- This will decrease blood supply which lead to ischemia
2- Dilation of RV with septum bowing will lead to decrease in LV size → ↓EDV→ ↓SV→ ↓CO, Eventually to Shock and it is called Obstructive Shock
3- if we will give patient fluid it will increase RV pressure leading to dilation and that will worsen symptoms

Pathophysiology:

- Massive PE causes an increase in PVR → right ventricular outflow obstruction → decrease left ventricular preload → Decrease CO.
- In patients without cardiopulmonary disease, occlusion of 25-30 % of the vascular bed → increase in Pulmonary artery pressure (PAP).
- Hypoxemia ensues → stimulating vasoconstriction → increase in PAP.
- More than 50% of the vascular bed has to be occluded before PAP becomes substantially elevated.
- When obstruction approaches 75%, the RV must generate systolic pressure in excess of 50mmHg to preserve pulmonary circulation.
- The normal RV is unable to accomplish this acutely and eventually fails.

In cor pulmonale there is pulmonary hypertension but the increase in pressure is chronic and gradual so the RV can accommodate. Also, the right ventricle is a volume chamber not a pressure chamber so it will not be able to overcome high pressure which will lead to its failure.



Diagnosis:

- ECG
- CXR
- ABG
- D-dimer
- Spiral CT
- V/Q
- Echo
- Angio

- The diagnosis of massive PE should be explored whenever oxygenation or hemodynamic parameters are severely compromised without explanation

D-dimer:

An elevated D-dimer is of limited value, as it may be raised in a variety of conditions including PE. If negative, you can rule out a clot/PE. but if it is positive, this does not help you.

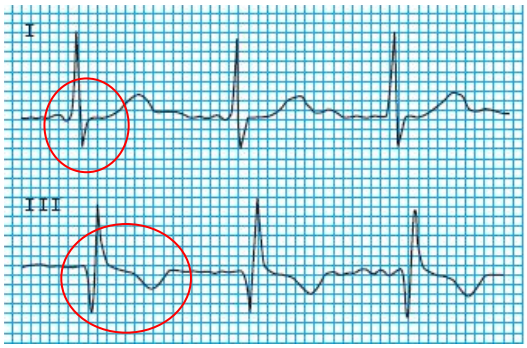
ABG:

- Significant hypoxemia is almost uniformly present when there is a hemodynamically significant PE

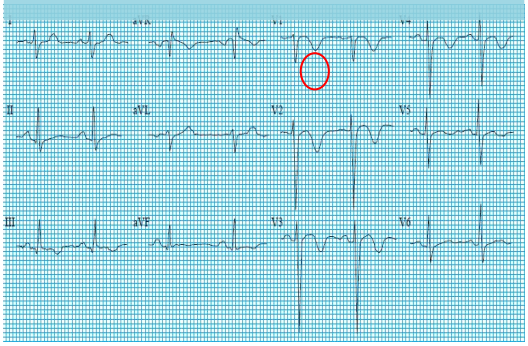
Low Oxygen sat on 100% O2 mask (Massive PE)

ECG: May suggest , but not diagnostic

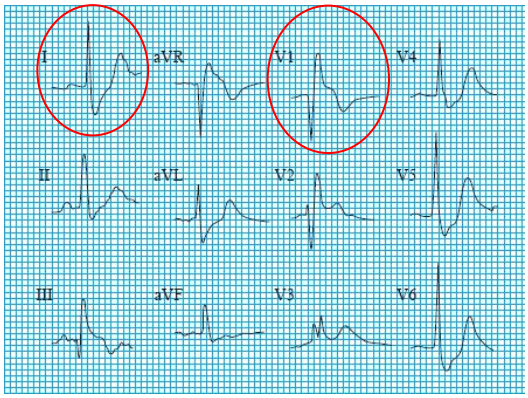
S1 Q3 T3 Pattern



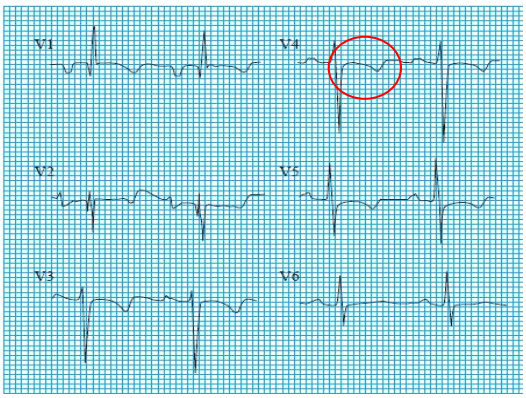
T-wave inversion



Rt. Bundle Branch Block

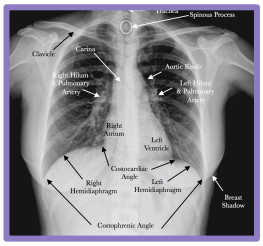


Rt. Ventricular Strain

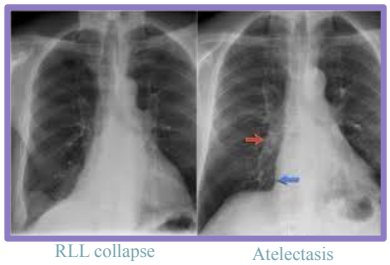


Chest X-ray

You will see
1. Atelectasis (may cause Raised hemidiaphragm)
2. Pleural effusion
3. Pleural based opacity

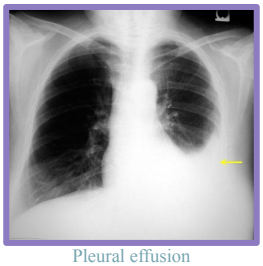


Normal


RLL collapse

Atelectasis

Collapse at atelectasis is due to decrease blood supply so the surfactant will decrease. If this happen to a small part it's atelectasis, if a full lobe it's collapse.


Pleural effusion

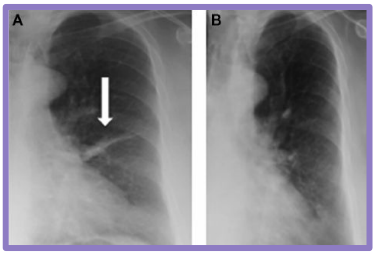
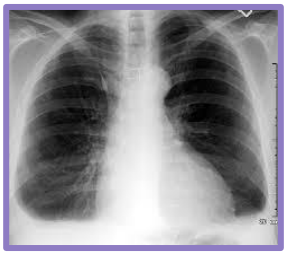
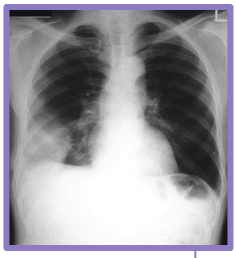


Plate atelectasis


Atelectasis

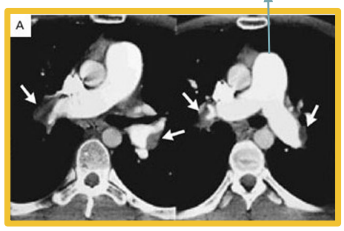

Bilateral pleural effusion

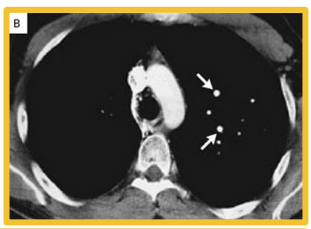
Chest radiograph showing Wedge shaped pulmonary infarct in right lower lobe

Spiral CT

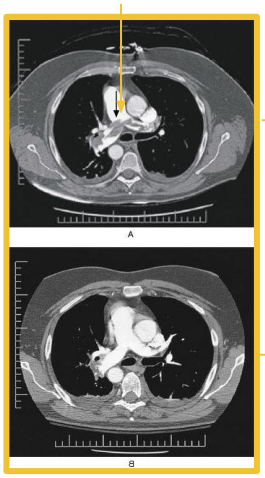
Has replaced V/Q scan.

Pulmonary artery larger than the aorta



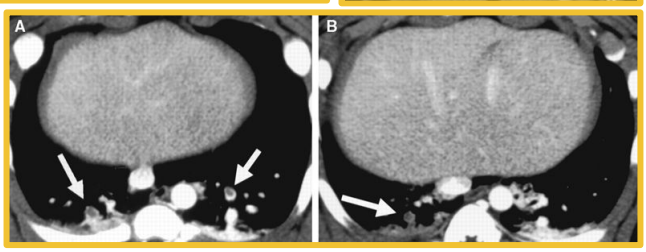


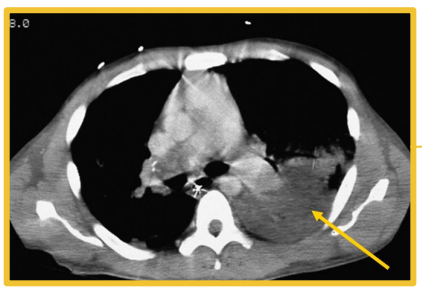
Saddle clot



Before

After

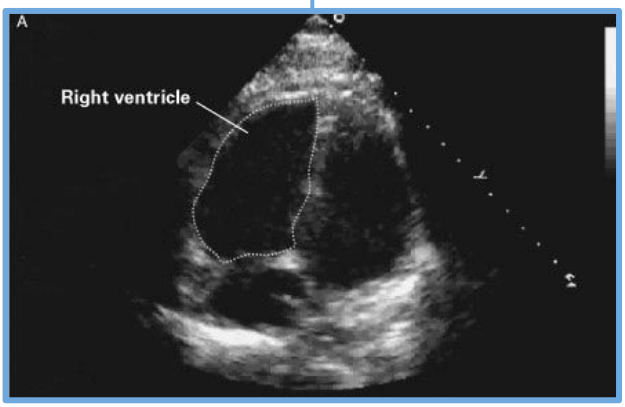



Tomographic scan showing infarcted left lung, large clot in right main pulmonary artery

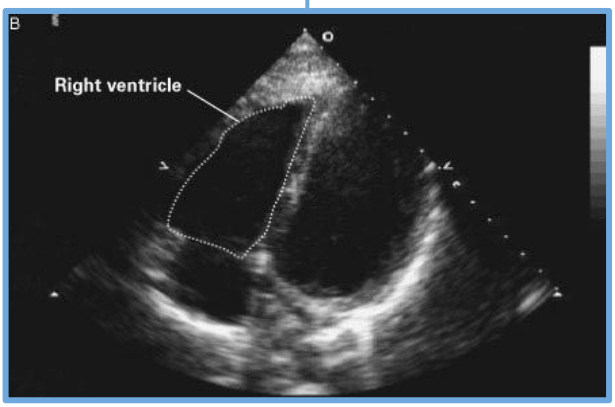
- **Computed tomographic pulmonary angiography (Spiral CT):** Data suggest that a negative Spiral CT is an adequate criterion for excluding PE in patients with a non-high clinical probability of PE. The first-line diagnostic test

Echo:

Before



After



What happens here is that when the volume increases in the RV, this will cause shifting of the septum towards the LV resulting in shrinking the size of the chamber so that's why it looks small on Echo; observe the difference after the treatment.

Chest radiographic findings in patients PE:

	COPD, %(n=21)	No prior cardiopulmonary disease, %(n=117)
Atelectasis or pulmonary parenchymal abnormality	76	68
Pleural effusion	52	48
Pleural-based opacity	33	35
Elevated diaphragm	14	24
Decreased pulmonary vascularity	38	21
Prominent central pulmonary artery	29	15
Cardiomegaly	19	12
Westermark's sign*	5	7
Pulmonary edema	14	4

This part was skipped by dr. Bahammam. More details about this test are discussed in Lung investigations lecture

The use of ventilation perfusion scan in diagnosing pulmonary embolism

For the results to be useful we need a healthy lung to start with, if a patient is already diagnosed with COPD the lung is destroyed so the V/Q is not helpful

High probability

VQ is diagnostic, Confirms PE
Start treatment

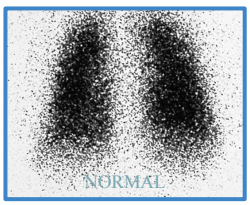
- =2 large segmental (>75% of a segment) perfusion defects without corresponding ventilation or radiographic abnormalities or substantially larger than matching ventilation or radiologic abnormalities.

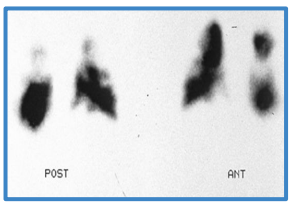
OR

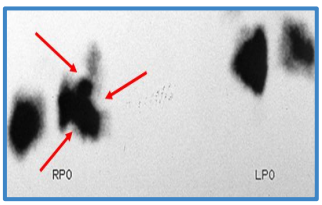
- =2 moderate segmental (>25% and <75% of a segment) perfusion defects without matching ventilation or chest radiographic abnormalities plus one large unmatched segmental defect.

OR

- =4 moderate segmental perfusion defects without matching ventilation or chest radiologic abnormalities.







Intermediate probability

Scans that do not fall into normal, very low, low, or high probability categories.

Low probability

If there is low or intermediate probability,we need to do further tests:

- Non-Segmental perfusion defects.

OR

- Single moderate mismatched segmental perfusion defect with normal chest radiograph/

OR

- Any perfusion defect with a substantially larger abnormality on chest radiograph.

OR

- Large or moderate segmental perfusion defects involving no more than four segments in one lung and no more than three segments in one lung region with matching or larger ventilation/radiographic abnormalities.

OR

- More than three small segmental perfusion defects (<25% of a segment) with a normal chest radiograph.

Very low probability

A NORMAL V/Q scan rules out PE, no further testing is needed.

- Three or fewer small segmental perfusion defects with a normal chest radiograph.
- Normal.
- No perfusion defects present.

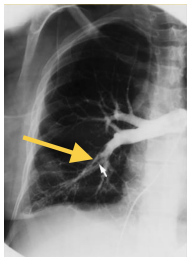
Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) results:

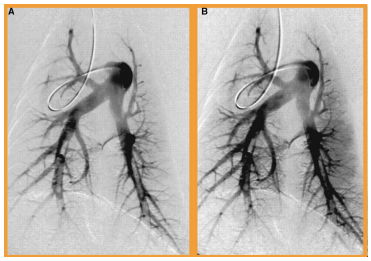
Prospective investigation of pulmonary embolism diagnosis results					
Scan category	PE present	PE absent	PE uncertain	No angiogram	Total
High probability In VQ it means he had PE & you should treat	102	14	1	7	124
Intermediate probability	105	217	9	33	364
Low probability	39	199	12	62	312
Near normal or normal No PE	5	50	2	74	131
Total	251	480	24	176	931

CT tells you if this PE or not, here it can tell you it's Intermediate or low, which is useless

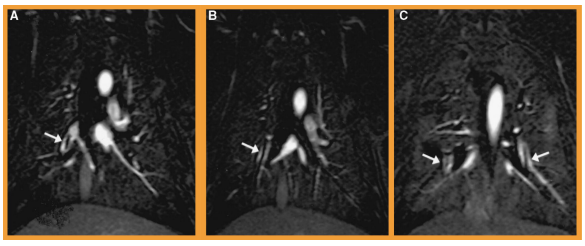
Pulmonary angiogram:

Wedge shaped
Dual blood supply,
elevated diaphragm



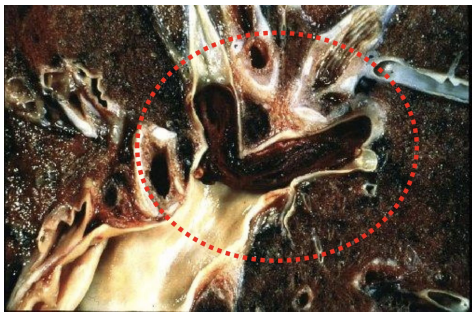


MRA with contrast:



All of the three above are NOT used anymore

Pulmonary Embolism:



Dosage and monitoring of anticoagulant therapy:

- Anticoagulation should be commenced immediately in patients suspected with PE
- We start with HEPARIN (either unfractionated or low-molecular-weight) + warfarin
- After initiating heparin therapy, repeat APTT every 6 h for first 24 h and then every 24 h when therapeutic APTT is achieved
- Warfarin 5 mg/d can be started on day 1 of therapy; there is no benefit from higher starting doses
- Platelet count should be monitored at least every 3 d during initial heparin therapy
- Heparin is usually continued for 5–7 d
- Heparin can be stopped after 4–5 d of warfarin therapy when INR is in 2.0–3.0 range
- Warfarin is continued for 6 weeks to 6 months (Regular measurement of the INR is required throughout the duration) Why?
    - narrow therapeutic index of warfarin
    - its propensity to interact with other drugs and food.
- We start the patient on both heparin and warfarin, because heparin has fast onset and will provide immediate results while the warfarin will take 3 to 4 days to start it effect. And then when we measure the INR and it's in the therapeutic range we stop the heparin and continue with the warfarin.

Important drug interactions with warfarin

Skipped by the doctor

Drugs that decrease warfarin requirement	*Drugs that increase warfarin requirement*
Phenylbutazone Metronidazole Trimethoprim-sulfamethoxazole Amiodarone Second- and third-generation cephalosporins Clofibrate Erythromycin Anabolic steroids Thyroxine	Barbiturates Carbamazepine Rifampin Penicillin Griseofulvin Cholestyramine

Complications of anticoagulation:

Heparin

Complication	Management
Bleeding	Stop heparin infusion. For severe bleeding, the anticoagulant effect of heparin can be reversed with intravenous protamine sulfate 1 mg/100 units of heparin bolus or 0.5 mg for the number of units given by constant infusion over the past hour; provide supportive care including transfusion and clot evacuation from closed body cavities as needed.
Heparin-induced thrombocytopenia and thrombosis	Carefully monitor platelet count during therapy. Stop-heparin for platelet counts <75,000. Replace heparin with direct inhibitors of thrombin like desirudin if necessary. These agents do not cause heparin-induced thrombocytopenia. Avoid platelet transfusion because of the risk for thrombosis.
Heparin-induced osteoporosis (therapy >1 mo)	LMWHs may have lower propensity to cause osteoporosis as compared with unfractionated heparin; consider LMWH if prolonged heparin therapy is necessary.

Warfarin

Complication	Management
Bleeding	Stop therapy. Administer vitamin K and fresh frozen plasma for severe bleeding; provide supportive care including transfusion and clot evacuation from closed body cavities as needed
Skin necrosis (rare)	Supportive care.
Teratogenicity	Do not use in pregnancy or in patients planning to become pregnant.

These four drugs are used nowadays for 6 months.

Table 11 Overview of phase III clinical trials with non-vitamin K-dependent new oral anticoagulants (NOACs) for the acute-phase treatment and standard duration of anticoagulation after VTE

Drug	Trial	Design	Treatments and dosage	Duration	Patients	Efficacy outcome (results)	Safety outcome (results)
Dabigatran		Double-blind, double-dummy	Enoxaparin/dabigatran (150 mg b.i.d.)[a] vs. enoxaparin/warfarin	6 months	2539 patients with acute VTE	Recurrent VTE or fatal PE: 2.4% under dabigatran vs. 2.1% under warfarin	Major bleeding: 1.6% under dabigatran vs. 1.9% under warfarin
	RE-COVER II[94]	Double-blind, double-dummy	Enoxaparin/dabigatran (150 mg b.i.d.)[a] vs. enoxaparin/warfarin	6 months	2589 patients with acute VTE	Recurrent VTE or fatal PE: 2.3% under dabigatran vs. 2.2% under warfarin	Major bleeding: 15 patients under dabigatran vs. 22 patients under warfarin
Rivaroxaban		Open-label	Rivaroxaban (15 mg b.i.d. for 3 weeks, then 20 mg o.d.) vs. enoxaparin/warfarin	3, 6, or 12 months	3449 patients with acute DVT	Recurrent VTE or fatal PE: 2.1% under rivaroxaban vs. 3.0% under warfarin	Major or CRNM bleeding: 8.1% under rivaroxaban vs. 8.1% under warfarin
	EINSTEIN-PE[96]	Open-label	Rivaroxaban (15 mg b.i.d. for 3 weeks, then 20 mg o.d.) vs. enoxaparin/warfarin	3, 6, or 12 months	4832 patients with acute PE	Recurrent VTE or fatal PE: 2.1% under rivaroxaban vs. 1.8% under warfarin	Major or CRNM bleeding: 10.3% under rivaroxaban vs. 11.4% under warfarin
Apixaban		Double-blind, double-dummy	Apixaban (10 mg b.i.d. for 7 days, then 5 mg b.i.d.) vs. enoxaparin/warfarin	6 months	5395 patients with acute DVT and/or PE	Recurrent VTE or fatal PE: 2.3% under apixaban vs. 2.7% under warfarin	Major bleeding: 0.6% under apixaban vs. 1.8% under warfarin
Endoxaban		Double-blind, double-dummy	LMWH/edoxaban (60 mg o.d.; 30 mg o.d. if creatinine clearance 30–60 ml/min or body weight <60 kg) vs. UFH or LMWH/warfarin	Variable, 3–12 months	8240 patients with acute DVT and/or PE	Recurrent VTE or fatal PE: 3.2% under edoxaban vs. 3.5% under warfarin	Major or CRNM bleeding: 8.5% under edoxaban vs. 10.3% under warfarin

b.i.d. = bis in die (twice daily); CRNM = clinically relevant non-major; DVT= deep vein thrombosis; o.d. = omni die (once daily); PE= pulmonary embolism; UFH = unfractionated heparin; VTE = venous thromboembolism.

Approved thrombolytics for pulmonary embolism:

- We only give thrombolytic to patients with MASSIVE PE & SHOCK.
- **Recombinant tissue-plasminogen activator** 100 mg as a continuous peripheral intravenous infusion administered over 2 h
- **Streptokinase** 250,000 IU as loading dose over 30 min, followed by 100,000 U/h for 24 h
- **Urokinase** 4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h for 12-24 h

Indications for thrombolytic therapy in pulmonary embolism: (Signs of massive PE)

- **Hemodynamic instability** shock and hypotension
- Hypoxia on 100% oxygen
- Right ventricular dysfunction by echocardiography

Contraindications: Skipped by the doctor

Relative	Absolute
Recent surgery within last 10 d Previous arterial punctures within 10 d Neurosurgery within 6 mo Bleeding disorder (thrombocytopenia, renal failure, liver failure) Ophthalmologic surgery within 6 wk Hypertension >200 mm Hg systolic or 110 mm Hg diastolic Placement of central venous catheter within 48 h Hypertensive retinopathy with hemorrhages or exudates Intracerebral aneurysm or malignancy Cardiopulmonary resuscitation within 2 wk Cerebrovascular disease Major internal bleeding within the last 6 mo Pregnancy and the 1st 10 d postpartum Infectious endocarditis Severe trauma within 2 mo Pericarditis	Active internal bleeding

Other Treatment Modalities:

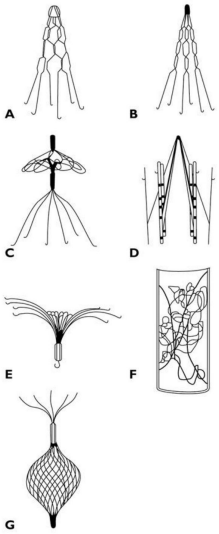
If thrombolytics have failed, this is rescue therapy

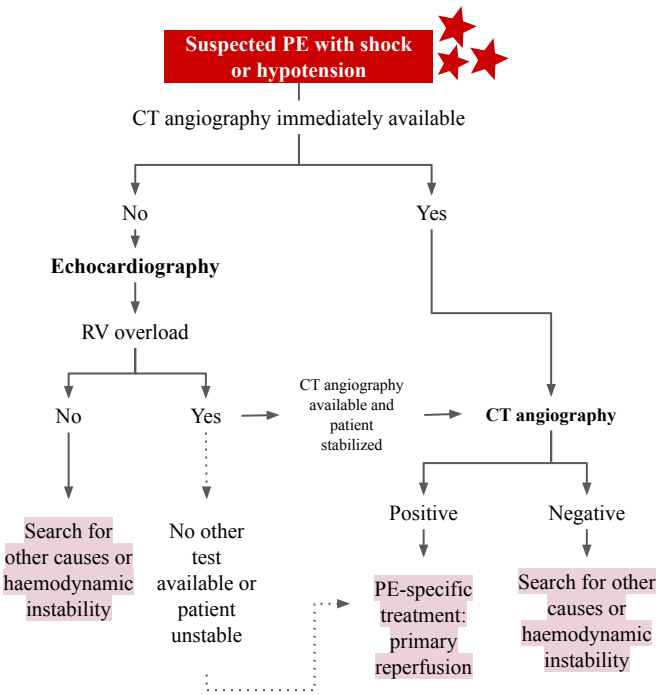
- Surgical embolectomy
- Percutaneous catheter-directed treatment

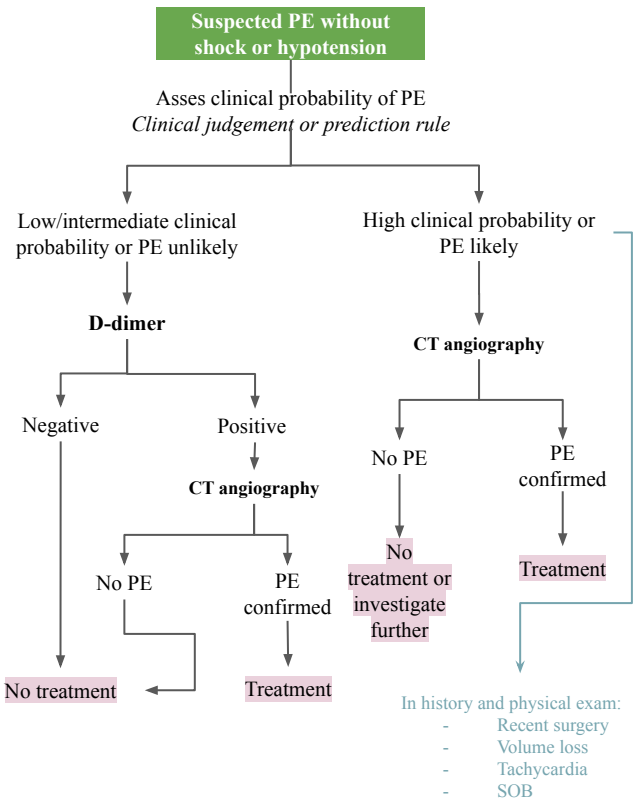
Indications for inferior vena caval (IVC) filters

Doctor said this is not used anymore

- Anticoagulation contraindicated (eg, patients with multiple trauma, active bleeding)
- Failure of antithrombotic therapy
- Complications from anticoagulant therapy preclude further use
- Prophylaxis against embolism from preexisting deep vein thrombosis in patients with poor cardiopulmonary reserve
- Prophylaxis against embolism in patients at high risk to develop deep vein thrombosis
- Patients with recurrent pulmonary embolism undergoing thromboendarterectomy







Conclusions:

- PE is common and under-recognized serious medical problem
- Early diagnosis and treatment is essential for good outcome
- High index of suspicion is needed in high risk patients

Summary

PE is a medical emergency!
Early diagnosis and management is crucial for reducing mortality

Risk factors for DVT and PE include:

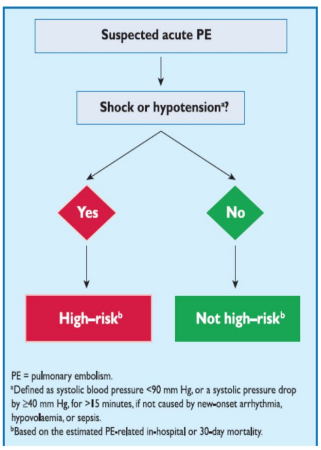
Stasis	Injury to venous intima	Coagulation problems
General anesthesia	Lower limb/ pelvic injury or surgery	Congestive Heart Failure
Prolonged immobility	Pregnancy/ Postpartum	Oral contraceptive pill
Advanced age	Malignancy	Obesity

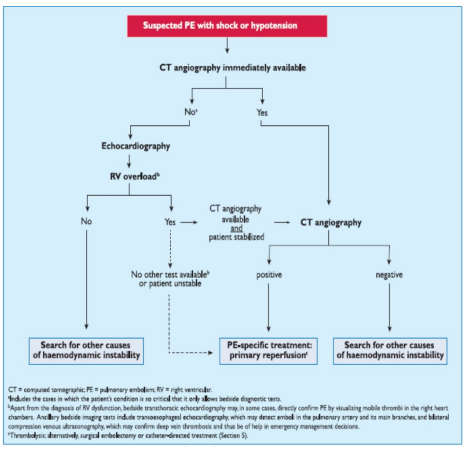
Clinical Feature:

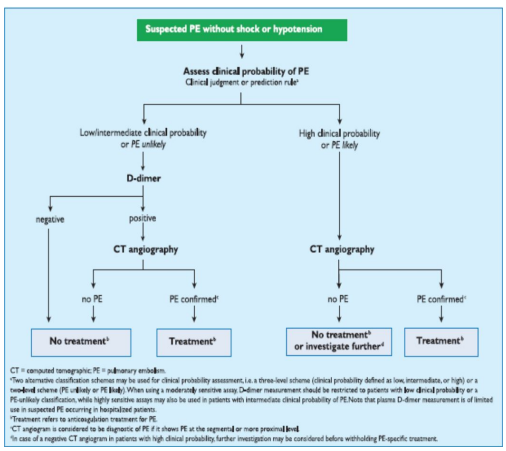
- Sudden onset dyspnea
- Pleuritic chest pain
- Hemoptysis

Investigations:

- Fist step is to determine whether the patient is stable or not
- The following investigative guidelines should be followed based on patient risk







Diagnosis can only be achieved by **Spiral CT**

Pharmacological Treatment:

Normal PE	Massive PE
• Heparin • Warfarin • NOACs (eg. Dabigatran, Rivaroxaban)	• Recombinant tissue-plasminogen activator • Streptokinase • Urokinase

Questions

1. **Which of the following is the best diagnostic test for pulmonary embolism?**
   A. V/Q Scan
   B. Spiral CT
   C. CXR
   D. D-dimer

2. **In which type of P.E are thrombolytics indicated?**
   A. Massive P.E
   B. Acute small P.E
   C. Acute medium P.E
   D. Chronic P.E

3. **Which of the following is a risk factor for P.E?**
   A. Local anesthesia
   B. Congestive heart failure
   C. Infective endocarditis
   D. Analgesics

4. **Patient presents to the ER complaining of dyspnea, chest pain and mild hemoptysis. HR and BP are normal, D-dimer was positive and CT confirmed pulmonary embolism. What is the appropriate next step in management?**
   A. Give O2 and I.V steroids
   B. Give analgesic and discharge
   C. Start patient on heparin
   D. Take for emergency cardiac surgery

5. **A pregnant lady presented to the ER with mild chest pain and was diagnosed to have P.E. Which of the following medications is contraindicated for her?**
   A. Warfarin
   B. Heparin
   C. Aspirin
   D. Paracetamol

6. **A patient complained of mild chest pain, and shortness of breathing during inspiration. He was diagnosed with PE. What is the most likely source of the embolus?**
   A. Renal arteries
   B. Upper extremities
   C. Lower extremities
   D. Axilla

7. **Which of the following have the greatest risk for PE?**
   A. DVT above the knee.
   B. DVT below the knee.
   C. Renal artery thrombus.
   D. Normal delivery in healthy woman**.**

Questions

8. A lady in her late 50s is having recurrent PE for the last 18 months. Which of the following is most probably true about this patient.

A. She's on oral contraceptives to control pregnancy.
B. Recurrent PE is due to her advanced age.
C. Recent lower limb injury with major surgery fixation.
D. She could have malignancy somewhere.

9. A 29-year-old male known diabetes and dyslipidemia. He presented to the ER with sudden SOB for the last 2 hours. Patient's body temperature is 40.2 and on physical examination there's track marks which suggest that the patient is a drug abuser. The patient was diagnosed with PE. What is the most likely source of the embolus?

A. Fat embolism due to long bone fracture.
B. DVT due to sedentary lifestyle
C. Air embolism due to trauma.
D. Septic embolism due to septicemia.

10. Which of the following is right regarding the treatment of PE?

A. Circulatory shock should be treated with inotropic agents.
B. All patients with PE should receive high flow oxygen.
C. Heparin is better than NOAC.
D. Warfarin can be stopped after 4-5 days of Heparin therapy initiation**.**

11. Which of the following is NOT true regarding PE?

A. Early diagnosis and treatment will not affect prognosis.
B. PE is common and under-recognized serious medical problem.
C. High index of suspicion is needed in high risk patients. Acute massive PE leads to hemodynamic instability.
D. DVT is the common source of embolus in PE

Answers:
1.B
2.A
3.B
4.C
5.A
6.C
7.A
8.D
9.D
10.B
11.A

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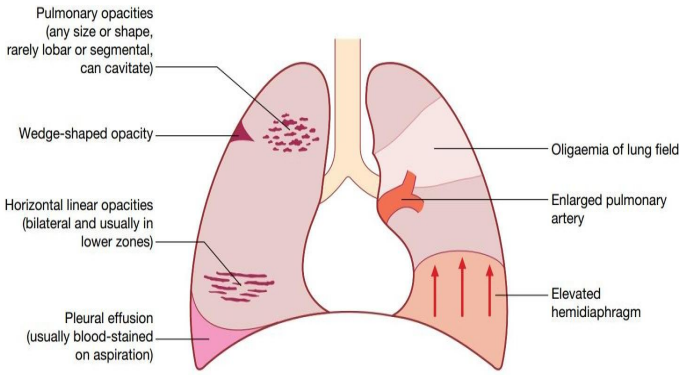

Fig. 19.67 Features of pulmonary thromboembolism/infarction on chest X-ray.

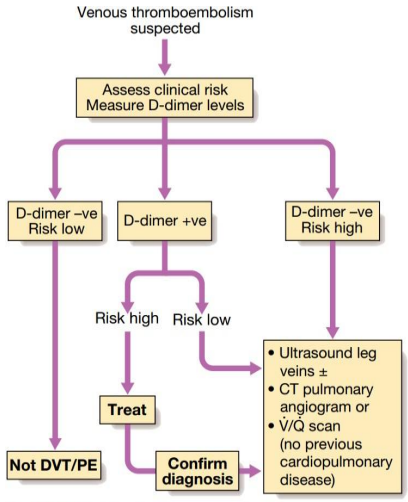

Fig. 19.68 **Algorithm for the investigation of patients with suspected pulmonary thromboembolism.** Clinical risk is based on the presence of risk factors for venous thromboembolism and the probability of another diagnosis.

Summary

1. Pulmonary embolism is a medical emergency
2. Diagnosis of DVT or PE is an indication for treatment
3. Lower extremities are the main source of emboli.
4. In severe cases acute cor pulmonale may result
5. Clinical symptoms become more overt as the size of dead space in lung increases.
6. Symptoms of PE are not specific.
7. Most often PE is silent.
8. Dyspnea, pleuritic chest pain , tachypnea are the most common manifestations.
9. Recurrences are common.
10. Spiral CT is the test of choice in diagnosing PE.
11. DVT is diagnosed by ultrasound and clinical suspicion.
12. V/Q scan plays an important role in diagnosing PE if spiral CT is contraindicated.
13. Pulmonary angiography can make a definite diagnosis but, it is invasive.
14. Start therapeutic heparin as initial treatment. Also start warfarin at the same time.

24.17 Factors predisposing to venous thrombosis

Patient factors

- Increasing age
- Obesity
- Varicose veins
- Previous DVT
- Family history, especially of unprovoked VTE when young
- Pregnancy/puerperium
- Oestrogen-containing oral contraceptives and HRT
- Immobility, e.g. long-distance travel (> 4 hrs)
- IV drug use (femoral vein)

Surgical conditions

- Major surgery, especially if > 30 mins' duration
- Abdominal or pelvic surgery, especially for cancer
- Major lower limb orthopaedic surgery, e.g. joint replacement and hip fracture surgery

Medical conditions

- Myocardial infarction/heart failure
- Inflammatory bowel disease
- Malignancy
- Nephrotic syndrome
- Pneumonia
- Neurological conditions associated with immobility, e.g. stroke, paraplegia, Guillain–Barré syndrome

Haematological disorders

- Polycythaemia rubra vera
- Essential thrombocythaemia
- Deficiency of anticoagulants: antithrombin, protein C, protein S
- Paroxysmal nocturnal haemoglobinuria
- Gain-of-function prothrombotic mutations: factor V Leiden, prothrombin gene *G20210A*
- Myelofibrosis

Antiphospholipid syndrome

- Lupus anticoagulant (more strongly associated with thrombosis than anticardiolipin antibodies)
- Anticardiolipin antibody

19.93 Features of pulmonary thromboemboli

	Acute massive PE	Acute small/medium PE	Chronic PE
Pathophysiology	Major haemodynamic effects: ↓cardiac output; acute right heart failure	Occlusion of segmental pulmonary artery → infarction ± effusion	Chronic occlusion of pulmonary microvasculature, right heart failure
Symptoms	Faintness or collapse, crushing central chest pain, apprehension, severe dyspnoea	Pleuritic chest pain, restricted breathing, haemoptysis	Exertional dyspnoea. Late symptoms of pulmonary hypertension or right heart failure
Signs	Major circulatory collapse: tachycardia, hypotension, ↑JVP, RV gallop rhythm, loud P_2, severe cyanosis, ↓urinary output	Tachycardia, pleural rub, raised hemidiaphragm, crackles, effusion (often blood-stained), low-grade fever	May be minimal early in disease. Later: RV heave, loud P_2. Terminal: signs of right heart failure
Chest X-ray	Usually normal. May be subtle oligaemia	Pleuropulmonary opacities, pleural effusion, linear shadows, raised hemidiaphragm	Enlarged pulmonary artery trunk, enlarged heart, prominent right ventricle
ECG	$S_1Q_3T_3$ anterior T-wave inversion, RBBB	Sinus tachycardia	RV hypertrophy and strain
Arterial blood gases	Markedly abnormal with ↓PaO_2 and ↓$PaCO_2$. Metabolic acidosis	May be normal or ↓PaO_2 or ↓$PaCO_2$	Exertional ↓PaO_2 or desaturation on formal exercise testing
Alternative diagnoses	Myocardial infarction, pericardial tamponade, aortic dissection	Pneumonia, pneumothorax, musculoskeletal chest pain	Other causes of pulmonary hypertension

(JVP = jugular venous pressure; PE = pulmonary embolism; RBBB = right bundle branch block; RV = right ventricular)

19.94 Risk factors for venous thromboembolism

Surgery

- Major abdominal/pelvic surgery
- Hip/knee surgery
- Post-operative intensive care

Obstetrics

- Pregnancy/puerperium

Cardiorespiratory disease

- COPD
- Congestive cardiac failure
- Other disabling disease

Lower limb problems

- Fracture
- Varicose veins
- Stroke/spinal cord injury

Malignant disease

- Abdominal/pelvic
- Advanced/metastatic
- Concurrent chemotherapy

Miscellaneous

- Increasing age
- Previous proven VTE
- Immobility
- Thrombotic disorders (p. 1054)
- Trauma