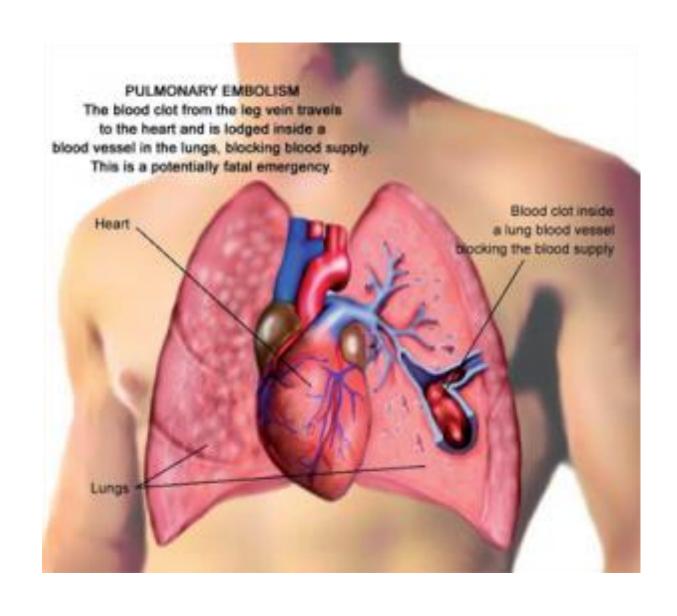
# Pulmonary embolism

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#### Venous thromboembolism:

- pulmonary embolism (PE)
- deep vein thrombosis (DVT)
- 1% of all patients admitted to hospital
- 5% of in-hospital mortality
- A common mode of death in patients with cancer, stroke and pregnancy.



# Pathophysiology of pulmonary embolism

- Ventilation-perfusion mismatch and ischaemia to the peripheral pulmonary lung tissues.
- Acute increase in pulmonary vascular resistance which increase right ventricular load and may reduce cardiac output.

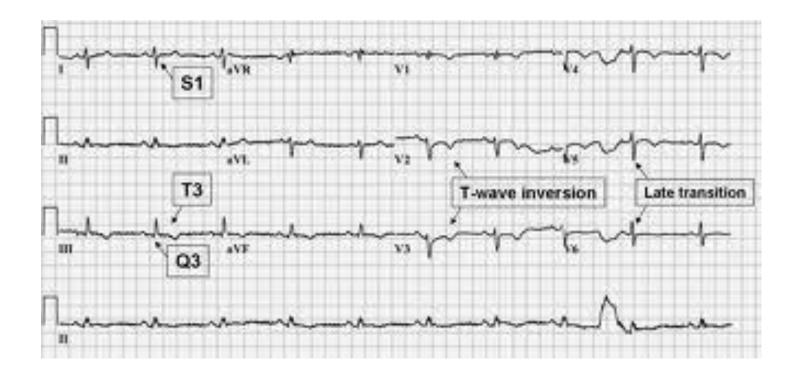
	Risk factors for pulmonary embolism
Surgery	<ul> <li>Major abdominal and pelvic surgery</li> <li>Hip and knee surgery</li> <li>Postoperative intensive care</li> </ul>
Obstetric	Pregnancy and puerperium
Cardiopulmonary	<ul><li>COPD</li><li>Congestive heart failure</li></ul>
Lowerlimb problem	<ul> <li>Fracture</li> <li>Stroke</li> <li>Spinal cord injury</li> <li>Varicose veins</li> </ul>
Malignancy	<ul><li>Abdominal and pelvic malignancy</li><li>Advanced/metastatic</li></ul>
Miscellaneous	<ul> <li>Increasing age</li> <li>Immobility</li> <li>previous VTE</li> <li>Thrombotic disorders</li> </ul>

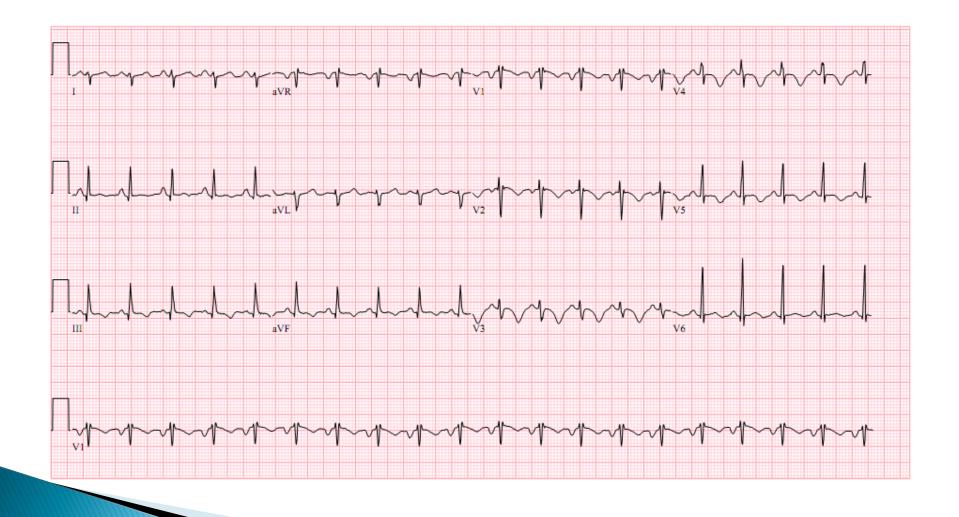
### Clinical features

- The clinical presentation varies depending on the number and size of the embolus and on the underlying cardiopulmonary reserve.
- It is possible to recognize three major categories of cases:
- Acute massive PE
- Acute small and medium PE
- Chronic thromboembolism

# Clinical features Acute massive PE

- A big thrombus obstructing a major pulmonary artery causing low cardiac output and acute right heart failure.
- Sudden collapse (fainting), crushing central chest pain and severe dyspnoea.
- Examination would reveal tachypnoea, tachycardia, hypotension (or cardiogenic shock), raised JVP and cyanosis.
- CXR is commonly normal (or show subtle oligaemia).
- ► ECG: tachycardia and may show S<sub>1</sub>Q<sub>3</sub>T<sub>3</sub>, anterior T-wave inversion or RBBB
- Arterial BGA: hypoxaemia and hypocapnea
- Differential diagnosis: acute myocardial infarction (MI), pericardial tamponade and aortic dissection.



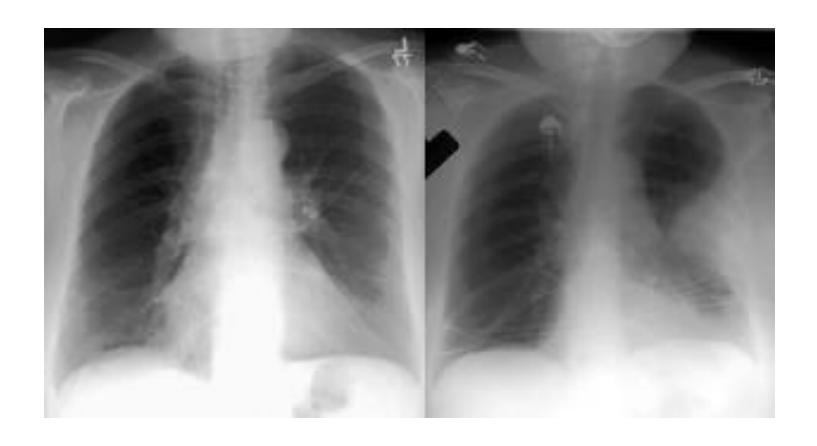


### **Clinical features**

#### Acute small or medium PE

- Occlusion of segmental pulmonary artery leading to pulmonary infarction with or without pleural effusion.
- Dyspnoea, pleuritic chest pain and haemoptysis.
- Tachycardia, low grade fever, but normal BP. Pleural rub or crackles can be heard. Dullness and diminished breathing at the lung base may indicate pleural effusion or elevated dome of the diaphragm.
- CXR may show opacities (of any size or shape); more specifically it may show horizontal linear opacities or wedge shaped pleural based opacities. Pleural effusion or raised hemidiaphragm may be noticed.
- **ECG**: sinus tachycardia
- Arterial BGA: normal or show mild hypoxaemia and hypocapnea
- sential diagnosis: pneumonia





# Clinical features

#### Chronic thromboembolism

- Chronic occlusion of pulmonary microvasculature caused by multiple small emboli or a sequel to previous organized thrombus, resulting in chronic pulmonary hypertension.
- The patient presents with exertional dyspnoea and features of right heart failure.
- ▶ ECG: right ventricular hypertrophy and strain.
- Arterial BGA: exertional hypoxaemia

# Consider the diagnosis of VTE in any patient presenting with:

- New or worsening dyspnoea
- Chest pain
- Sustained hypotension
   without alternative obvious cause
   particularly in patients who have risk factors

# **Diagnosis**

- Three questions should be in mind on facing a patient with suspected VTE:
- ▶ Is the clinical presentation consistent with PTE?
- Does the patient have risk factors?
- Any alternative diagnosis that can explain the presentation?
- The ECG may show tachycardia only, the CXR is commonly normal. <u>Both investigations are more useful in excluding other diagnosis</u> like MI (ECG), pneumonia and pneumothorax (CXR)

Well's clinical probability score for	
pulmonary embolism	
Clinical signs of deep vein thrombosis	+ 3
Alternative diagnosis less likely than pulmonary embolism	+ 3
Previous pulmonary embolism or deep vein thrombosis	+ 1.5
Heart rate >100 beats per minute	+ 1.5
Recent surgery or immobilization (within the last 30 d)	+ 1.5
Haemoptysis	+ 1
Cancer (treated within the last 6 mo)	+ 1
Clinical Probability of Pulmonary Embolism	score
Low	0-1
Intermediate	2-6
High	≥7

### **D-dimer**

- D-dimer is a specific degradation product of cross linked fibrin. Apart from PE, it also rises in MI, pneumonia and sepsis.
- Low D-dimer has a high negative predicted value and other investigations are unnecessary if the clinical probability is low.
- D-dimer is not useful in intermediate and high risk patients because a further investigation is mandatory even if it is normal

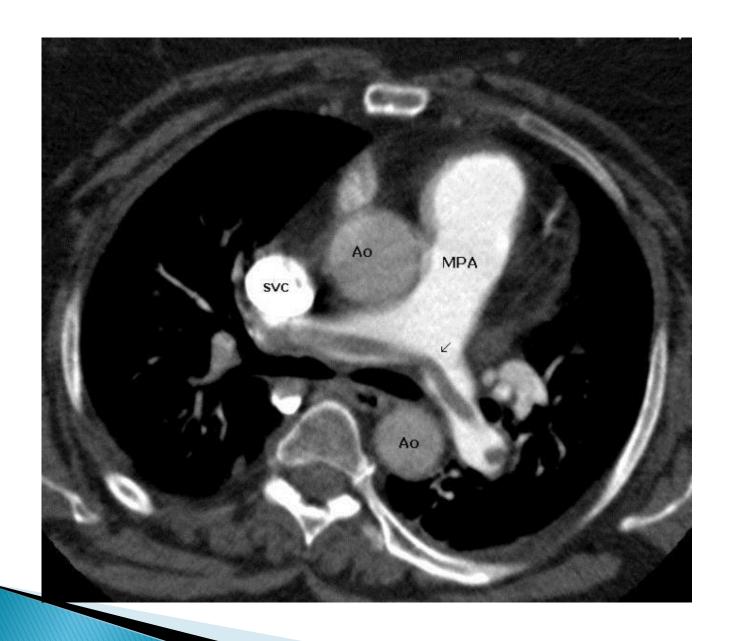
# **Imaging**

- CT pulmonary angiography (CTPA)
- Ventilation-perfusion scan (V/Q scan)
- Colour Doppler ultrasound of the leg

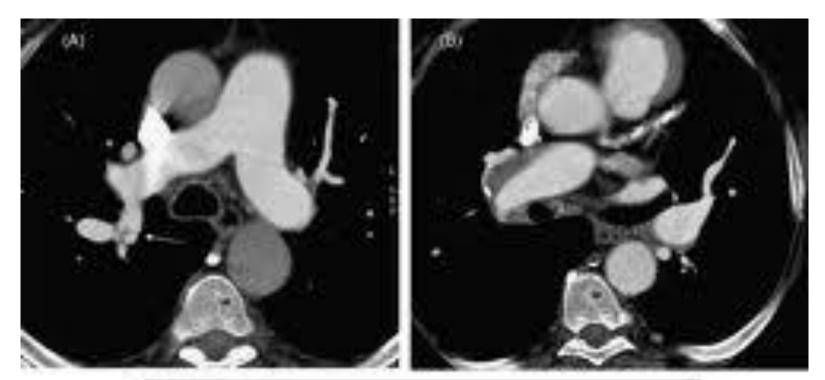
# CT pulmonary angiography (CTPA)

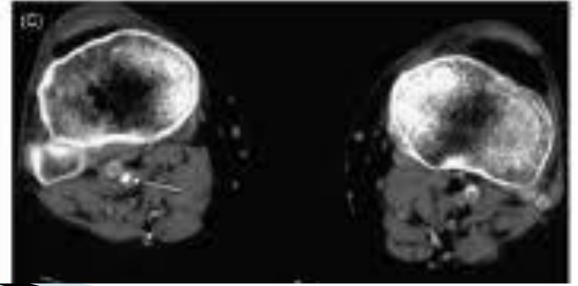
- It visualizes the distribution and extent of emboli in positive cases.
- It may also prove alternative diagnosis
- Simultaneous visualization of femoral and popliteal veins for DVT improves sensitivity.
- Can be performed safely in pregnant women (with foetal shielding).





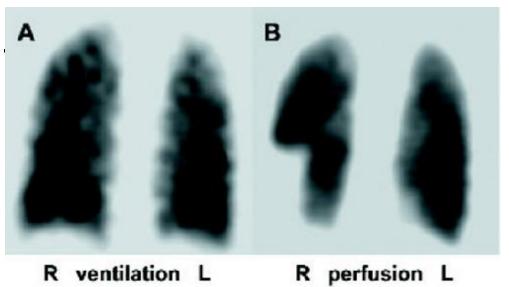






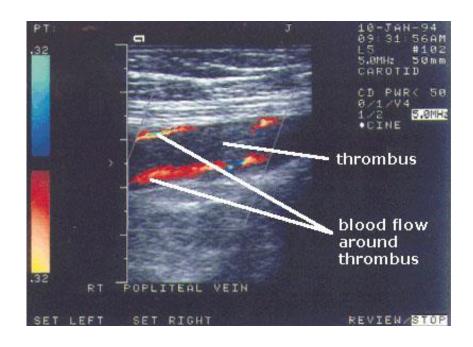
# Ventilation-perfusion scan (V/Q scan)

- Less commonly used.
- It is more useful in patients without pre existing cardiopulmonary disease and normal CXR; otherwise the interpretation of the results can be difficult.



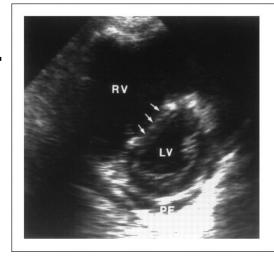
# Colour Doppler ultrasound of the leg

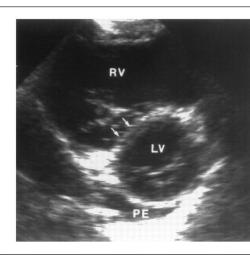
- The investigation of choice in the assessment of suspected DVT.
- It can be performed in patients with suspected pulmonary embolism to prove the presence of thrombus in the leg veins.



# **Echocardiography**

- It is helpful in the evaluation of patients with acute circulatory collapse.
- Acute right ventricular dilatation is usually present in massive PE
- A thrombus may be visible.
- Alternative diagnosis, like heart failure and pericardial tamponade can be excluded





# Management General measures

- Oxygen therapy for hypoxaemic patients
- Circulatory shock should be treated with intravenous fluids. Enotropic agents are of limited value
- Opiates may be necessary to relieve pain and distress
- Resuscitation by external cardiac massage in moribund patient may be successful
- Diuretics and vasodilators should be avoided

# **Anticoagulation**

- Subcutaneous low molecular weight heparin (LMWH) is preferred
- Fondaparinux is a pentasaccharide that is also approved for treatment of VTE.
- Unfractionated heparin can also be used, but it should be administered as continuous intravenous infusion (through infusion pump) and requires adjustment of APTT
- The duration of heparin (or fondaparinux) treatment should be at least 5 days, during which oral warfarin is commenced

# **Anticoagulation**

- Patients with a persistent risk factor or a history of previous thrombosis should receive warfarin for life.
- Those with identifiable and reversible risk factor require 3 months therapy.
- If the condition is idiopathic or the risk factor is weak, anticoagulation for 6 months is recommended.
- Warfarin is teratogenic, so VTE should be treated with LMWH during pregnancy (giving prophylactic dose after the initial therapeutic dosing).

# Thrombolytic therapy

- Thrombolysis is indicated in any patient presenting with acute massive PE accompanied by cardiogenic shock.
- Less certain indications include right ventricular dilatation and hypokinesia on echocardiography, severe hypoxaemia or high troponin level
- Patients with haemorrhagic risks should be excluded, as there is a risk of intracranial haemorrhge.

### **Caval filters**

Inferior vena caval filter is indicted in the following situations:

- When anticoagulation is contraindicated
- Massive haemorrhage on anticoagulation
- Recurrent PE despite anticoagulation





# **Prognosis**

- The case mortality rate of acute pulmonary embolism ranges from 1% 60%
- The immediate mortality is greatest in those with cardiogenic shock or right ventricular dysfunction.
- Recurrence is possible especially in the first 6-12 months after the initial event.
- The majority regain normal right ventricular function within 3 weeks, however by 2 years, 4% of patients have persistent pulmonary hypertension