PE: Paritial or complete occlusion of a pulmonary arterial branch by blood clot [part of clot deatach & travel in blood to block vessels in lung]

- VTE = DVT + PE
- VTE is the 3rd cause of vascular death after CVD & strokes.

** Source of empoli =>

[1] Thrombotic: *Most of them originate in the deep veins of calf, then popliteal & femoral veins (lower extremity)
- calf-limited (below the knee) → minimal embolic risk
* May originate from atypical sites ⇒ upper extremity associated with central venous cath. or intravascular cardiac or thoracic outlet obstruction or effort thrombosis.

[2] Non-Thrombotic: Fat embolism [fractures] / Aminotic fluid [pregnant]
Air embolism [divers] / Tumor embolism
Septic pulmonary embolism [infection]

** Risk factors =>

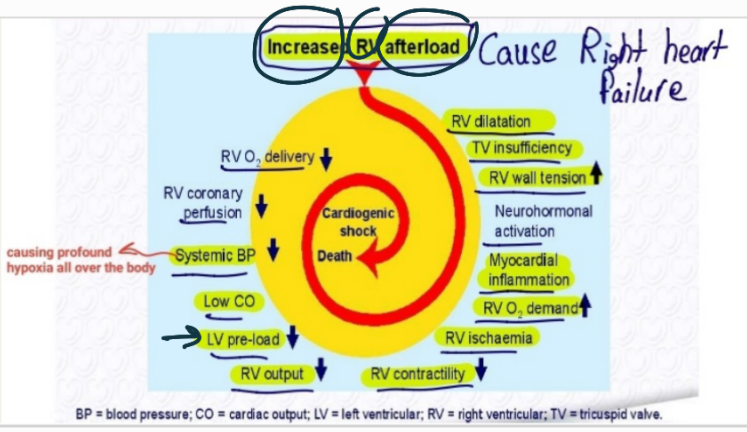
Strong (odd ratio >10)	Moderate (OR 2-9)		Weak (OR<2)
① Fracture of lower limb	• Autoimmune/infection	• Thrombophilia	• Bed >3 days
② previous VTE	• Blood transfusion	• Hormone replacement therapy/OC	• DM / HTN
③ Spinal cord injury	• Central venous lines	• postpartum period	• Travel
④ Hospitalization (3 months)	• IV cath./congestive HF	• Cancer/paralytic stroke	• ↑ age
⑤ Hip or Knee replacement.	• Chemotherapy	• Sup. V. thromposis	• obesity/pregnancy
⑥ Major Trauma	• Arthroscopic Knee surgery		• Varicose veins
⑦ MI (within previous 3 months)			

** Inherited thrombophilias **

1) Anti-thrombin (Most thrombogenic)
2) protein C / protein S deficiency
3) Factor 5 Leiden (Most common)
4) prothrombin
5) Hyper-Homo-cysteinemia
6) ↑ Factor 8

** Emboli travel via veins to right heart => reach pulmonary arterial system.
↳ can be asymptomatic or hemodynamic collapse (vary widely)
Depend on many factors
① size, location of emboli
② coexisting cardiopulmonary disease
③ Hypoxic responses
④ rate of resolution of emboli

Hemodynamic collapse =>



** Gas exchange abnormalities < Hypoxemia, ↑ A-a gradient

- The mechanism of hypoxemia in PE is V/Q mismatch not shunt
- Suggest massive PE: ① ↑ dead space ② Respiratory alkalosis from hyperventilation

Sudden Onset

Symptoms	Frequency (%)
Dyspnea	73
Pleuritic chest pain	66
Cough	37
Leg swelling	33
*Hemoptysis → An indication of lung infarction (ischemic pulmonary parenchymal necrosis)	13
Wheezing	9
Chest pain	4

Most common

Rare

* 27%. Asymptomatic

Signs	
*Respiratory rate ≥20/min tachypnea	70
Crackles	51
Heart rate ≥100/min	30
Third or fourth heart sound	26
Loud pulmonary component of second heart sound → due to Pul. HTN	23
Temperature > 38.5° C mild fever	7
Pleural rub	3

▷ Management
- Assess clinical probability
- Assess risk of mortality
- Investigation
- Treatment

[1] Assess risk of probability

- Wells rule / Revised geneva score

[2] Assess risk of mortality

- High risk : Hemodynamically unstable / early mortality 15%.
- Non-High risk : Intermediate or low risk.

Hemodynamically unstable
- Cardic arrest : need resuscitation
- Obstructive shock : systolic BP<90, vasopressors needed (adrenaline(NE) - NOT improved with saline
- + End organ hypoperfusion.
- Persistant hypotension (systolic BP<90, for 15 min)

Table 9 Classification of PE based on early mortality risk

Early mortality risk		Indicators of risk			
		Haemo-dynamic instability	Clinical parameters of PE severity/ comorbidity: PESI III–V or sPESI≥1	RV dysfunction on TTE or CTPA	Elevated cardiac troponin levels
High		+	(+)	+	(+)
Intermediate	Intermediate–high	-	+	+	+
	Intermediate–low	-	+	One (or none) positive	
Low		-	-	-	Assessment optional; if assessed, negative

Investigations:

- TTE

[TransThoracic Echo]

Gold standered

Figure 3 Diagnostic algorithm for suspected high-risk PE

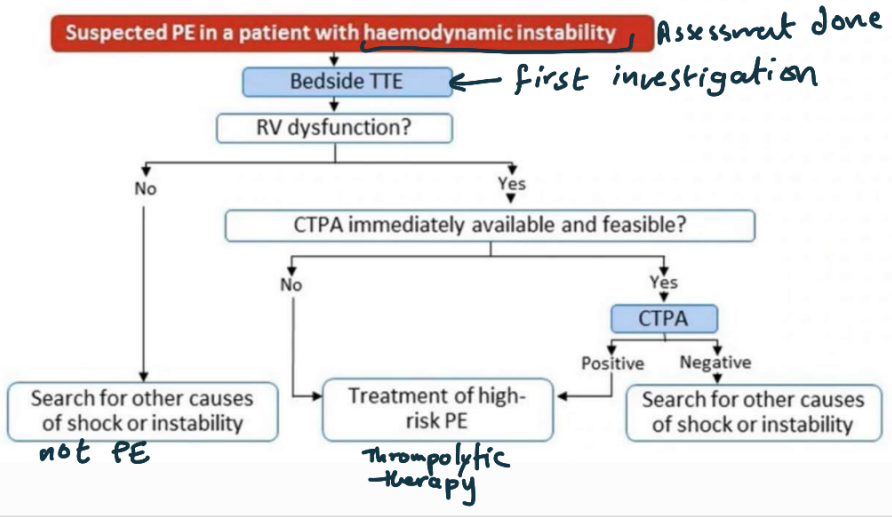
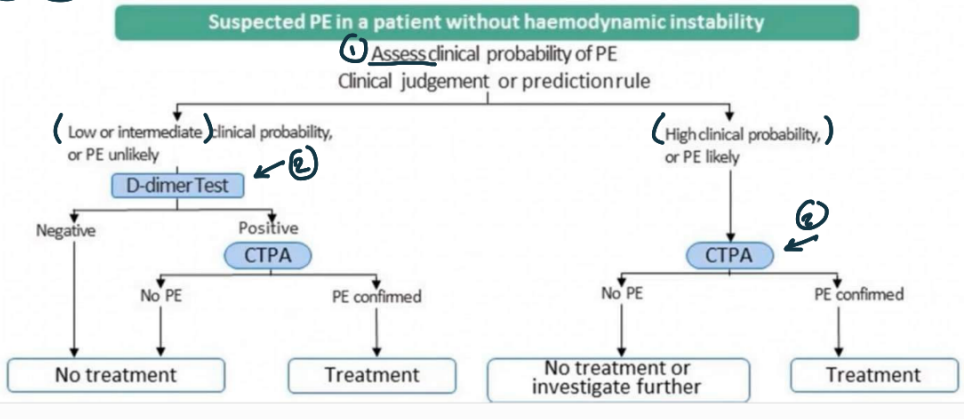


Figure 4 Diagnostic algorithm for suspected PE without haemodynamic instability



- D-Dimer

Rule out PE in low & moderate risk.

- useful for outpatient-emergency / may be elevated in infection / pregnancy / elderly
- Adjusted with age = pt. age X 10 => age 50 & below = .5 (cutoff point)

60 = .6 / 70 = .7

- **V/Q scan** : another assessment tool that can be use to detect PE, we use it if CT can't be done.
  - Renal impairment
  - IV contrast allergies
  - pregnancy
  - Hospital resources.

- **Spiral CT** : large doses of contrast with rapid rate (timed).
- **ECG** : useless, just we use it with SOB / chest pain pts. to rule out MI.
- **CXR** (not useful)
- **Echocardiogram** : suspected massive PE
- **Troponin** : indicate RV dysfunction

[4] Treatment

3 phases → ① Initial (5-10 days) ② long term (3-6 months)

③ extended (more than 3-6 months)

** NOACs recommended => Rivaroxaban / Dabigtran / Apixaban / Edoxapan

* Alternative is warfarine [multiple limitation =>

① variability in doses ② food & drug interaction

③ slow onset & offset action ④ ↑risk of ICH

⑤ requiring frequent monitoring ⑥ reversal with vit.K is slow]

#NOAC not used <
- CKD with Ccl less than 30
- Pregnancy & lactation
- Hepatic impairment
- Antiphospholipid syndrome

* provoke PE (known the cause) = 3 months 1B

* unprovoke PE → low or moderate Bleeding = >3 months 2B
  → High bleeding = 3 months 1B

- unprovoke PE who stop anticoagulant => Aspirin

▷ Risk factors for VTE recurrance (longer period of coagulation) :-

① idiopathic presentation ② primary DVT ③ ↑ age / ↑ thrombophilia

④ proximal DVT < femoral / iliac ⑤ cancer ⑥ ♂

▷ special cases use doppler US for follow up (No need to treat) :-

1- pt with isolated subsegmental PE

2- pt with isolated distal DVT (under knee).

3- IVC filter → failure of anticoagulation or acute PE with contraindication of anticoagulation (upper GI bleeding)