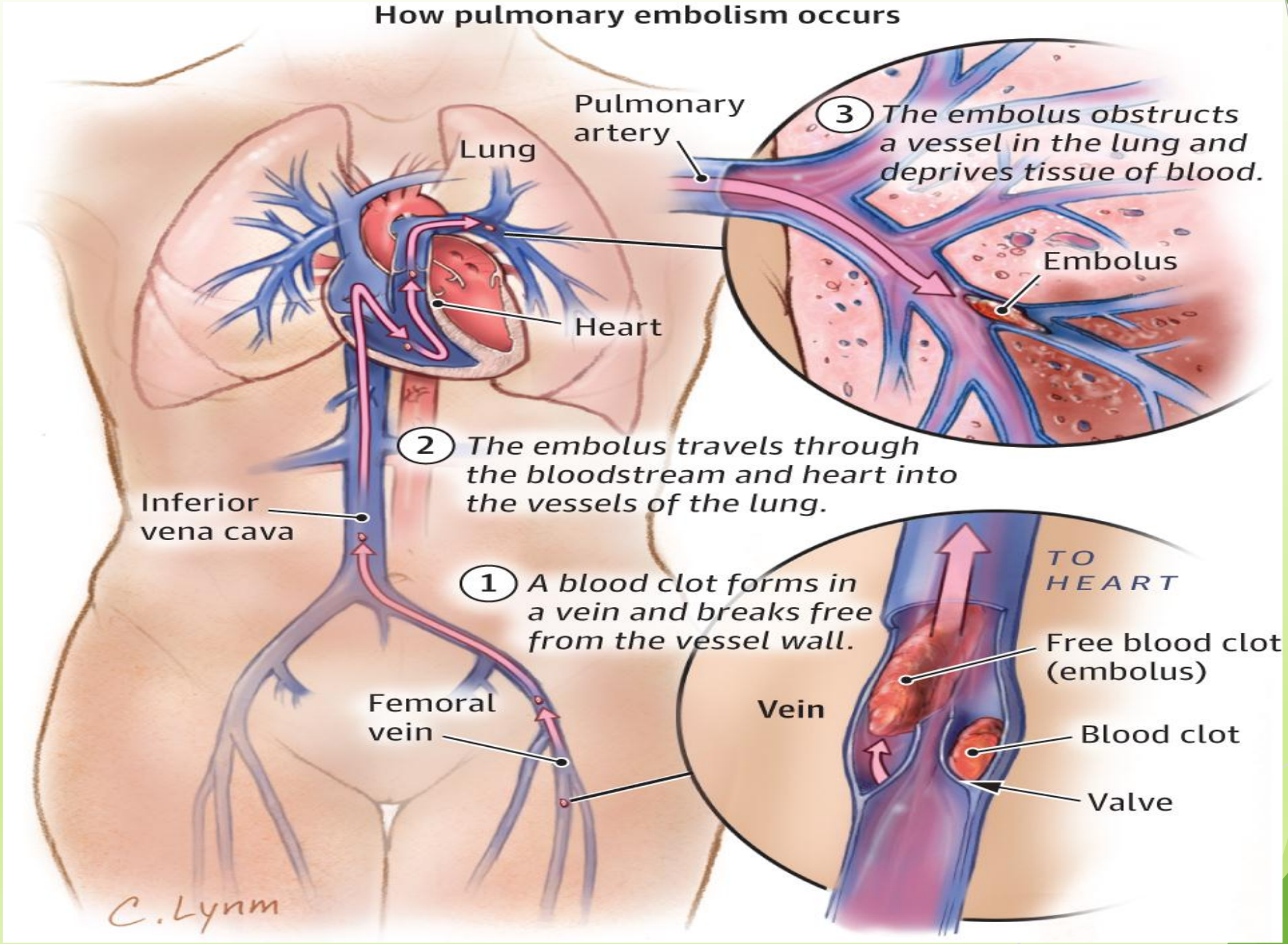
How pulmonary embolism occurs
Pulmonary artery
Lung
Heart
3 The embolus obstructs a vessel in the lung and deprives tissue of blood.
Embolus
2 The embolus travels through the bloodstream and heart into the vessels of the lung.
Inferior vena cava
1 A blood clot forms in a vein and breaks free from the vessel wall.
TO HEART
Free blood clot (embolus)
Vein
Blood clot
Valve
Femoral vein
C. Lynn

Epidemiology

In epidemiological studies, annual incidence rates for PE range from 39-115 per100 000 population; for DVT, incidence rates range from 53-162 per 100 000 population

34% died suddenly or within a few hours of the acute event, before therapy could be initiated or take effect.

Table 3 Predisposing factors for venous thromboembolism (data modified from Rogers *et al.*[23] and Anderson and Spencer[24])

Strong risk factors (OR > 10)
Fracture of lower limb
Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months)
Hip or knee replacement
Major trauma
Myocardial infarction (within previous 3 months)
Previous VTE
Spinal cord injury

Moderate risk factors (OR 2–9)
Arthroscopic knee surgery
Autoimmune diseases
Blood transfusion
Central venous lines
Intravenous catheters and leads
Chemotherapy
Congestive heart failure or respiratory failure
Erythropoiesis-stimulating agents
Hormone replacement therapy (depends on formulation)
In vitro fertilization
Oral contraceptive therapy
Post-partum period
Infection (specifically pneumonia, urinary tract infection, and HIV)
Inflammatory bowel disease
Cancer (highest risk in metastatic disease)
Paralytic stroke
Superficial vein thrombosis
Thrombophilia

Weak risk factors (OR < 2)
Bed rest >3 days
Diabetes mellitus
Arterial hypertension
Immobility due to sitting (e.g. prolonged car or air travel)
Increasing age
Laparoscopic surgery (e.g. cholecystectomy)
Obesity
Pregnancy
Varicose veins

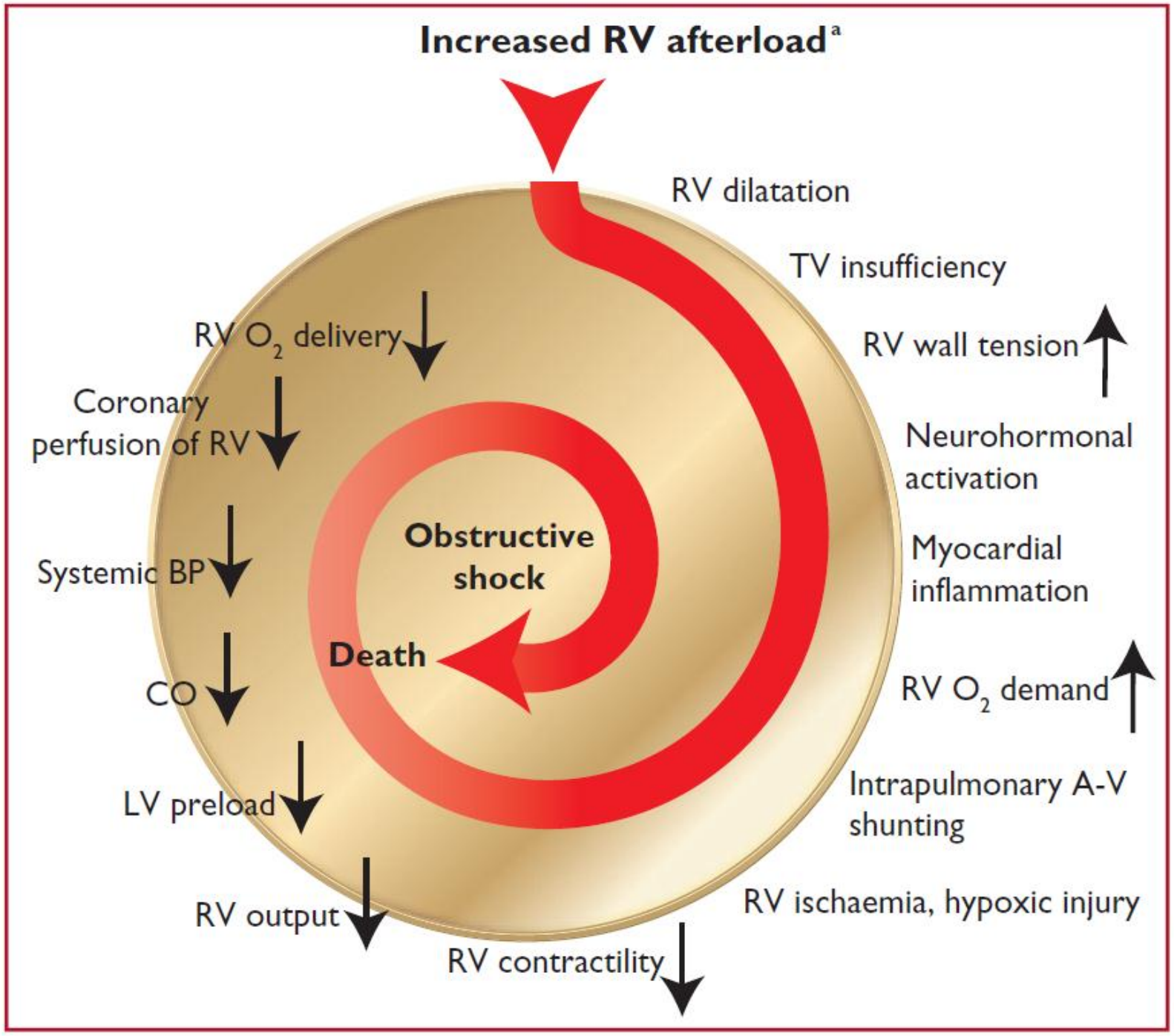
Increased RV afterload[a]
RV dilatation
TV insufficiency
RV wall tension
Neurohormonal activation
Myocardial inflammation
RV O_2 demand
Intrapulmonary A-V shunting
RV ischaemia, hypoxic injury
RV contractility
RV output
LV preload
CO
Systemic BP
Coronary perfusion of RV
RV O_2 delivery
Obstructive shock
Death
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Table 4 Definition of haemodynamic instability, which delineates acute high-risk pulmonary embolism (one of the following clinical manifestations at presentation)

(1) Cardiac arrest	(2) Obstructive shock[68–70]	(3) Persistent hypotension
Need for cardiopulmonary resuscitation	Systolic BP < 90 mmHg or vasopressors required to achieve a BP ≥90 mmHg despite adequate filling status	Systolic BP < 90 mmHg or systolic BP drop ≥40 mmHg, lasting longer than 15 min and not caused by new-onset arrhythmia, hypovolaemia, or sepsis
	And	
	End-organ hypoperfusion (altered mental status; cold, clammy skin; oliguria/anuria; increased serum lactate)	

BP = blood pressure.

Table 1. Simplified Wells Score for Assessment of the Pretest Clinical Probability of Pulmonary Embolism.*

Variable	Points
Clinical signs or symptoms of deep-vein thrombosis	3.0
Alternative diagnosis less likely than pulmonary embolism	3.0
Heart rate >100 beats/min	1.5
Immobilization or surgery in the previous 4 wk	1.5
Previous venous thromboembolism	1.5
Hemoptysis	1.0
Active cancer	1.0

* A total score of 4.0 or lower indicates that pulmonary embolism is unlikely, and a score higher than 4.0 indicates that pulmonary embolism is likely. This table was adapted with permission from Wells et al.[8]

Table 5 The revised Geneva clinical prediction rule for pulmonary embolism

Items	Clinical decision rule points	
	Original version[91]	Simplified version[87]
Previous PE or DVT	3	1
Heart rate		
75–94 b.p.m.	3	1
≥95 b.p.m.	5	2
Surgery or fracture within the past month	2	1
Haemoptysis	2	1
Active cancer	2	1
Unilateral lower-limb pain	3	1
Pain on lower-limb deep venous palpation and unilateral oedema	4	1
Age >65 years	1	1
Clinical probability		
Three-level score		
Low	0–3	0–1
Intermediate	4–10	2–4
High	≥11	≥5
Two-level score		
PE-unlikely	0–5	0–2
PE-likely	≥6	≥3

Clinical presentation

The clinical signs and symptoms of acute PE are non-specific. In most cases, PE is suspected in a patient with dyspnea, chest pain, pre syncope or syncope, hemoptysis.

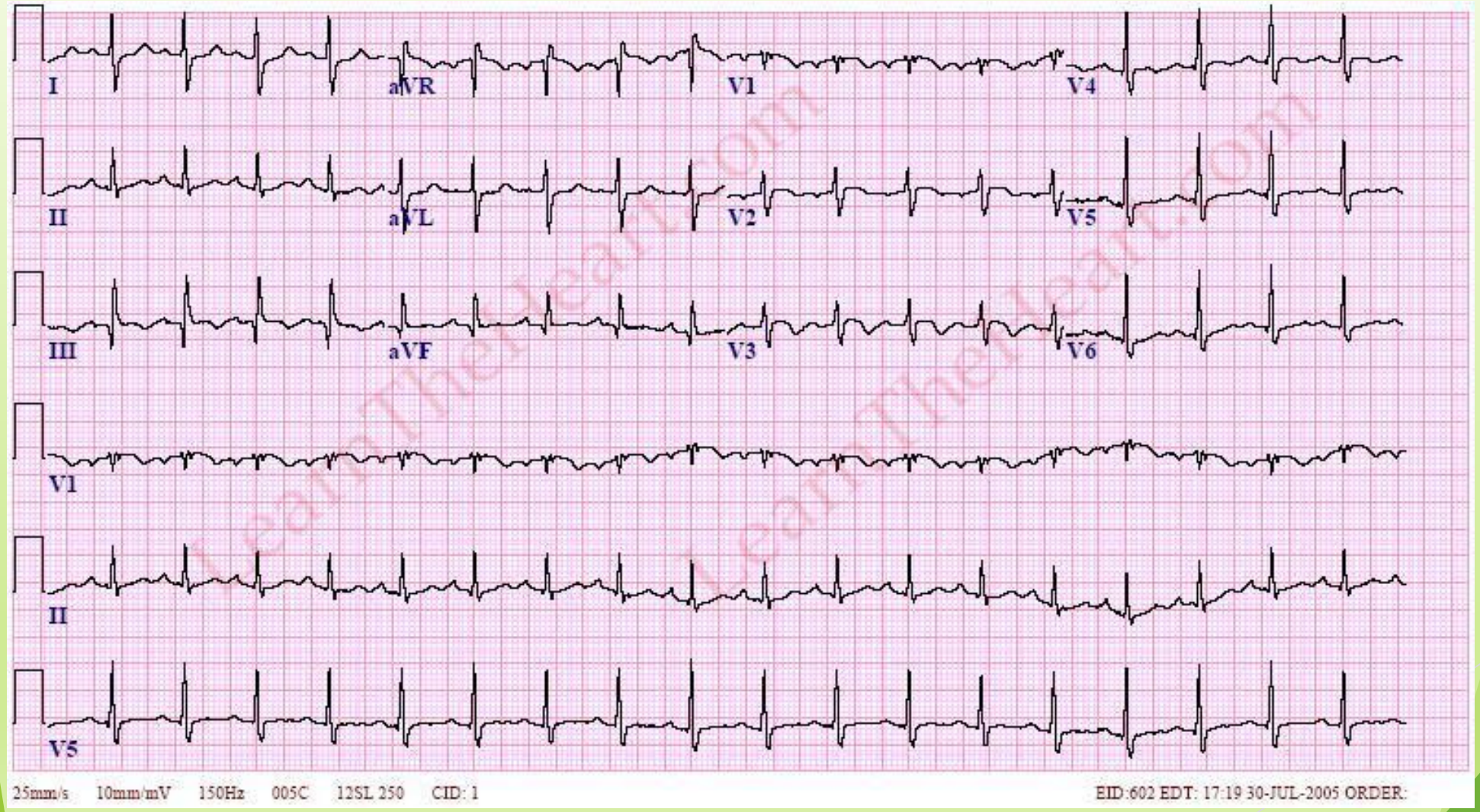
Hemodynamic instability is a rare but important form of clinical presentation, as it indicates central or extensive PE with severely reduced hemodynamic reserve.

Syncope may occur, and is associated with a higher prevalence of hemodynamic instability and RV dysfunction. acute PE may be a frequent finding in patients presenting with syncope (17%), even in the presence of an alternative explanation.

Age-adjusted D-dimer cut-offs

The use of age-adjusted cut offs may improve the performance of D-dimer testing in the elderly. A multinational prospective management study evaluated a previously validated age-adjusted cut-off (, age * 10 mcg/L, for patients aged >50 years) in a cohort of 3346 patients.

Pulmonary Embolism ECG



it is estimated that about 80% of patients with acute PE had an abnormal CXR. The most common abnormality noted from 4 studies was cardiomegaly[

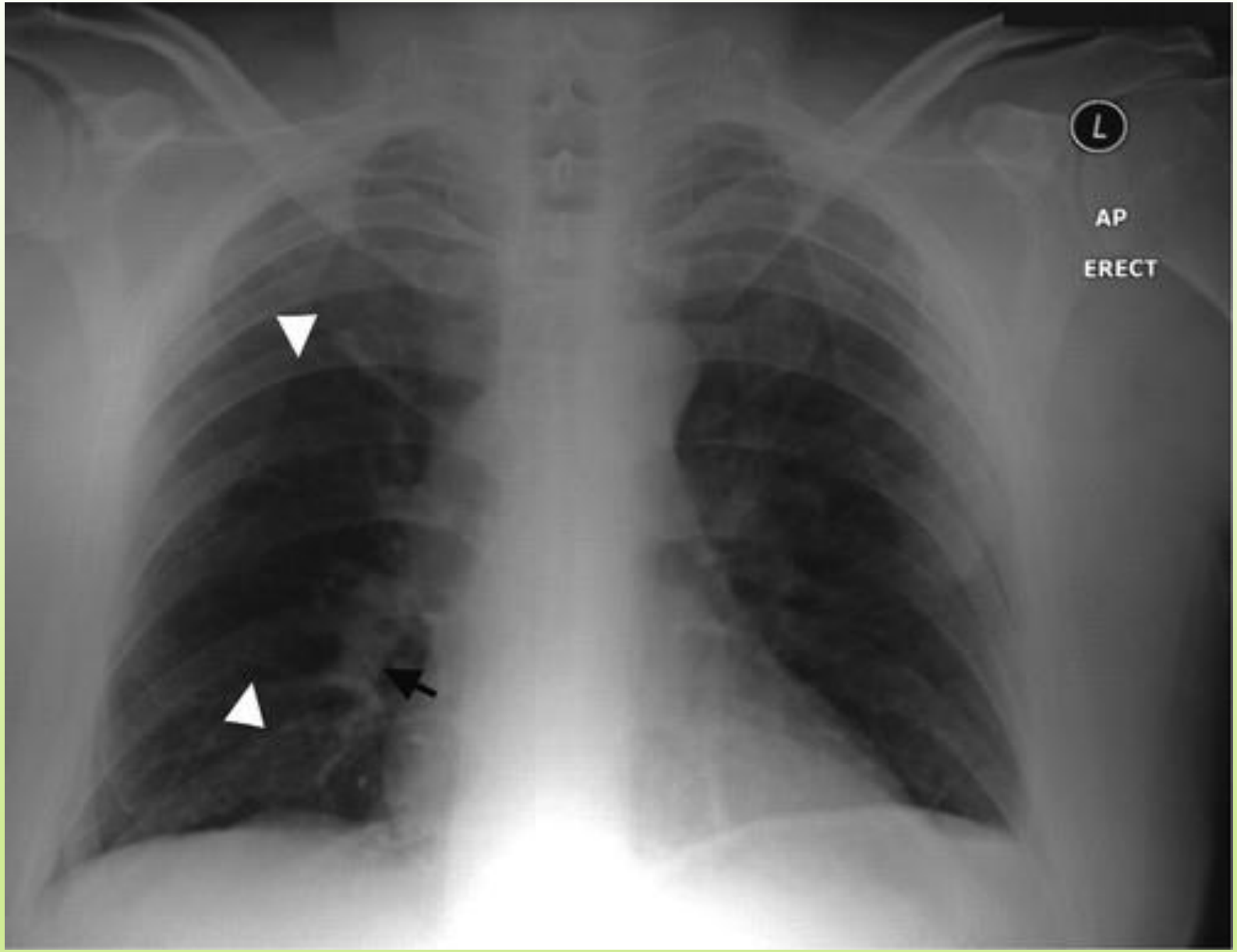
The other signs frequently noted were pulmonary infitrates, atelectasis, pleural effusion, pulmonary congestion, elevated hemidiapgram, Palla's sign, Westermark sign and Hampton's hump

Table 6 Imaging tests for diagnosis of pulmonary embolism

	Strengths	Weaknesses/limitations	Radiation issues[a]
CTPA	• Readily available around the clock in most centres • Excellent accuracy • Strong validation in prospective management outcome studies • Low rate of inconclusive results (3–5%) • May provide alternative diagnosis if PE excluded • Short acquisition time	• Radiation exposure • Exposure to iodine contrast: ○ limited use in iodine allergy and hyperthyroidism ○ risks in pregnant and breastfeeding women ○ contraindicated in severe renal failure • Tendency to overuse because of easy accessibility • Clinical relevance of CTPA diagnosis of subsegmental PE unknown	• Radiation effective dose 3–10 mSv[b] • Significant radiation exposure to young female breast tissue
Planar V/Q scan	• Almost no contraindications • Relatively inexpensive • Strong validation in prospective management outcome studies	• Not readily available in all centres • Interobserver variability in interpretation • Results reported as likelihood ratios • Inconclusive in 50% of cases • Cannot provide alternative diagnosis if PE excluded	• Lower radiation than CTPA, effective dose ~2 mSv[b]
V/Q SPECT	• Almost no contraindications • Lowest rate of non-diagnostic tests (<3%) • High accuracy according to available data • Binary interpretation ('PE' vs. 'no PE')	• Variability of techniques • Variability of diagnostic criteria • Cannot provide alternative diagnosis if PE excluded • No validation in prospective management outcome studies	• Lower radiation than CTPA, effective dose ~2 mSv[b]
Pulmonary angiography	• Historical gold standard	• Invasive procedure • Not readily available in all centres	• Highest radiation, effective dose 10–20 mSv[b]

Westermark's sign

Westermark's sign refers to a focal area of enhanced or increased translucency due to oligaemia, which occurs due to impaired vascularisation of the lung due to primary mechanical obstruction or reflex vasoconstriction.

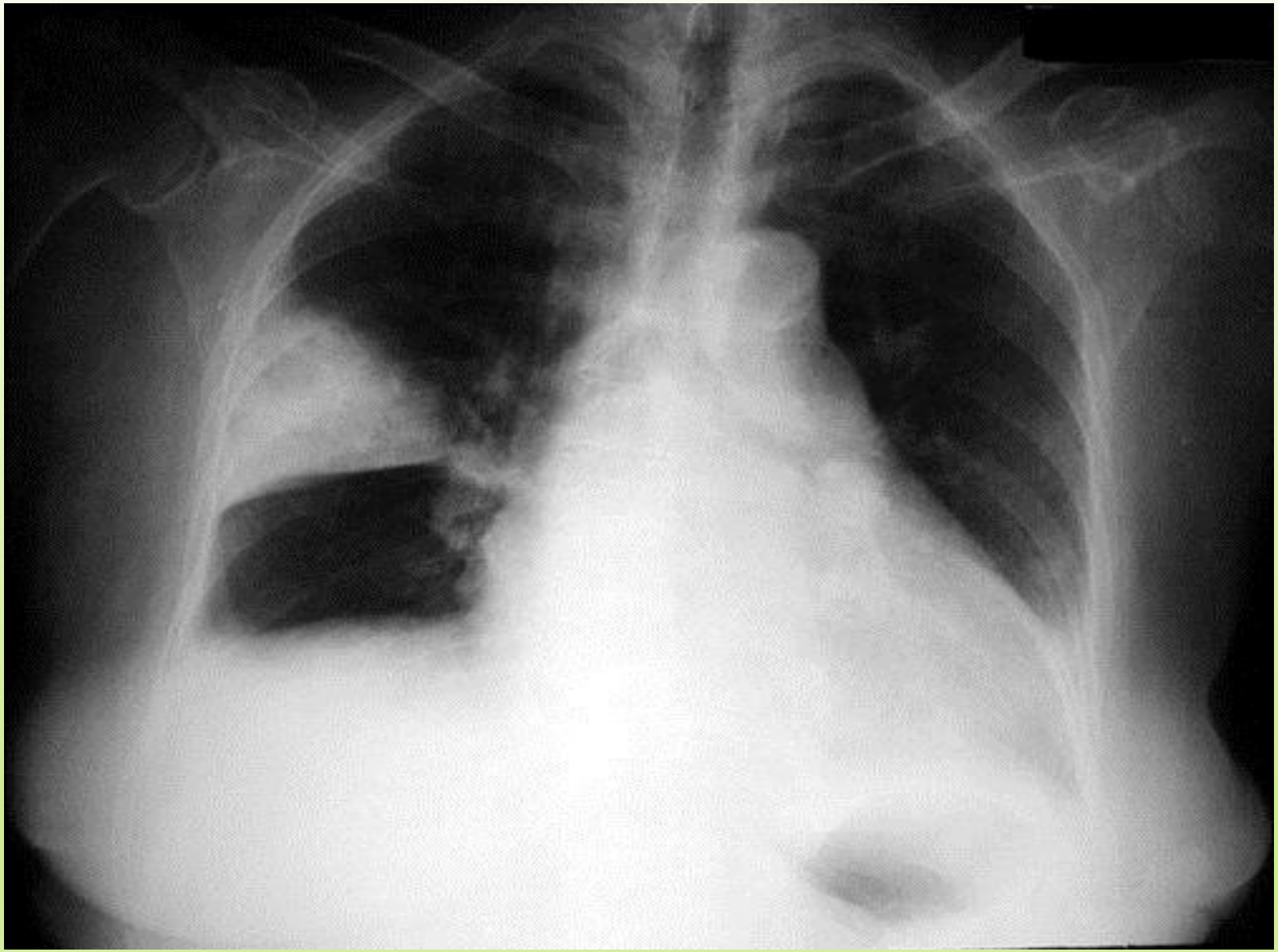

L
AP
ERECT

Palla's sign

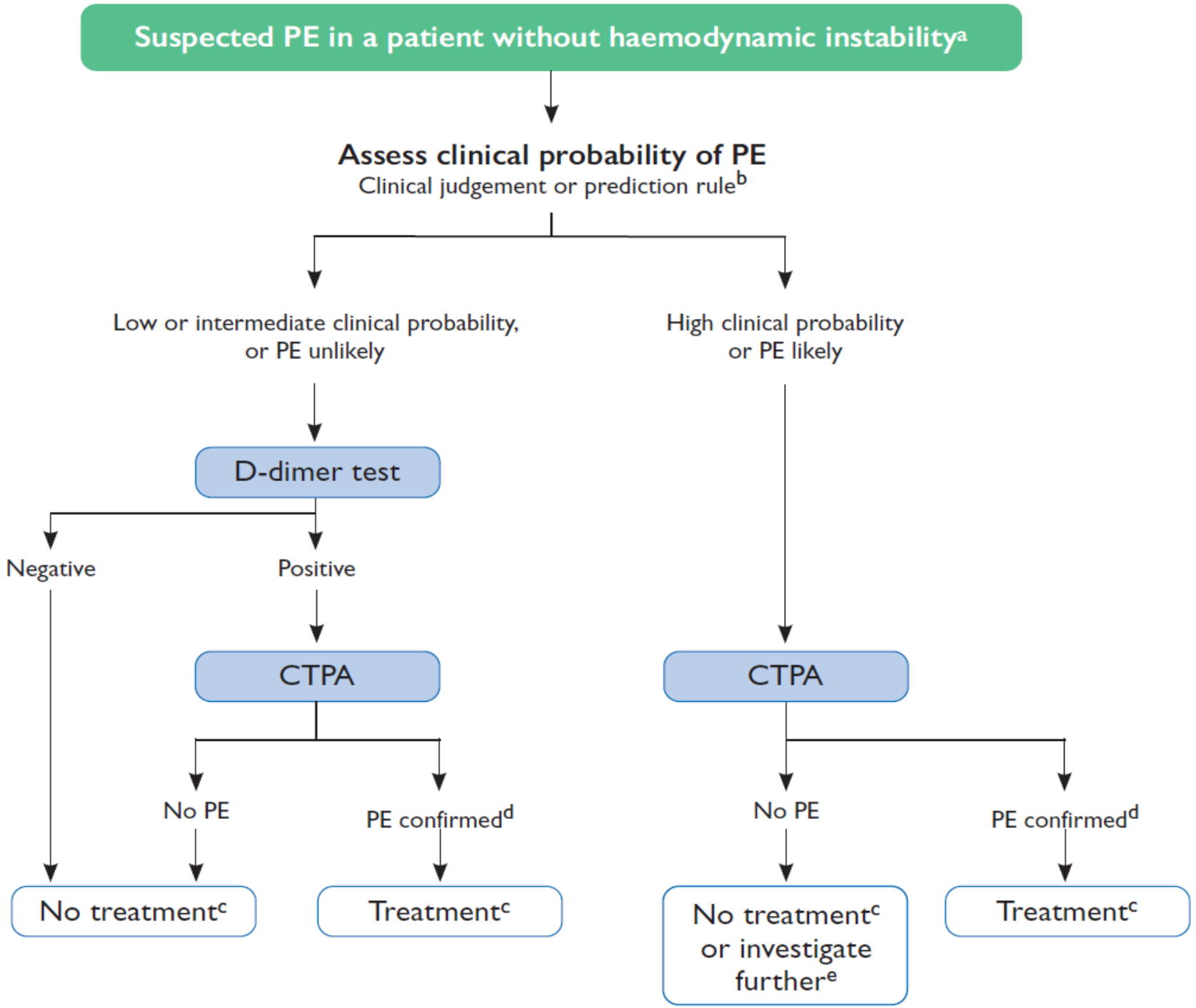
Palla's sign refers to an enlargement of the right descending pulmonary artery proximal to a cut off of the pulmonary artery due to acute pulmonary embolism

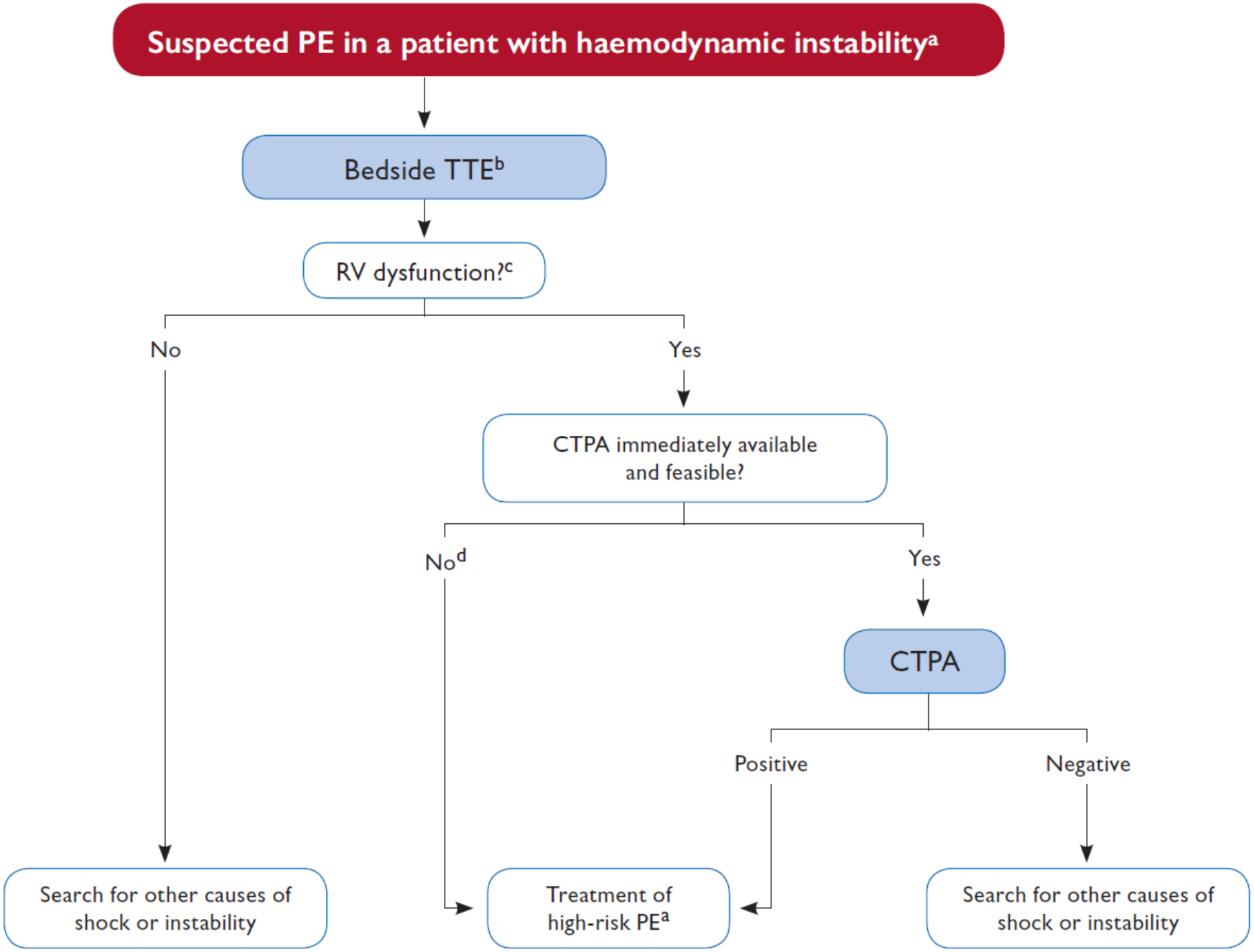
Hampton's hump

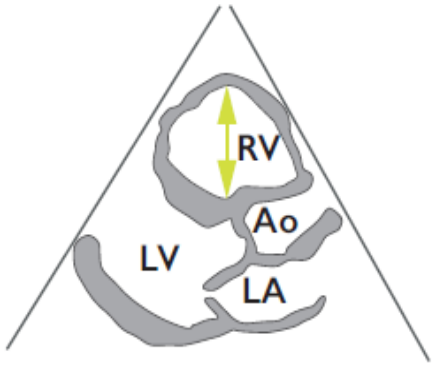
Hampton's hump is seen on the chest radiograph as a wedge- shaped opacity with a rounded convex apex directed towards the hilum.



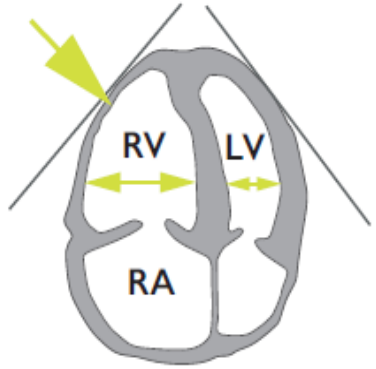
D-dimer		
Plasma D-dimer measurement, preferably using a highly sensitive assay, is recommended in outpatients/emergency department patients with low or intermediate clinical probability, or those that are PE-unlikely, to reduce the need for unnecessary imaging and irradiation.[101–103,122,164,171,173,174]	I	A
As an alternative to the fixed D-dimer cut-off, a negative D-dimer test using an age-adjusted cut-off (age × 10 μg/L, in patients aged >50 years) should be considered for excluding PE in patients with low or intermediate clinical probability, or those that are PE-unlikely.[106]	IIa	B
As an alternative to the fixed or age-adjusted D-dimer cut-off, D-dimer levels adapted to clinical probability[c] should be considered to exclude PE.[107]	IIa	B
D-dimer measurement is not recommended in patients with high clinical probability, as a normal result does not safely exclude PE, even when using a highly sensitive assay.[175,176]	III	A


Suspected PE in a patient without haemodynamic instability[a]
Assess clinical probability of PE
Clinical judgement or prediction rule[b]
Low or intermediate clinical probability, or PE unlikely
High clinical probability or PE likely
D-dimer test
Negative
Positive
CTPA
No PE
PE confirmed[d]
No treatment[c]
Treatment[c]
CTPA
No PE
PE confirmed[d]
No treatment[c] or investigate further[e]
Treatment[c]

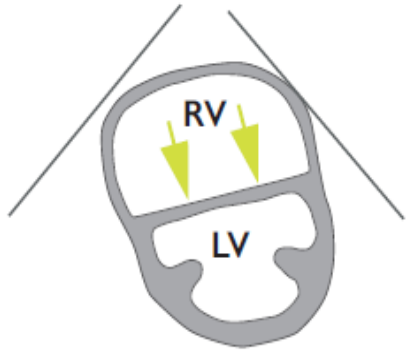

Suspected PE in a patient with haemodynamic instability[a]
Bedside TTE[b]
RV dysfunction?[c]
No
Yes
CTPA immediately available and feasible?
No[d]
Yes
CTPA
Positive
Negative
Search for other causes of shock or instability
Treatment of high-risk PE[a]
Search for other causes of shock or instability



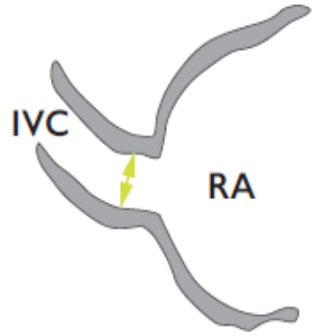
A. Enlarged right ventricle, parasternal long axis view



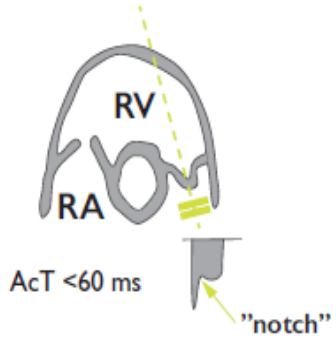
B. Dilated RV with basal RV/LV ratio >1.0, and McConnell sign (arrow), four chamber view

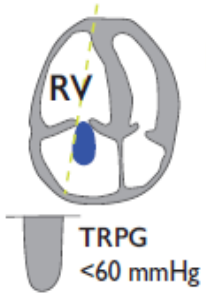


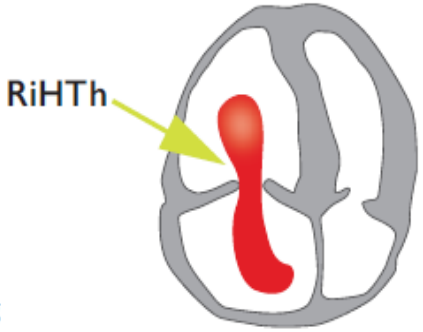
C. Flattened intraventricle septum (arrows) parasternal short axis view



D. Distended inferior vena cava with diminished inspiratory collapsibility, subcostal view

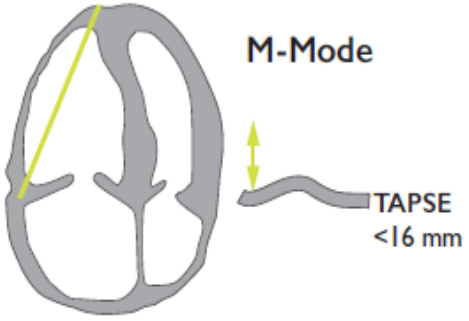




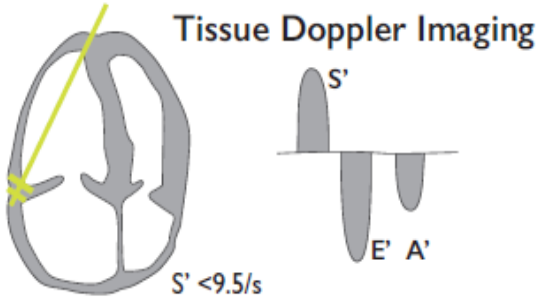


E. 60/60 sign: coexistence of acceleration time of pulmonary ejection <60 ms and midsystolic "notch" with mildy elevated (<60 mmHg) peak systolic gradient at the tricuspic valve

F. Right heart mobile thrombus detected in right heart cavities (arrow)



G. Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<16 mm)



H. Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s)

Suspected PE with haemodynamic instability		
In suspected high-risk PE, as indicated by the presence of haemodynamic instability, bedside echocardiography or emergency CTPA (depending on availability and clinical circumstances) is recommended for diagnosis.[169]	I	C
It is recommended that i.v. anticoagulation with UFH, including a weight-adjusted bolus injection, be initiated without delay in patients with suspected high-risk PE.	I	C
Suspected PE without haemodynamic instability		
The use of validated criteria for diagnosing PE is recommended.[12]	I	B
Initiation of anticoagulation is recommended without delay in patients with high or intermediate clinical probability of PE while diagnostic workup is in progress.	I	C

CTPA		
It is recommended to reject the diagnosis of PE (without further testing) if CTPA is normal in a patient with low or intermediate clinical probability, or who is PE-unlikely.[101,122,164,171]	I	A
It is recommended to accept the diagnosis of PE (without further testing) if CTPA shows a segmental or more proximal filling defect in a patient with intermediate or high clinical probability.[115]	I	B
It should be considered to reject the diagnosis of PE (without further testing) if CTPA is normal in a patient with high clinical probability or who is PE-likely.[171]	IIa	B
Further imaging tests to confirm PE may be considered in cases of isolated subsegmental filling defects.[115]	IIb	C
CT venography is not recommended as an adjunct to CTPA.[115,164]	III	B
V/Q scintigraphy		
It is recommended to reject the diagnosis of PE (without further testing) if the perfusion lung scan is normal.[75,122,134,174]	I	A
It should be considered to accept that the diagnosis of PE (without further testing) if the V/Q scan yields high probability for PE.[134]	IIa	B
A non-diagnostic V/Q scan should be considered as exclusion of PE when combined with a negative proximal CUS in patients with low clinical probability, or who are PE-unlikely.[75,122,174]	IIa	B

Table 7 Original and simplified Pulmonary Embolism Severity Index

Parameter	Original version[226]	Simplified version[229]
Age	Age in years	1 point (if age >80 years)
Male sex	+10 points	—
Cancer	+30 points	1 point
Chronic heart failure	+10 points	1 point
Chronic pulmonary disease	+10 points	
Pulse rate ≥110 b.p.m.	+20 points	1 point
Systolic BP <100 mmHg	+30 points	1 point
Respiratory rate >30 breaths per min	+20 points	—
Temperature <36°C	+20 points	—
Altered mental status	+60 points	—
Arterial oxyhaemo-globin saturation <90%	+20 points	1 point

	Risk strata[a]	
	Class I: ≤65 points very low 30 day mortality risk (0—1.6%) **Class II: 66—85 points** low mortality risk (1.7—3.5%)	**0 points =** 30 day mortality risk 1.0% (95% CI 0.0—2.1%)
	Class III: 86—105 points moderate mortality risk (3.2—7.1%) **Class IV: 106—125 points** high mortality risk (4.0—11.4%) **Class V: >125 points** very high mortality risk (10.0—24.5%)	**≥1 point(s) =** 30 day mortality risk 10.9% (95% CI 8.5—13.2%)

BP = blood pressure; b.p.m. = beats per minute; CI = confidence interval.
[a]Based on the sum of points.

Table 8 **Classification of pulmonary embolism severity and the risk of early (in-hospital or 30 day) death**

Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/or comorbidity: PESI class III–V or sPESI ≥I	RV dysfunction on TTE or CTPA[b]	Elevated cardiac troponin levels[c]
High		+	(+)[d]	+	(+)
Intermediate	Intermediate–high	-	+[e]	+	+
	Intermediate–low	-	+[e]	One (or none) positive	
Low		-	-	-	Assesment optional; if assessed, negative

Treatment in the acute phase

Table 9 Treatment of right ventricular failure in acute high-risk pulmonary embolism

Strategy	Properties and use	Caveats
Volume optimization		
Cautious volume loading, saline, or Ringer's lactate, ≤500 mL over 15–30 min	Consider in patients with normal–low central venous pressure (due, for example, to concomitant hypovolaemia)	Volume loading can over-distend the RV, worsen ventricular interdependence, and reduce CO[239]
Vasopressors and inotropes		
Norepinephrine, 0.2–1.0 μg/kg/min[a] [240]	Increases RV inotropy and systemic BP, promotes positive ventricular interactions, and restores coronary perfusion gradient	Excessive vasoconstriction may worsen tissue perfusion
Dobutamine, 2–20 μg/kg/min[241]	Increases RV inotropy, lowers filling pressures	May aggravate arterial hypotension if used alone, without a vasopressor; may trigger or aggravate arrhythmias
Mechanical circulatory support		
Veno–arterial ECMO/extracorporeal life support[251,252,258]	Rapid short-term support combined with oxygenator	Complications with use over longer periods (>5–10 days), including bleeding and infections; no clinical benefit unless combined with surgical embolectomy; requires an experienced team

CO = cardiac output; BP = blood pressure; ECMO = extracorporeal membrane oxygenation; RV = right ventricle/ventricular.
[a]Epinephrine is used in cardiac arrest.

Table 10 Thrombolytic regimens, doses, and contraindications

Molecule	Regimen	Contraindications to fibrinolysis
rtPA	100 mg over 2 h	**Absolute**
	0.6 mg/kg over 15 min (maximum dose 50 mg)[a]	History of haemorrhagic stroke or stroke of unknown origin
Streptokinase	250 000 IU as a loading dose over 30 min, followed by 100 000 IU/h over 12–24 h	Ischaemic stroke in previous 6 months
	Accelerated regimen: 1.5 million IU over 2 h	Central nervous system neoplasm
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h over 12–24 h	Major trauma, surgery, or head injury in previous 3 weeks
	Accelerated regimen: 3 million IU over 2 h	Bleeding diathesis
		Active bleeding
		Relative
		Transient ischaemic attack in previous 6 months
		Oral anticoagulation
		Pregnancy or first post-partum week
		Non-compressible puncture sites
		Traumatic resuscitation
		Refractory hypertension (systolic BP >180 mmHg)
		Advanced liver disease
		Infective endocarditis
		Active peptic ulcer

6.6 Recommendations for acute-phase treatment of high-risk pulmonary embolism[a]

Recommendations	Class[b]	Level[c]
It is recommended that anticoagulation with UFH, including a weight-adjusted bolus injection, be initiated without delay in patients with high-risk PE.	I	C
Systemic thrombolytic therapy is recommended for high-risk PE. [282]	I	B
Surgical pulmonary embolectomy is recommended for patients with high-risk PE, in whom thrombolysis is contraindicated or has failed.[d] [281]	I	C
Percutaneous catheter-directed treatment should be considered for patients with high-risk PE, in whom thrombolysis is contraindicated or has failed.[d]	IIa	C
Norepinephrine and/or dobutamine should be considered in patients with high-risk PE.	IIa	C
ECMO may be considered, in combination with surgical embolectomy or catheter-directed treatment, in patients with PE and refractory circulatory collapse or cardiac arrest.[d] [252]	IIb	C

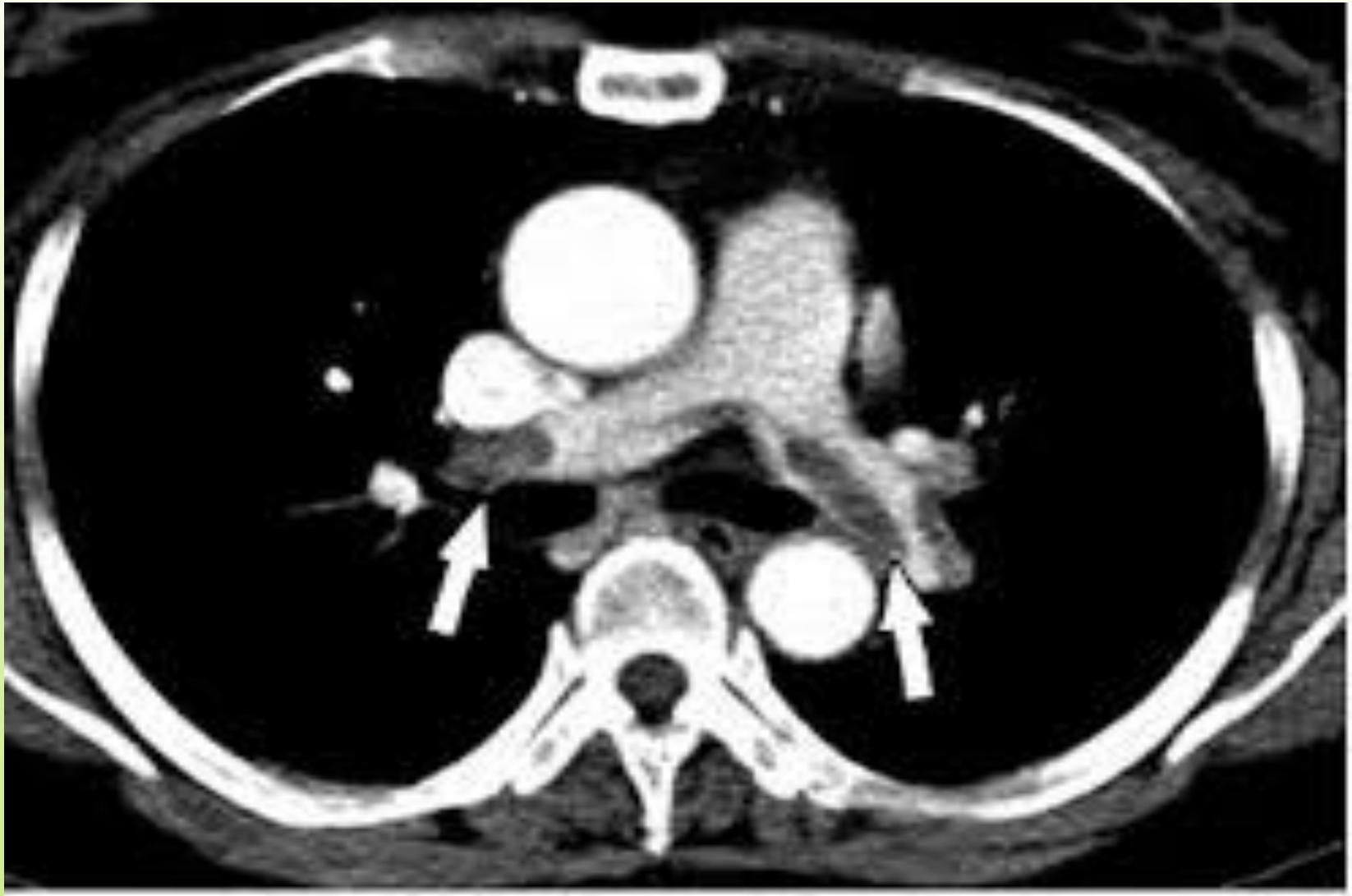
6.7 Recommendations for acute-phase treatment of intermediate- or low-risk pulmonary embolism

Recommendations	Class[a]	Level[b]
Initiation of anticoagulation		
Initiation of anticoagulation is recommended without delay in patients with high or intermediate clinical probability of PE,[c] while diagnostic workup is in progress.	I	C
If anticoagulation is initiated parenterally, LMWH or fondaparinux is recommended (over UFH) for most patients.[262,309–311]	I	A
When oral anticoagulation is started in a patient with PE who is eligible for a NOAC (apixaban, dabigatran, edoxaban, or rivaroxaban), a NOAC is recommended in preference to a VKA.[260,261,312–314]	I	A
When patients are treated with a VKA, overlapping with parenteral anticoagulation is recommended until an INR of 2.5 (range 2.0–3.0) is reached.[315,316]	I	A
NOACs are not recommended in patients with severe renal impairment,[d] during pregnancy and lactation, and in patients with antiphospholipid antibody syndrome.[260,261,312–314]	III	C

Reperfusion treatment		
Rescue thrombolytic therapy is recommended for patients with haemodynamic deterioration on anticoagulation treatment.[282]	I	B
As an alternative to rescue thrombolytic therapy, surgical embolectomy[e] or percutaneous catheter-directed treatment[e] should be considered for patients with haemodynamic deterioration on anticoagulation treatment.	IIa	C
Routine use of primary systemic thrombolysis is not recommended in patients with intermediate- or low-risk PE.[c,f 179]	III	B

6.9 Recommendations for inferior vena cava filters

Recommendations	Class[a]	Level[b]
IVC filters should be considered in patients with acute PE and absolute contraindications to anticoagulation.	IIa	C
IVC filters should be considered in cases of PE recurrence despite therapeutic anticoagulation.	IIa	C
Routine use of IVC filters is not recommended.[302–304]	III	A



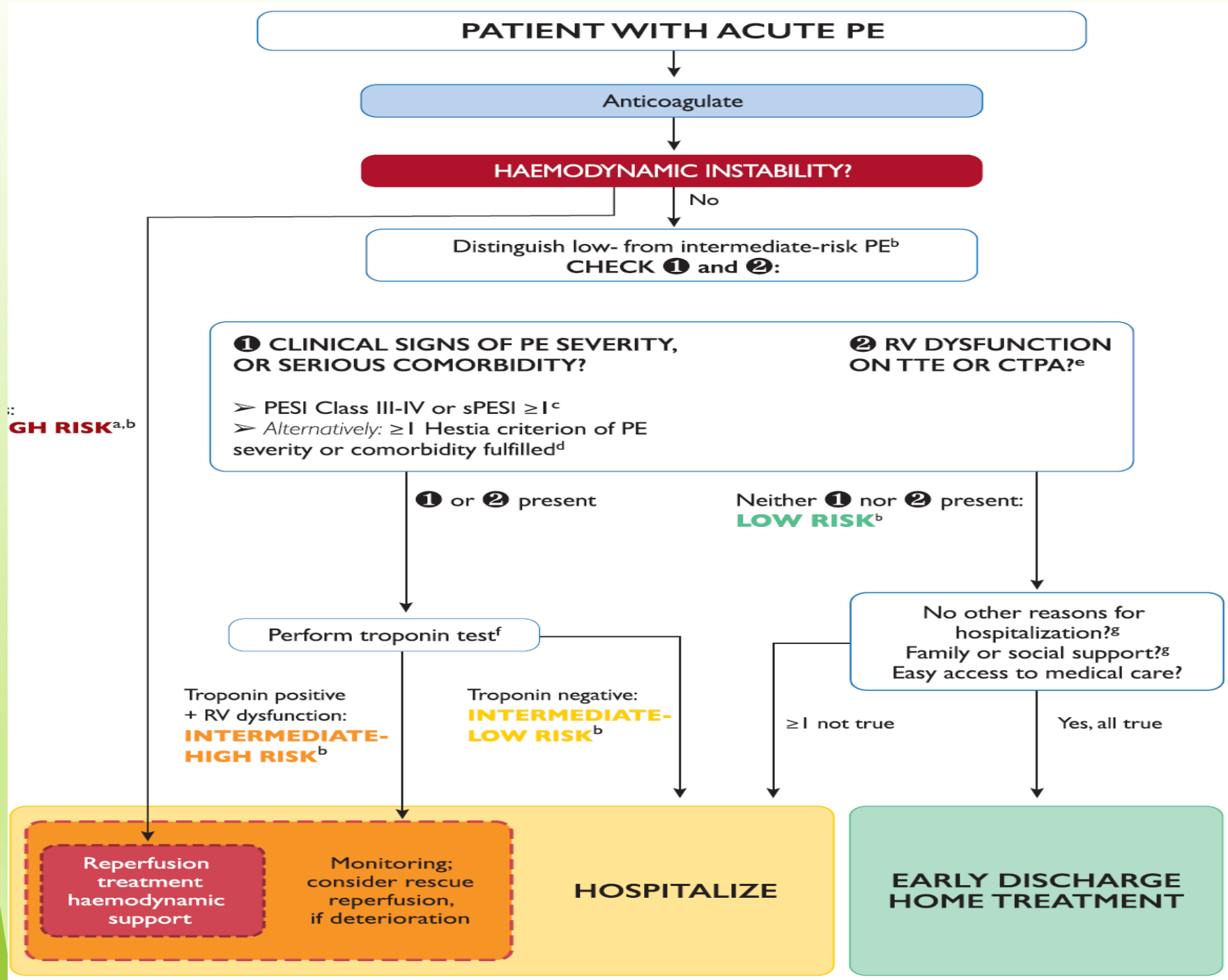

PATIENT WITH ACUTE PE
Anticoagulate
HAEMODYNAMIC INSTABILITY?
No
Distinguish low- from intermediate-risk PE[b]
CHECK ❶ and ❷:
❶ CLINICAL SIGNS OF PE SEVERITY, OR SERIOUS COMORBIDITY?
➢ PESI Class III-IV or sPESI ≥1[c]
➢ Alternatively: ≥1 Hestia criterion of PE severity or comorbidity fulfilled[d]
❷ RV DYSFUNCTION ON TTE OR CTPA?[e]
GH RISK[a,b]
❶ or ❷ present
Neither ❶ nor ❷ present:
LOW RISK[b]
Perform troponin test[f]
No other reasons for hospitalization?[g]
Family or social support?[g]
Easy access to medical care?
Troponin positive + RV dysfunction:
INTERMEDIATE-HIGH RISK[b]
Troponin negative:
INTERMEDIATE-LOW RISK[b]
≥1 not true
Yes, all true
Reperfusion treatment haemodynamic support
Monitoring; consider rescue reperfusion, if deterioration
HOSPITALIZE
EARLY DISCHARGE HOME TREATMENT

Table 11 **Categorization of risk factors for venous thromboembolism based on the risk of recurrence over the long-term**

Estimated risk for long-term recurrence[a]	Risk factor category for index PE[b]	Examples[b]
Low (<3% per year)	Major transient or reversible factors associated with >10-fold increased risk for the index VTE event (compared to patients without the risk factor)	• Surgery with general anaesthesia for >30 min • Confined to bed in hospital (only "bathroom privileges") for ≥3 days due to an acute illness, or acute exacerbation of a chronic illness • Trauma with fractures
Intermediate (3–8% per year)	Transient or reversible factors associated with ≤10-fold increased risk for first (index) VTE	• Minor surgery (general anaesthesia for <30 min) • Admission to hospital for <3 days with an acute illness • Oestrogen therapy/contraception • Pregnancy or puerperium • Confined to bed out of hospital for ≥3 days with an acute illness • Leg injury (without fracture) associated with reduced mobility for ≥3 days • Long-haul flight
	Non-malignant persistent risk factors	• Inflammatory bowel disease • Active autoimmune disease
	No identifiable risk factor	
High (>8% per year)		• Active cancer • One or more previous episodes of VTE in the absence of a major transient or reversible factor • Antiphospholipid antibody syndrome

8.4 Recommendations for the regimen and duration of anticoagulation after pulmonary embolism in patients without cancer

Recommendations	Class[a]	Level[b]
Therapeutic anticoagulation for ≥ 3 months is recommended for all patients with PE.[347]	I	A
Patients in whom discontinuation of anticoagulation after 3 months is recommended		
For patients with first PE/VTE secondary to a major transient/reversible risk factor, discontinuation of therapeutic oral anticoagulation is recommended after 3 months.[331,340,341]	I	B
Patients in whom extension of anticoagulation beyond 3 months is recommended		
Oral anticoagulant treatment of indefinite duration is recommended for patients presenting with recurrent VTE (that is, with at least one previous episode of PE or DVT) not related to a major transient or reversible risk factor.[358]	I	B
Oral anticoagulant treatment with a VKA for an indefinite period is recommended for patients with antiphospholipid antibody syndrome.[359]	I	B

Patients in whom extension of anticoagulation beyond 3 months should be considered[c,d]		
Extended oral anticoagulation of indefinite duration should be considered for patients with a first episode of PE and no identifiable risk factor.[330,331,347,351–353]	IIa	A
Extended oral anticoagulation of indefinite duration should be considered for patients with a first episode of PE associated with a persistent risk factor other than antiphospholipid antibody syndrome.[330,352,353]	IIa	C
Extended oral anticoagulation of indefinite duration should be considered for patients with a first episode of PE associated with a minor transient or reversible risk factor.[330,331,352]	IIa	C
NOAC dose in extended anticoagulation[e]		
If extended oral anticoagulation is decided after PE in a patient without cancer, a reduced dose of the NOACs apixaban (2.5 mg b.i.d.) or rivaroxaban (10 mg o.d.) should be considered after 6 months of therapeutic anticoagulation.[352,353]	IIa	A

8.6 Recommendations for the regimen and the duration of anticoagulation after pulmonary embolism in patients with active cancer

Recommendations	Class[a]	Level[b]
For patients with PE and cancer, weight-adjusted subcutaneous LMWH should be considered for the first 6 months over VKAs.[360–363]	IIa	A
Edoxaban should be considered as an alternative to weight-adjusted subcutaneous LMWH in patients without gastrointestinal cancer.[366]	IIa	B
Rivaroxaban should be considered as an alternative to weight-adjusted subcutaneous LMWH in patients without gastrointestinal cancer.[367]	IIa	C
For patients with PE and cancer, extended anticoagulation (beyond the first 6 months)[c] should be considered for an indefinite period or until the cancer is cured.[378]	IIa	B
In patients with cancer, management of incidental PE in the same manner as symptomatic PE should be considered, if it involves segmental or more proximal branches, multiple subsegmental vessels, or a single subsegmental vessel in association with proven DVT.[376,377]	IIa	B

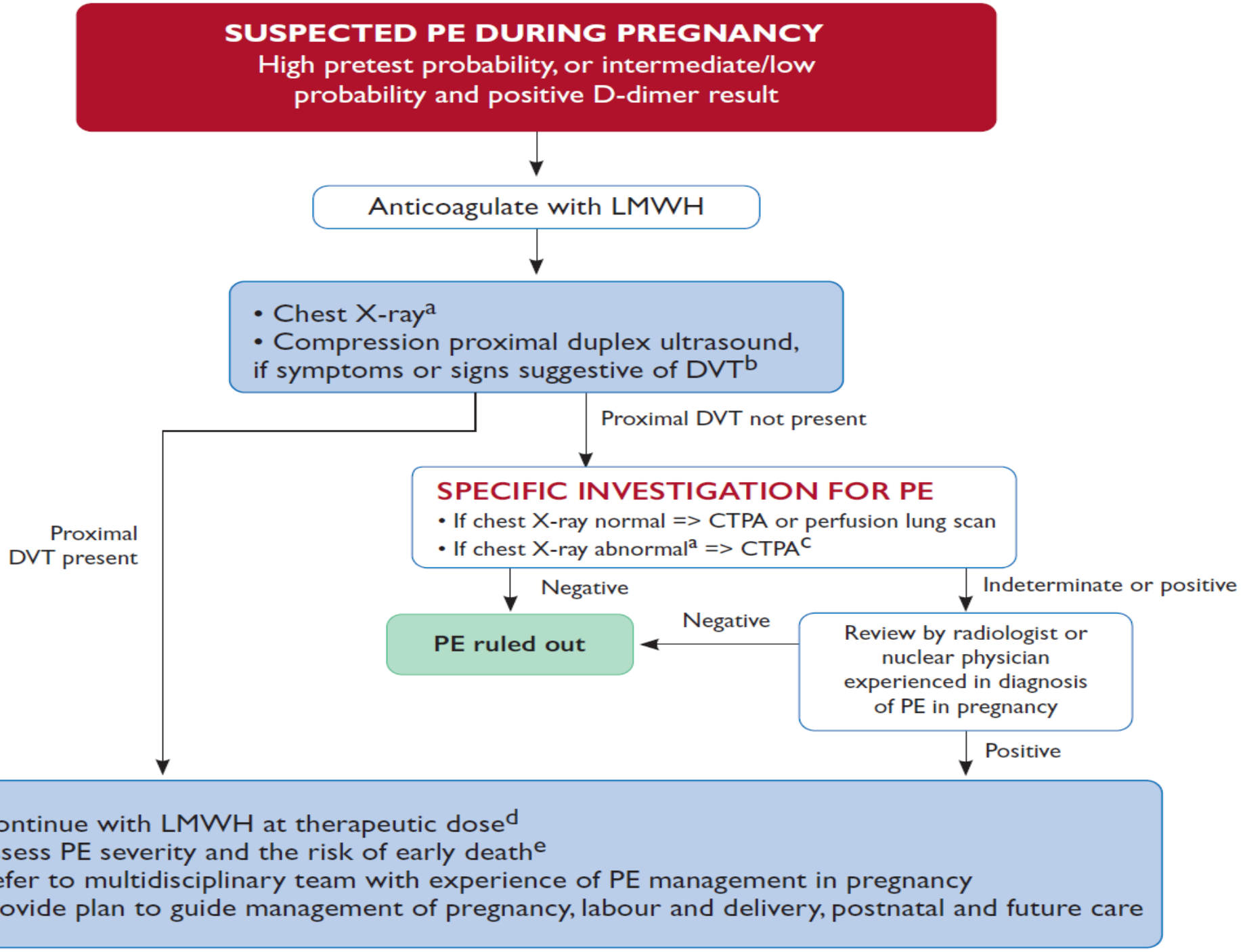

SUSPECTED PE DURING PREGNANCY
High pretest probability, or intermediate/low probability and positive D-dimer result
Anticoagulate with LMWH
• Chest X-ray[a]
• Compression proximal duplex ultrasound, if symptoms or signs suggestive of DVT[b]
Proximal DVT not present
Proximal DVT present
SPECIFIC INVESTIGATION FOR PE
• If chest X-ray normal => CTPA or perfusion lung scan
• If chest X-ray abnormal[a] => CTPA[c]
Negative
Indeterminate or positive
PE ruled out
Negative
Review by radiologist or nuclear physician experienced in diagnosis of PE in pregnancy
Positive
• Continue with LMWH at therapeutic dose[d]
• Assess PE severity and the risk of early death[e]
• Refer to multidisciplinary team with experience of PE management in pregnancy
• Provide plan to guide management of pregnancy, labour and delivery, postnatal and future care

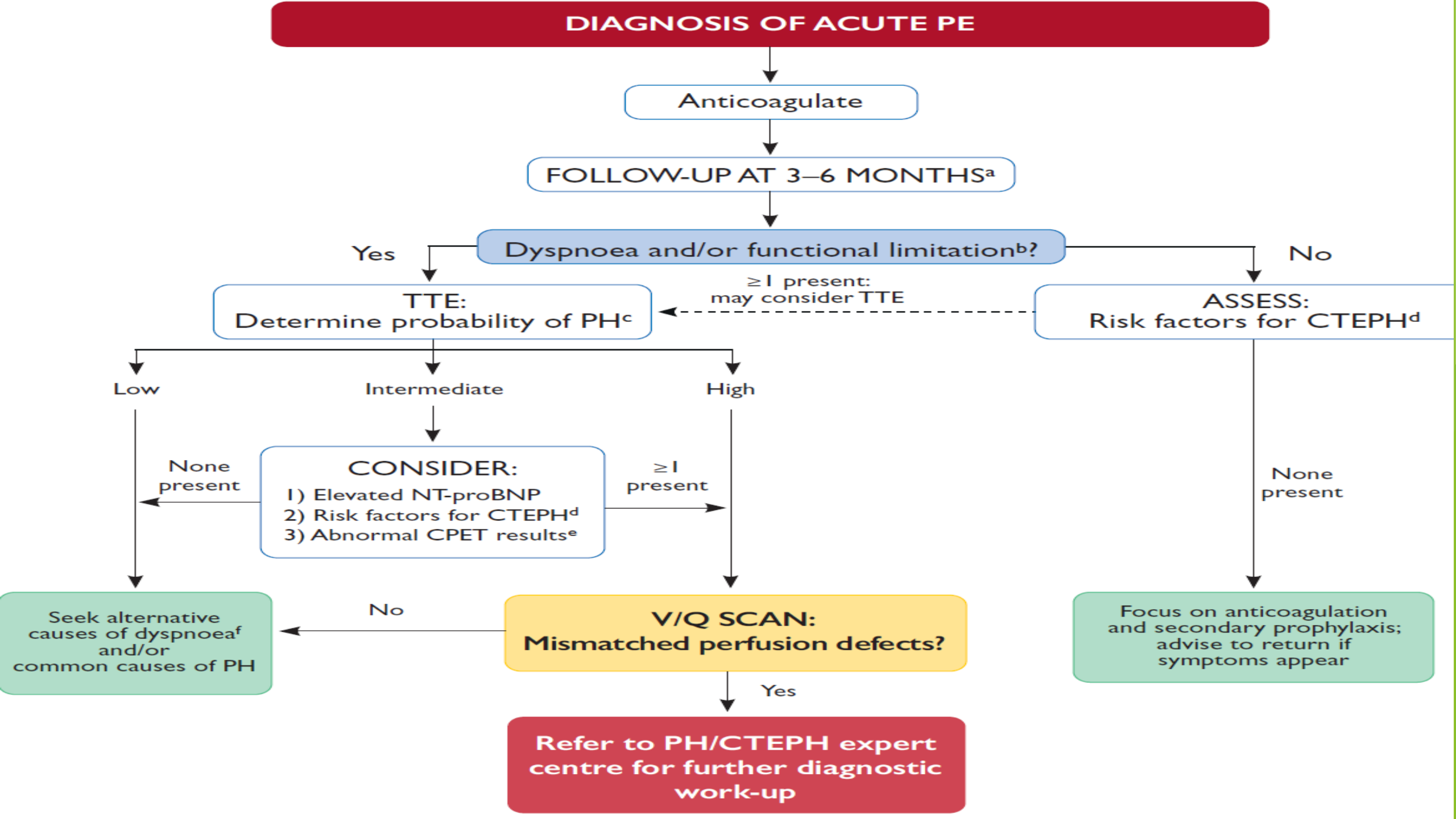
Table 12 Estimated amounts of radiation absorbed in procedures used to diagnose pulmonary embolism (based on various references[385,392–398])

Test	Estimated foetal radiation exposure (mGy)[a]	Estimated maternal radiation exposure to breast tissue (mGy)[a]
Chest X-ray	<0.01	<0.1
Perfusion lung scan with technetium-99m-labelled albumin		
Low dose: ~40 MBq	0.02–0.20	0.16–0.5
High dose: ~200 MBq	0.20–0.60	1.2
Ventilation lung scan	0.10–0.30	<0.01
CTPA	0.05–0.5	3–10

9.5 Recommendations for pulmonary embolism in pregnancy

Recommendations	Class[a]	Level[b]
Diagnosis		
Formal diagnostic assessment with validated methods is recommended if PE is suspected during pregnancy or in the post-partum period.[388,391]	I	B
D-dimer measurement and clinical prediction rules should be considered to rule out PE during pregnancy or the post-partum period.[388,391]	IIa	B
In a pregnant patient with suspected PE (particularly if she has symptoms of DVT), venous CUS should be considered to avoid unnecessary irradiation.[388]	IIa	B
Perfusion scintigraphy or CTPA (with a low-radiation dose protocol) should be considered to rule out suspected PE in pregnant women; CTPA should be considered as the first-line option if the chest X-ray is abnormal.[385,386]	IIa	C

Treatment		
A therapeutic, fixed dose of LMWH based on early pregnancy body weight is the recommended therapy for PE in the majority of pregnant women without haemodynamic instability.[408,410]	I	B
Thrombolysis or surgical embolectomy should be considered for pregnant women with high-risk PE.[421]	IIa	C
Insertion of a spinal or epidural needle is not recommended, unless ≥24 h have passed since the last therapeutic dose of LMWH.	III	C
Administration of LMWH is not recommended within 4 h of removal of an epidural catheter.	III	C
NOACs are not recommended during pregnancy or lactation.	III	C


DIAGNOSIS OF ACUTE PE
Anticoagulate
FOLLOW-UP AT 3–6 MONTHS[a]
Dyspnoea and/or functional limitation[b]?
Yes
No
≥1 present: may consider TTE
TTE: Determine probability of PH[c]
ASSESS: Risk factors for CTEPH[d]
Low
Intermediate
High
None present
CONSIDER:
1) Elevated NT-proBNP
2) Risk factors for CTEPH[d]
3) Abnormal CPET results[e]
≥1 present
None present
Seek alternative causes of dyspnoea[f] and/or common causes of PH
No
V/Q SCAN: Mismatched perfusion defects?
Yes
Focus on anticoagulation and secondary prophylaxis; advise to return if symptoms appear
Refer to PH/CTEPH expert centre for further diagnostic work-up