Acute Cor Pulmonale

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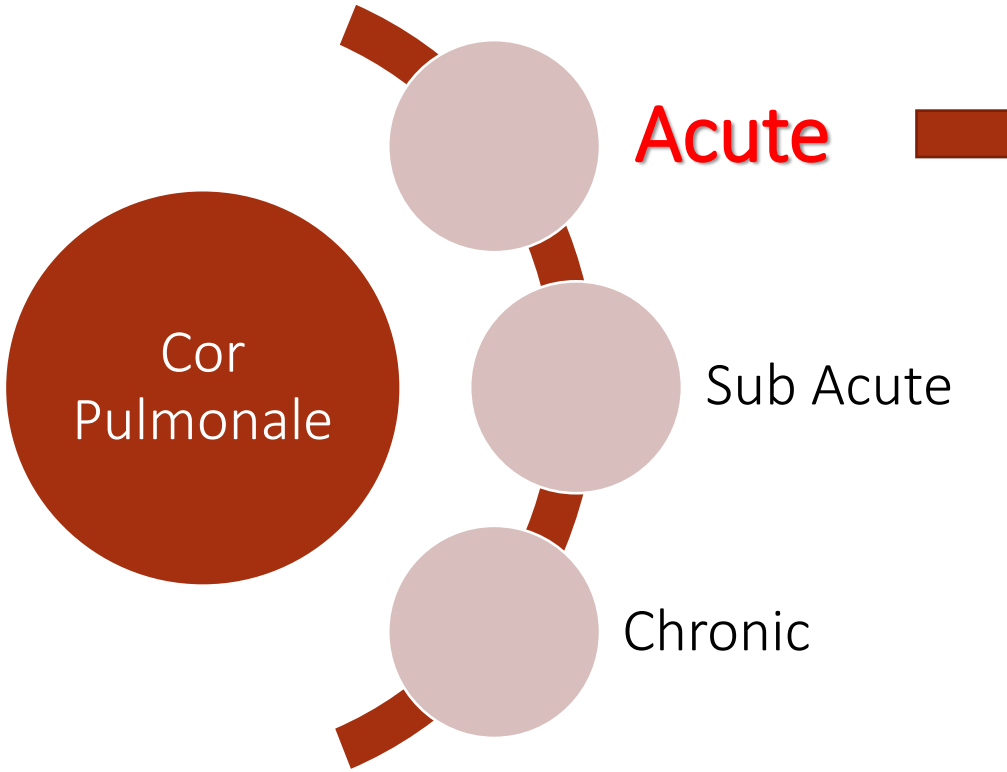
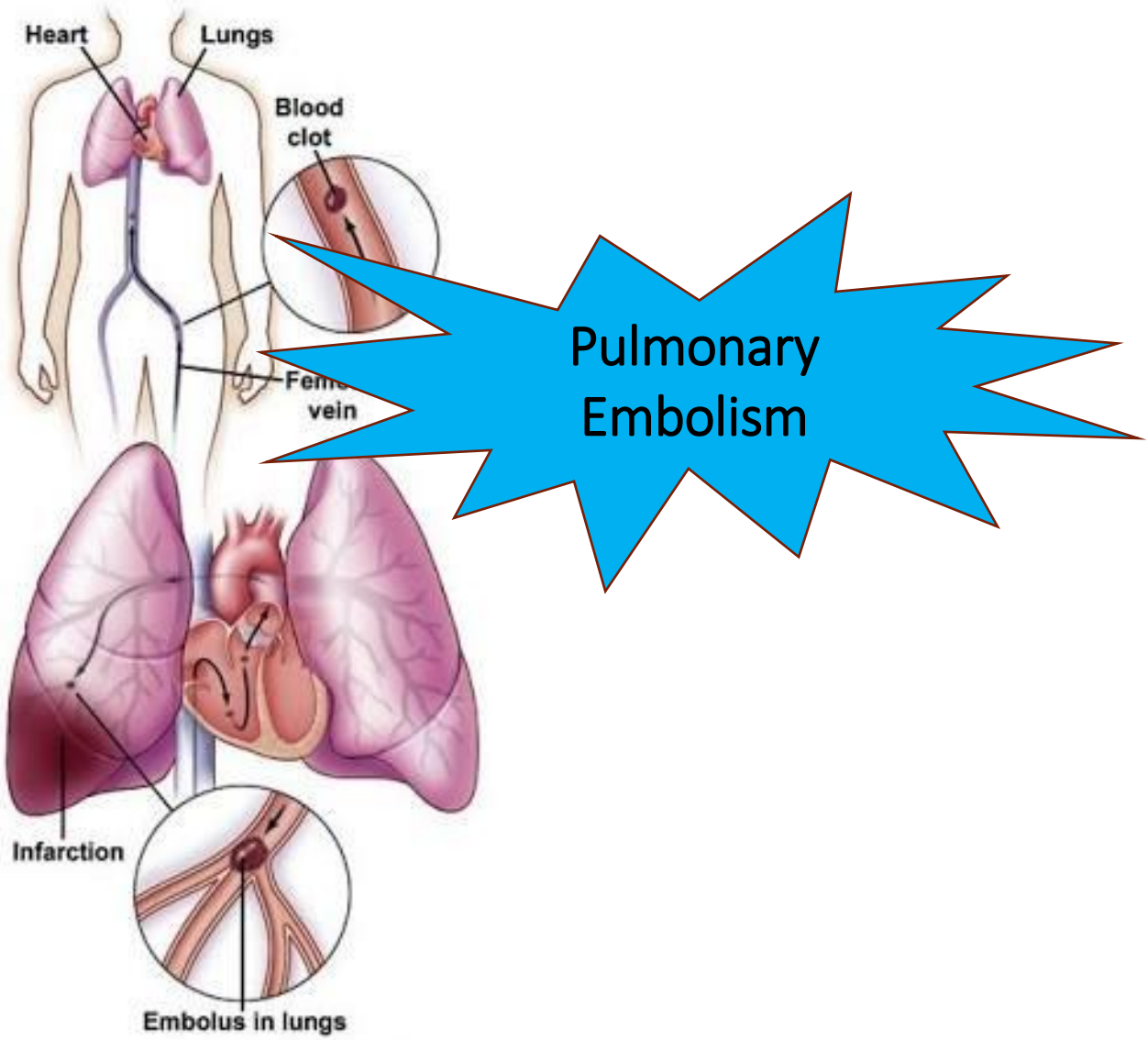
Cor Pulmonale (Pulmonary Heart Disease)



Goodwin, 1956



Acute Cor Pulmonale

Acute cor pulmonale is a form of ***acute right heart failure*** produced by a sudden increase in resistance to blood flow in the pulmonary circulation.

In clinical setting acute cor pulmonale mainly observed as a complication *Acute Respiratory Distress Syndrome* and *Massive Pulmonary Embolism*

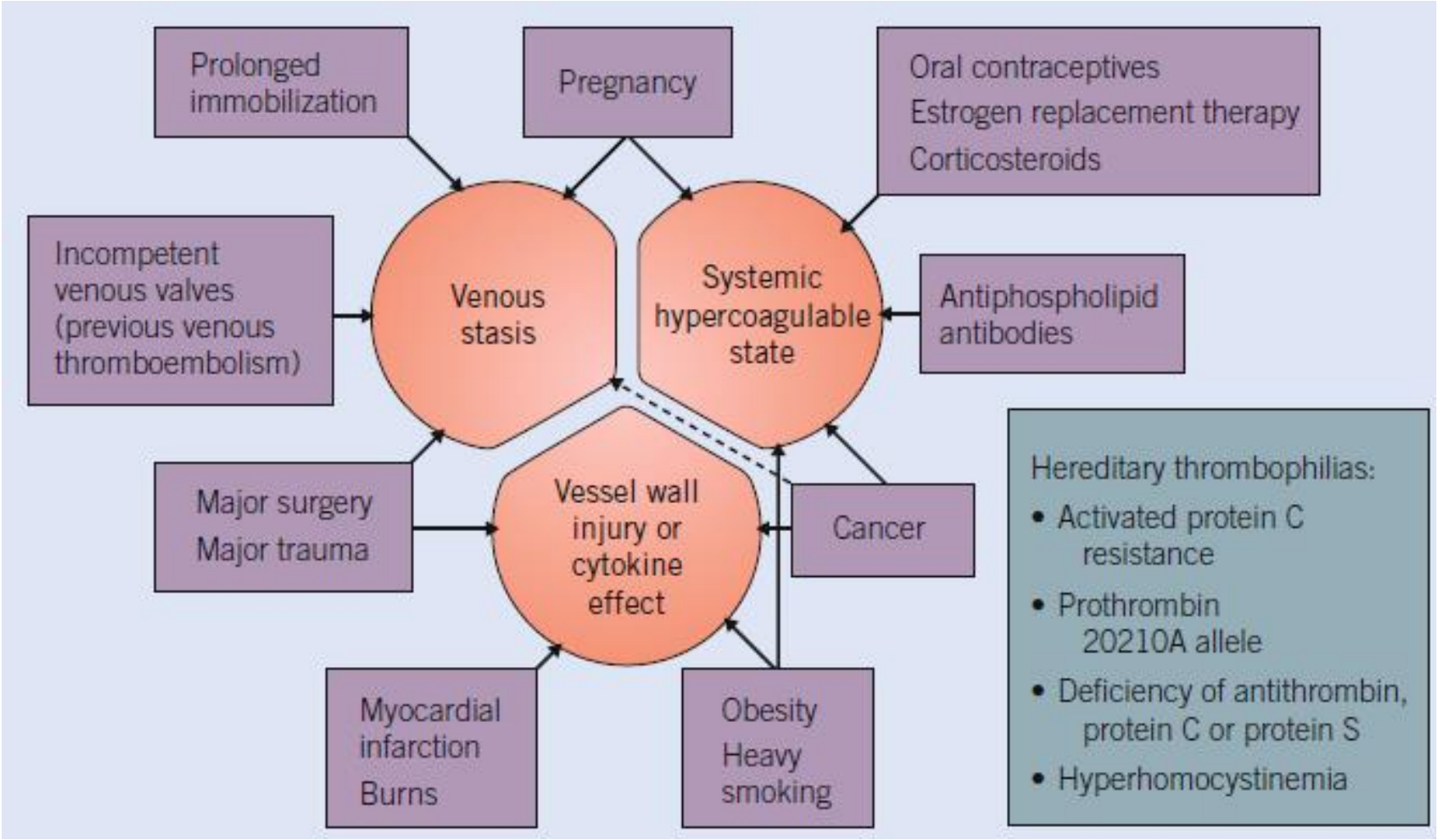
Jardin F, 2009

Pulmonary Embolism

- Pulmonary embolism (PE) and deep venous thrombosis are two clinical presentations of venous thromboembolism and share the same risk factors and predisposing conditions. In most cases, PE is a consequence of deep venous thrombosis of the lower extremities.
- VTE may be lethal in the acute phase or lead to chronic disease and disability.
- It is the third most frequent cardiovascular disease with an overall annual incidence of 100–200 per 100 000 inhabitants.
- Non Thromboembolic causes of PE are rare.

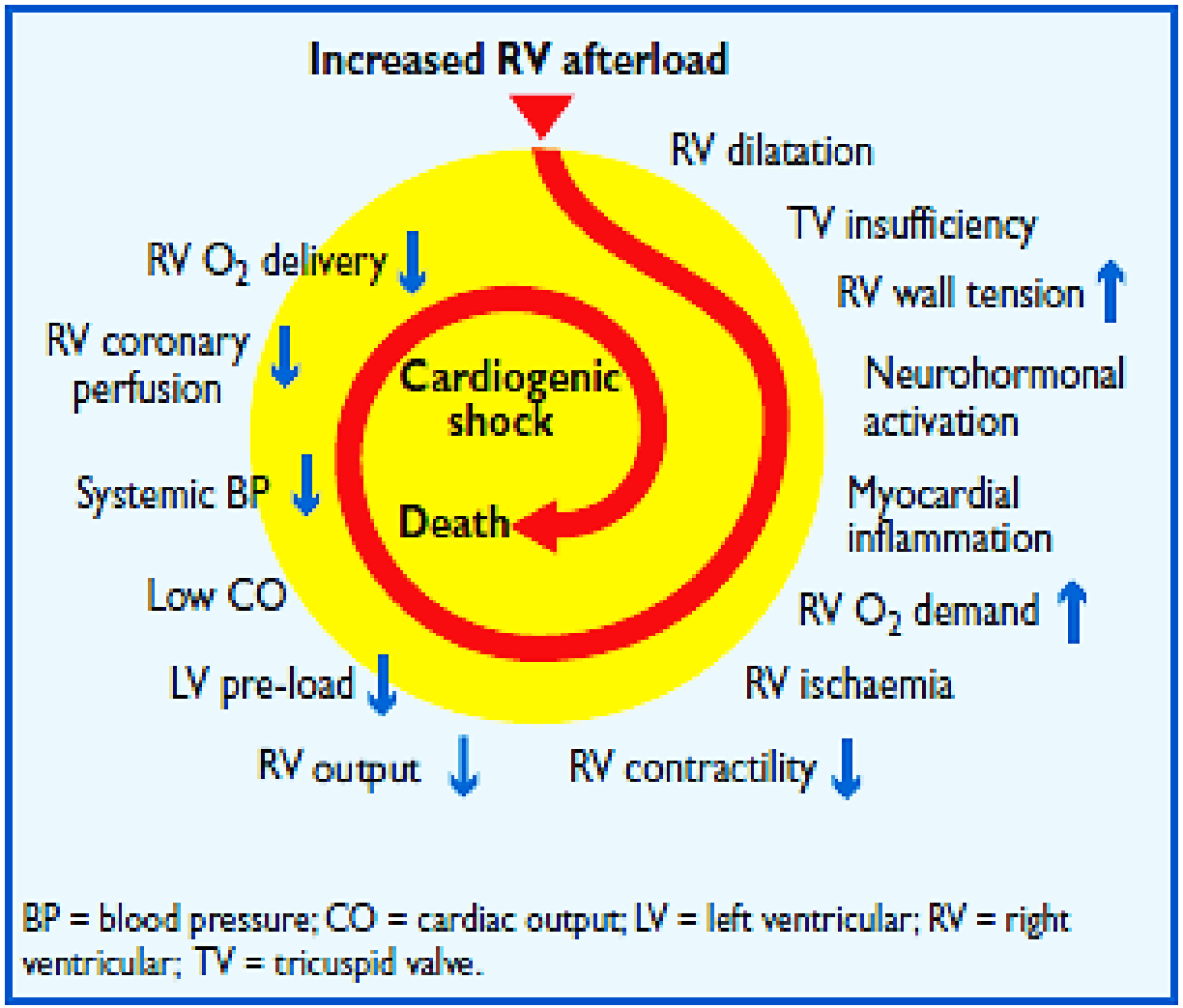
Konstantinides,2014

Pathogenesis of Veno thromboembolism



Konstantinides,2010

Pathophysiology



BP = blood pressure; CO = cardiac output; LV = left ventricular; RV = right ventricular; TV = tricuspid valve.

Konstantinides,2014

Diagnosis

Dx

- Clinical Presentation
- Assesment of Clinical Probability
- D-Dimer
- CT Angiography
- Echocardiography
- Pulmonary Angiography
- MRA
- Lung Scintigraphy

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%

Konstantinides,2014

Well Score

Items	Clinical decision rule points	
Wells rule	**Original version[95]**	**Simplified version[107]**
Previous PE or DVT	1.5	1
Heart rate ≥100 b.p.m.	1.5	1
Surgery or immobilization within the past four weeks	1.5	1
Haemoptysis	1	1
Active cancer	1	1
Clinical signs of DVT	3	1
Alternative diagnosis less likely than PE	3	1
Clinical probability		
Three-level score		
Low	0–1	N/A
Intermediate	2–6	N/A
High	≥7	N/A
Two-level score		
PE unlikely	0–4	0–1
PE likely	≥5	≥2

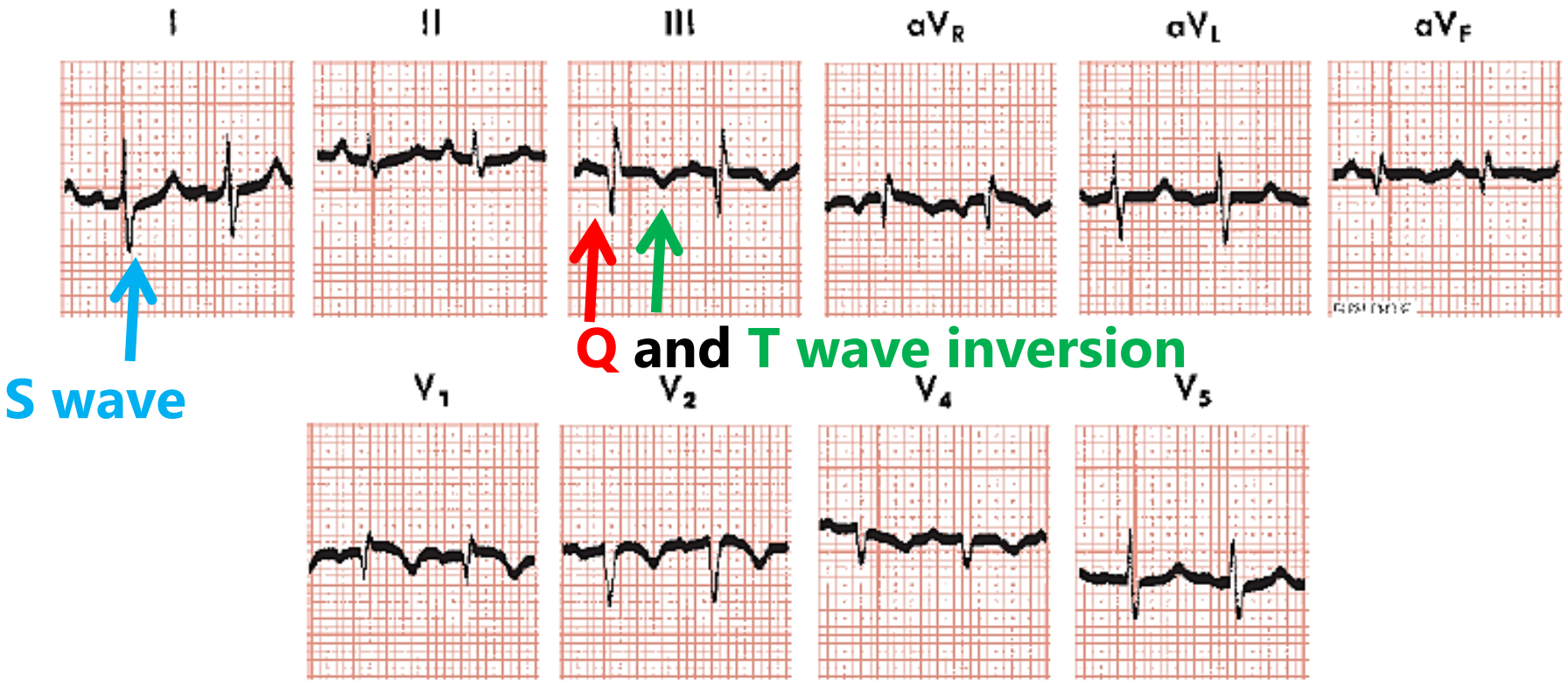
Konstantinides,2014

Revised Geneva Score

Revised Geneva score	Original version[93]	Simplified version[108]
Previous PE or DVT	3	1
Heart rate 75–94 b.p.m. ≥95 b.p.m.	 3 5	 1 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

Konstantinides,2014

Electrocardiography (ECG) feature



Features occasionally seen with PE include :

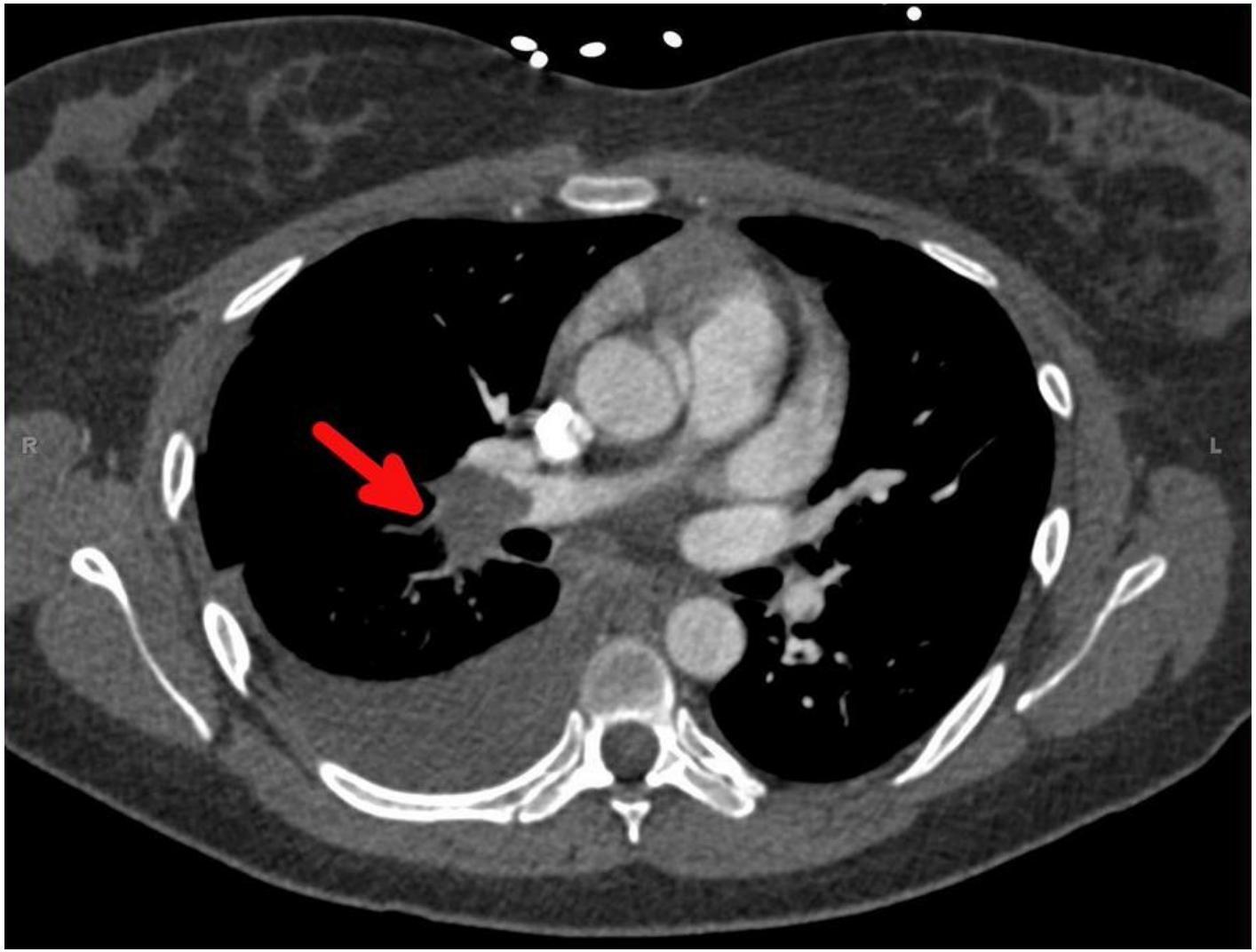
1. Sinus tachycardia
2. S waves in lead I with Q waves and T wave inversions in lead III (SIQIIITIII pattern)
3. Slow R wave progression with T wave inversions in chest leads V1 to V4 resulting from acute right ventricular overload.

Goldberger,2006

Echocardiography

- Dilatation of the right ventricle
- RV dysfunction in some cases with preservation of the motility of the apex (Mc Conell sign)
- Dilatation of the IVC with lack of collapse during inspiration
- Flattening of the interventricular septum suggesting right ventricu lar pressure overload
- Pulmonary hypertension based on the jet of tricuspid regurgitation

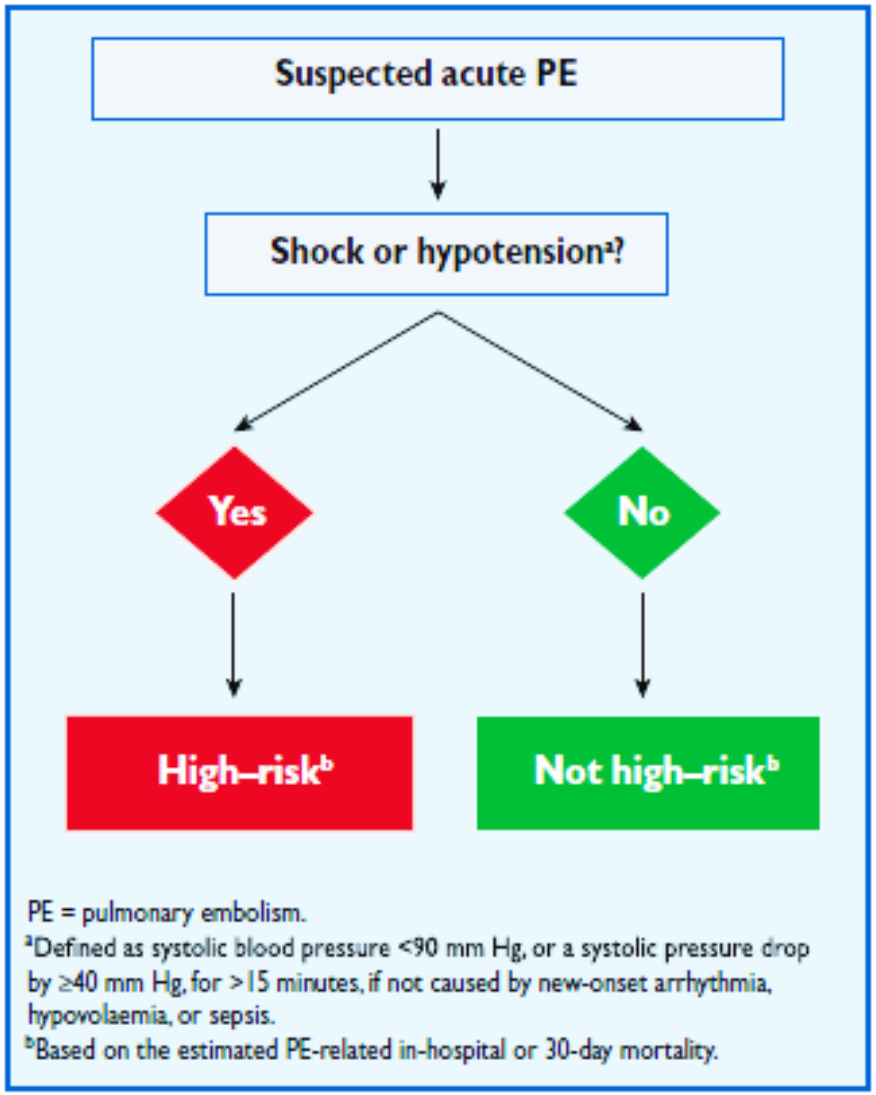
CT angiography



Computed tomography angiography (CTA) of the chest (*transverse view*) demonstrating thrombus in the *right* pulmonary artery (*red arrow*)

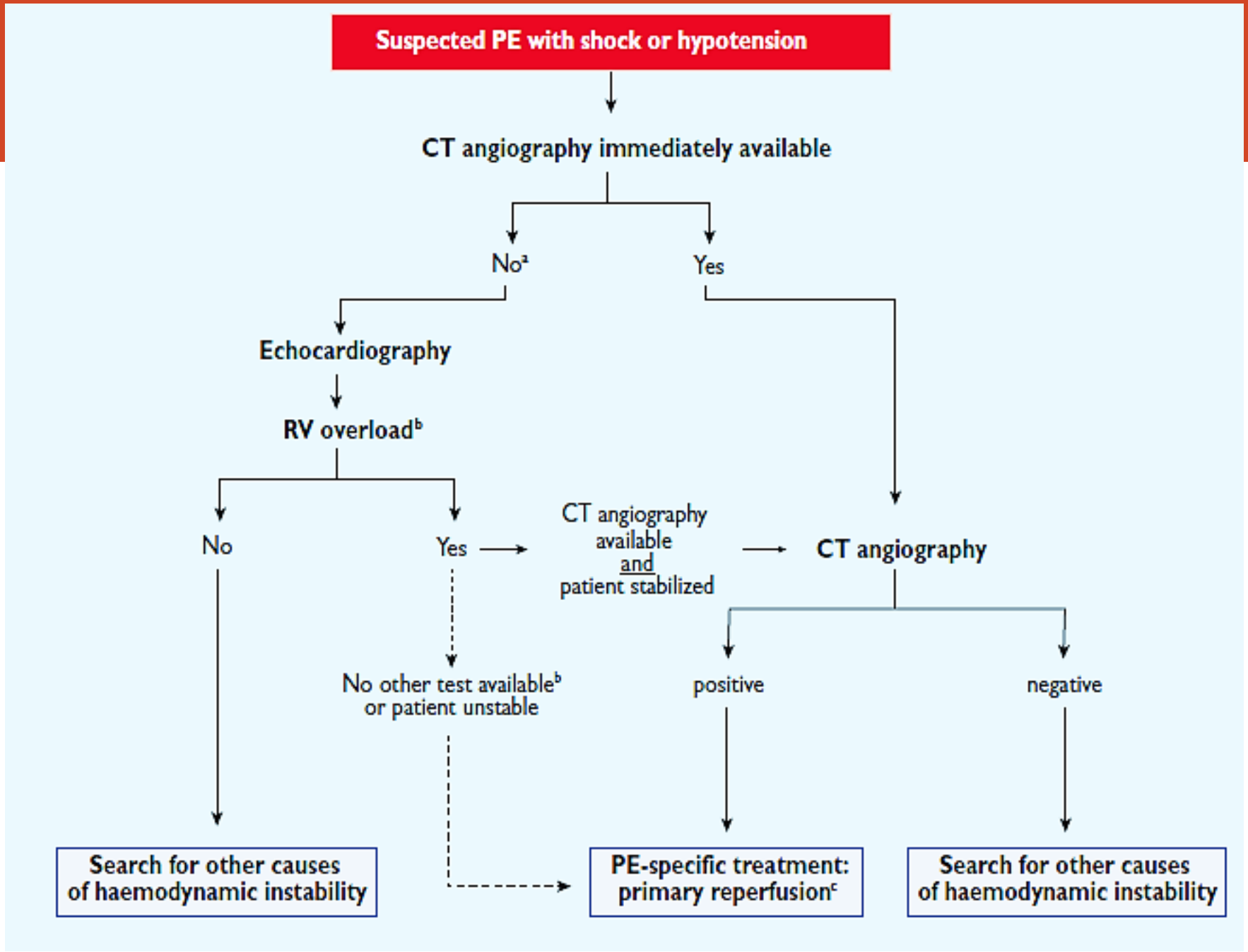
Sritika Tapha,2016

Diagnostic Strategy



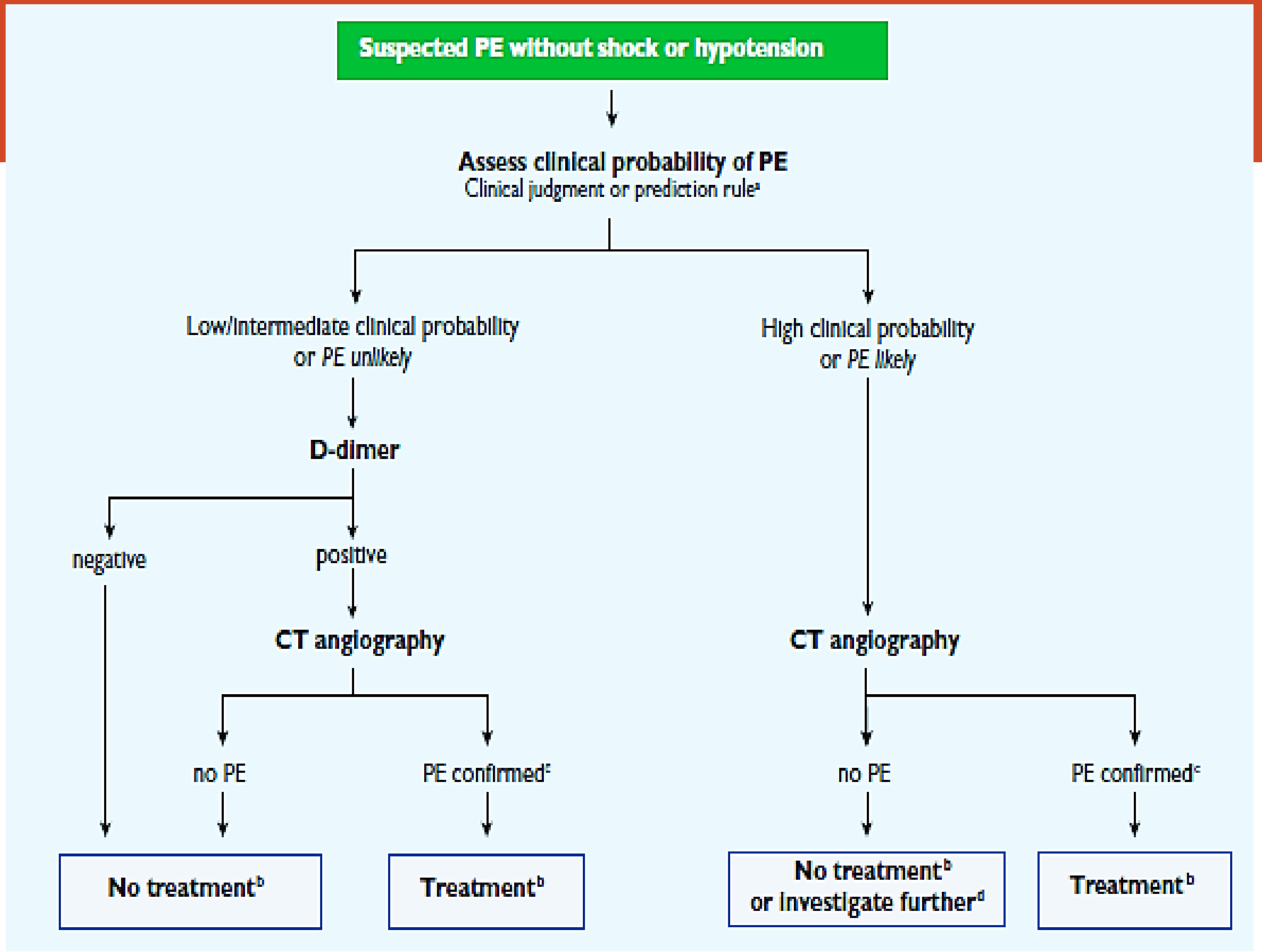
Konstantinides,2014

Diagnostic Strategy cont'd



Konstantinides,2014

Diagnostic Strategy cont'd



Konstantinides,2014

Treatment

T X	Haemodynamic and respiratory support
	Anticoagulation
	Thrombolytic
	Surgical embolectomy
	Percutaneous catheter directed treatment
	Vena cava filter

Anticoagulation

- Preventing early death and recurrent symptomatic or fatal VTE.
- Acute-phase treatment consists of administering parenteral anticoagulation (UFH, LMWH or fondaparinux] over the first 5–10 days.
- Parenteral heparin should overlap with the initiation of a vitamin K antagonist (VKA); alternatively, it can be followed by administration of one of the new oral anticoagulants (NOAC)

	Dosage	Interval
Enoxaparin	1.0 mg/kg or 1.5 mg/kg[a]	Every 12 hours Once daily[a]
Tinzaparin	175 U/kg	Once daily
Dalteparin	100 IU/kg[b] or 200 IU/kg[b]	Every 12 hours[b] Once daily[b]
Nadroparin[c]	86 IU/kg or 171 IU/kg	Every 12 hours Once daily
Fondaparinux	5 mg (body weight <50 kg); 7.5 mg (body weight 50–100 kg); 10 mg (body weight >100 kg)	Once daily

Konstantinides,2014

Secondary Prophylaxis (Oral Anticoagulation)

1.	First episode with transient risk factor (trauma, surgery, immobilization, pregnancy, contraceptive use, or hormonal replacement therapy	3 months
2.	First episode, unprovoked (no transient risk factor), or with low-risk thrombophilia (e.g., heterozygous activated protein C resistance, G20210A prothrombin mutation)	At least 3 months, preferably 6-12 months; consider indefinite treatment in selected patients with low bleeding risk
3.	First episode with homozygous or combined thrombophilia, antiphospholipid syndrome	At least 12 months
4.	Recurrent venous thromboembolism, active cancer	Indefinte anticoagulation

Konstantinides,2010

Thrombolysis

- It restores pulmonary perfusion more rapidly than anticoagulation
- The greatest benefit is observed when treatment is initiated within 48 hours of symptom onset, but thrombolysis can still be useful in patients who have had symptoms for 6–14 days
- It carries a risk of major bleeding, including intracranial haemorrhage
- An alternative approach may consist of local, catheter-delivered, ultrasound-assisted thrombolysis using small doses of a thrombolytic agent

Streptokinase	250 000 IU as a loading dose over 30 min, followed by 100 000 IU/h over 12–24 h
	Accelerated regimen: 1.5 million IU over 2 h
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h over 12–24 h
	Accelerated regimen: 3 million IU over 2 h
rtPA	100 mg over 2 h
	or 0.6 mg/kg over 15 min (maximum dose 50 mg)

rtPA = recombinant tissue plasminogen activator.

Adam Torbicky,2008

Thrombolysis Contraindication

Absolute

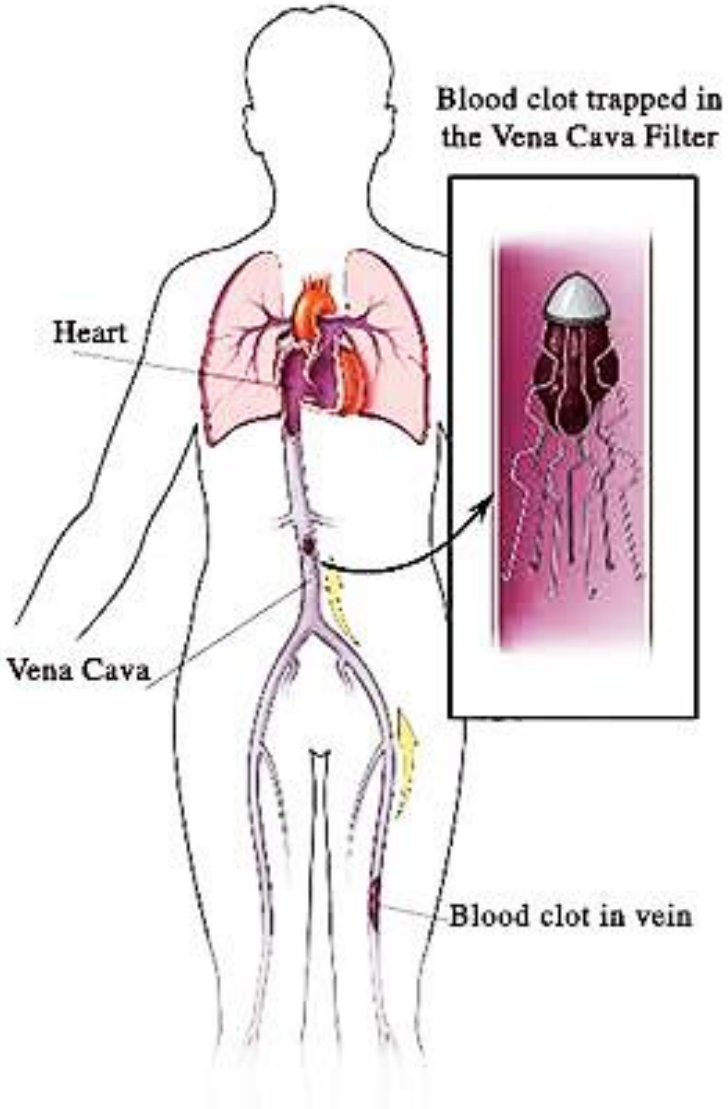
- Haemorrhagic stroke or stroke of unknown origin at any time
- Ischaemic stroke in preceding 6 months
- Central nervous system damage or neoplasms
- Recent major trauma/surgery/head injury (within preceding 3 weeks)
- Gastrointestinal bleeding within the last month
- Known bleeding

Relative

- Transient ischaemic attack in preceding 6 months
- Oral anticoagulant therapy
- Pregnancy or within 1 week post partum
- Non-compressible punctures
- Traumatic resuscitation
- Refractory hypertension (systolic blood pressure >180 mmHg)
- Advanced liver disease
- Infective endocarditis
- Active peptic ulcer

Adam Torbicky,2008

Venous Filter



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29 September 2018

WORLD HEART FEDERATION

World Heart Day
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HARI JANTUNG SEDUNIA