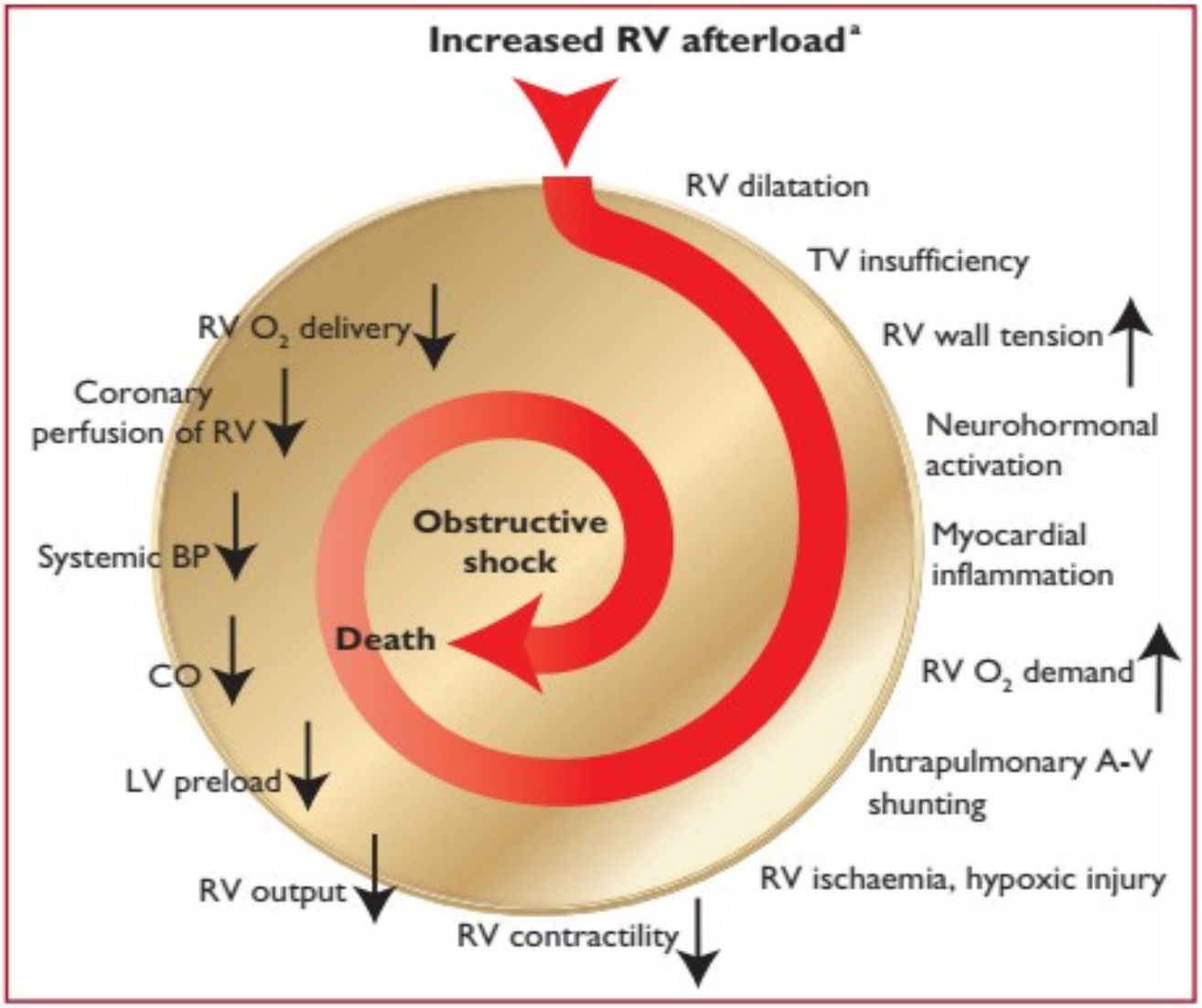
SIMA 23
Σι-μα Ιητηρ(ιατρός) Μινωϊκή Κρήτη

ΙΑΤΡΙΚΟΣ ΣΥΛΛΟΓΟΣ ΗΡΑΚΛΕΙΟΥ

Κλινική εικόνα και αντιμετώπιση Πνευμονικής Εμβολής

Κωνσταντίνος Καραγιάννης
Πνευμονολόγος - Φυματιολόγος
Επιμελητής Β'

PE can kill!!!



Konstantinides SV et al, *Eur Heart J 2020*

PE can kill!!!

Table 6: Total non-fatal venous thromboembolism (VTE) events, VTE-related deaths, and associated outcomes across all six European Union countries modeled* in 2004.

Event, n (95% CI)	Community-acquired	Hospital-acquired	Total
Non-fatal VTE event			
Deep-vein thrombosis	200,482 (172,548–226,239)	265,233 (209,844–332,407)	465,715 (404,664–538,189)
Pulmonary embolism	86,511 (73,967–99,626)	209,471 (153,817–273,371)	295,982 (242,450–360,363)
VTE-related deaths†	108,535 (77,243–178,968)	261,477 (211,782–325,823)	370,012 (300,193–483,108)
Treated VTE†	8,124 (6,151–10,470)	18,349 (12,422–25,695)	26,473 (19,158–35,271)
Untreated VTE	63,541 (41,574–114,074)	153,853 (110,943–211,670)	217,394 (154,910–317,068)
Sudden death	36,870 (25,467–60,724)	89,275 (64,718–117,822)	126,145 (92,352–170,949)

Deaths from PE

- *7% diagnosed PE*
- *34% sudden fatal PE*
- *59% undiagnosed (untreated) PE*

Cohen AT et al, *Thromb Haemost 2007*

Early treatment saves lives!

CHEST

Original Research

ANTITHROMBOTIC THERAPY

Early Anticoagulation Is Associated With Reduced Mortality for Acute Pulmonary Embolism

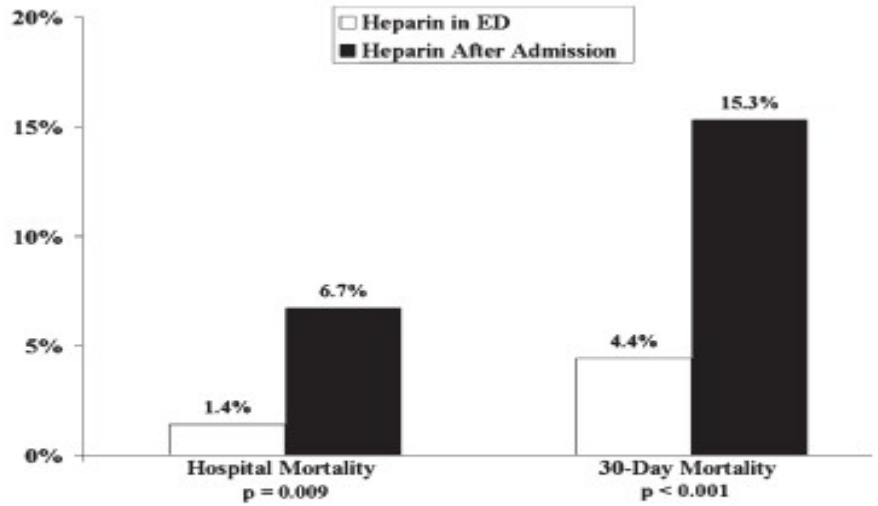
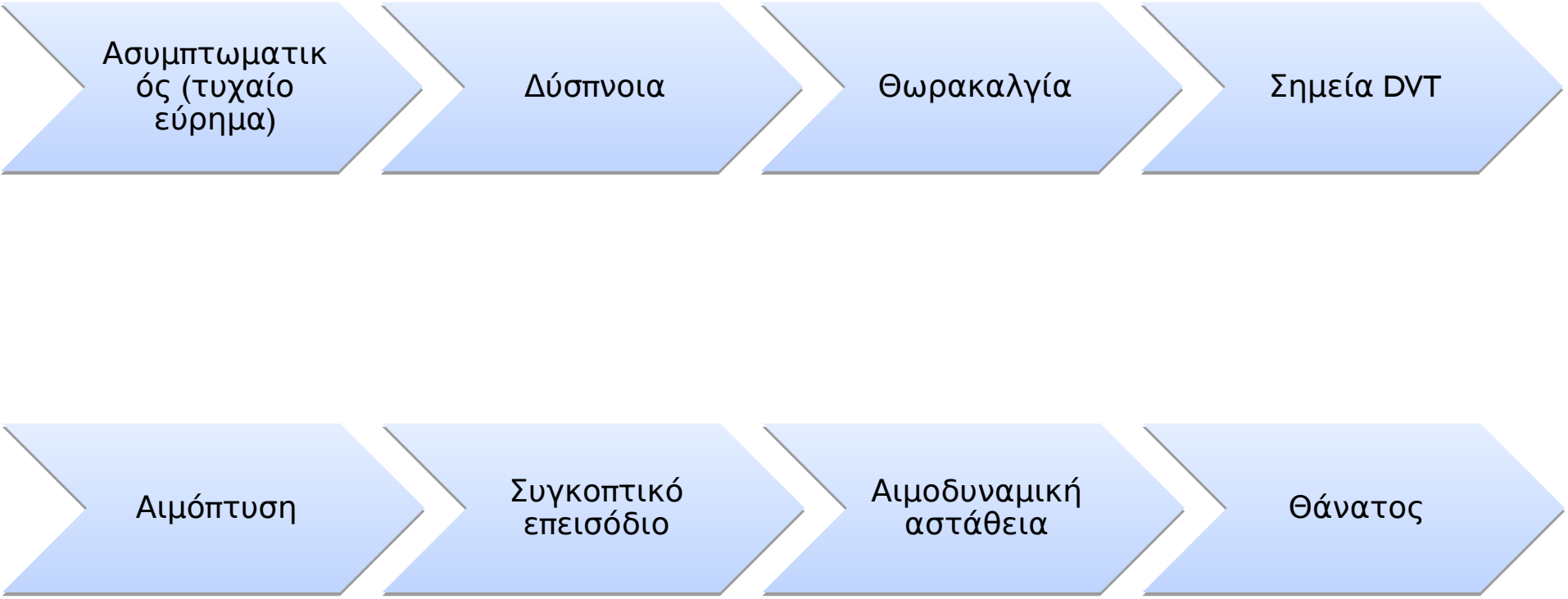


FIGURE 1. Hospital and 30-day mortality rates for patients who received heparin in the ED compared with those who received heparin after admission.

Smith SB et al, *Chest 2010*

Clinical presentation



Clinical presentation (symptoms)

Table 4 Symptoms of Pulmonary Embolism

	PE No Prior CPD N = 127-133 n (%)	No PE No Prior CPD N = 361-366 n (%)	PE All Patients N = 184-191 n (%)	No PE All Patients N = 622-632 n (%)
Dyspnea				
Dyspnea (rest or exertion)	97 (73)	248 (68)	151 (79)	459 (73)
Dyspnea (at rest)#	73 (55)	167 (46)	117 (61)	338 (54)
Dyspnea (exertion only)#	21 (16)	73 (20)	31 (16)	111 (18)
Orthopnea (≥2-pillow)	37 (28)	88 (24)	69 (36)	220 (35)
Pleuritic pain	58 (44)	207 (57)^	89 (47)	376 (59)^
Chest pain (not pleuritic)	25 (19)	80 (22)	33 (17)	130 (21)
Cough	45 (34)*	103 (28)**	82 (43)†	248 (39)††
Wheezing	27 (21)	66 (18)	58 (31)	193 (31)
Calf or thigh swelling	52 (41)	62 (17)^^	72 (39)	126 (20)^^
Calf and thigh swelling	9 (7)	14 (4)	15 (8)	35 (6)
Calf or thigh pain	56 (44)	83 (23)^^	78 (42)	156 (25)^^^
Calf and thigh pain	22 (17)	24 (7)^^	30 (16)	61 (10)^^^

Stein PD et al, *Am J Med 2007*

Clinical presentation - Syncope

Prevalence of Pulmonary Embolism among Patients Hospitalized for Syncope

Paolo Prandoni, M.D., Ph.D., Anthonie W.A. Lensing, M.D., Ph.D., Martin H. Prins, M.D., Ph.D., Maurizio Ciammaichella, M.D., Marica Perlati, M.D., Nicola Mumoli, M.D., Eugenio Bucherini, M.D., Adriana Visonà, M.D., Carlo Bova, M.D., Davide Imberti, M.D., Stefano Campostrini, Ph.D., and Sofia Barbar, M.D., for the PESIT Investigators*

CONCLUSIONS

Pulmonary embolism was identified in nearly one of every six patients hospitalized for a first episode of syncope. (Funded by the University of Padua; PESIT Clinical-Trials.gov number, NCT01797289.)

Stein PD et al, *NEJM 2016*

JAMA Internal Medicine | Original Investigation | LESS IS MORE

Prevalence of Pulmonary Embolism in Patients With Syncope

Giorgio Costantino, MD; Martin H. Ruwald, MD, PhD; James Quinn, MD; Carlos A. Camargo Jr, MD, DrPH; Frederik Dalgaard, MD; Gunnar Gislason, MD, PhD; Tadahiro Goto, MD, MPH; Kohei Hasegawa, MD, MPH; Padma Kaul, PhD; Nicola Montano, MD, PhD; Anna-Karin Numé, MD; Antonio Russo, MD; Robert Sheldon, MD, PhD; Monica Solbiati, MD; Benjamin Sun, MD; Giovanni Casazza, PhD

CONCLUSIONS AND RELEVANCE Pulmonary embolism was rarely identified in patients with syncope. Although PE should be considered in every patient, not all patients should undergo evaluation for PE.

Clinical presentation (signs)

Table 6 Signs of Pulmonary Embolism

	PE	No PE	PE All Patients N = 184-191 n (%)	No PE All Patients N = 602-629 n (%)
General				
Tachypnea (≥20/min)			108 (57)	296 (47)§
Tachycardia (>100/min)			49 (26)	98 (16)§
Diaphoresis			8 (4)	40 (6)
Cyanosis			1 (1)	1 (0)
Temperature > 38.5°C (>10			3 (2)	14 (2)
Cardiac examination (abnorma			42 (22)	72 (12)‖
Increased P2†			22 (15)	27 (5)‖
Right ventricular lift‡			8 (5)	9 (2)#
Jugular venous distension			25 (13)	50 (8)**
Lung examination (abnormal)			70 (37)	227 (36)
Rales (crackles)			40 (21)	112 (18)
Wheezes			6 (3)	54 (9)#
Rhonchi			9 (5)	32 (5)
Decreased breath sounds			40 (21)	109 (17)
Pleural friction rub			2/ (1)	5 (1)
DVT signs††				
Calf or thigh			90 (47)	146 (23)‖
Calf and thigh			23 (12)	30 (5)‖

CLINICAL SIGNIFICANCE

- In patients with pulmonary embolism only in the segmental pulmonary branches, generally recognized symptoms may be mild or absent, leading to underdiagnosis.
- Absence of symptoms is most common in patients with pulmonary embolism only in the segmental pulmonary branches, but this also occurs in patients with proximal pulmonary embolism.
- A low-probability objective clinical assessment does not exclude pulmonary embolism, even in the proximal pulmonary arteries.

Stein PD et al, *Am J Med 2007*

Clinical presentation - ABGs

Table 2 Arterial Blood Gases and Alveolar-Arterial Oxygen Difference While Breathing Room Air

	PE No Prior CPD N = 48 n (%)	No PE No Prior CPD N = 88 n (%)	PE All Patients N = 74 n (%)	No PE All Patients N = 186 n (%)
Pao_2 (mm Hg)				
≤49	1 (2)	2 (2)	4 (5)	17 (9)
50-59	6 (13)	12 (14)	12 (16)	32 (17)
60-69	15 (31)	14 (16)*	20 (27)	35 (19)
70-79	8 (17)	13 (15)	14 (19)	32 (17)
≥80	18 (38)	47 (53)	24 (32)	70 (38)
$Paco_2$ (mm Hg)				
≤35	30 (63)	39 (44)*	42 (57)	65 (35)‡
36-39	12 (25)	17 (19)	18 (24)	39 (21)
≥40	6 (13)	32 (36)	14 (19)	82 (44)§
pH (units)				
<7.35	0 (0)	7 (8)*	0 (0)	13 (7)†
7.35-7.45	29 (60)	60 (68)	41 (55)	131 (70)†
>7.45	19 (40)	21 (24)	33 (45)	42 (23)§
A-a O_2 difference (mm Hg)				
≤20	17 (35)	44 (50)	24 (32)	70 (38)
21-30	4 (8)	10 (11)	5 (7)	32 (17)*
31-40	11 (23)	13 (15)	18 (24)	30 (16)
41-50	9 (19)	13 (15)	14 (19)	32 (17)
51-60	5 (10)	6 (7)	10 (14)	17 (9)
≥61	2 (4)	2 (2)	3 (4)	5 (3)

Stein PD et al, *Am J Med 2007*

Clinical presentation - ECG

Table 2 Frequency of ECG findings for all patients and controls, and for subgroups by clot load and absence of cardiorespiratory disease

ECG finding				
Patients				
Normal ECG				
Any abnormality				
Sinus tachycardia				
RBBB				
RV strain				
RAD				
P pulmonale	1 (0.5) 0 (0.0)	1 (1.3) 0 (0.0)	0 (0.0) 0 (0.0)	1 (1.1) 0 (0)
S1Q3T3	7 (3.7) 1 (0.5)	5 (6.6) 1 (1.3)	2 (1.8) 0 (0.0)	4 (4.6) 0 (0)
Clockwise rotation	38 (20.1) 029 (15.3)	15 (19.7) 12 (15.8)	23 (20.4) 17 (15.0)	15 (17.2) 16 (18)
Atrial tachyarrhythmias	19 (10.1) 24 (12.7)	7 (9.2) 9 (11.8)	12 (10.6) 15 (13.3)	6 (6.9) 11 (13)

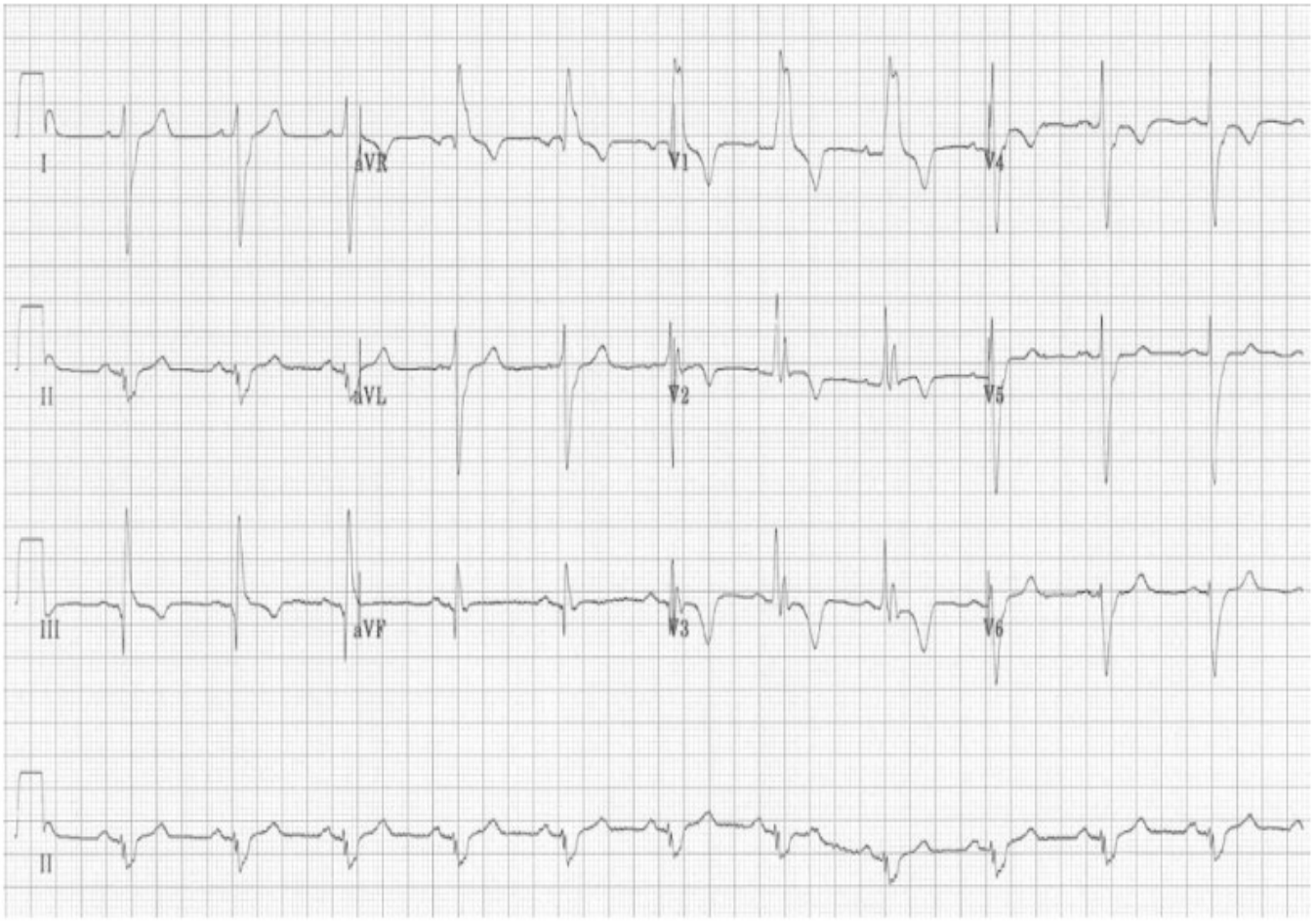
RAD, right axis deviation; RBBB, right bundle branch block; RV, right ventricular.

Main messages

- The ECG finding that best predicted pulmonary embolism (PE) in our study was right ventricular (RV) strain pattern.
- S1Q3T3 was uncommon in our study.
- An ECG showing RV strain when present in a breathless patient is highly suggestive of PE.
- Many of the other ECG changes that have been described in PE occur too infrequently to be of predictive value.

Thomson D et al, *Postgrad Med J 2019*

Clinical presentation - ECG

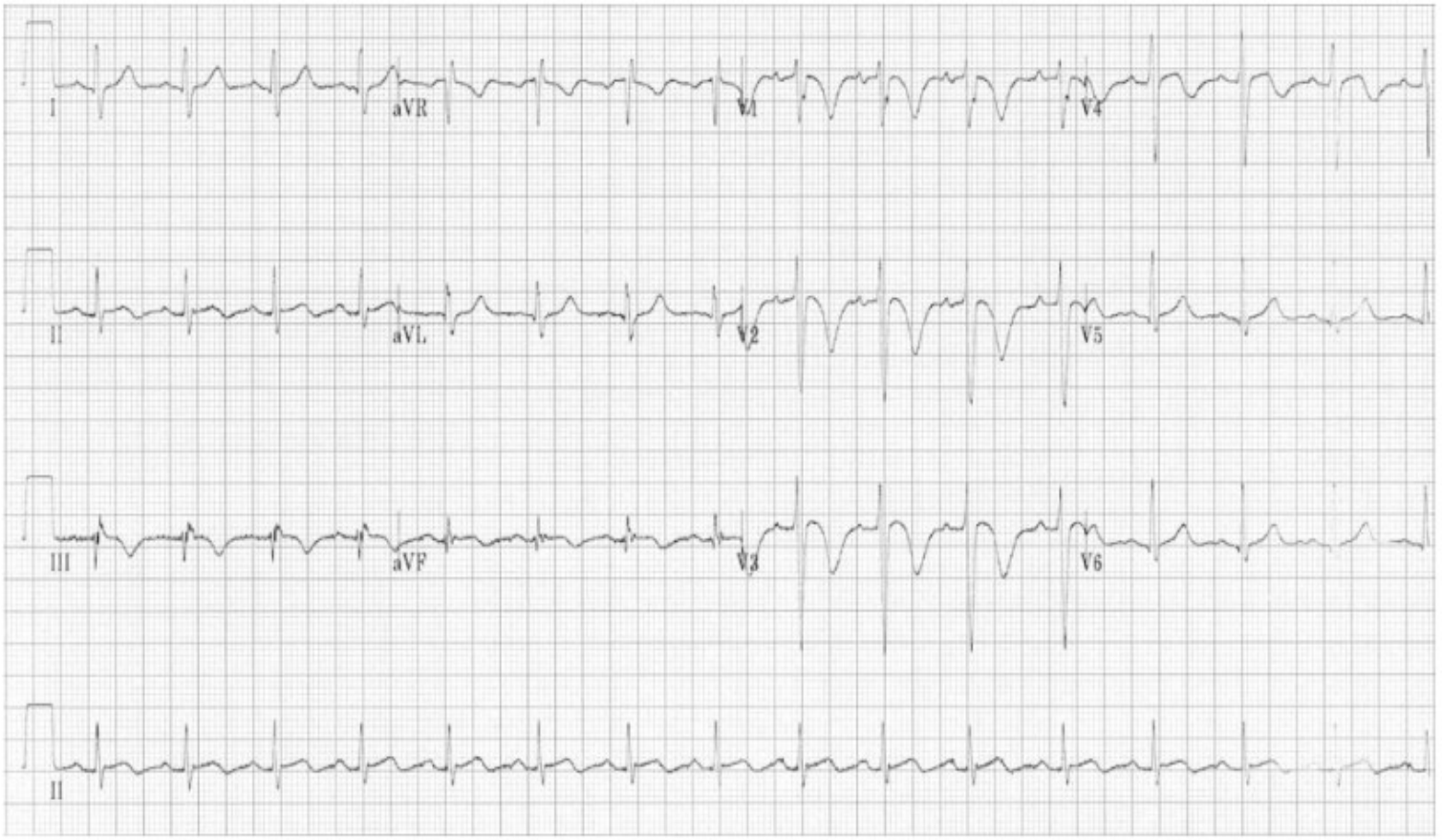


Σπάνιο

- SiQiiiTiii
- RBBB
- Rt axis

https://litfl.com/ecg-changes-in-pulmonary-embolism/

Clinical presentation - ECG



- Sinus tachy
- T-wave inversion V1-4 and inferior (II, III, aVF)

https://litfl.com/ecg-changes-in-pulmonary-embolism/

Clinical presentation - CXR

Table 2

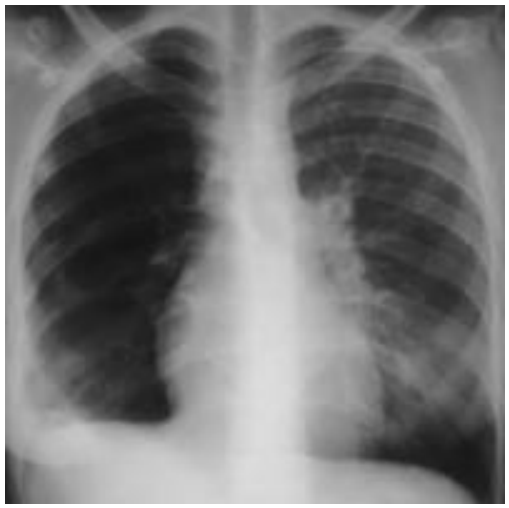
Chest Radiographic Findings for the Right Hemithorax in 259 Patients with the Angiographic Diagnosis of Right-sided PE and 680 Patients in Whom PE Was Angiographically Excluded

Finding	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	*P* Value
Oligemia (Westermark sign)	14	92	38	76	<.05*
Vascular redistribution	10	87	21	74	NS
Pleural-based areas of increased opacity (Hampton hump)	22	82	29	76	NS
Pleural effusion	36	70	28	76	NS
Elevated diaphragm	20	85	30	76	NS

* Patient with oligemia in the right hemithorax more likely to have PE.

Worsley DF et al, *Radiology 1993*

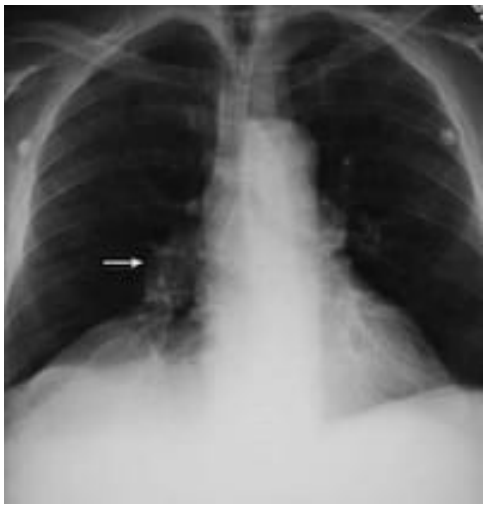
Clinical presentation - CXR

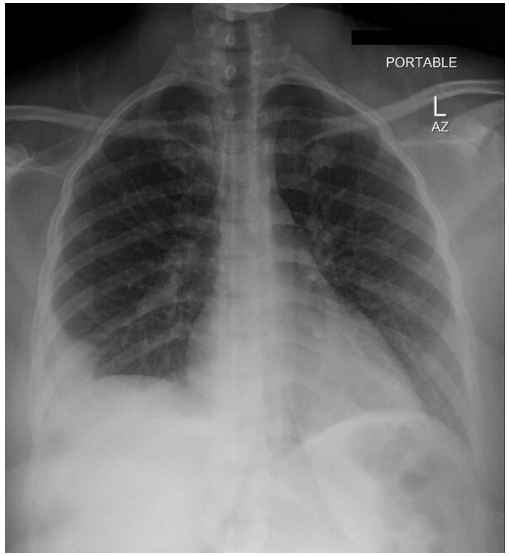


Westermark sign

- Sens 14%
- Spe 92%

Fleishner sign

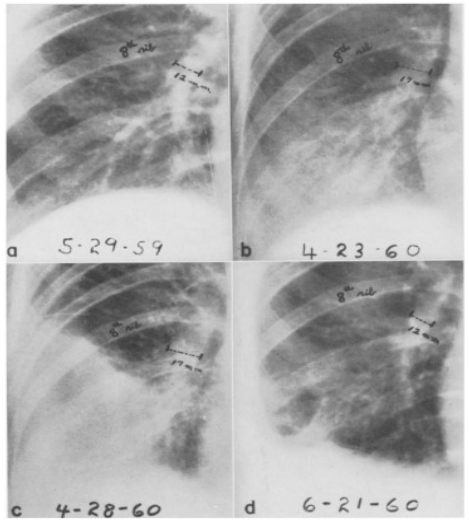




Hampton Hump

- Sens 22%
- Spe 82%

Chang sign



https://litfl.com/cxr-eponyms-in-pulmonary-embolism/

Clinical presentation – to summarize..

- Μη ειδική συμπτωματολογία των ασθενών με ΠΕ
- Κλινικά σημεία με πολύ χαμηλή ευαισθησία
- Υψηλό επίπεδο υποψίας – σε ποιους ασθενείς θα εφαρμόσουμε το πρωτόκολλο διερεύνησης ΠΕ
- Diagnostic tests overuse vs PE misdiagnosis (increased mortality)

General Considerations – Predisposing factors

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

Moderate risk factors (OR 2–9)
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

Permanent vs Temporary
Major vs Minor

Diagnosis – Pre test probability

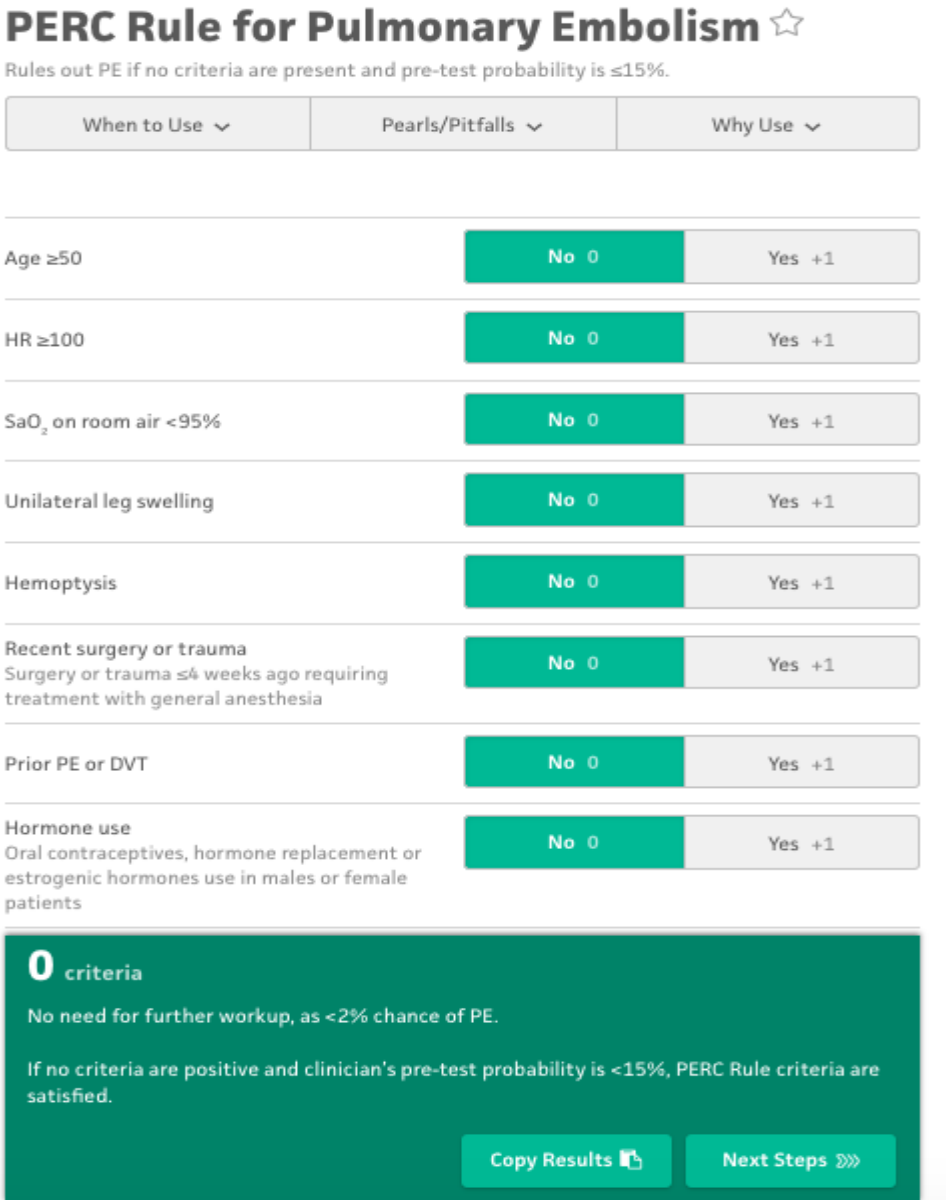
Wells score		Revised Geneva score	
Variable	Points	Variable	Points
Predisposing factors		Predisposing factors	
Previous DVT or PE	+1.5	Age >65	+1
Recent surgery/immobilization	+1.5	Previous DVT or PE	+3
Cancer	+1	Surgery or fracture Within 1 month	+2
		Active malignancy	+2
Symptoms		Symptoms	
Haemoptysis	+1	Unilateral lower limb pain	+3
		Haemoptysis	+2
Clinical signs		Clinical signs	
Heart rate >100/min	+1.5	Heart rate	+3
		75-94/min	+5
		≥ 95/min	
Clinical signs of DVT	+3		
Clinical judgement			
Alternative diagnosis less than PE	+3	Pain on lower limb deep vein at palpation and unilateral oedema	+4
Clinical probability	Total	Clinical probability	Total
Low	0-1	Low	0-3
Intermediate	2-6	Intermediate	4-10
High	≥ 7	High	≥ 11
Clinical probability (2 levels)			
PE unlikely	0-4		
PE likely	0-4		

Proportion of pts with confirmed PE

Low – 10%, Moderate – 30%, High – 65%

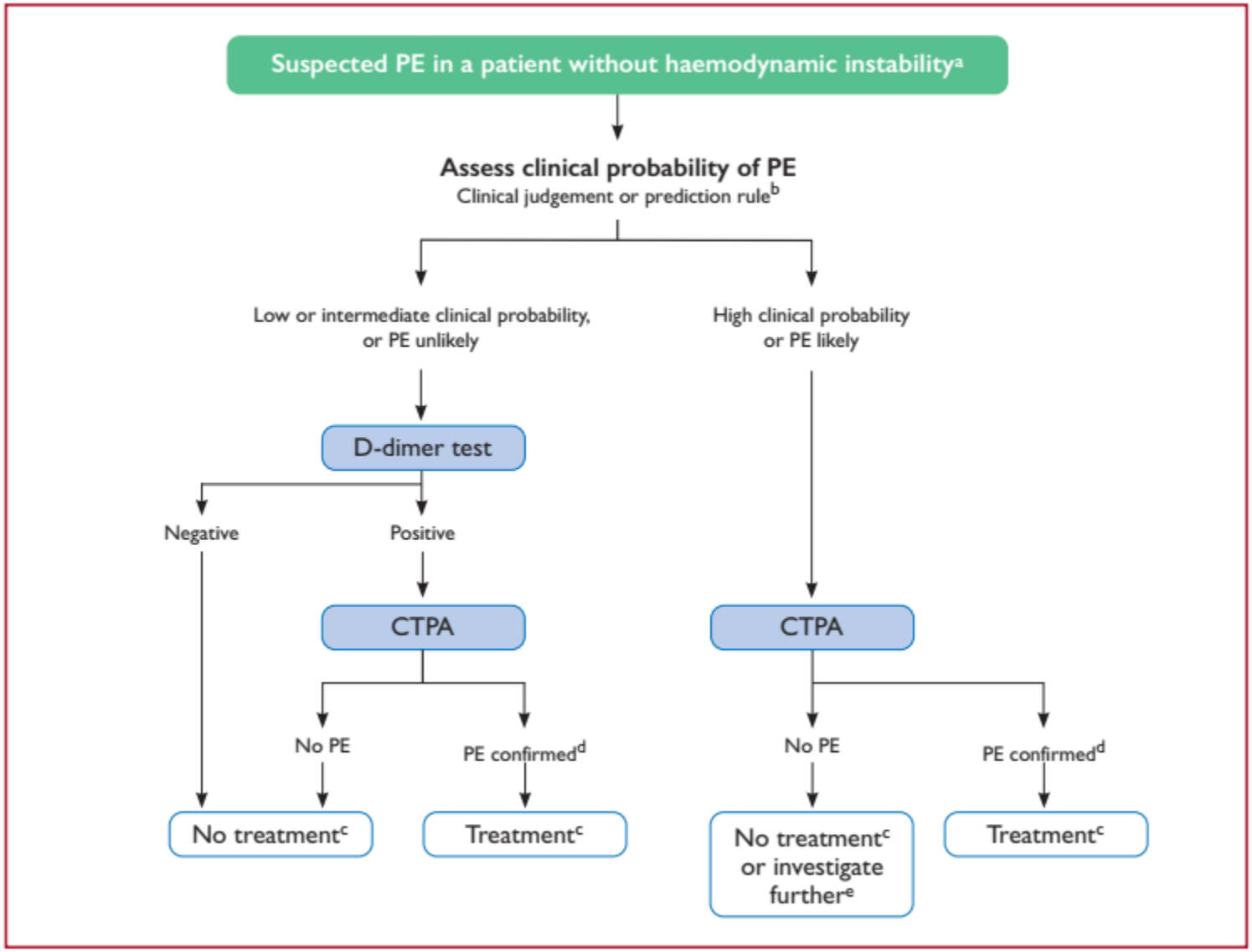
Konstantinides SV et al, *Eur Heart J 2020*

Diagnosis – PERC score



Rule out PE
Avoiding overuse of diagnostic tests

Diagnostic strategies



Konstantinides SV et al, *Eur Heart J 2020*

Diagnosis – D - dimers

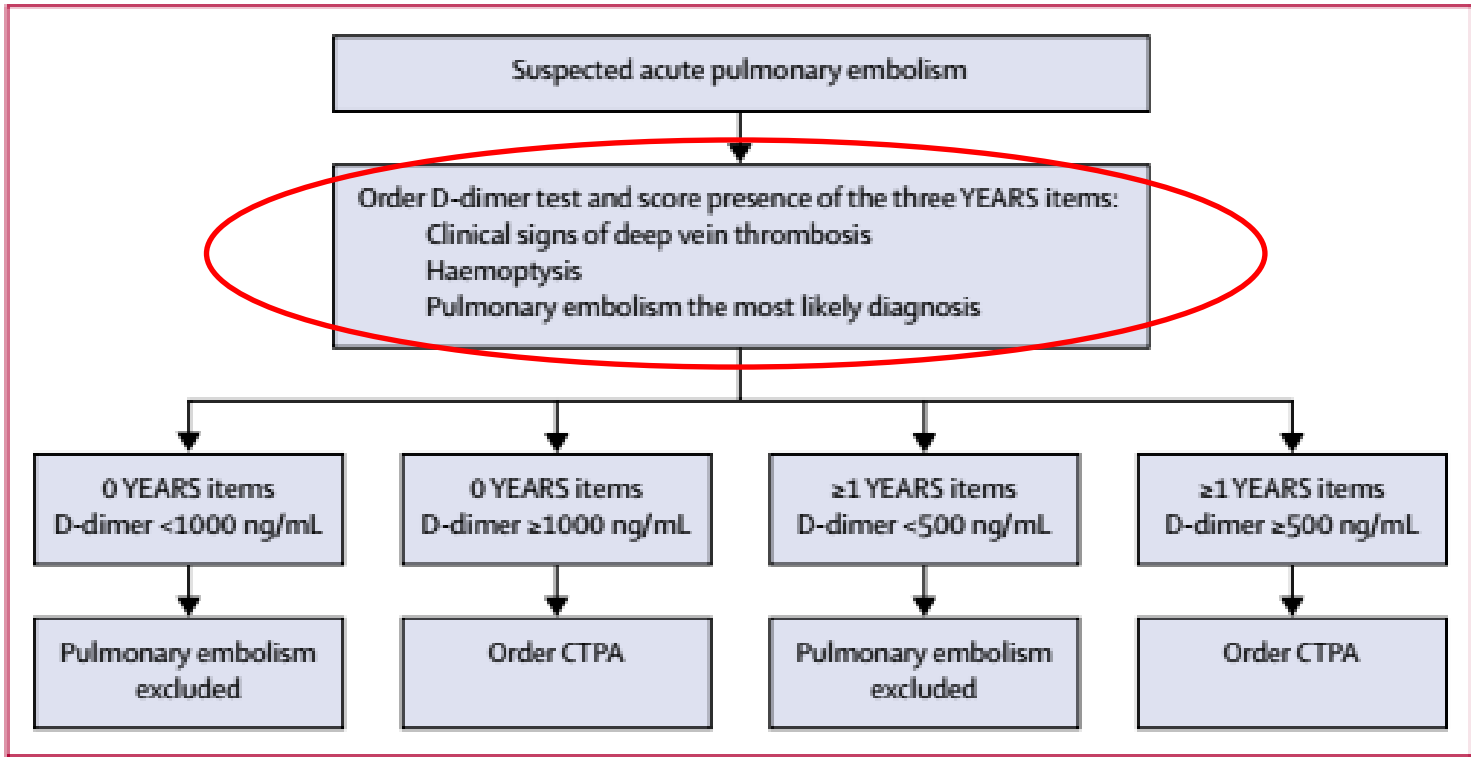
- High sensitivity, Low specificity
- Elisa-derived assays – sensitivity ≥ 95%
- Low or intermediate pre-test probability + negative Elisa D-dimer =

  Exclusion of PE

- Age-adjusted D-dimer cut offs (age x 10 mg/L, for pts > 50 years)
- Clinical probability adapted D-dimer cut offs

Konstantinides SV et al, *Eur Heart J 2020*

Diagnosis – D - dimers

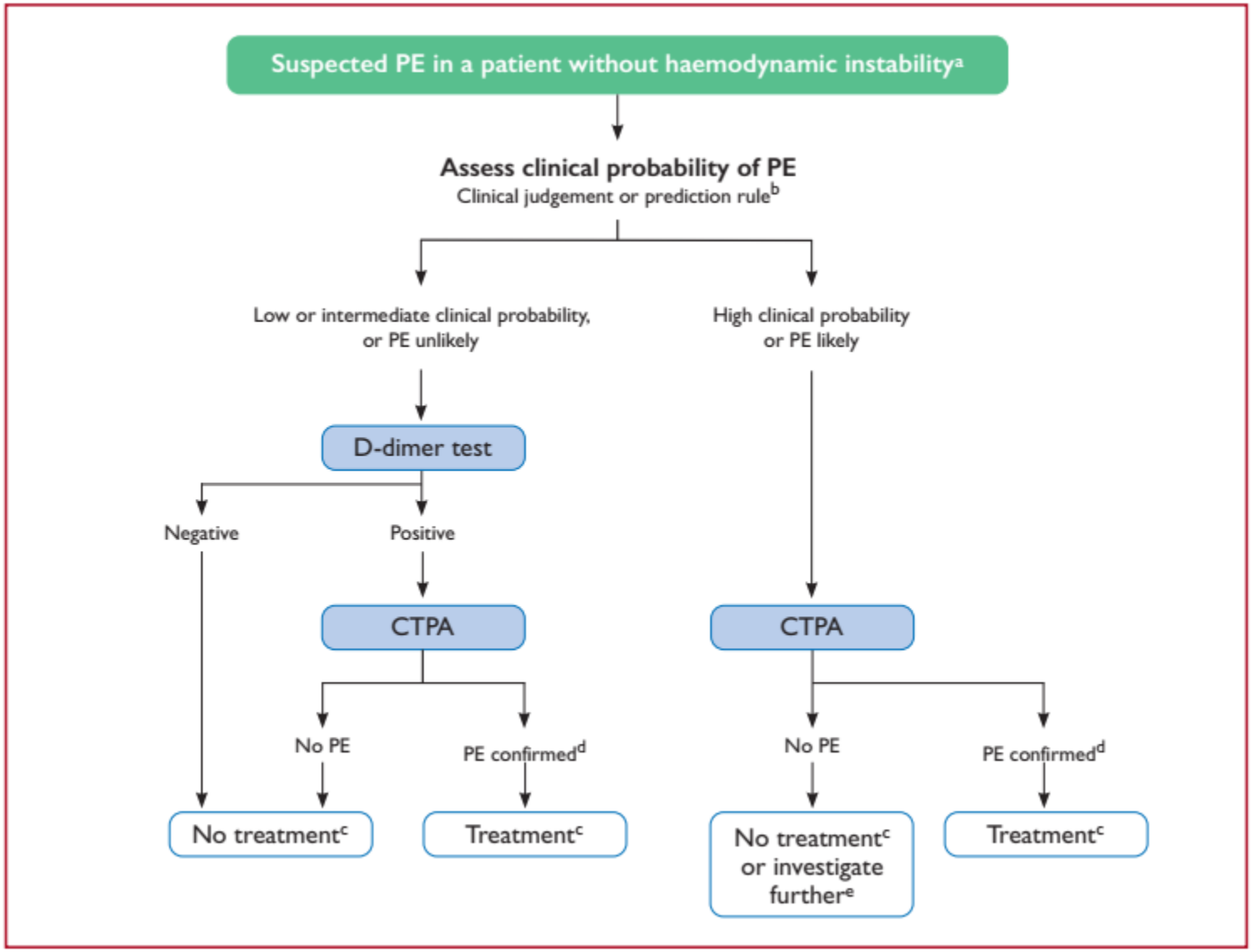


remained untreated, 18 patients were diagnosed with symptomatic venous thromboembolism during 3-month follow-up (0·61%, 95% CI 0·36–0·96) of whom six had fatal pulmonary embolism (0·20%, 0·07–0·44). CTPA was not indicated in 1651 (48%) patients with the YEARS algorithm compared with 1174 (34%) patients, if Wells' rule and fixed D-dimer threshold of less than 500 ng/mL would have been applied, a difference of 14% (95% CI 12–16).

Interpretation In our study pulmonary embolism was safely excluded by the YEARS diagnostic algorithm in patients with suspected pulmonary embolism. The main advantage of the YEARS algorithm in our patients is the absolute 14% decrease of CTPA examinations in all ages and across several relevant subgroups.

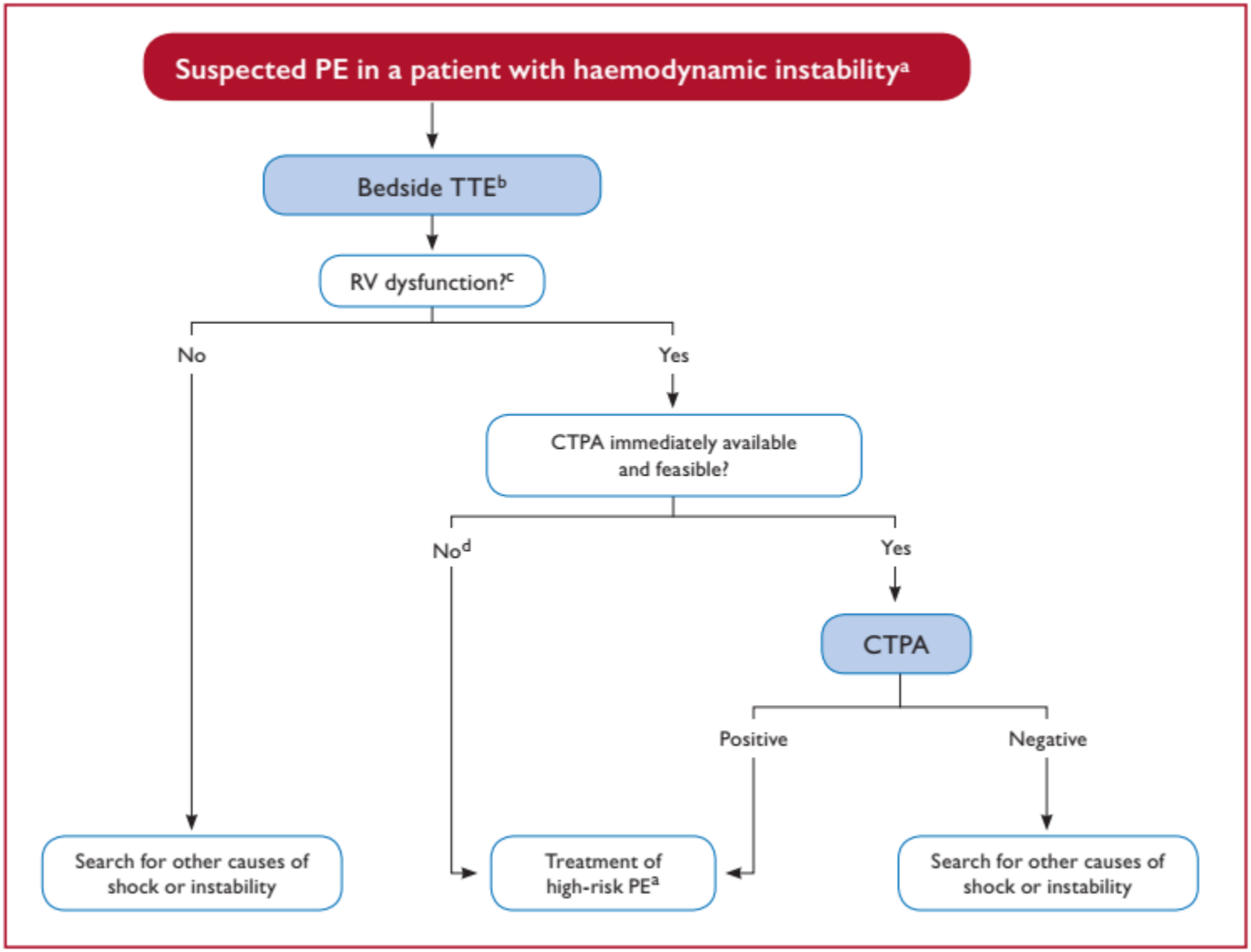
van der Hulleet T et al, *Lancet 2017*

Diagnostic strategies



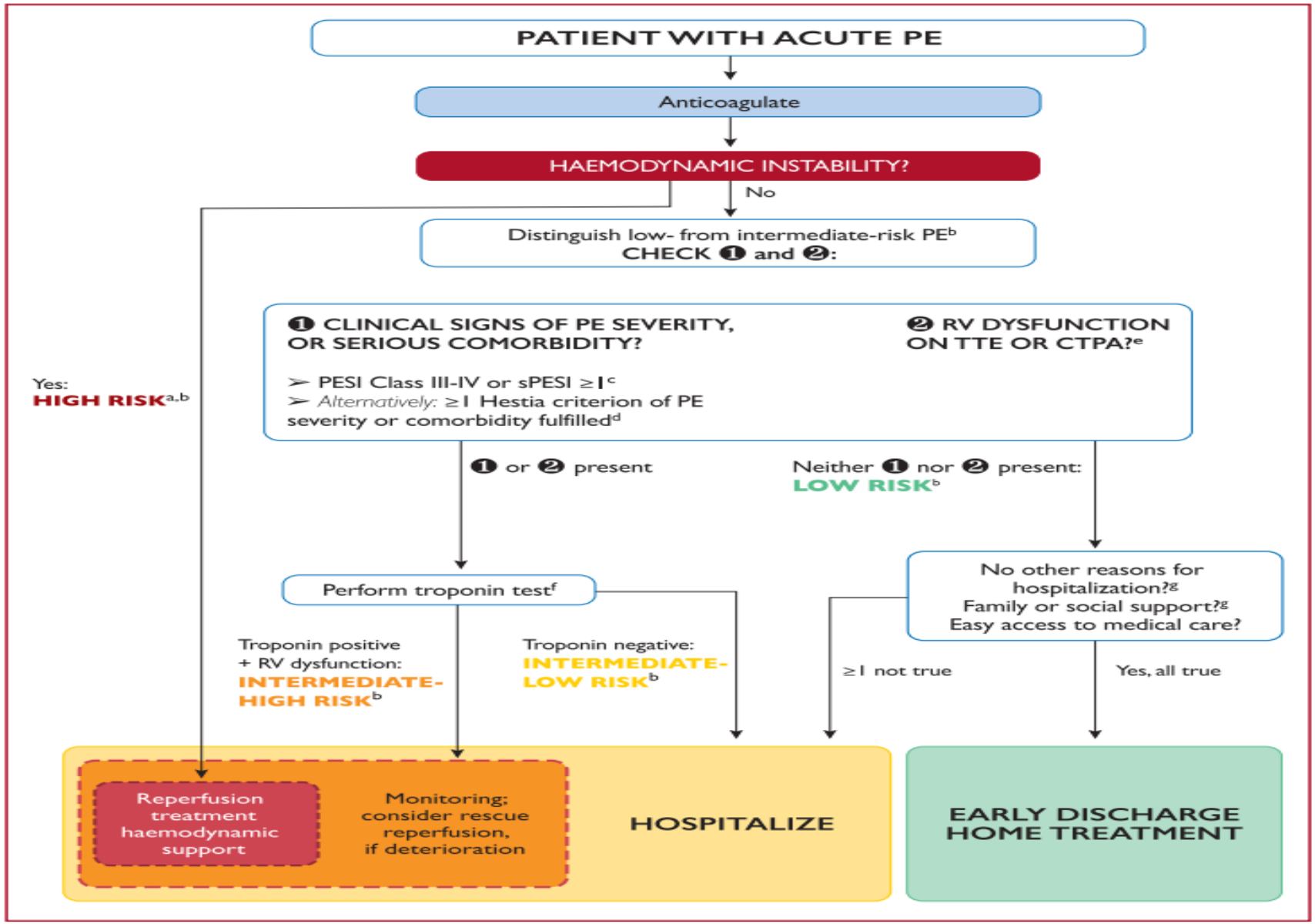
Konstantinides SV et al, *Eur Heart J 2020*

Diagnostic strategies



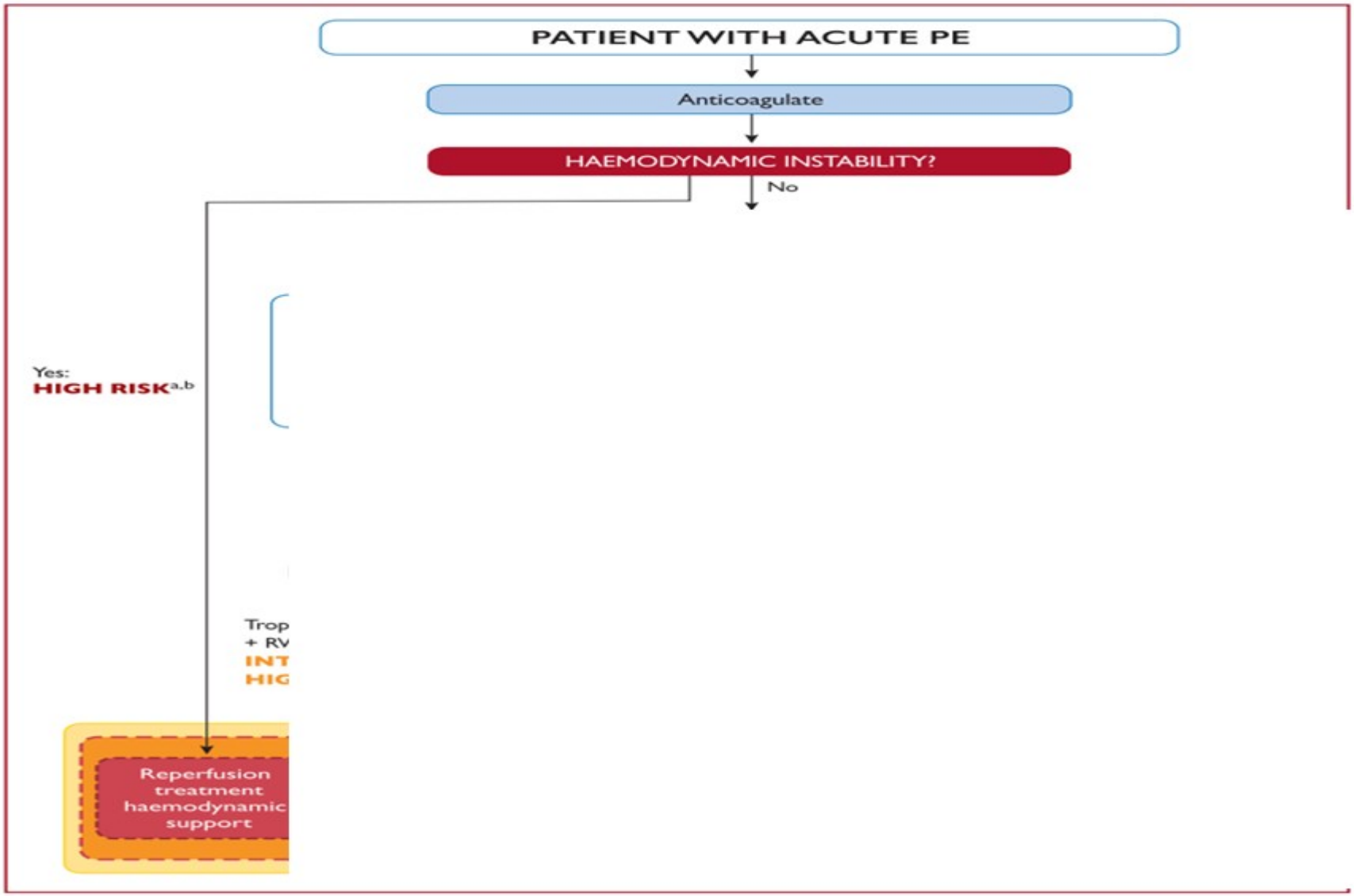
Konstantinides SV et al, *Eur Heart J 2020*

Treatment strategies



Konstantinides SV et al, *Eur Heart J 2020*

Treatment strategies – High risk



Konstantinides SV et al, *Eur Heart J 2020*

Treatment strategies – High risk

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)	

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

Konstantinides SV et al, *Eur Heart J 2020*

Treatment strategies – High risk

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

Konstantinides SV et al, *Eur Heart J 2020*

Treatment strategies – High risk

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

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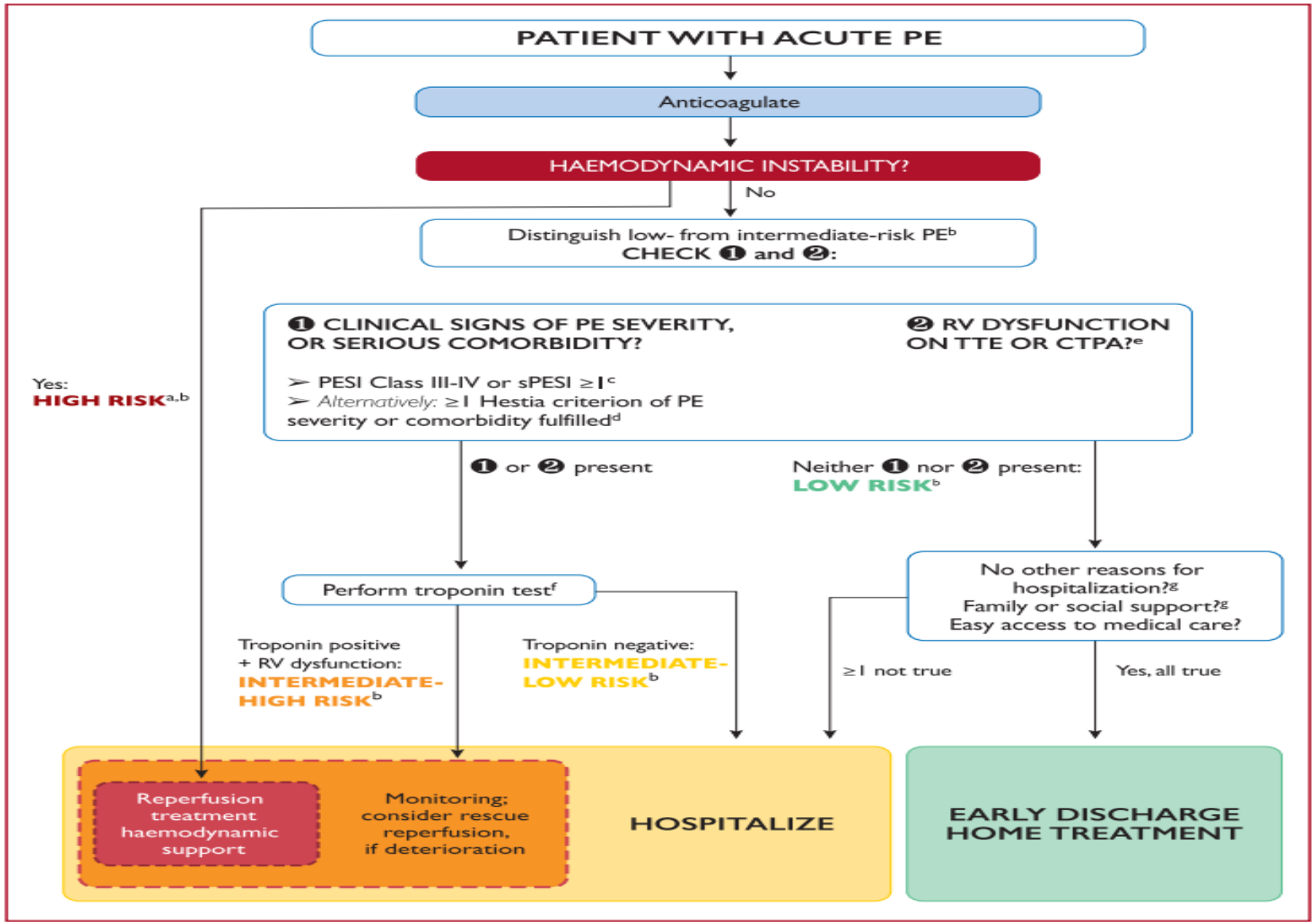
Treatment strategies



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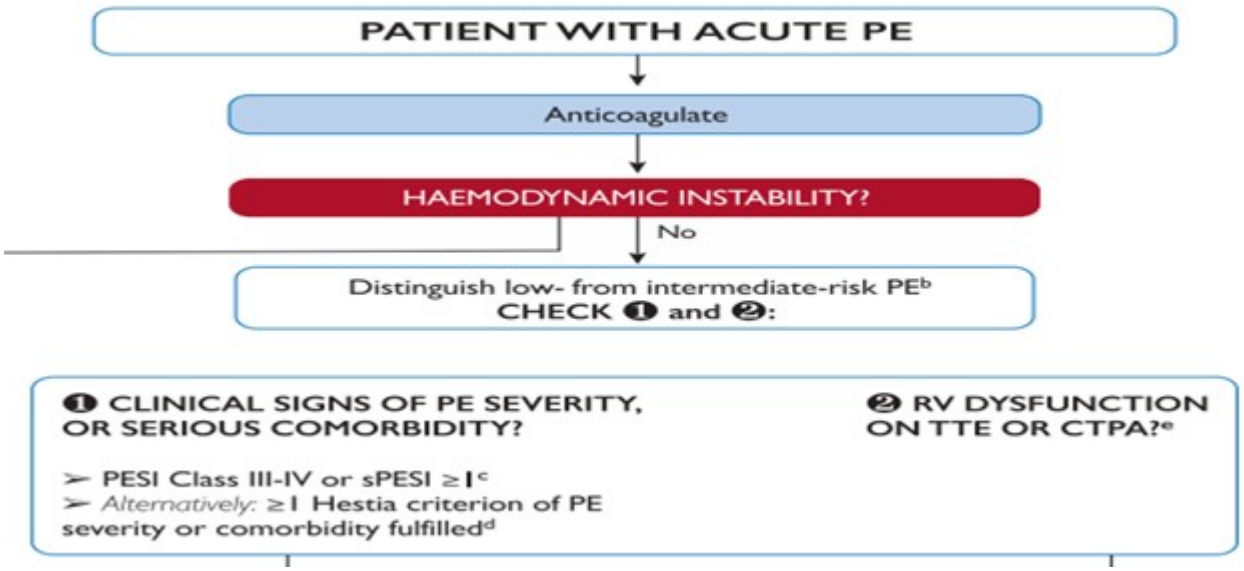
Treatment strategies



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Treatment in the acute phase – low, intermediate risk PE

Anticoagulation

- UFH
- LMWH and fondaparinux (lower risk of major bleeding and HIT than UFH)
- Non vitamin K antagonists oral anticoagulants (NOACs/DOACs)
- Vitamin K antagonists

Treatment strategies

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

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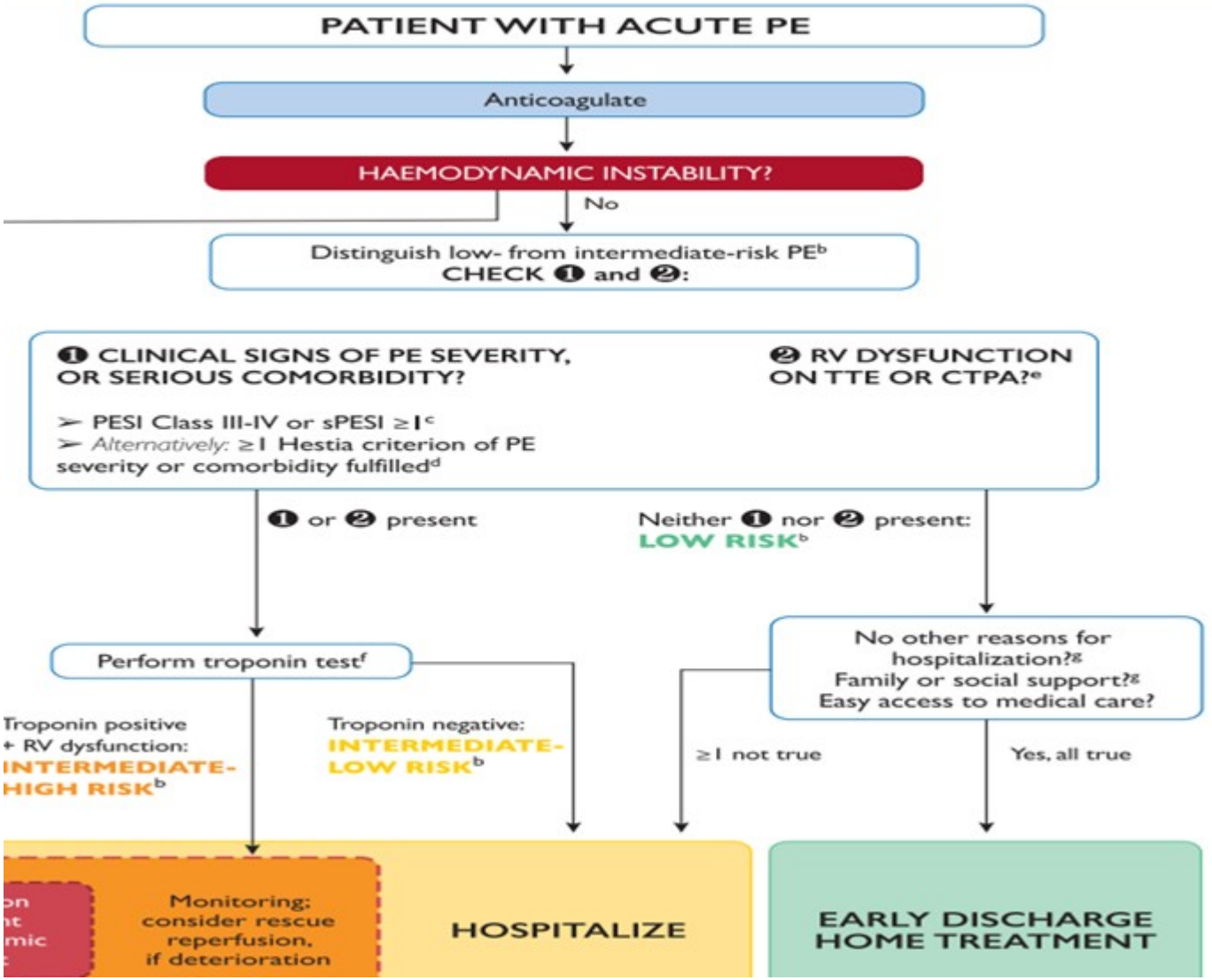
Treatment strategies



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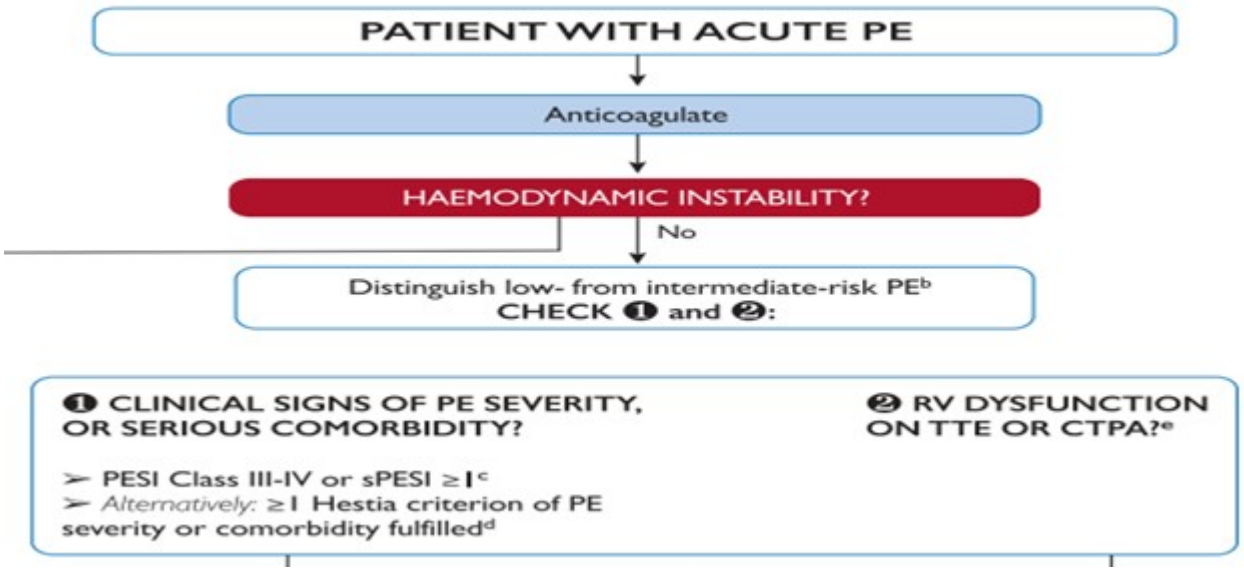
Treatment strategies



Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/ or comorbidity: PESI class III–V or sPESI ≥1	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

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Treatment strategies

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 oxyhaemoglobin 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%)

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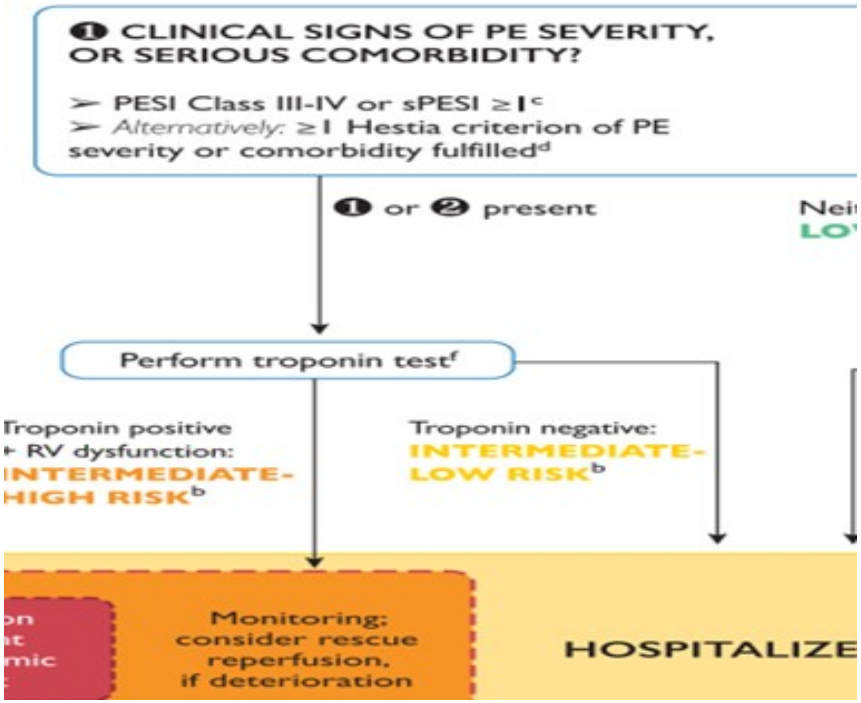
Treatment strategies



Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/or comorbidity: PESI class III–V or sPESI ≥1	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

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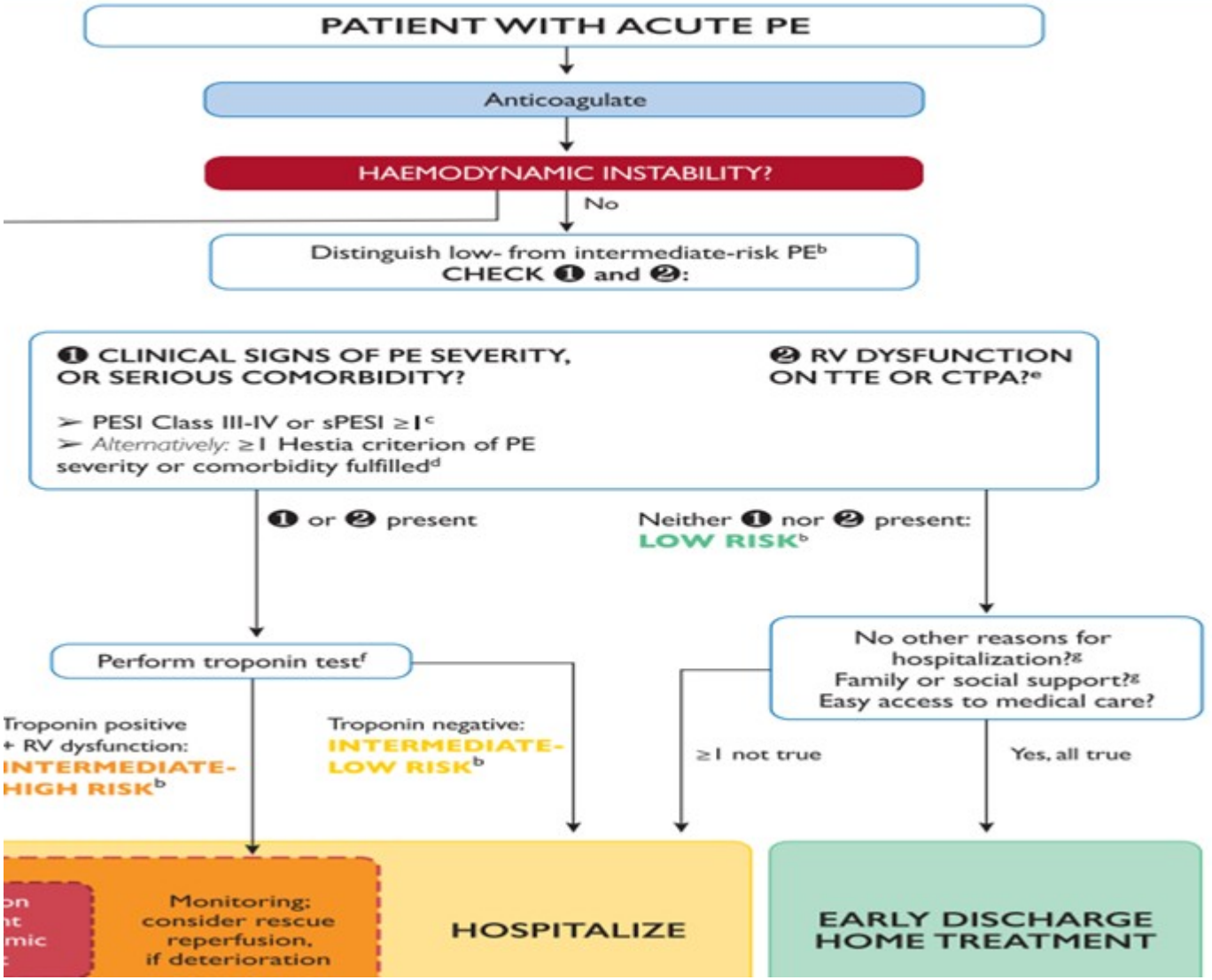
Treatment strategies



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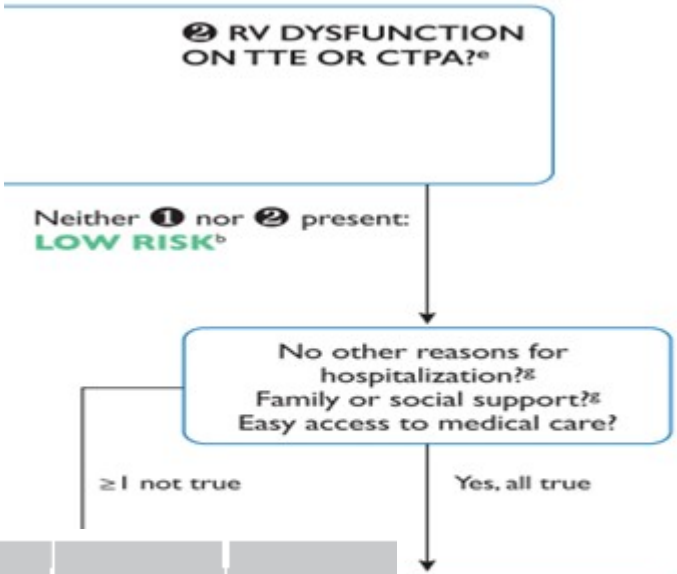
Treatment strategies

Supplementary Table 12 Hestia exclusion criteria for outpatient management

Criterion/question
Is the patient haemodynamically unstable?[a]
Is thrombolysis or embolectomy necessary?
Active bleeding or high risk of bleeding?[b]
More than 24 h of oxygen supply to maintain oxygen saturation >90%?
Is PE diagnosed during anticoagulant treatment?
Severe pain needing i.v. pain medication for more than 24 h?
Medical or social reason for treatment in the hospital for >24 h (infection, malignancy, or no support system)?
Does the patient have a CrCl of <30 mL/min?[c]
Does the patient have severe liver impairment?[d]
Is the patient pregnant?
Does the patient have a documented history

Hestia exclusion criteria for outpatient managemer
not be treated at home.



Recommendation	Class[a]	Level[b]
Carefully selected patients with low-risk PE should be considered for early discharge and continuation of treatment at home, if proper outpatient care and anticoagulant treatment can be provided.[c] 178,206,317–319	IIa	A

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Treatment strategies

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

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Treatment strategies

Table 1. Treatment with anticoagulant therapy in patients with pulmonary embolism.

Drug	Dose	Special Consideration	Specific Patient Characteristics	Reversal Agents
UFH	80 unit/kg IV bolus, followed by an 18-unit/kg/h infusion;	**Avoid** HIT; Osteopenia Pronounced drag-drag interactions	Overt haemodynamic instability; (CrCl) ≤ 30 mL/min; Pragnancy; Severe obesity	Protamine sulfate
LMWH	1 mg/kg twice daily 1.5 mg/kg once daily	**Avoid** with severe renal impairment	Pragnancy; Obesity;	Protamine sulfate
VKA	**Warfarin** 5 mg/day once daily 4 mg/day once day-patients > 70 years	Cross the placenta-**contraindicated in pregnancy**	Antiphospholipid syndrome; Mechanical heart valves; Extremely reduced renal function; Severe mitral stenosis;	4F-PCC 4 or FFP
Apiksaban	10 mg twice daily for 7 days followed by 5 mg twice daily	**Avoid in** CrCl < 15 mL/min Severe hepatic impairment	Previous GI bleeding or high risk of bleeding; Patients with CA; Eldery patients;	Andexanet
Rivaroxaban	15 mg- twice daily (3 weeks) then 20 mg once daily (at least 6 months)	**Avoid in** CrCl < 30 mL/min; (FDA) CrCl < 15 mL/min (EMA).	Low risk of bleeding and without gastrointestinal tumours; Patient preference—a single dose regimen;	Andexanet
Dabigatran	150 mg—twice daily 110 mg—twice daily for patients ≥ 80 years	**Avoid in** CrCl < 30 mL/min.; Concomitant treatment with P-gp inhibitors in patients with CrCl < 50 mL/min; Reduce dose to 110 mg for patients ≥ 80 years or ≥75 years with at least one bleeding risk factor;	Can be removed by hemodialysis in patients with severe renal impairment;	Idarucizumab
Edoxaban	60 mg—once daily 30 mg—once daily if body weight ≤ 60 kg	**Avoid** CrCl < 15 mL/min. Severe hepatic dysfunction	Low risk of bleeding and without gastrointestinal tumours	Andexanet
Fondaparinux	5 mg subQ daily <50 kg 7.5 mg subQ daily—50–100 kg 10 mg subQ daily >100 kg	**Avoid** CrCl < 30 mL/min	HIT (off lable); alergy of LMWH	Factor VIIa

Lasica R et al, *Pharmac 2022*

Take home messages

- Σημαντική η κλινική υποψία αφού τα συμπτώματα/σημεία – μη ειδικά
- ΤΤΕ σε αιμοδυναμική αστάθεια
- Έναρξη αντιπηκτικής αγωγής με την υποψία (intermediate or high pre test probability)
- PESI score and RV dysfunction – κατηγοριοποίηση κινδύνου/βαρύτητας νόσου

SIMA 23
Σι-μα Ιητηρ(ιατρός) Μινωϊκή Κρήτη

ΙΑΤΡΙΚΟΣ ΣΥΛΛΟΓΟΣ ΗΡΑΚΛΕΙΟΥ

Κλινική εικόνα και αντιμετώπιση Πνευμονικής Εμβολής

Κωνσταντίνος Καραγιάννης
Πνευμονολόγος - Φυματιολόγος
Επιμελητής Β'