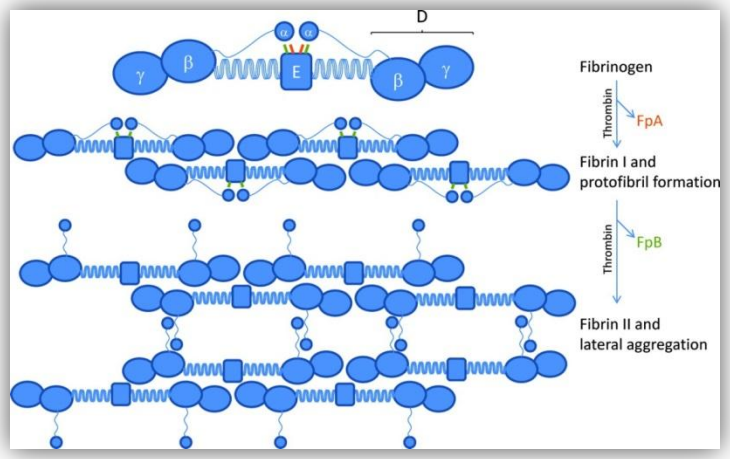
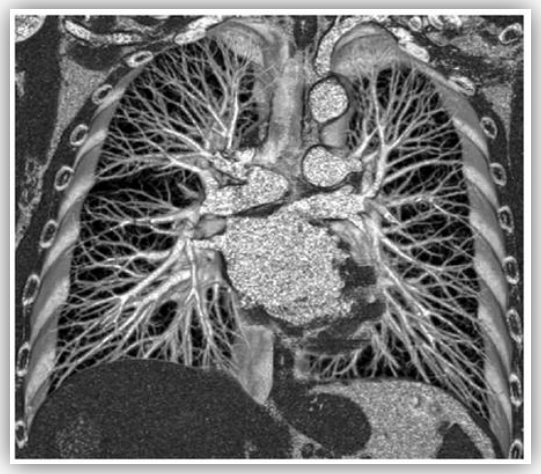
"Thromboembolism - An Update"





Hans Peter Kohler, MD, MACP
Professor of Medicine,
Secretary General,
International Society of Internal Medicine (ISIM)
Department of Internal Medicine,
Tiefenauspital, Inselgruppe AG
&
Experimental Haemostasis Group,
Department of Clinical Research,
University of Bern, Switzerland

International Society
of Internal Medicine

Risk factors for VTE

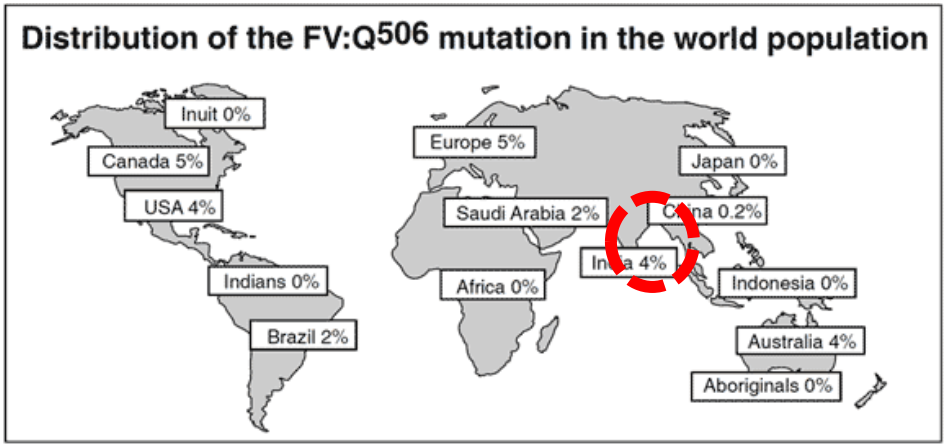
Table 1 Risk factors for venous thrombosis

Acquired	Inherited	Mixed/unknown
Immobilization	Antithrombin deficiency	High levels of factor VIII
Plaster cast	Protein C deficiency	High levels of factor IX
Trauma	Protein S deficiency	High levels of factor XI
Major surgery	Factor V Leiden (FVL)	High levels of fibrinogen
Orthopedic surgery	Prothrombin 20210A	High levels of TAFI
Malignancy	Dysfibrinogenemia	Low levels of TFPI
Oral contraceptives	Factor XIII 34val	APC-resistance in the absence of FVL
Hormonal replacement therapy	Fibrinogen (G) 10034T	
Antiphospholipid syndrome	Non-O blood group	Hyperhomocysteinemia
Myeloproliferative disorders		High levels of PCI (PAI-3)
Polycythemia vera		
Central venous catheters		
Age		
Obesity		

TAFI, thrombin-activatable fibrinolysis inhibitor; TFPI, tissue factor pathway inhibitor; PCI, protein C inhibitor; PAI-3, plasminogen-activator inhibitor-3.

J Thromb Haemost 2009; **7** (Suppl. 1): 301–4.

Genetic risk for VTE



Relative risk

- AT-III Deficiency
- Protein C Deficiency
- Protein S Deficiency

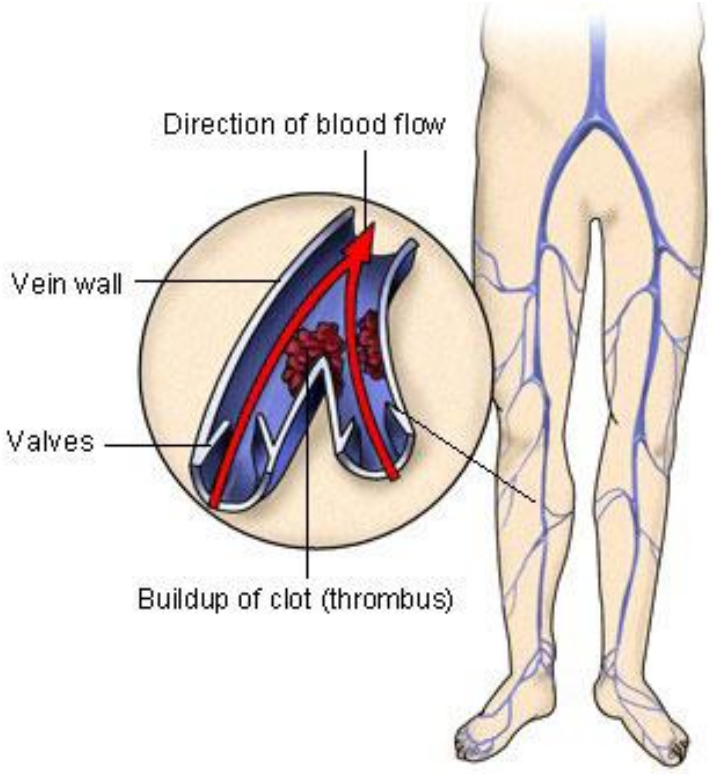
FV Leiden

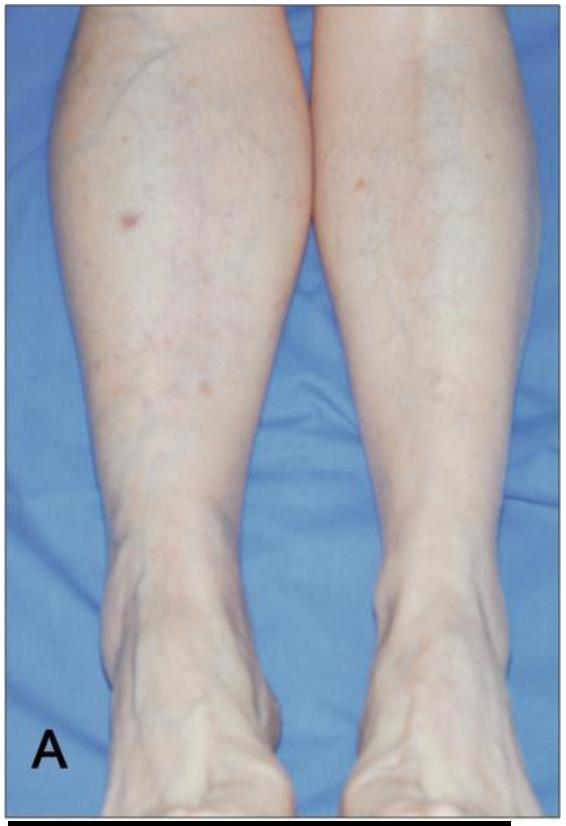
Prothrombin Mutation

Other common polymorphisms

Prevalence

Clinical signs and symptoms of deep vein thrombosis

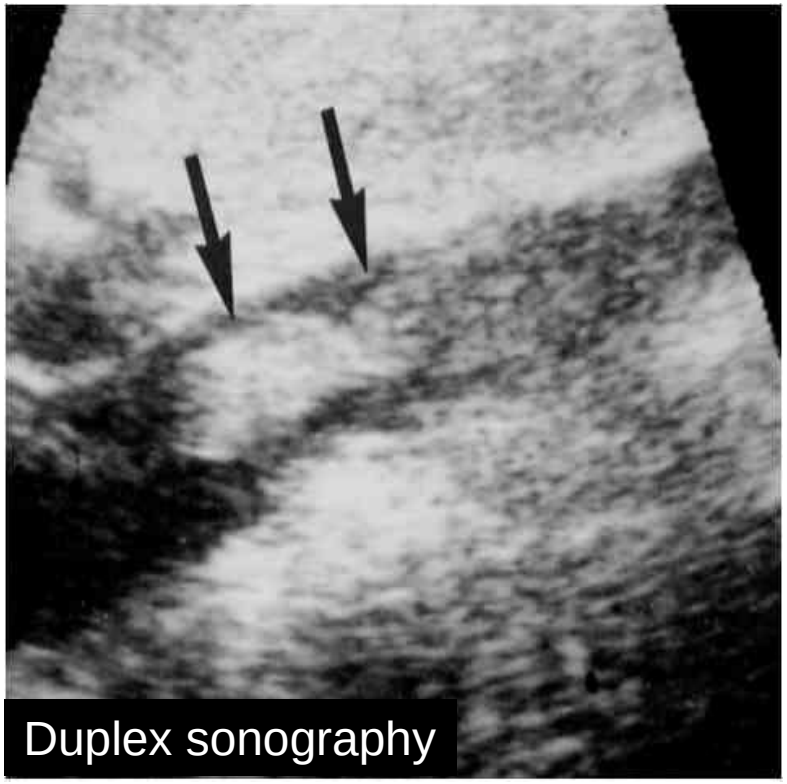




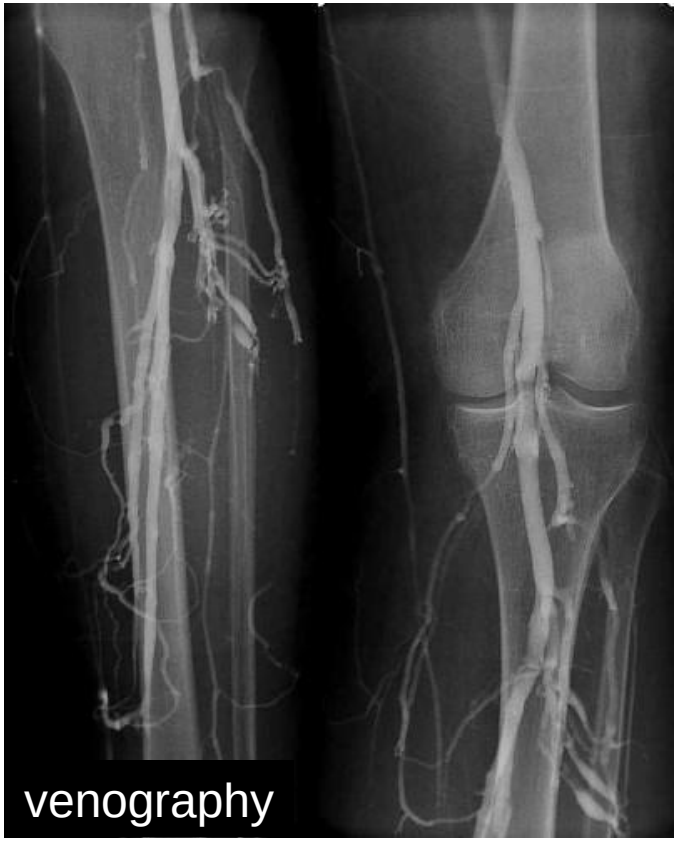
Deep vein thrombosis

-Swelling of the leg
-Pain or tenderness in the leg
-Feeling of increased warmth in the area of the leg that is swollen or that hurts
-Red or discolored skin.

Diagnosis of deep vein thrombosis (DVT)

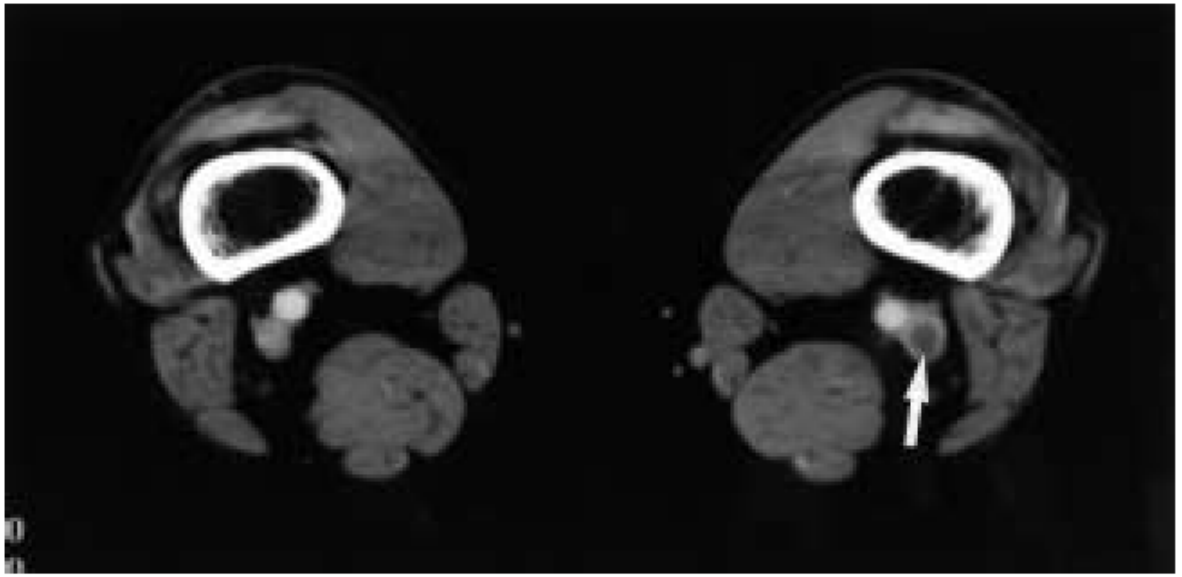


Duplex sonography



venography

CT Venography (optional)



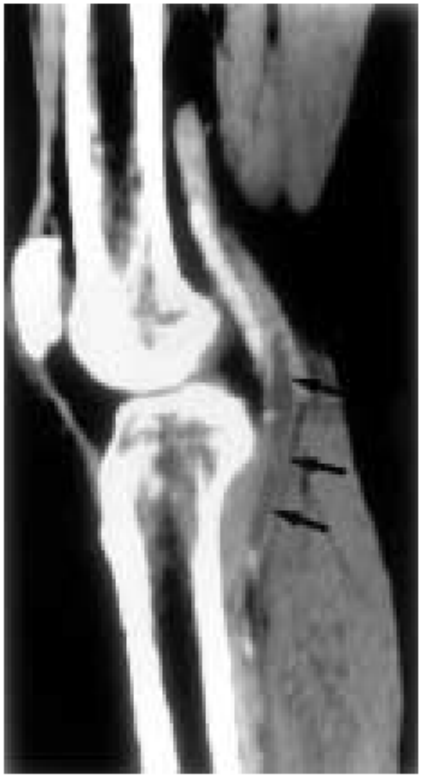


Fig. 3. A 47-year-old man with proven pulmonary embolism after air travel.
a: Contrast enhanced multi-slice helical CT venography shows thrombus (*arrow*) in the left popliteal vein.
b: Parasagittal multiplanar reformatted CT image shows the 5-cm-long thrombus (*arrows*) in the left popliteal vein.

a | b

Radiation Medicine: Vol. 22 No. 2, 77–81 p.p., 2004
Jonetz-Mentzel L et al. Zentralbl Chir 2002; 127: 755±759

Diagnosis of pulmonary embolism

Pulmonary embolism (PE): an *unsuspected killer*?

-**Third most common cardiovascular disease**

-Morbidity/mortality of missed PE is high: ± 25%

-The optimal strategy at individual institutions is dependent on local ***availability***, ***expertise***, and ***cost***

-**Clinical signs and symptoms are unspecific...**

-The diagnosis of PE remains difficult...

-**Easy to miss!!!**

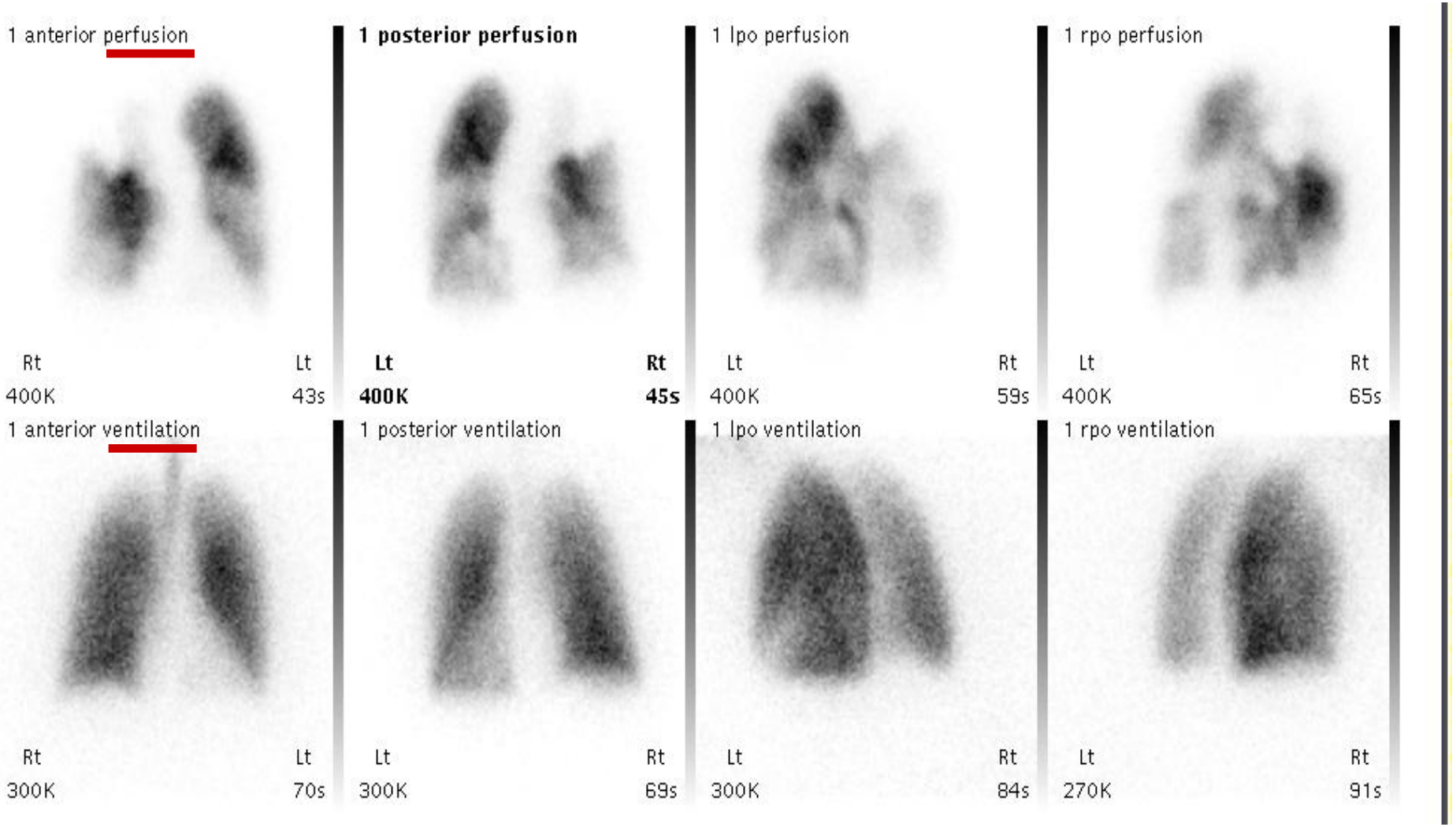
Prevalence of symptomes and signs

Table 6 **Prevalence of symptoms and signs in patients with suspected PE according to final diagnosis**

	PE confirmed (n = 219)	PE excluded (n = 546)
Symptoms		
Dyspnoea	80%	59%
Chest pain (pleuritic)	52%	43%
Chest pain (substernal)	12%	8%
Cough	20%	25%
Haemoptysis	11%	7%
Syncope	19%	11%
Signs		
Tachypnoea (≥20/min)	70%	68%
Tachycardia (>100/min)	26%	23%
Signs of DVT	15%	10%
Fever (>38.5°C)	7%	17%
Cyanosis	11%	9%

Data are form references 53 and 55.
DVT = deep vein thrombosis.

Nuclear imaging (lung scintigraphy)



Major problem: many results are not conclusive!!!

Nuclear imaging: some major problems...

-High percentage of **indeterminate results**
-**Poor inter-observer correlation**!
-Definitive exclusion of PE is only possible in the small minority of patients
-**False positive**? (pneumonia, fluid, etc.), **especially in older patients with multiple diagnosis** and involvement of the lung.

Sometimes useful for:
-otherwise healthy, young subjects without concurrent cardio-pulmonary disease
-subjects with impaired renal function....

Current Status of Ventilation-Perfusion Scintigraphy for Suspected Pulmonary Embolism

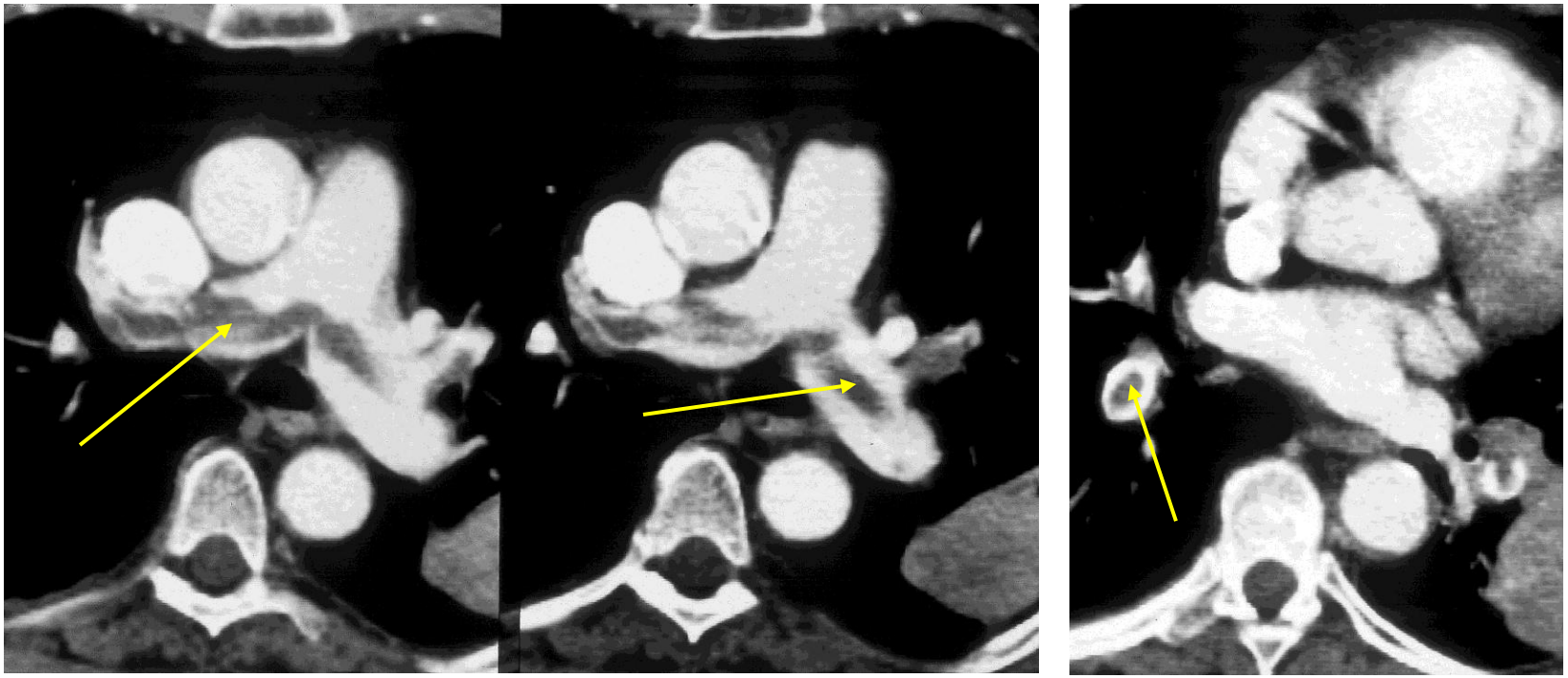
OBJECTIVE. The purpose of this article is to outline recent progress made in ventilation-perfusion (V/Q) scintigraphy imaging techniques and the interpretation systems used for the diagnosis of pulmonary embolism (PE). Various state-of-the-art approaches that can be selected according to the needs dictated by the medical practice environment and specific patient groups are presented.

CONCLUSION. Although advances in tomographic imaging have certainly improved the sensitivity of V/Q scans for the diagnosis of PE, they may lead to overdiagnosis by revealing small and clinically insignificant PEs.

Integral to V/Q scan interpretation is the pretest clinical assessment of pretest probability **and the presence or absence of prior cardiopulmonary disease**.

AJR:208, March 2017

Advantages of **spiral computed tomography**



-Readily available
-**Direct visualisation of the clot**
-visualisation of mediastinal and parenchymal structures
-Majority of patients with suspected PE receive another diagnosis (aortic dissection, pneumonia, pneumothorax, cancer etc.)

Pretest probability for VTE: Geneva / Wells Score

Table 2. **The Revised Geneva Score***

Variable	Regression Coefficients	Points
Risk factors		
Age > 65 y	0.39	1
Previous DVT or PE	1.05	3
Surgery (under general anesthesia) or fracture (of the lower limbs) within 1 mo	0.78	2
Active malignant condition (solid or hematologic malignant condition, currently active or considered cured < 1 y)	0.45	2
Symptoms		
Unilateral lower-limb pain	0.97	3
Hemoptysis	0.74	2
Clinical signs		
Heart rate		
75–94 beats/min	1.20	3
≥95 beats/min	0.67	5
Pain on lower-limb deep venous palpation and unilateral edema	1.34	4
Clinical probability		
Low		0–3 total
Intermediate		4–10 total
High		≥11 total

* DVT = deep venous thrombosis; PE = pulmonary embolism.

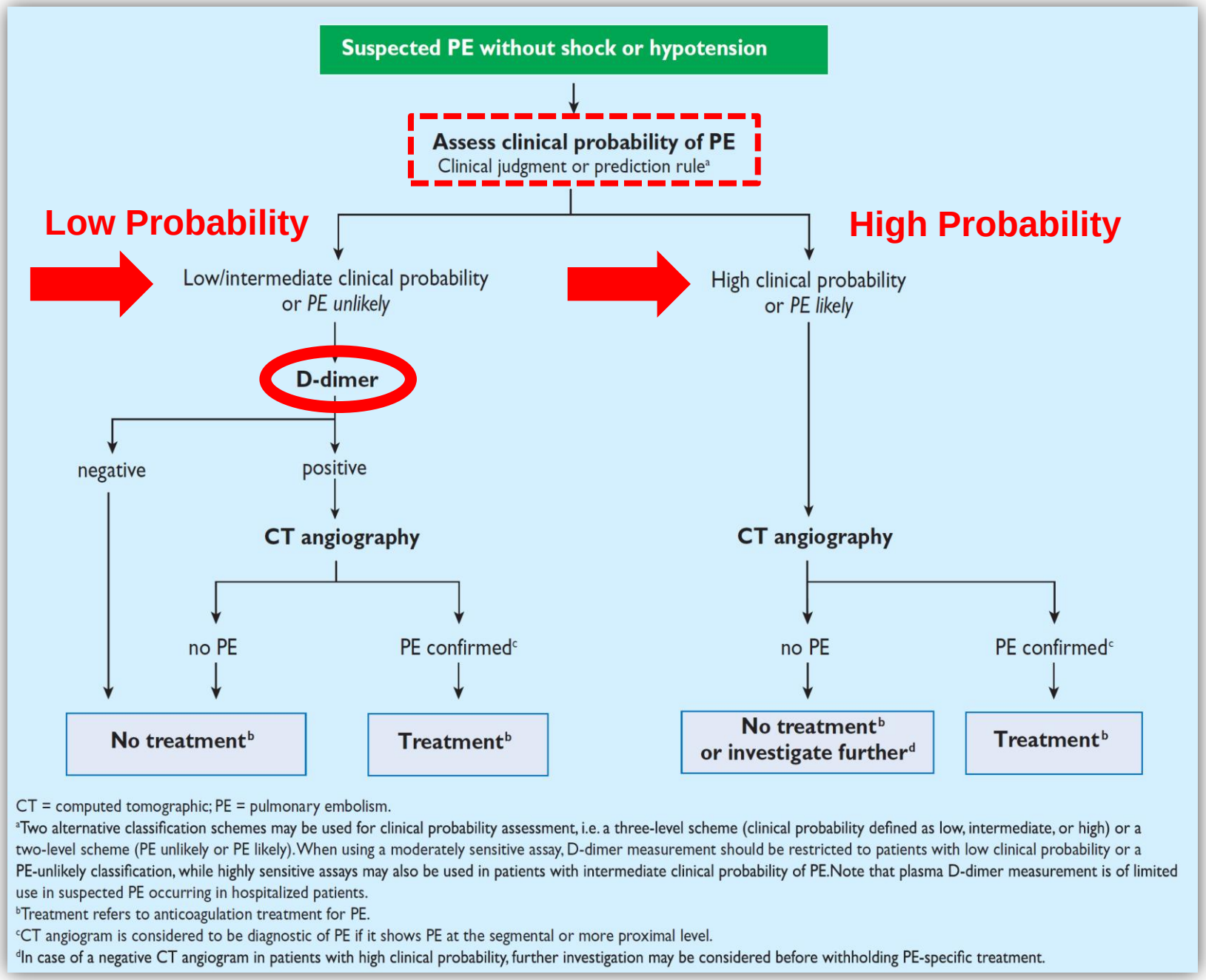
Ann Intern Med. 2006;144:165-171.

Table 1

Wells Scoring Criteria: Clinical Probability[22]

Criteria Scoring	Score (Max Score=12.5)
Clinical signs of DVT	+3.0
PE is more likely than the alternate diagnosis	+3.0
Tachycardia more than 100 beats/min	+1.5
Surgery/immobilization in previous 4 wk	+1.5
Previously diagnosed PE/DVT	+1.5
Hemoptysis	+1.0
Active malignant disease	+1.0
Traditional classification[22]	
High risk	>6.0
Moderate risk	2.0-6.0
Low risk	<2.0
Alternative classification[26]	
PE likely	>4.0
PE unlikely	<4.0

Abbreviations: DVT, deep vein thrombosis; PE, pulmonary embolism.



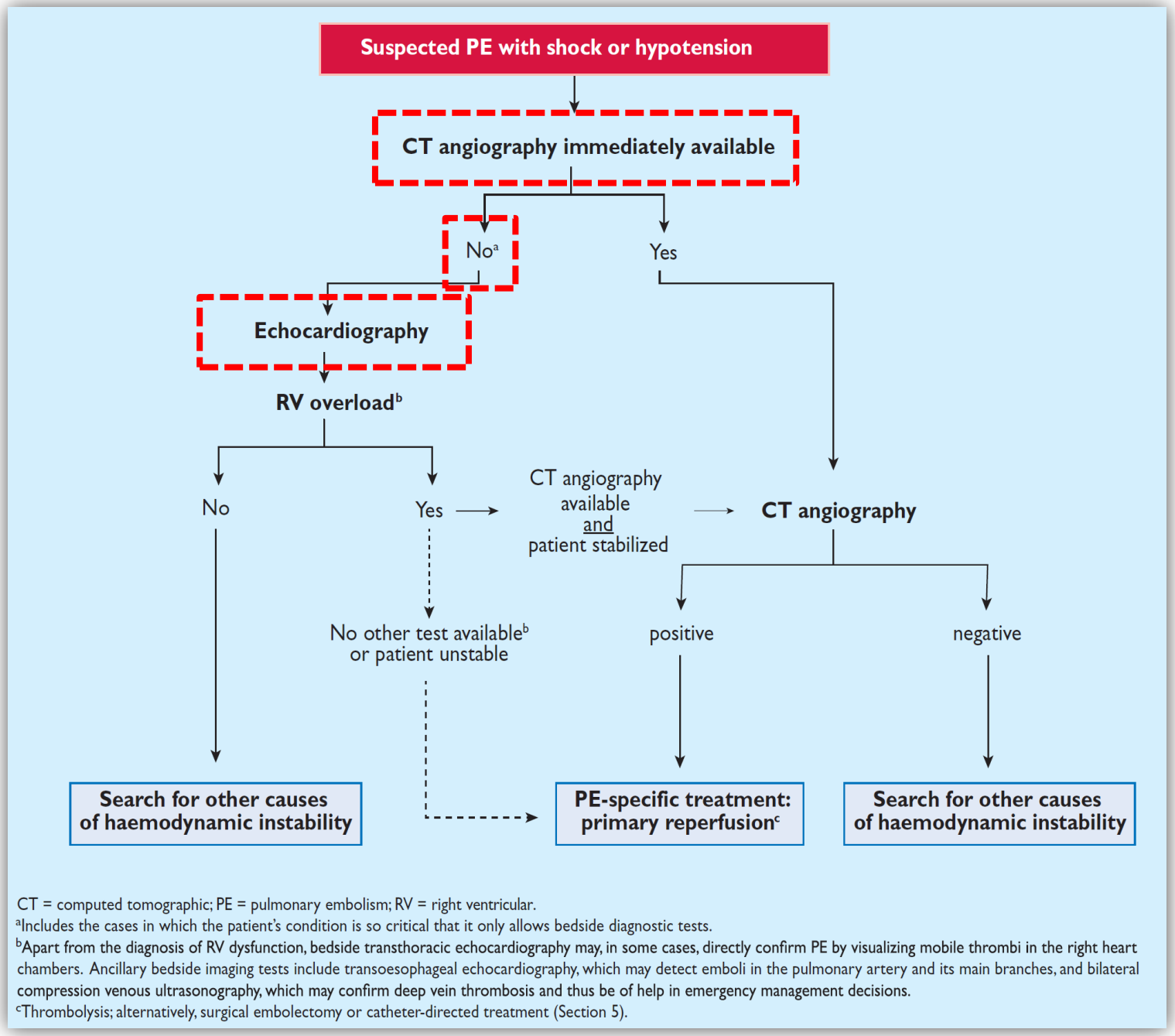
CT = computed tomographic; PE = pulmonary embolism.

[a]Two alternative classification schemes may be used for clinical probability assessment, i.e. a three-level scheme (clinical probability defined as low, intermediate, or high) or a two-level scheme (PE unlikely or PE likely). When using a moderately sensitive assay, D-dimer measurement should be restricted to patients with low clinical probability or a PE-unlikely classification, while highly sensitive assays may also be used in patients with intermediate clinical probability of PE. Note that plasma D-dimer measurement is of limited use in suspected PE occurring in hospitalized patients.

[b]Treatment refers to anticoagulation treatment for PE.

[c]CT angiogram is considered to be diagnostic of PE if it shows PE at the segmental or more proximal level.

[d]In case of a negative CT angiogram in patients with high clinical probability, further investigation may be considered before withholding PE-specific treatment.



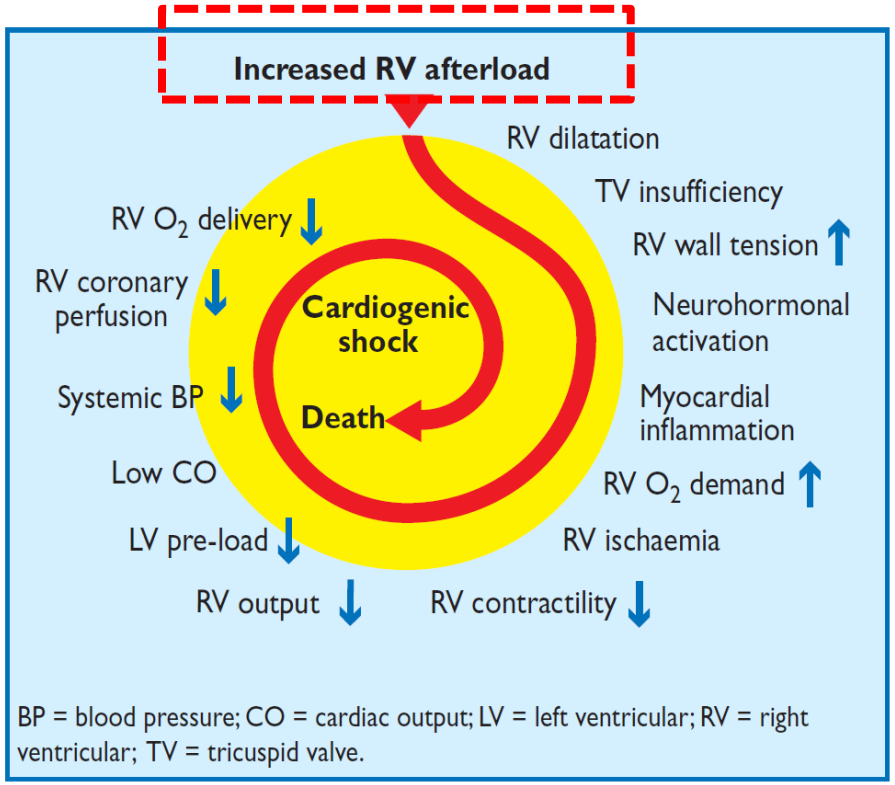
CT = computed tomographic; PE = pulmonary embolism; RV = right ventricular.

[a]Includes the cases in which the patient's condition is so critical that it only allows bedside diagnostic tests.

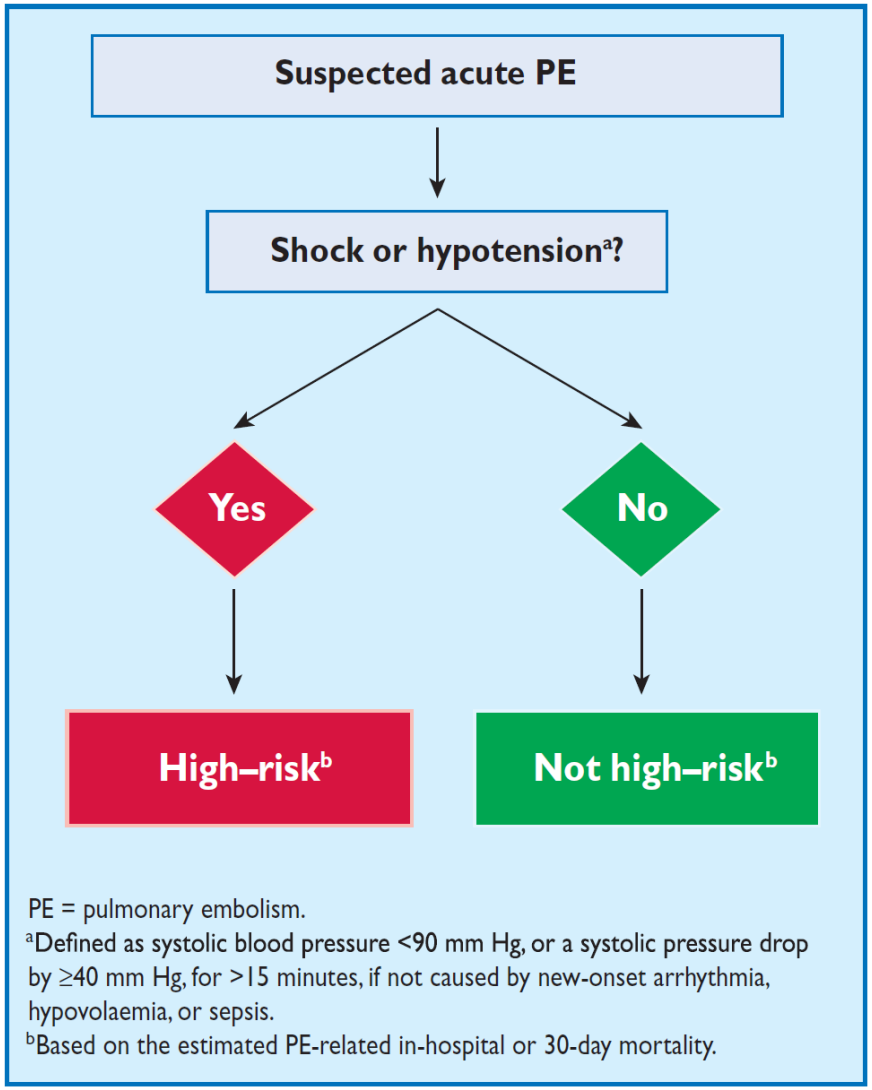
[b]Apart from the diagnosis of RV dysfunction, bedside transthoracic echocardiography may, in some cases, directly confirm PE by visualizing mobile thrombi in the right heart chambers. Ancillary bedside imaging tests include transoesophageal echocardiography, which may detect emboli in the pulmonary artery and its main branches, and bilateral compression venous ultrasonography, which may confirm deep vein thrombosis and thus be of help in emergency management decisions.

[c]Thrombolysis; alternatively, surgical embolectomy or catheter-directed treatment (Section 5).

Risk for morbidity and mortality



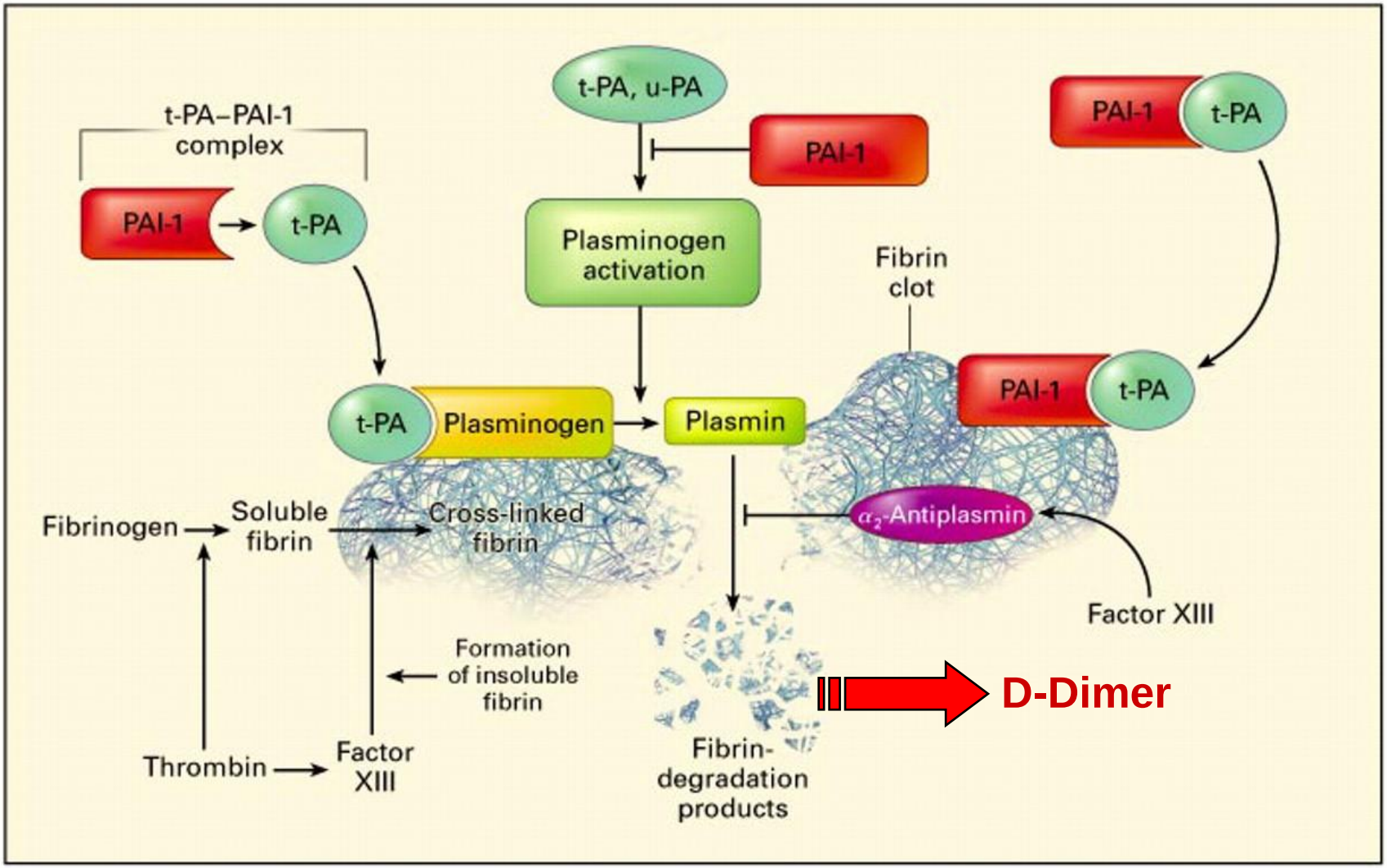
BP = blood pressure; CO = cardiac output; LV = left ventricular; RV = right ventricular; TV = tricuspid valve.



PE = pulmonary embolism.
[a]Defined as systolic blood pressure <90 mm Hg, or a systolic pressure drop by ≥40 mm Hg, for >15 minutes, if not caused by new-onset arrhythmia, hypovolaemia, or sepsis.
[b]Based on the estimated PE-related in-hospital or 30-day mortality.

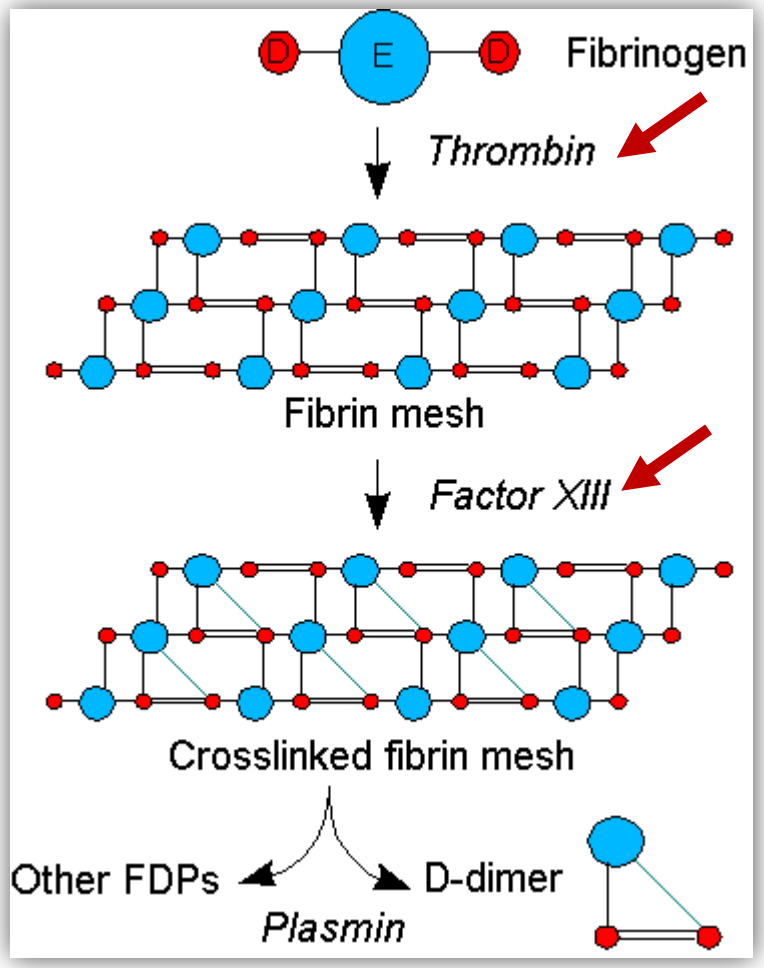
European Heart Journal (2014) **35**, 3033–3080
doi:10.1093/eurheartj/ehu283

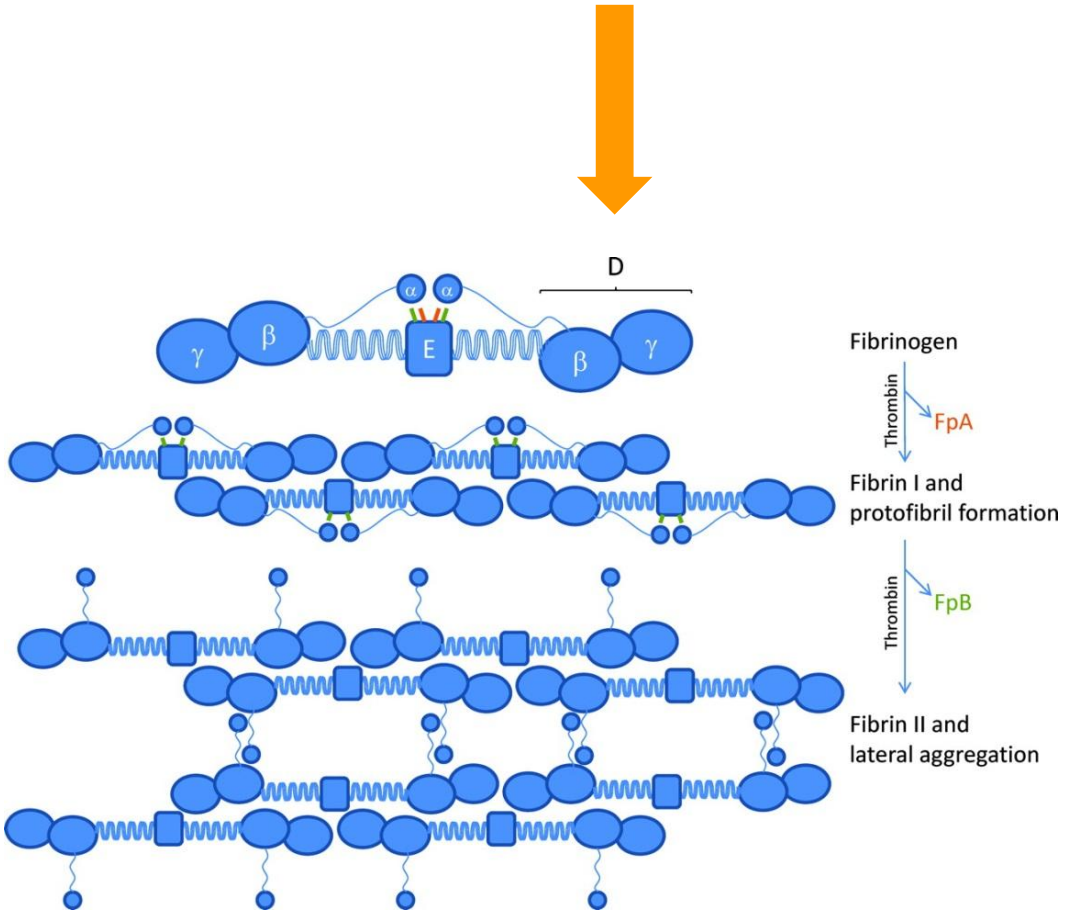
D-Dimer (Fibrin degradation products)



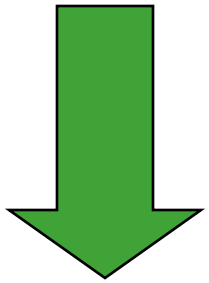
Kohler et al. N Engl J Med 342:1792-1801, 2000

D-Dimer


D
E
D
Fibrinogen
Thrombin
Fibrin mesh
Factor XIII
Crosslinked fibrin mesh
Other FDPs
D-dimer
Plasmin


D
α
α
γ
β
E
β
γ
Fibrinogen
Thrombin
FpA
Fibrin I and
protofibril formation
Thrombin
FpB
Fibrin II and
lateral aggregation

Value of D-Dimer testing:



D-Dimer levels not increased (≤ **500µg/L**):

⇒ **no significant fibrin formation in the body**

⇒ **No VTE**

BUT !.....

Case 1

-A 60 year old man with intermediate pretest probability for PE

-No shock or hypotension

-**Clinical signs for PE since 5 days…**

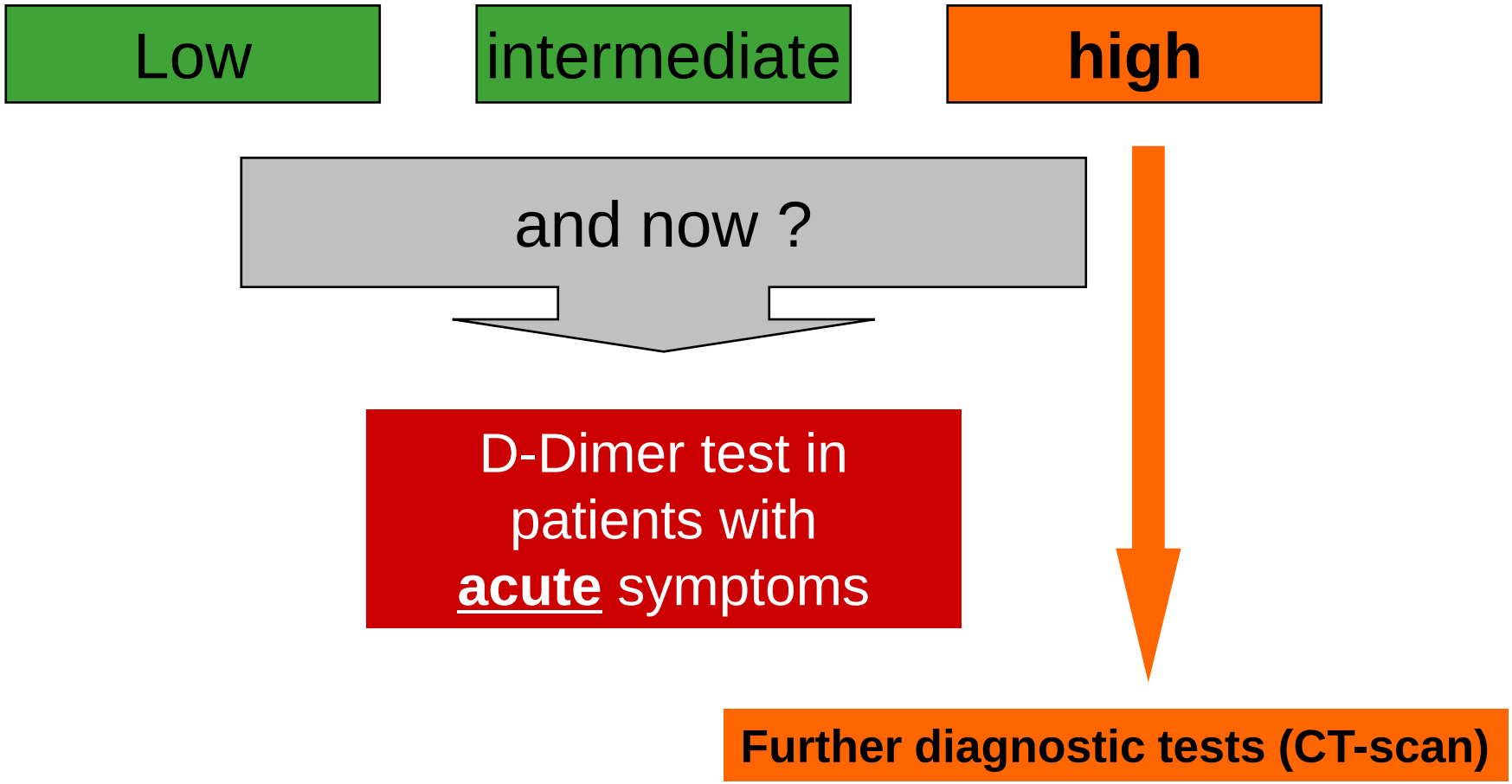
-D-Dimer: **475 µg/L**

What are you going to do?

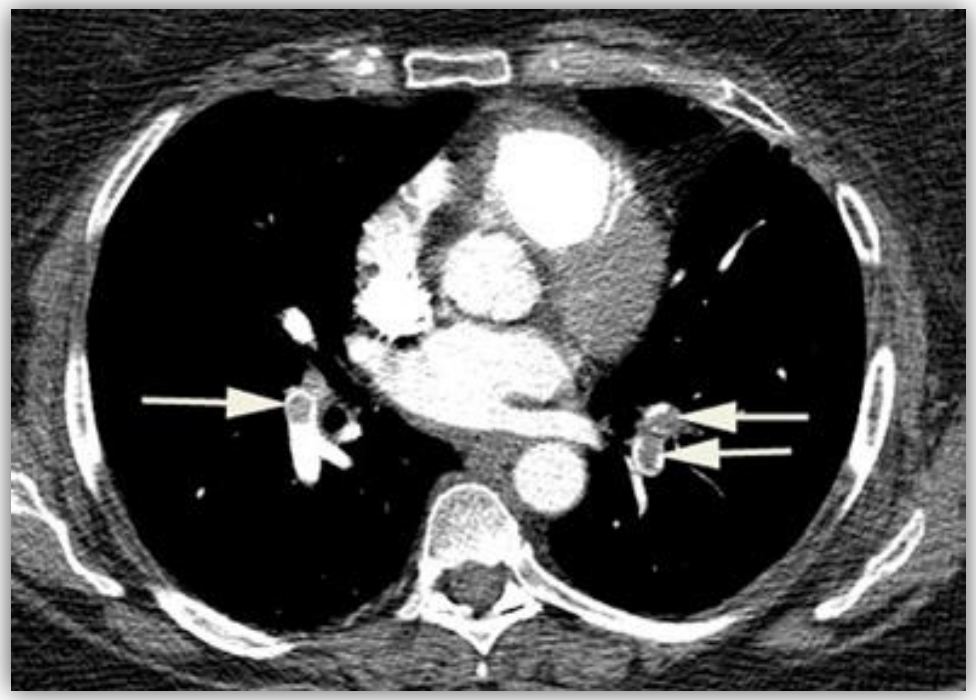
-Further investigations (hearth failure, chest infection etc.)

-CT scan to exclude pulmonary embolism despite low D-Dimer level?

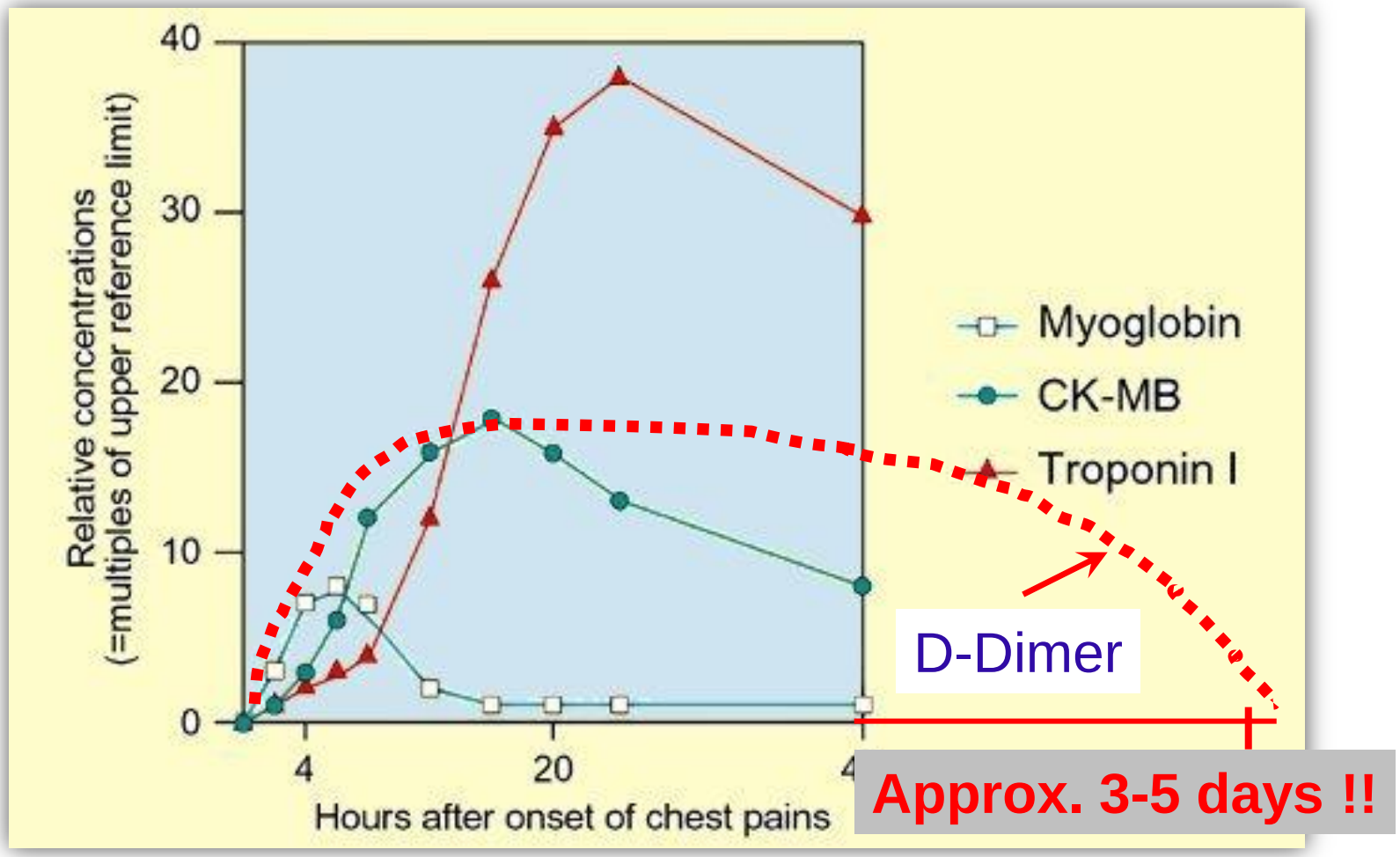
Clinical pretest probability



PE despite low D-Dimer…



D-Dimer testing only for acute clinical symptoms



Case 2

-A 55 year old woman with intermediate pretest probability for PE

-D-Dimer: **750 µg/L**

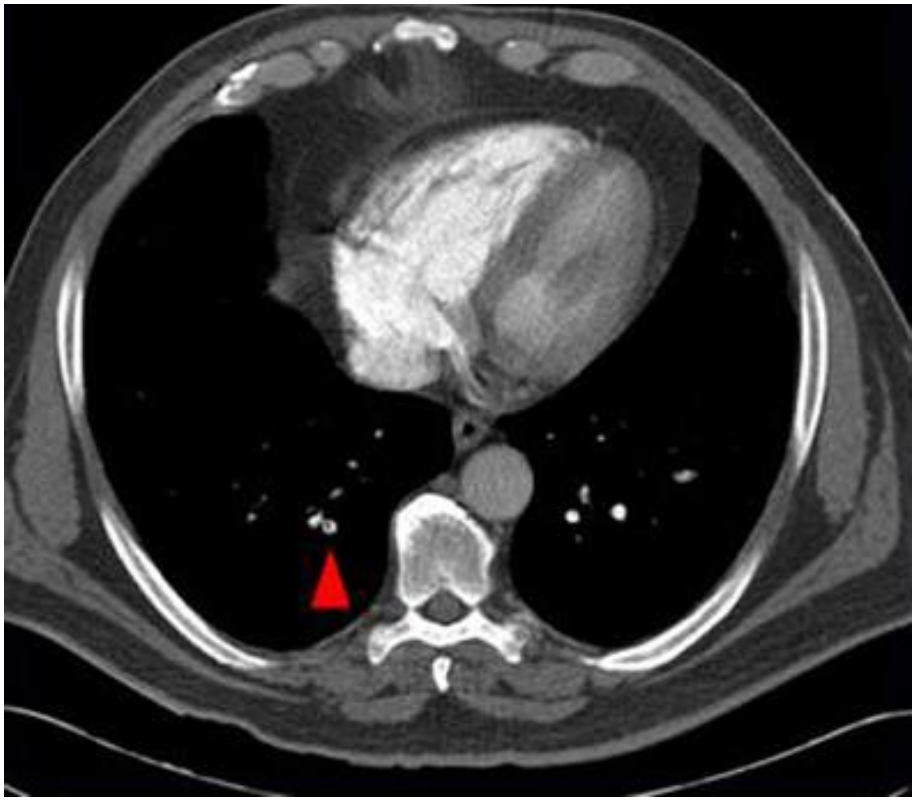
-CT scan shows **subsegmental** PE (**SSPE**)

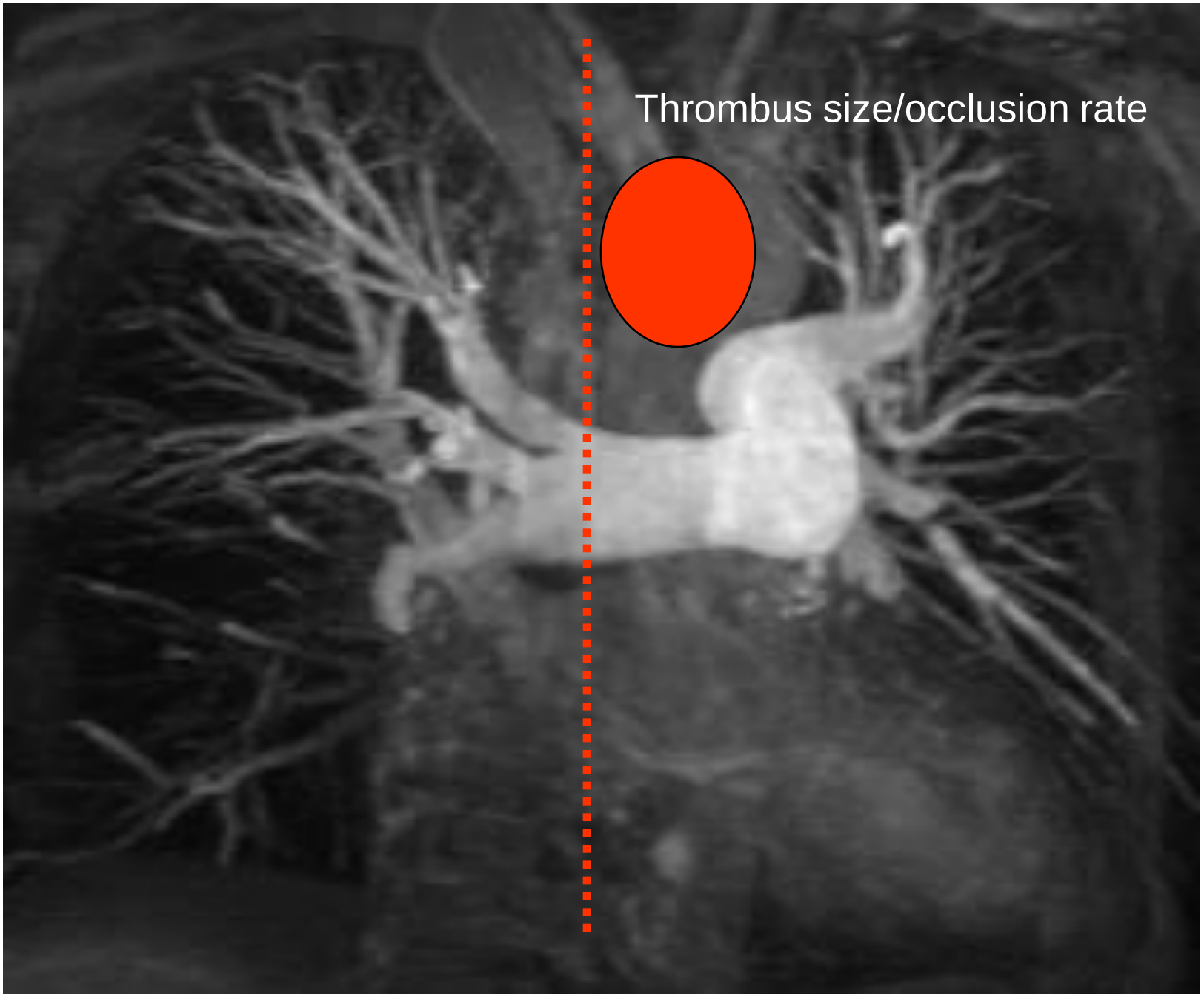
What are you going to do?

-Further tests?

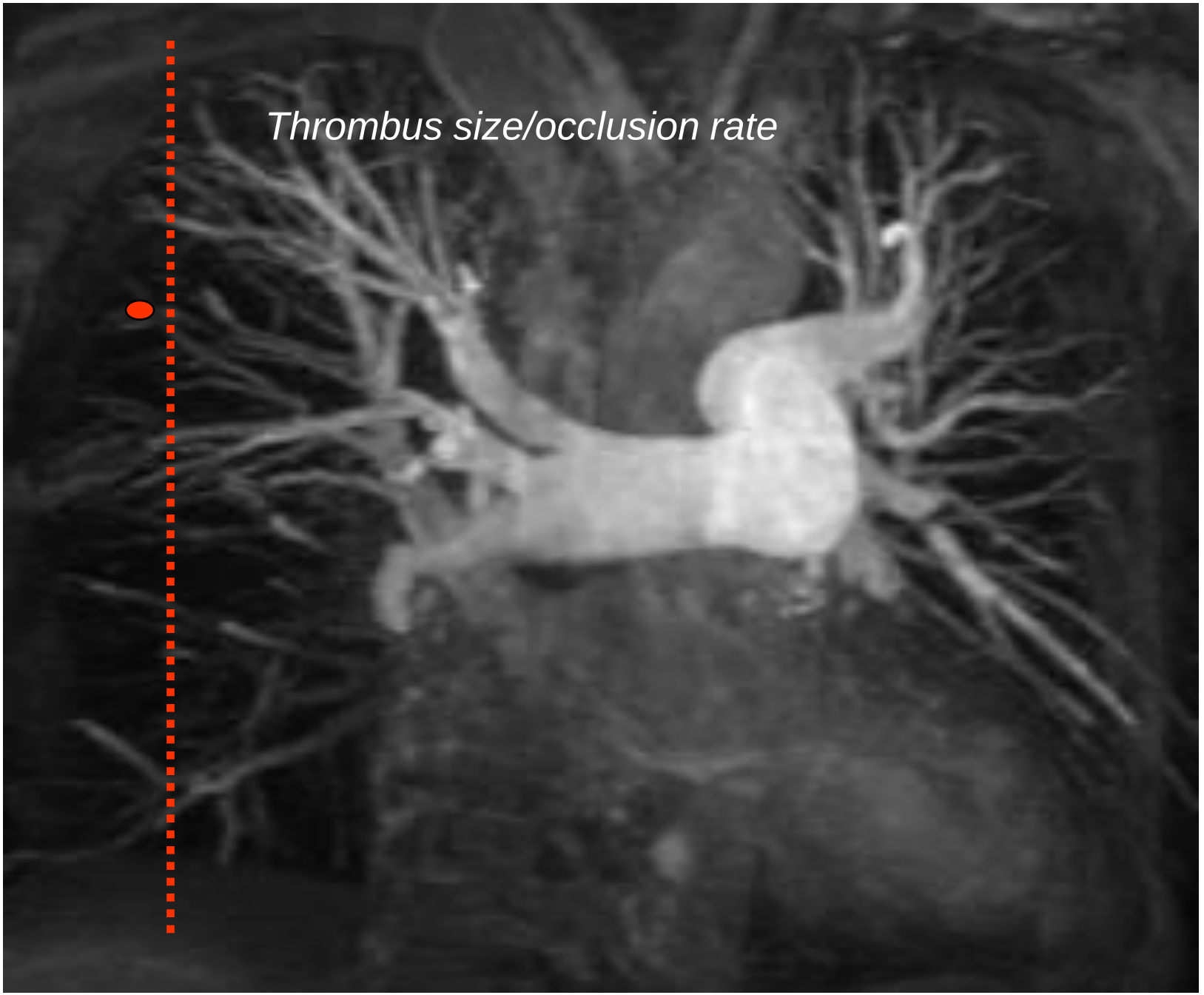
-Duplex ultrasonography for DVT?

-Anticoagulation therapy?




Thrombus size/occlusion rate


Thrombus size/occlusion rate


Thrombus size/occlusion rate

Journal of Thrombosis and Haemostasis, **10**: 1486–1490 DOI: 10.1111/j.1538-7836.2012.04804.x

REVIEW ARTICLE

Symptomatic subsegmental pulmonary embolism: what is the next step?

M. CARRIER,*† M. RIGHINI‡ and G. LE GAL§

**Thrombosis Program, Division of Hematology, Department of Medicine, University of Ottawa, Ottawa; †Clinical Epidemiology Program, The Ottawa Hospital Research Institute, Ottawa, Canada; ‡Division of Angiology and Hemostasis, Geneva University Hospital and Faculty of Medicine, Geneva, Switzerland; and §Brest University Hospital, Brest, France*

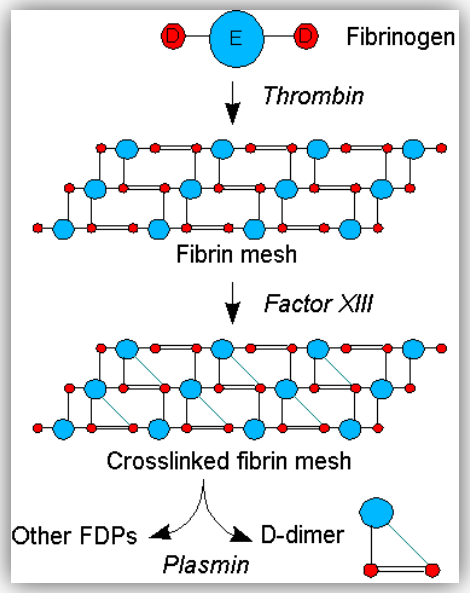
2012; **10**: 1486–90.

Conclusions:

1. SSPE might be clinically unimportant…..

2. Deep vein thrombosis (DVT) should probably be excluded….

D-Dimer levels in pregnancy

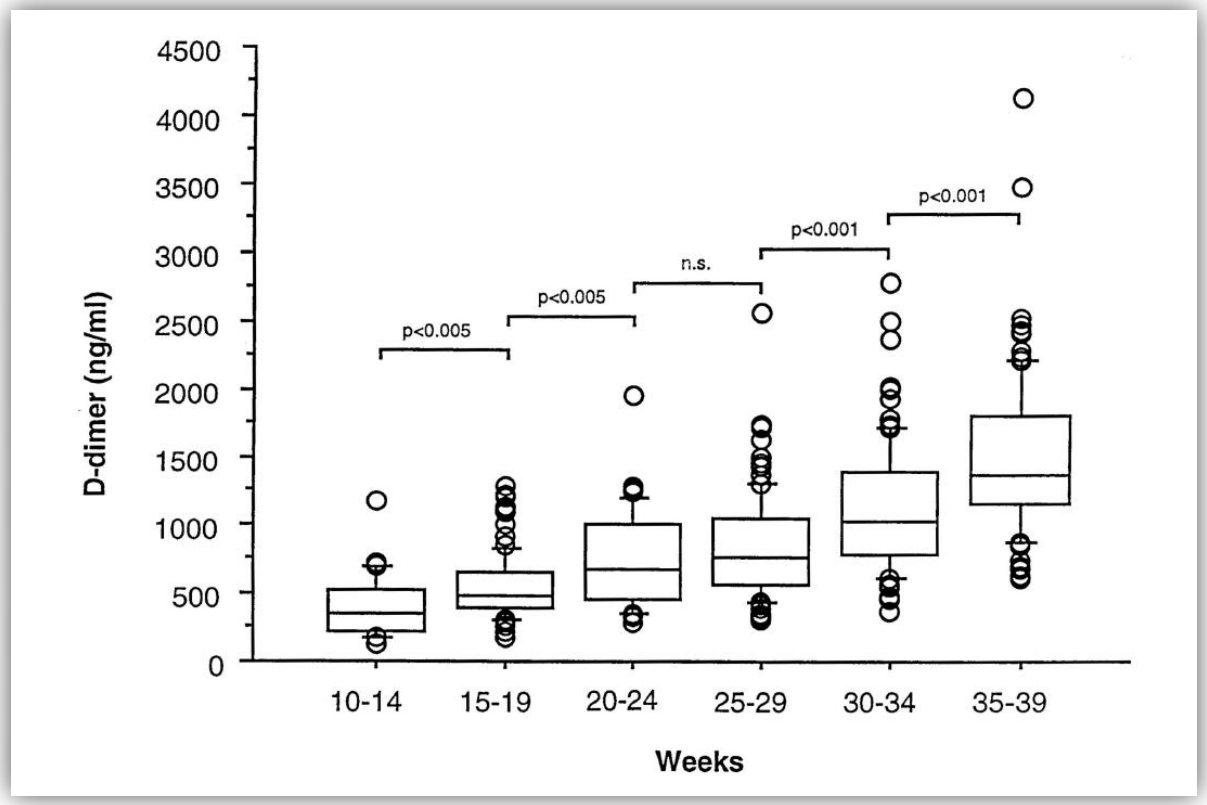


British Journal of Haematology, 2001, **115**, 150–152

SHORT REPORT

TAFI antigen and D-dimer levels during normal pregnancy and at delivery

PATRICK CHABLOZ,[1] GUIDO REBER,[2] FRANÇOISE BOEHLEN,[2] PATRICK HOHLFELD[1] AND PHILIPPE DE MOERLOOSE[2]
[1]*Department of Gynaecology and Obstetrics, Lausanne University Hospital, Lausanne, and* [2]*Division of Angiology and Haemostasis, Geneva University Hospital, Geneva, Switzerland*

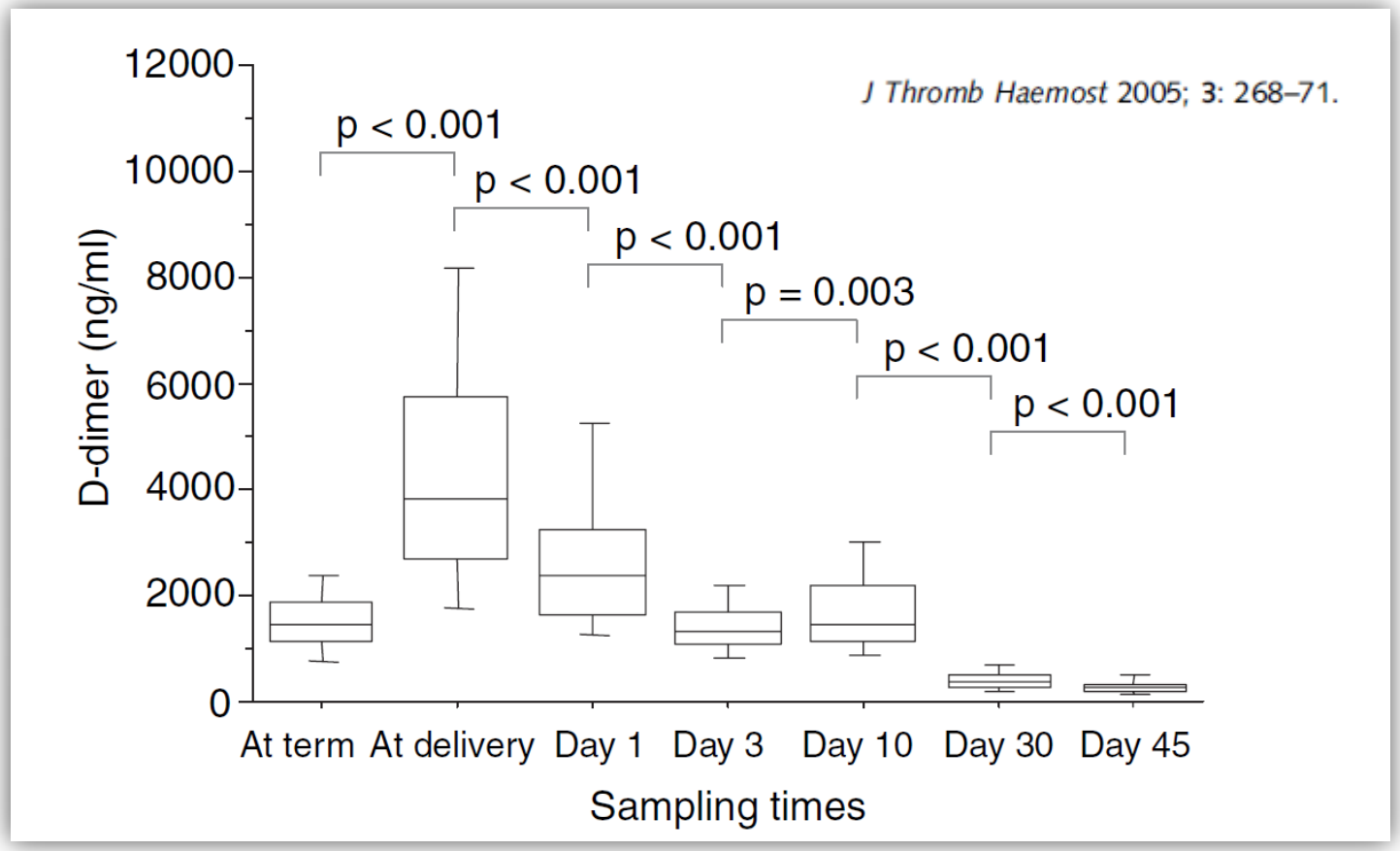


ORIGINAL ARTICLE

D-dimer levels during delivery and the postpartum

M. EPINEY,* F. BOEHLEN,† M. BOULVAIN,* G. REBER,† E. ANTONELLI,* M. MORALES,* O. IRION* and P. DE MOERLOOSE†

*Department of Obstetrics and Gynecology and †Division of Angiology and Haemostasis, University Hospitals of Geneva and Faculty of Medicine, Geneva, Switzerland



Venous thromboembolism diagnosis: unresolved issues

Marc Righini[1]; Grégoire Le Gal[2]; Henri Bounameaux[1]

[1]Division of Angiology and Hemostasis, Geneva University Hospital and Faculty of Medicine, Geneva, Switzerland; [2] Ottawa Health Research Institute, Ottawa, Canada

Thromb Haemost 2015; 113: 1184–1192

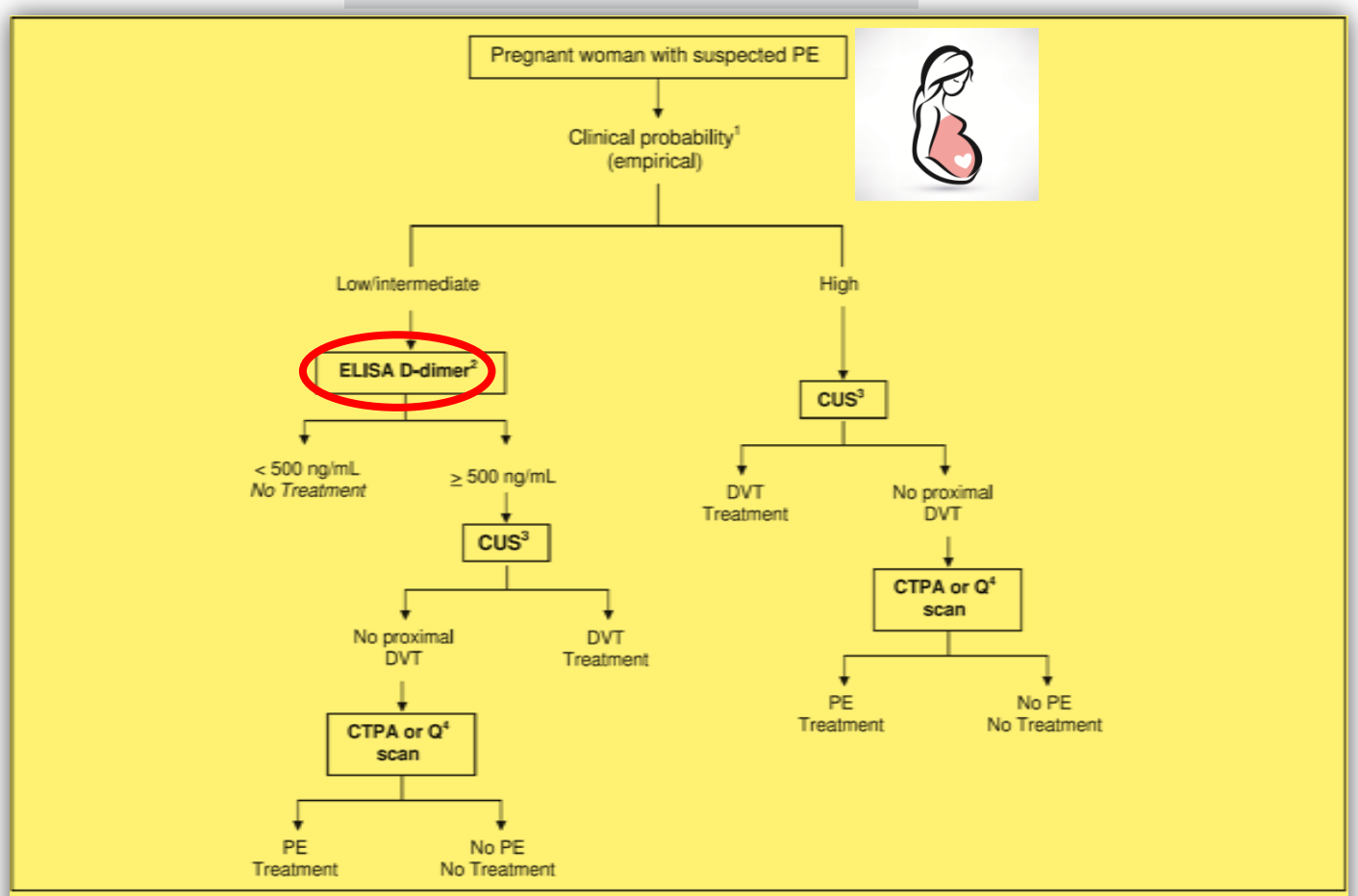
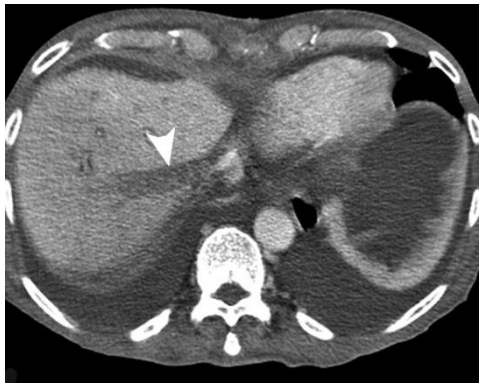
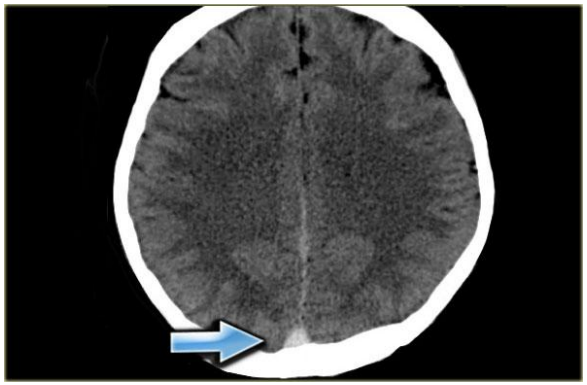


Figure 3: Proposed diagnostic algorithm for suspected PE in pregnant women. [1]Assessment of clinical probability is empirical as no usual clinical prediction rule as the Wells score or the Geneva rule has been validated in pregnant women. [2]A D-dimer level below the usual cut-off of 500 ng/ml should allow to rule out PE in pregnant women, even if this has never been formally validated in a prospective management outcome study. [3]Although the rate of positive finding is lower in patients without leg symptoms, the presence of a proximal DVT in a patient with suspected PE allows to rule in PE diagnosis and avoids the need for a radiating test. [4]While Q scan is the most often used imaging test to rule out PE in pregnant women, formal validation is poor. Data regarding ventilation perfusion lung scan are also scarce. CTPA is increasingly used but is associated with the concerns of maternal radiation. Please note that this proposed strategy has not been validated.

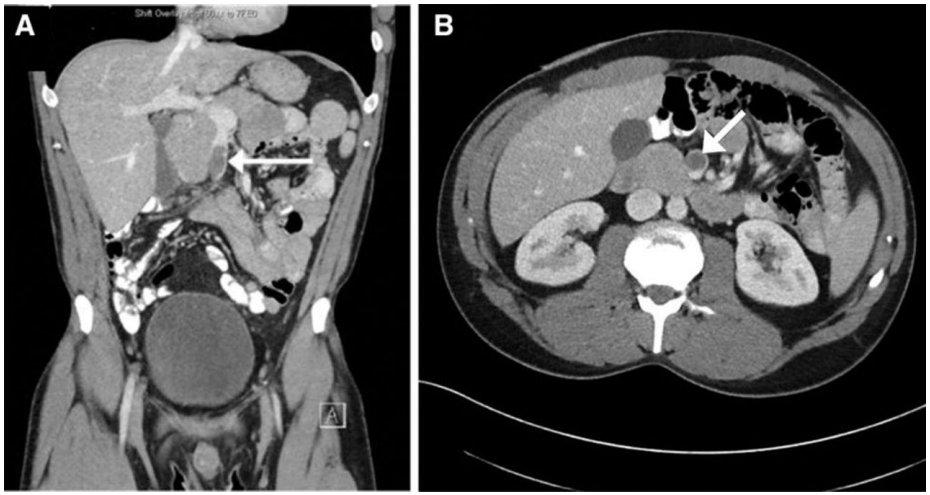
Unusual site thrombosis



Portal vein thrombosis



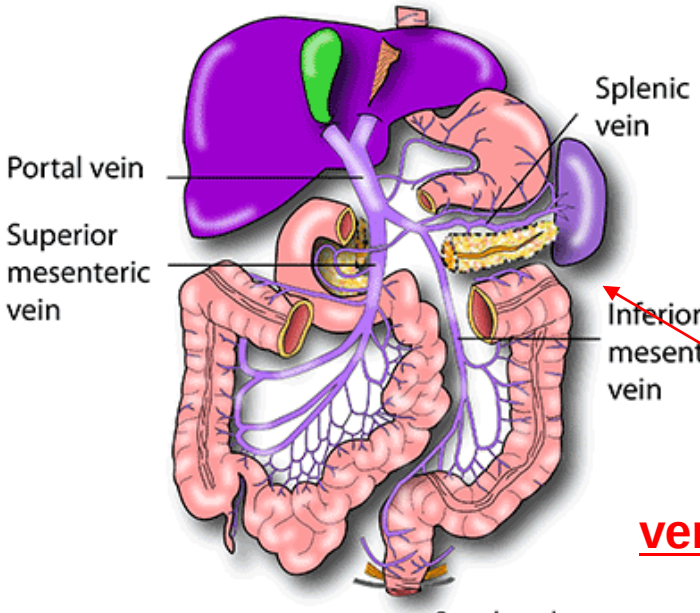
Sinus vein thrombosis

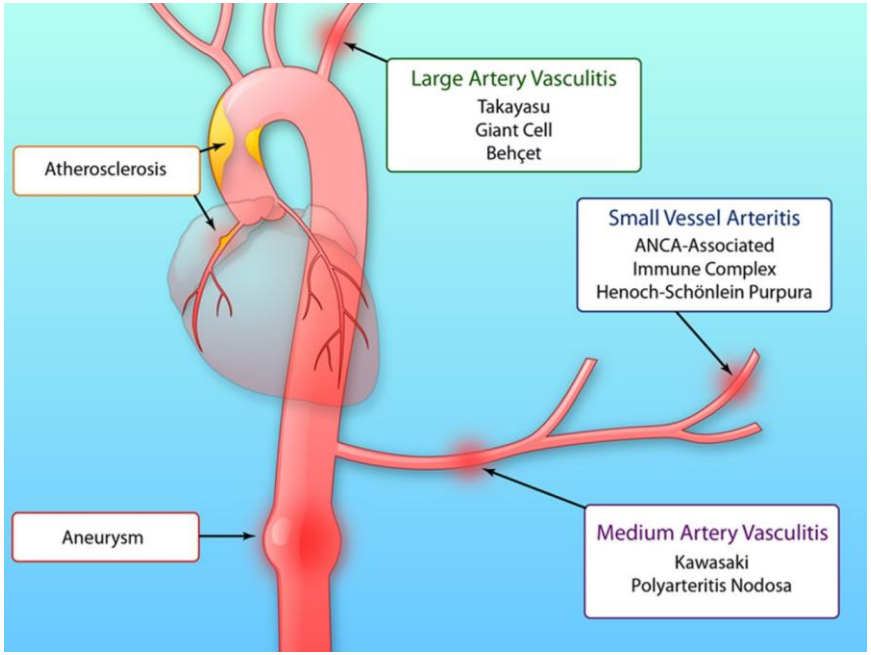


Mesenteric vein thrombosis

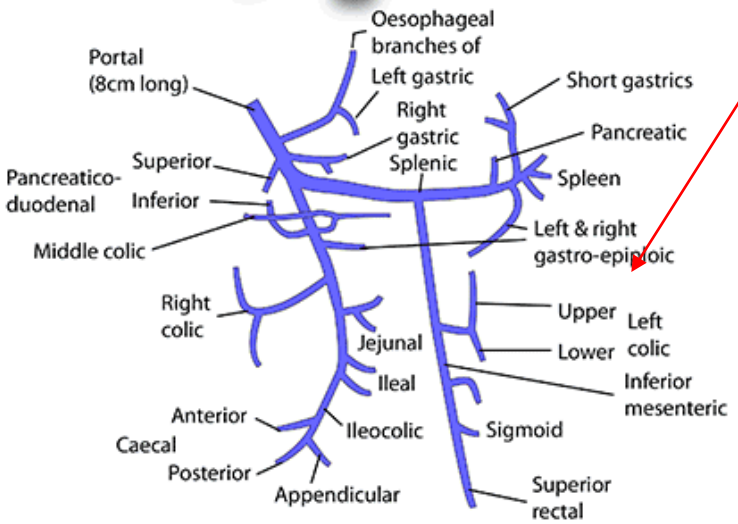
Arterial thrombosis

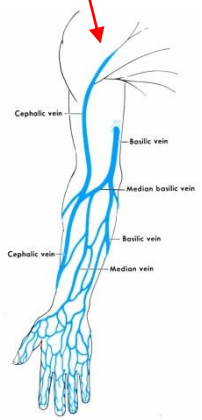
Atheromatosis, cardiac embolism, vasculitis

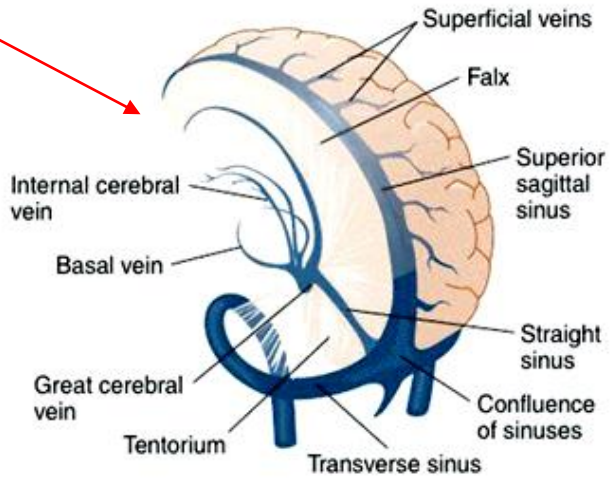




Unusual site
venous thrombosis







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Full Length Article

A multicenter prospective study of risk factors and treatment of unusual site thrombosis☆

Kim Ma [a,*], Phillip Wells [b], Charlotte Guzman [c], David Anderson [d], Mark Blostein [a], Andrew Hirsch [e], Alejandro Lazo-Langner [f], Michael J. Kovacs [g], Marc Rodger [b], Vicky Tagalakis [h], Susan R. Kahn [h,*]

[a] Medicine, Division of Hematology, McGill University, Montreal, Canada
[b] Clinical Epidemiology, Ottawa Hospital Research Institute, Ottawa, Canada
[c] Clinical Epidemiology, Lady Davis Institute, Montreal, Canada
[d] Medicine and Clinical Epidemiology, Dalhousie University, Halifax, Canada
[e] Medicine, Respiratory Division, Jewish General Hospital, Montreal, Canada
[f] Medicine and Clinical Epidemiology, Division of Hematology, University of Western Ontario, London, Canada
[g] Medicine, Division of Hematology, University of Western Ontario, London, Canada
[h] Medicine and Clinical Epidemiology, Jewish General Hospital, Montreal, Canada

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152 patients were prospectively enrolled at 4 Canadian centers

Distribution of localization

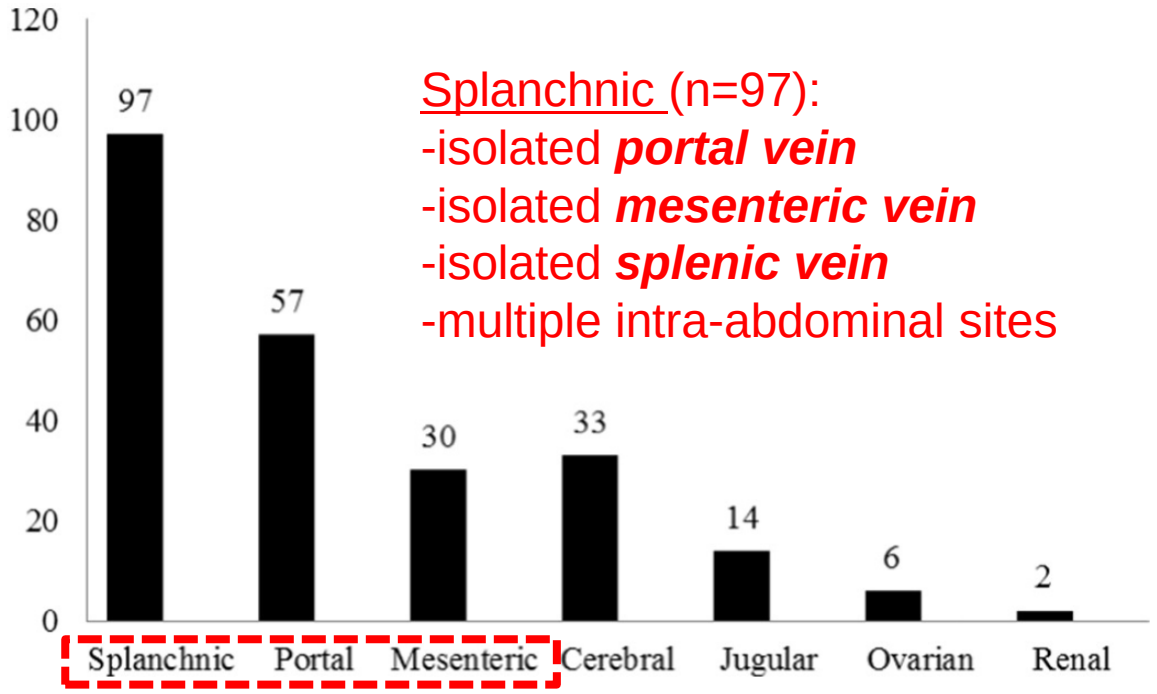

Fig. 1. Number of cases of USDVT by thrombosis site (n = 152). USDVT unusual site deep vein thrombosis. * Splanchnic vein thrombosis included: isolated portal vein (57), isolated mesenteric vein (30), isolated splenic vein (1), and thrombosis in multiple intra-abdominal sites (9).

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Characteristics of patients

Splanchnic vein thrombosis

Table 2
Baseline characteristics of patients with splanchnic vein thrombosis (n = 97).

Site of USDVT	Mesenteric	Portal	Other[a]	Total
Cases	30 (31)	57 (59)	10 (10)	97 (100.0)
Age, years (mean ± SD)	52.4 ± 18.1	56.9 ± 13.7	53.1 ± 9.3	55.1 ± 14.9
Sex				
Male	18 (60)	29 (51)	3 (30)	50 (52)
Female	12 (40)	28 (49)	7 (70)	47 (49)
Symptomatic VTE				
Yes	23 (77)	36 (63)	7 (70)	66 (68)
No	7 (23)	21 (37)	3 (30)	31 (32)
Risk factors for VTE				
Active malignancy[b]	9 (30)	18 (32)	1 (10)	28 (29)
Major surgery[c]	11 (37)	12 (21)	2 (20)	25 (26)
Immobility[d]	4 (13)	9 (16)	2 (20)	15 (16)
Central venous catheter	1 (3)	4 (7)	0 (0)	5 (5)
Recent hospitalization[e]	15 (50)	27 (47)	7 (70)	49 (51)
Inflammatory disease	10 (33)	23 (40)	5 (50)	38 (39)
Estrogen exposure	3 (10)	3 (5)	1 (10)	7 (7)
History of VTE	3 (10)	8 (14)	3 (30)	14 (14)
Family history of VTE	6 (20)	8 (14)	1 (10)	15 (16)
Known thrombophilia	0 (0)	1 (2)	0 (0)	1 (1)

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Prevalence of thrombophilia
in unusual site thrombosis

Table 6
Prevalence of thrombophilia per site of VTE (n = 75)[a].

Thrombophilia	Mesenteric	Portal vein	Multiple abdominal VTE	Cerebral	Jugular	Other[b]	Total
No. of cases	13 (17)	29 (39)	7 (9)	19 (25)	4 (5)	3 (4)	75 (100)
No. of thrombophilia	4 (31)	10 (35)	3(43)	7 (37)	0 (0)	1 (33)	25 (33)
Factor V Leiden	0 (0)	1 (3)	2 (29)	3 (16)	0 (0)	1 (33)	7 (9)
G20210A Prothrombin gene mutation	1 (8)	1 (3)	1 (14)	1 (5)	0 (0)	0 (0)	3 (4)
Protein C deficiency	1 (8)	0 (0)	0 (0)	1 (5)	0 (0)	0 (0)	2 (3)
Protein S deficiency	1 (8)	1 (3)	0 (0)	0 (0)	0 (0)	0 (0)	2 (3)
Antithrombin deficiency	1 (8)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1)
Hyperhomocysteinemia	0 (0)	2 (7)	0 (0)	0 (0)	0 (0)	0 (0)	2 (3)
Lupus anticoagulant	0 (0)	1 (3)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1)
Anticardiolipin antibodies	0 (0)	1 (3)	0 (0)	2 (11)	0 (0)	0 (0)	3 (4)
JAK 2 mutation	0 (0)	3 (10)	0 (0)	0 (0)	0 (0)	0 (0)	3 (4)

33%

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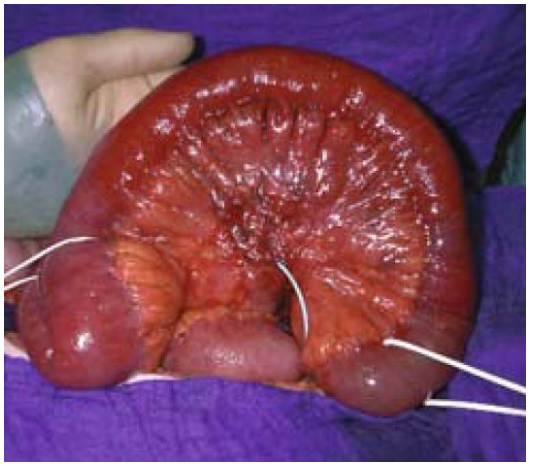
Splanchnic thrombosis

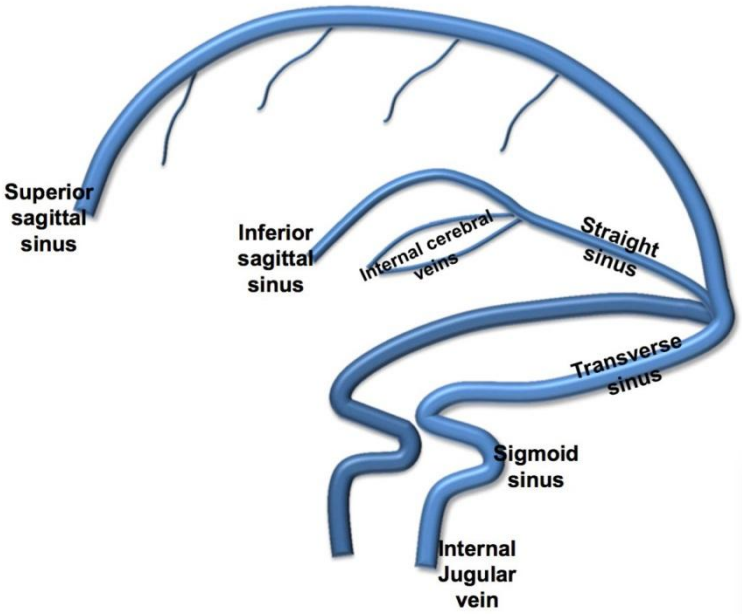
Causative factors of splanchnic thrombosis
Primary thrombosis (inherited or acquired prothrombotic conditions)
Factor V Leiden
Protein C deficiency
Protein S deficiency
Prothrombin gene mutations
Antithrombin III deficiency
Antiphospholipid antibody development
Homocystenemia
Pregnancy
Post-pregnancy
Neuroendocrine neoplasms
Endocrine active neoplasm
Polycythemia vera
Essential thrombocythemia
Paroxysmal nocturnal hemoglobinuria
Secondary thrombosis
Inflammatory
Diverticulitis
Crohn's disease
Ulcerative colitis
Pancreatitis
Peritonitis
Postoperative
Splenectomy
Visceral resections
Variceal ligation, embolization
Trauma
Blunt abdominal trauma
Direct pancreatic trauma
Direct mesenteric root trauma
Direct duodenal trauma

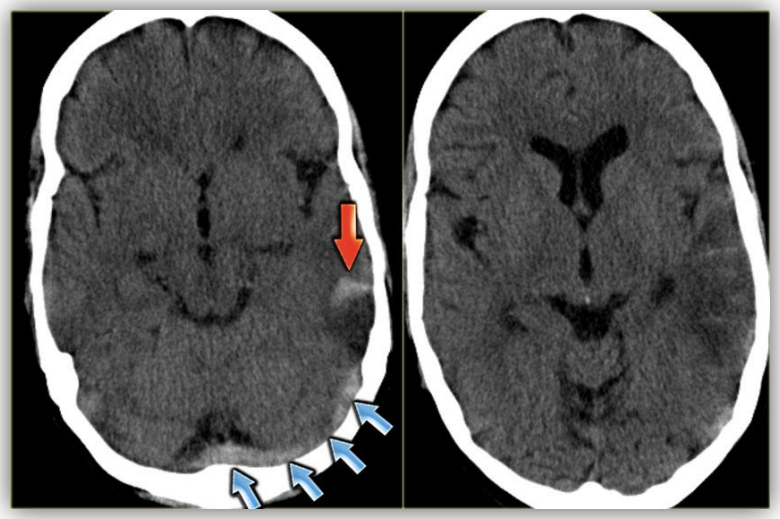


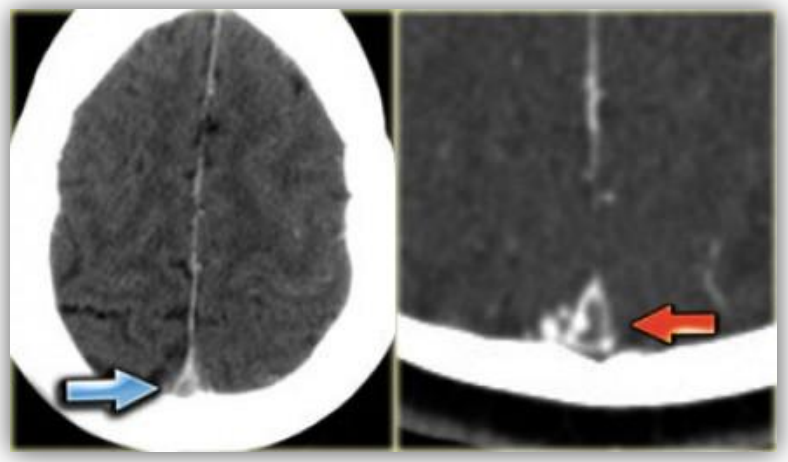
Thrombosis of superior mesenteric vein

Splanchnic venous thrombosis. Götz M. Richter, Jens Werner, p 639-48, from «General Surgery», Springer London, 2009

Cerebral venous sinus thrombosis







The incidence of cerebral venous thrombosis (CVT) is estimated to be **between 2 and 5 per million per year**

Cerebral venous sinus thrombosis

Cerebral Venous Sinus Thrombosis Incidence Is Higher Than Previously Thought

A Retrospective Population-Based Study

Sharon Devasagayam, MBBS; Ben Wyatt, MBBS; James Leyden, MBBS;
Timothy Kleinig, PhD, MBBS

Background and Purpose—The incidence of cerebral venous thrombosis (CVT) varies between studies, but it is estimated to be between 2 and 5 per million per year. A recent study in the Netherlands with comprehensive ascertainment suggested a much higher incidence. It is uncertain whether these differing estimates reflect the quality of ascertainment or true variation. The purpose of this study was to determine the incidence of CVT in Adelaide, using a novel clinical and radiological methodology.

Methods—We retrospectively identified CVT *International Classification of Diseases-coded* cases from all Adelaide public hospitals from 2005 to 2011. We also searched all neuroimaging studies (259 101) from these hospitals for text variations containing venous thromb. All potential cases were reviewed, and cases of incident CVT ascertained. Associations and outcomes were determined.

Results—Of 169 possible cases, 105 cases of CVT were confirmed (59 cases by both coding and neuroimaging, 40 from neuroimaging alone, and 6 from coding alone). In our population of 953 390 adults, this represented an incidence of 15.7 million per year (95% confidence interval, 12.9–19.0), the highest incidence reported. Of these cases, a possible procoagulant predisposition was identified in 48%. Fifty-five of 105 cases occurred in females. Relative risk of CVT in females of reproductive age was insignificantly higher than in males (1.18 [95% confidence interval, 0.94–1.48]).

Conclusions—Cerebral venous sinus thrombosis in our study was more common than previously reported, perhaps because of more complete ascertainment. Future CVT incidence studies should include comprehensive capture and review of neuroimaging. **(*Stroke*. 2016;47:2180-2182. DOI: 10.1161/STROKEAHA.116.013617.)**

Location of thrombosis

Table 2. Clinical and Radiological Manifestations

Clinical and Radiological Manifestations	
Median age, (interquartile range)	49, (40–61)
Sex, % female	52%
Potential procoagulant state identified	48%
Oral contraceptive use/HRT, % of women	31%
Pregnancy or puerperium, % of women	5%
Location of thrombus	
Multiple locations, %	57%
Superior sagittal sinus, %	16%
Transverse sinus, %	11%
Sigmoid sinus, %	9%
Cortical, %	5%
Cavernous sinus, %	1%
Straight sinus, %	1%
Mortality at discharge, %	9%
Mortality at follow-up (cumulative), %	12%

HRT indicates hormone replacement therapy.

Thrombophilia as risk factor for cerebral vein thrombosis

Table 1. Studies evaluating thrombophilia as risk factor for cerebral vein thrombosis

First author (reference)	Patients/controls	Risk factors analyzed	Main results
de Bruijn (7)	40/2248	FVL, OC. AT, PC, PS	OC associated with 13-fold increased risk; controls not tested for thrombophilia
Martinelli (8,9)	121/242	FVL, PT, OC, HHcy, AT, PC, PS APA	FVL associated with 5-fold, PT with 10-fold, HHcy with 4-fold and OC with 6-fold increased risk
Gadelha (10)	26/217	FVL, PT, OC	PT associated with 21-fold, OC with 8-fold increased risk
Rodrigues (11)	42/134	FVL, PT, OC	PT associated with 27-fold, OC with 10.5-fold increased risk
Bombeli (12)	51/120	FVL, PT, AT, PC, PS	Any inherited thrombophilia associated with 2.5-fold increased risk
Boncoraglio (13)	28/100	FVL, PT, HHcy	HHcy associated with 4-fold increased risk
Ventura (14)	30/40	FVL, PT, HHcy	PT associated with 16-fold, HHcy with 7-fold increased risk
Bugnicourt (15)	25/64	FVIII, VWF	FVIII and VWF plasma levels significantly higher in CVT
Voetsch (16)	23/123	H_2 aplotype in the *GPx-3* gene promoter	H_2 haplotype in the *GPx-3* gene promoter associated with 11-fold increased risk
Lichy (17)	77/203	FVL, PT, TAFI G-438A, PZ Intron FG79A	PT associated with 5-fold, FVL with 2-fold increased risk
Libourel (18)	63/209	FVL, PT, AT, PC, PS, FVIII, FIX, FXI, HHcy	The majority of CVT patients had single or multiple thrombophilic abnormalities

Risk: 4-27-fold increased

FVL: Factor V Leiden; **PT**: prothrombin G20210A mutation; **PC**: protein C deficiency; **PS**: protein S deficiency; **AT**: antithrombin deficiency; **APA**: antiphospholipid antibodies; **Hhcy**: hyperhomocysteinemia; **OC**: oral contraceptive; **GPx-3**: glutathione peroxidase; **TAFI**: thrombin activatable fibrinolysis inhibitor; **PZ**: protein Z; **CVT,** cerebral venous thrombosis;

(Blood. 2008;112:4818-4823)

Diagnosis and treatment of unusual site thrombosis

-**Various clinical symptoms…**
-Symptoms or signs of unusual site thrombosis differs significantly
-Wide range of imaging tests
-**No clear guidelines on treatment… (patient specific treatment!)**
-Choice of anticoagulant therapy (No data on NOAC`s…)
-Clinician are less experiences with unusual site thrombosis

-**Often incidental finding** in patients with unclear clinical symptoms

Duration of anticoagulation therapy

Table 4
Follow-up data.

Data	6 months	1 year	2 years
Number of patients attending visit[a] (baseline N = 152)	138 (91)	128 (84)	123 (81)
Deaths	9	9	4
Cause of death			
Malignancy[b]	8	8	4
Other	1	1	0
Ongoing anticoagulation	66 (48)	40 (31)	27 (22)

6 months (48%) **1 year (31%)** **2 years (22%)**

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