Pulmonary Embolism

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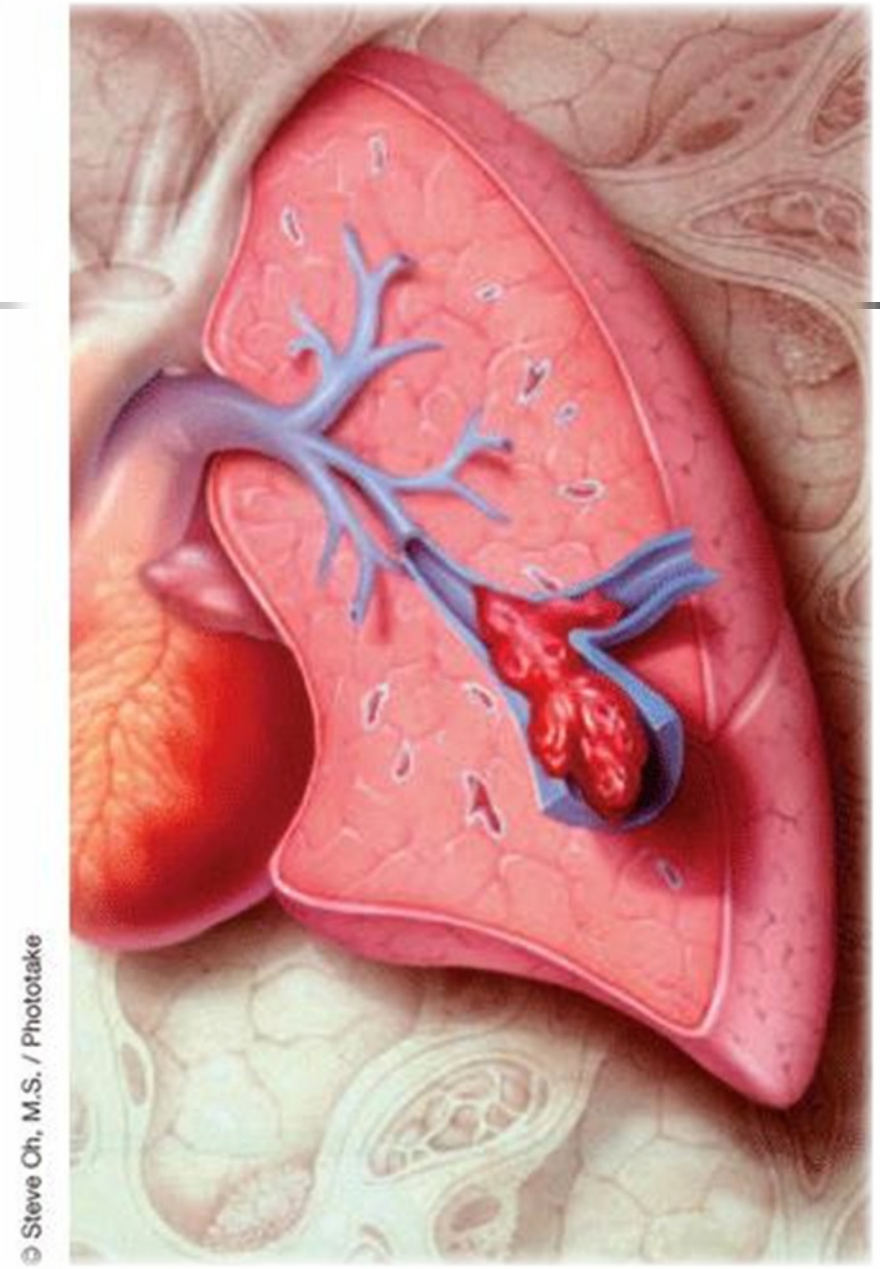
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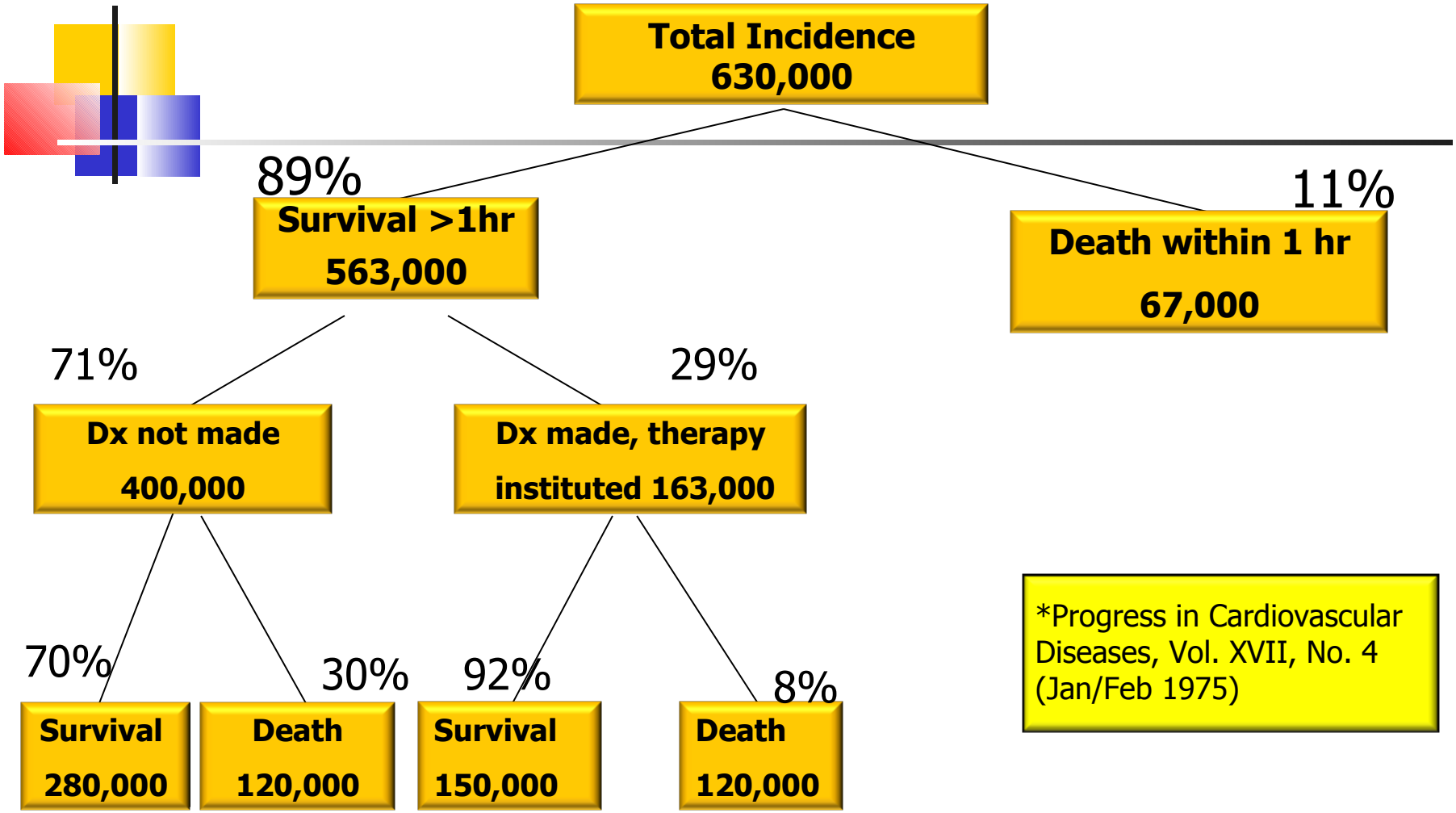
- 50,000 individuals die from PE each year in USA
- The incidence of PE in USA is 500,000 per year

Incidence of Pulmonary Embolism Per Year in the United States*



*Progress in Cardiovascular Diseases, Vol. XVII, No. 4 (Jan/Feb 1975)

Risk factor for venous thrombosis

- Stasis
- Injury to venous intima
- Alterations in the coagulation-fibrinolytic system

Source of emboli

- Deep venous thrombosis (>95%)
- Other veins:
  - Renal
  - Uterine
  - Right cardiac chambers

Risk factors for DVT

- General anesthesia
- Lower limb or pelvic injury or surgery
- Congestive heart failure
- Prolonged immobility
- Pregnancy
- Postpartum
- Oral contraceptive pills
- Malignancy
- Obesity
- Advanced age
- Coagulation problems

Clinical features

- Sudden onset dyspnea
- Pleuritic chest pain
- Hemoptysis
- Clinical clues cannot make the diagnosis of PE; their main value lies in suggesting the diagnosis

Signs or symptoms observed in patients with thromboembolism			
		Study	
		Stein et al., % (n= 117)	Anderson et al., % (n= 131)
Pulmonary embolism	Dyspnea	73	77
	Tachypnea	70	70
	Chest pain	66	55
	Cough	37	—
	Tachycardia	30	43
	Cyanosis	1	18
	Hemoptysis	13	13
	Wheezing	9	—
	Hypotension	—	10

Signs or symptoms observed in patients with thromboembolism			
		Study	
		Stein et al., % (n= 117)	Anderson et al., % (n= 131)
Pulmonary Embolism	Syncope	—	10
	Elevated jugular venous pulse	—	8
	Temperature >38.5°C	7	—
	S-3 gallop	3	5
	Pleural friction rub	3	2

Signs or symptoms observed in patients with thromboembolism			
		Study	
		Stein et al., % (n= 117)	Anderson et al., % (n= 131)
Deep vein thrombosis	Swelling	28	88*
	Pain	26	56
	Tenderness	—	55
	Warmth	—	42
	Redness	—	34
	Homan's sign	4	13
	Palpable cord	—	6

Massive Pulmonary Embolism

- It is a catastrophic entity which often results in acute right ventricular failure and death
- Frequently undiscovered until autopsy
- Fatal PE typically leads to death within one to two hours of the event

Pathophysiology

- Massive PE causes an increase in PVR $\longrightarrow$ right ventricular outflow obstruction $\longrightarrow$ decrease left ventricular preload $\rightarrow$ Decrease CO

- In patients without cardiopulmonary disease, occlusion of 25-30 % of the vascular bed $\longrightarrow$ increase in Pulmonary artery pressure (PAP)

- Hypoxemia ensues $\longrightarrow$ stimulating vasoconstriction $\rightarrow$ increase in PAP

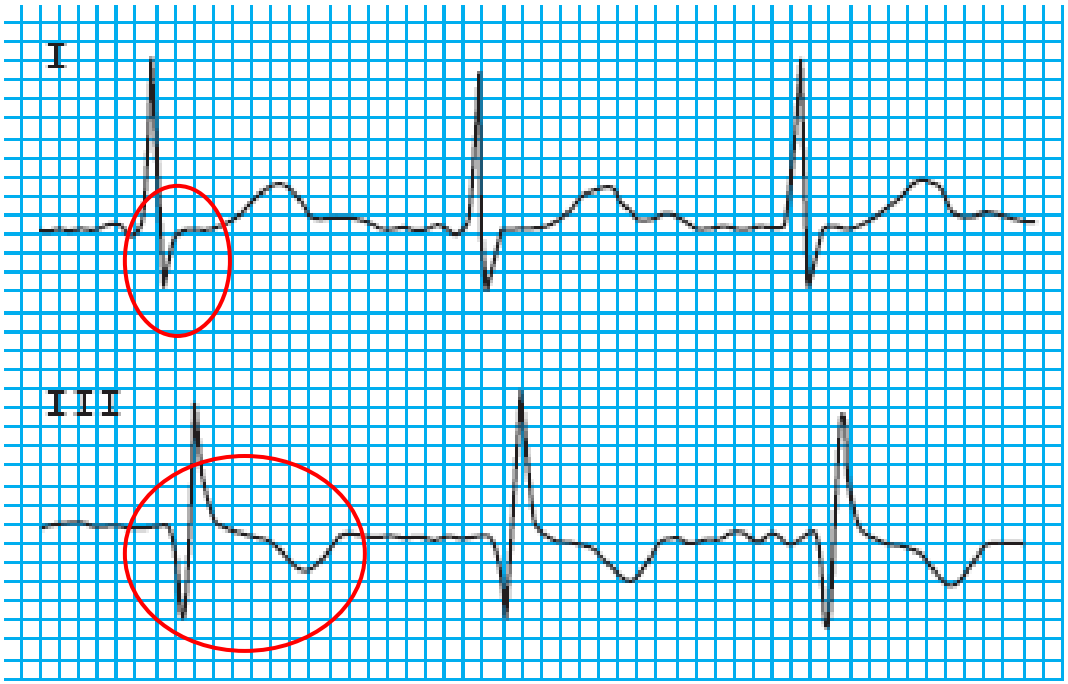
Pathophysiology

- More than 50% of the vascular bed has to be occluded before PAP becomes substantially elevated
- When obstruction approaches 75%, the RV must generate systolic pressure in excess of 50mmHg to preserve pulmonary circulation
- The normal RV is unable to accomplish this acutely and eventually fails

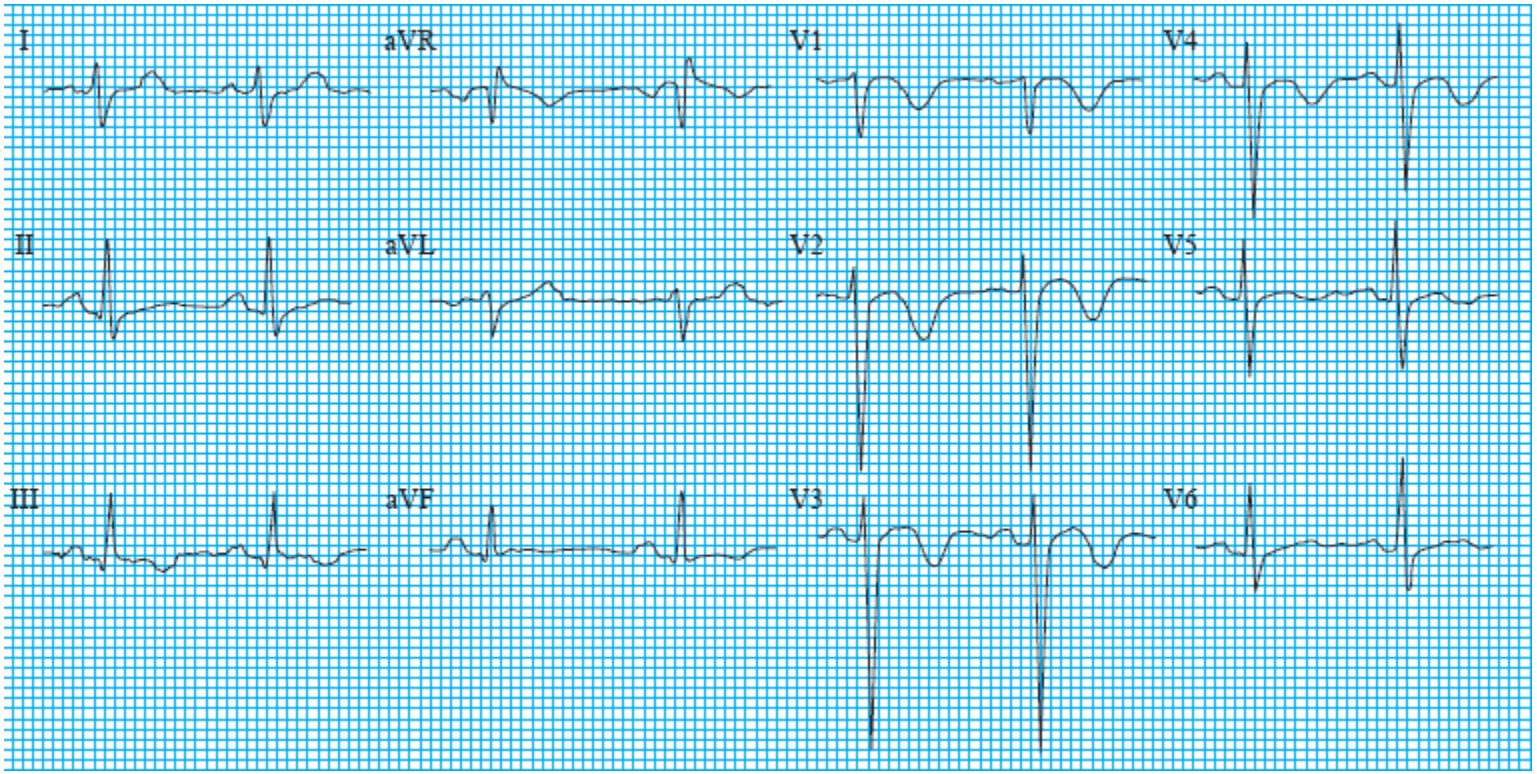
Diagnosis

- CXR
- ABG:
- ECG
- V/Q
- Spiral CT
- Echo
- Angio
- Fibrin Split Products/D-dimer

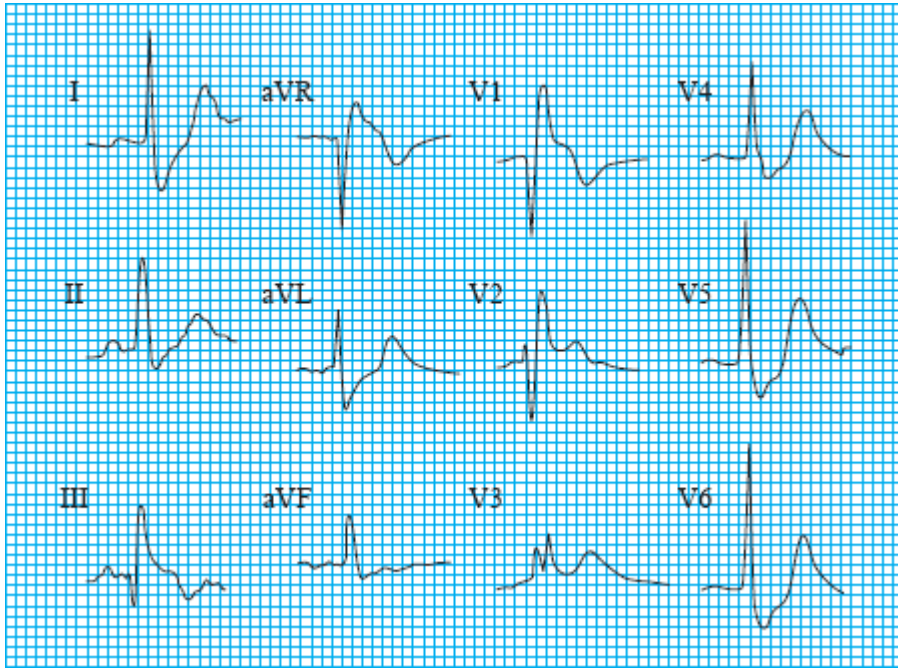
S1 Q3 T3 Pattern



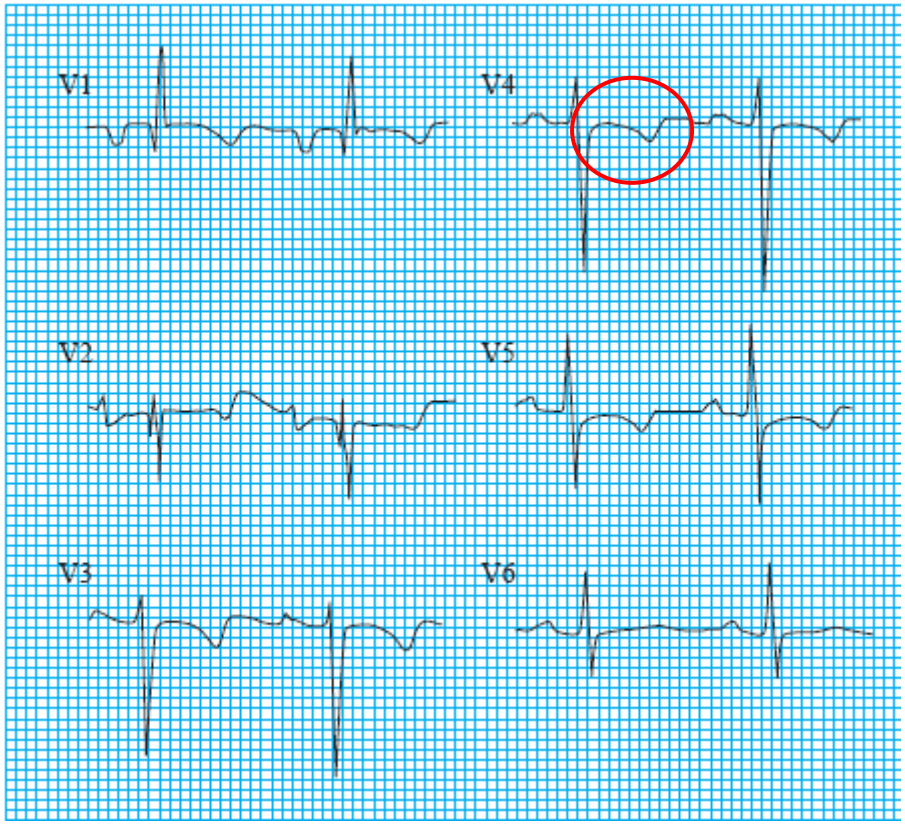
T-wave inversion



Rt. Bundle Branch Block



Rt. Ventricular Strain

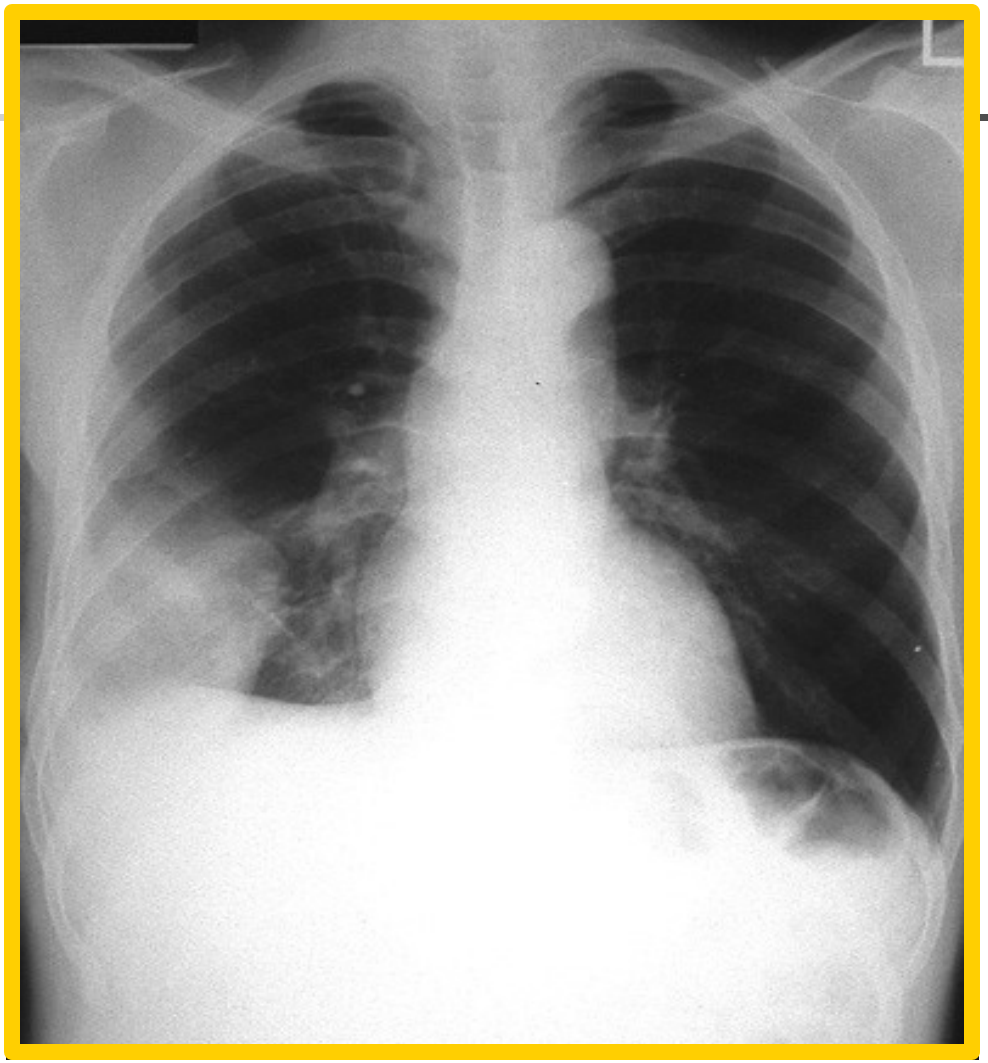


Diagnosis

The diagnosis of massive PE should be explored whenever oxygenation or hemodynamic parameters are severely compromised without explanation

- CXR
- ABG:
  - Significant hypoxemia is almost uniformly present when there is a hemodynamically significant PE
- V/Q
- Spiral CT
- Echo
- Angio

Chest radiograph showing pulmonary infarct in right lower lobe



Chest radiographic findings in patients with pulmonary embolism

	COPD, % (n= 21)	No prior cardiopulmonary disease, % (n= 117)
Atelectasis or pulmonary parenchymal abnormality	76	68
Pleural effusion	52	48
Pleural-based opacity	33	35
Elevated diaphragm	14	24
Decreased pulmonary vascularity	38	21
Prominent central pulmonary artery	29	15
Cardiomegaly	19	12
Westermark's sign*	5	7
Pulmonary edema	14	4

The use of ventilation perfusion scan in diagnosing pulmonary embolism

High probability

=2 large segmental (>75% of a segment) perfusion defects without corresponding ventilation or radiographic abnormalities or substantially larger than matching ventilation or radiologic abnormalities

OR

=2 moderate segmental (>25% and <75% of a segment) perfusion defects without matching ventilation or chest radiographic abnormalities plus one large unmatched segmental defect

OR

=4 moderate segmental perfusion defects without matching ventilation or chest radiologic abnormalities

The use of ventilation perfusion scan in diagnosing pulmonary embolism

Intermediate probability

Scans that do not fall into normal, very low, low, or high probability categories

The use of ventilation perfusion scan in diagnosing pulmonary embolism

Low probability

Nonsegmental perfusion defects

OR

Single moderate mismatched segmental perfusion defect with normal chest radiograph

OR

Any perfusion defect with a substantially larger abnormality on chest radiograph

OR

Large or moderate segmental perfusion defects involving no more than four segments in one lung and no more than three segments in one lung region with matching or larger ventilation/radiographic abnormalities

OR

More than three small segmental perfusion defects (<25% of a segment) with a normal chest radiograph

The use of ventilation perfusion scan in diagnosing pulmonary embolism

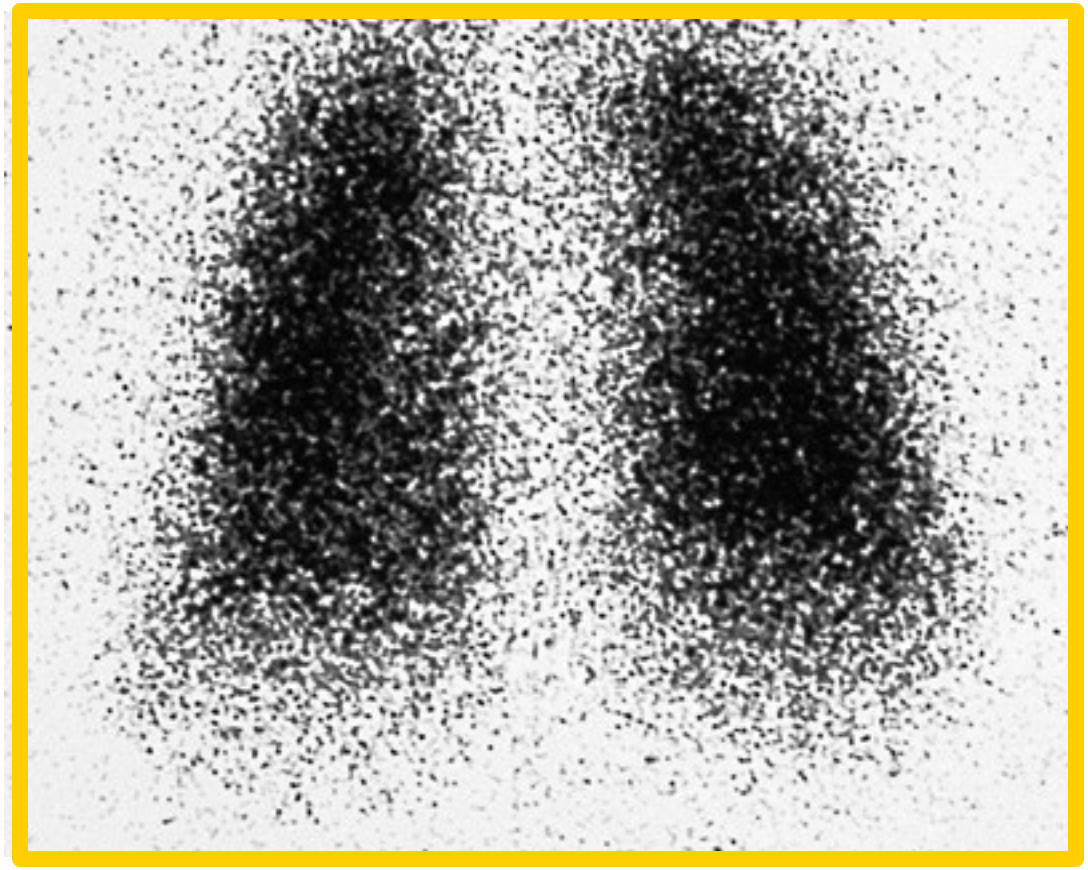
Very low probability

Three or fewer small segmental perfusion defects with a normal chest radiograph

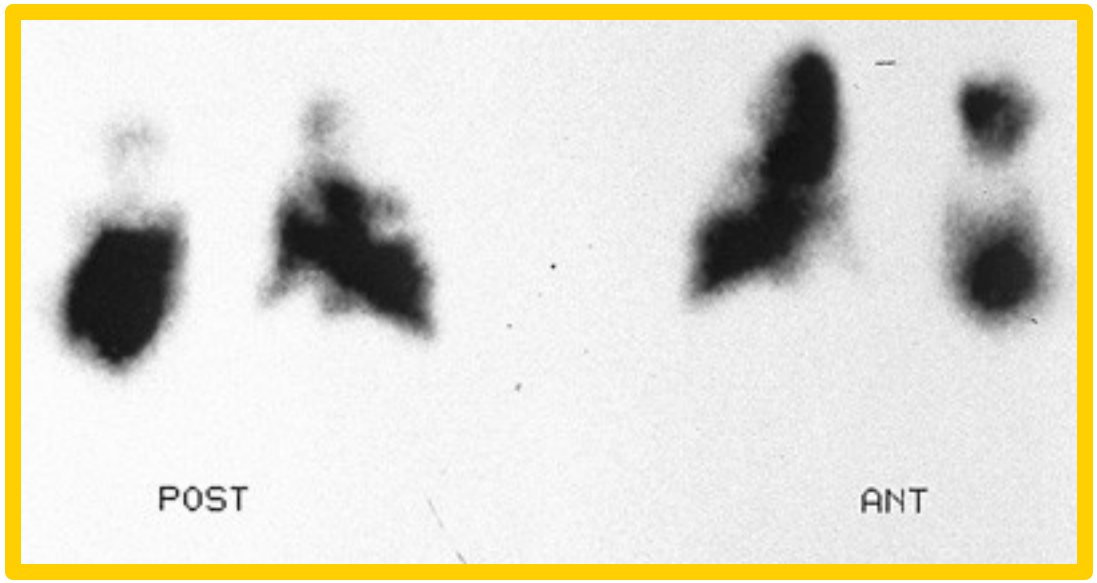
Normal

No perfusion defects present

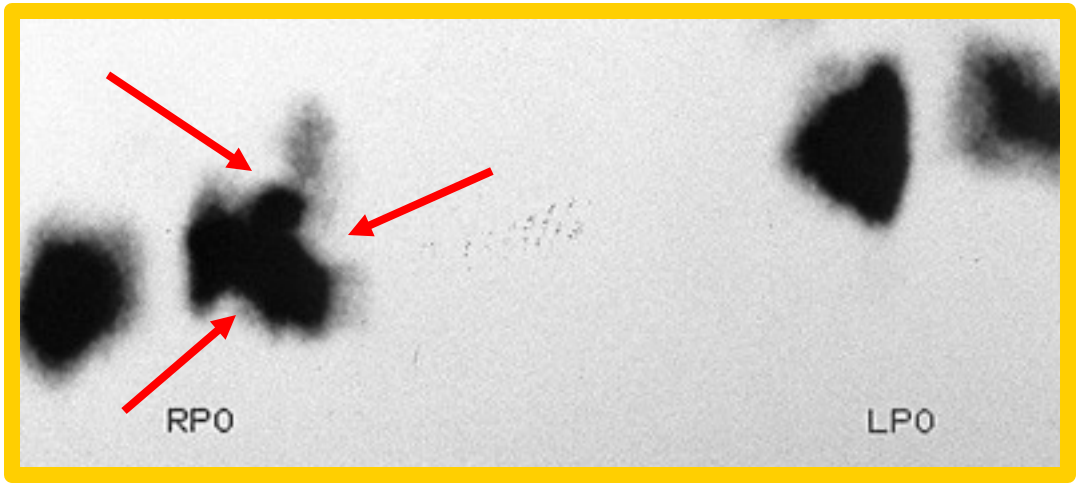
High-probability ventilation-perfusion scan



High-probability ventilation-perfusion scan



High-probability ventilation-perfusion scan

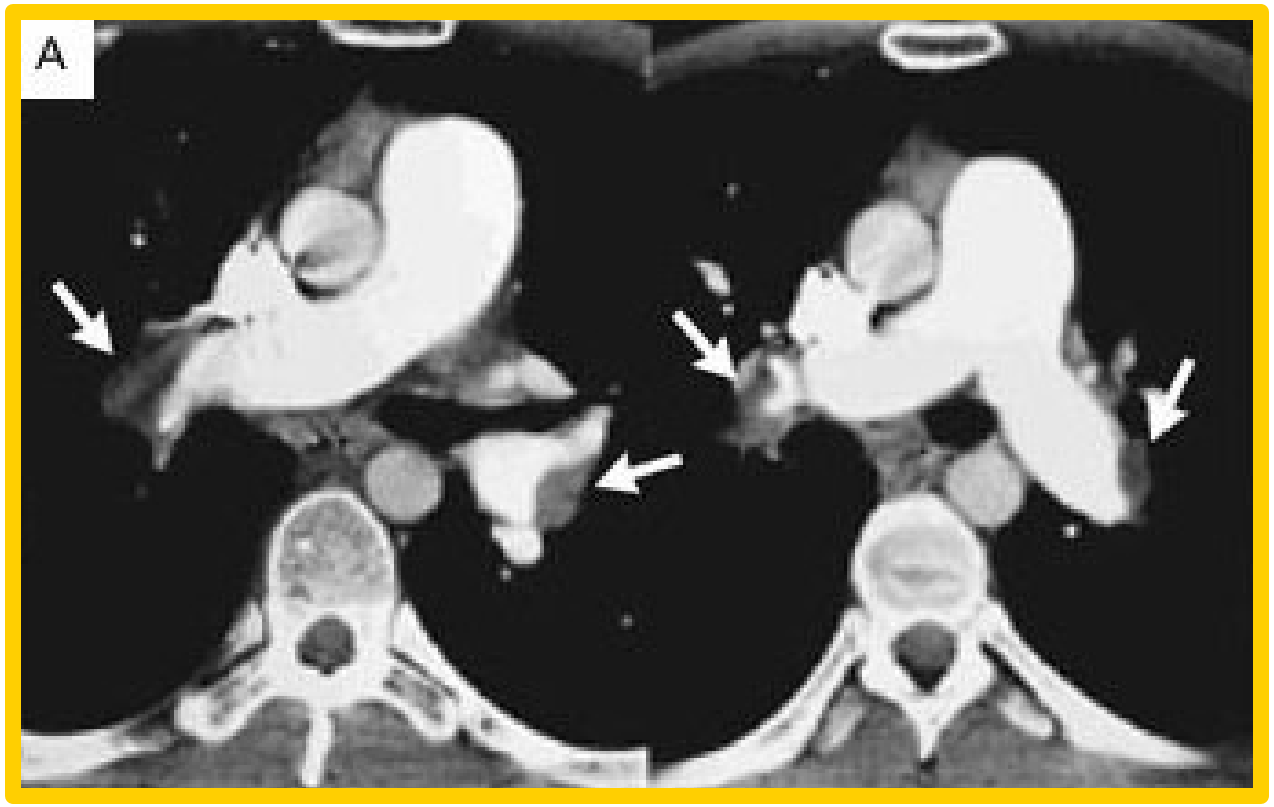


Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) results

Prospective investigation of pulmonary embolism diagnosis results

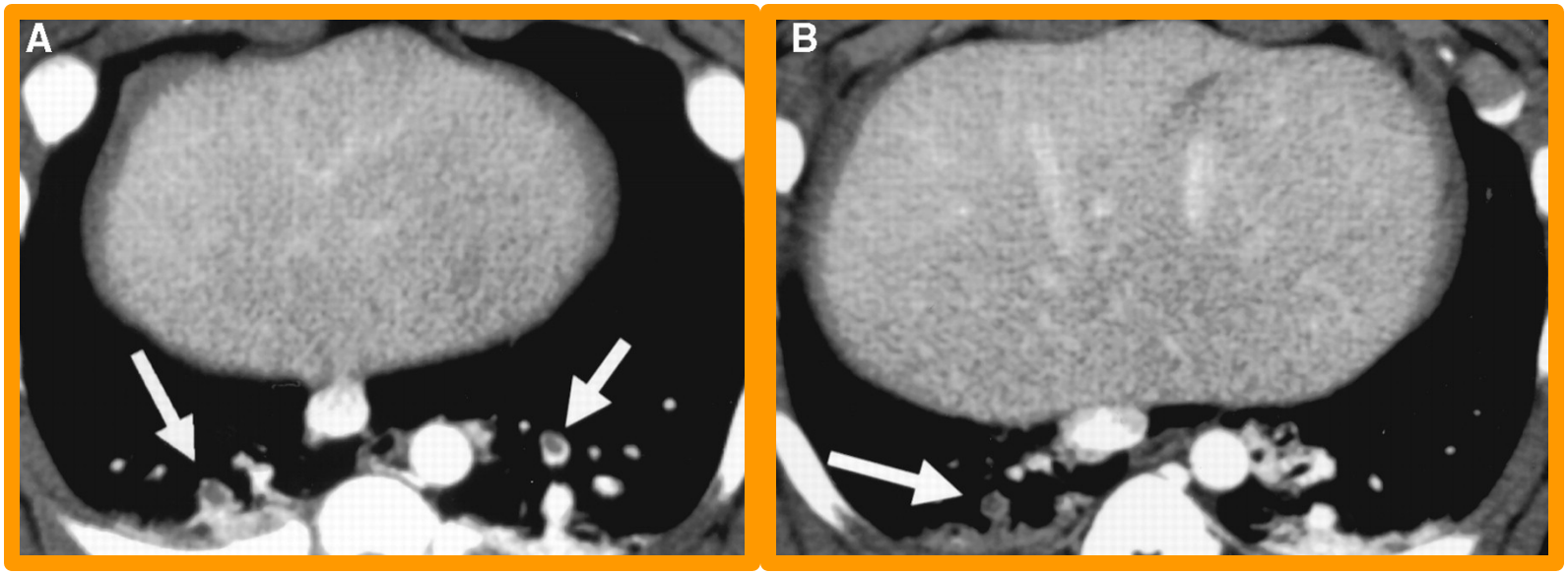
Scan category	PE present	PE absent	PE uncertain	No angiogram	Total
High probability	102	14	1	7	124
Intermediate probability	105	217	9	33	364
Low probability	39	199	12	62	312
Near normal or normal	5	50	2	74	131
Total	251	480	24	176	931

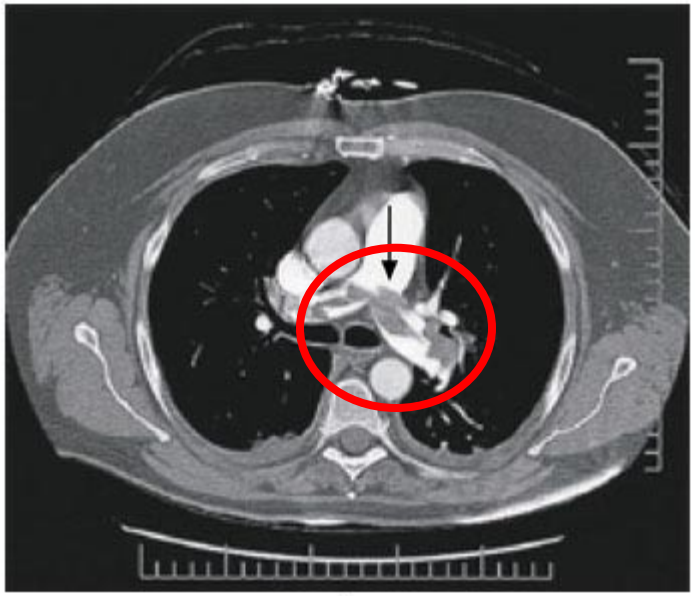
Spiral CT



Spiral CT

Spiral CT





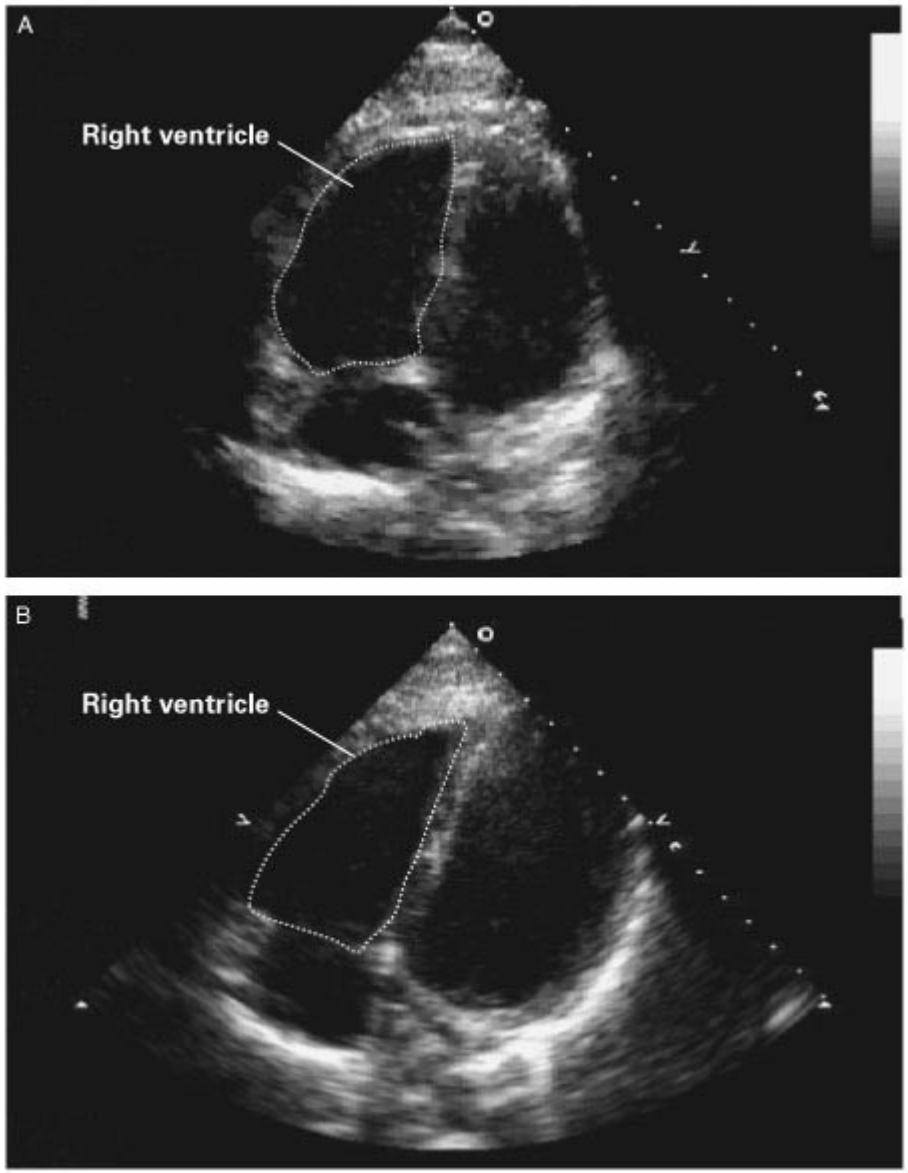
A

Before

B

After

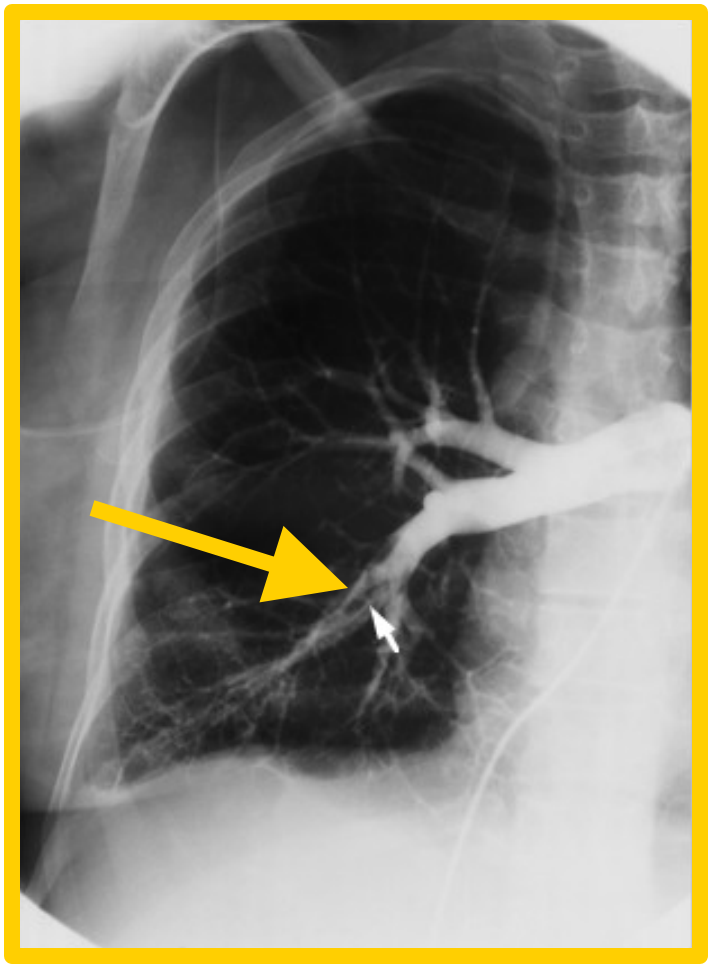
Tomographic scan showing infarcted left lung, large clot in right main pulmonary artery



Before

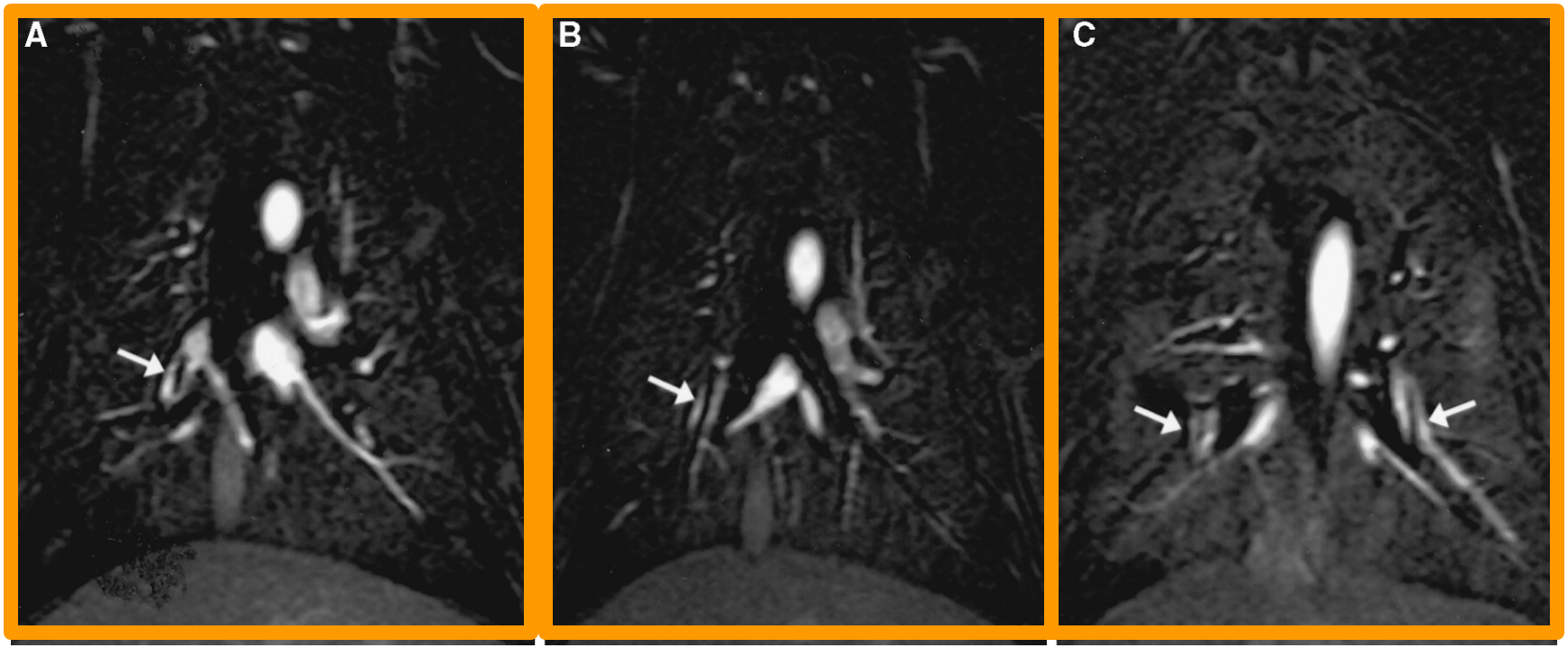
After

Pulmonary angiogram

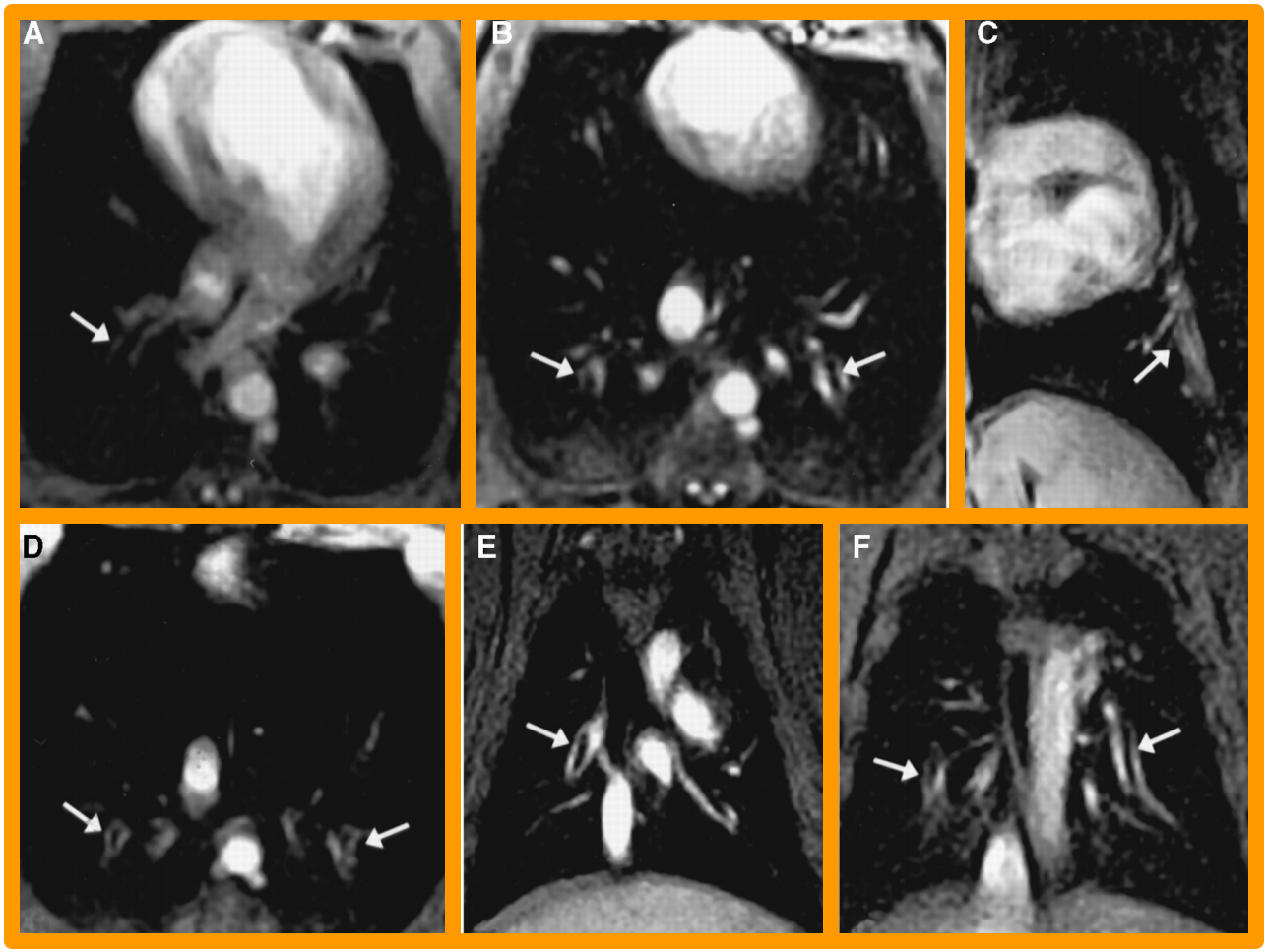


Pulmonary Angiogram

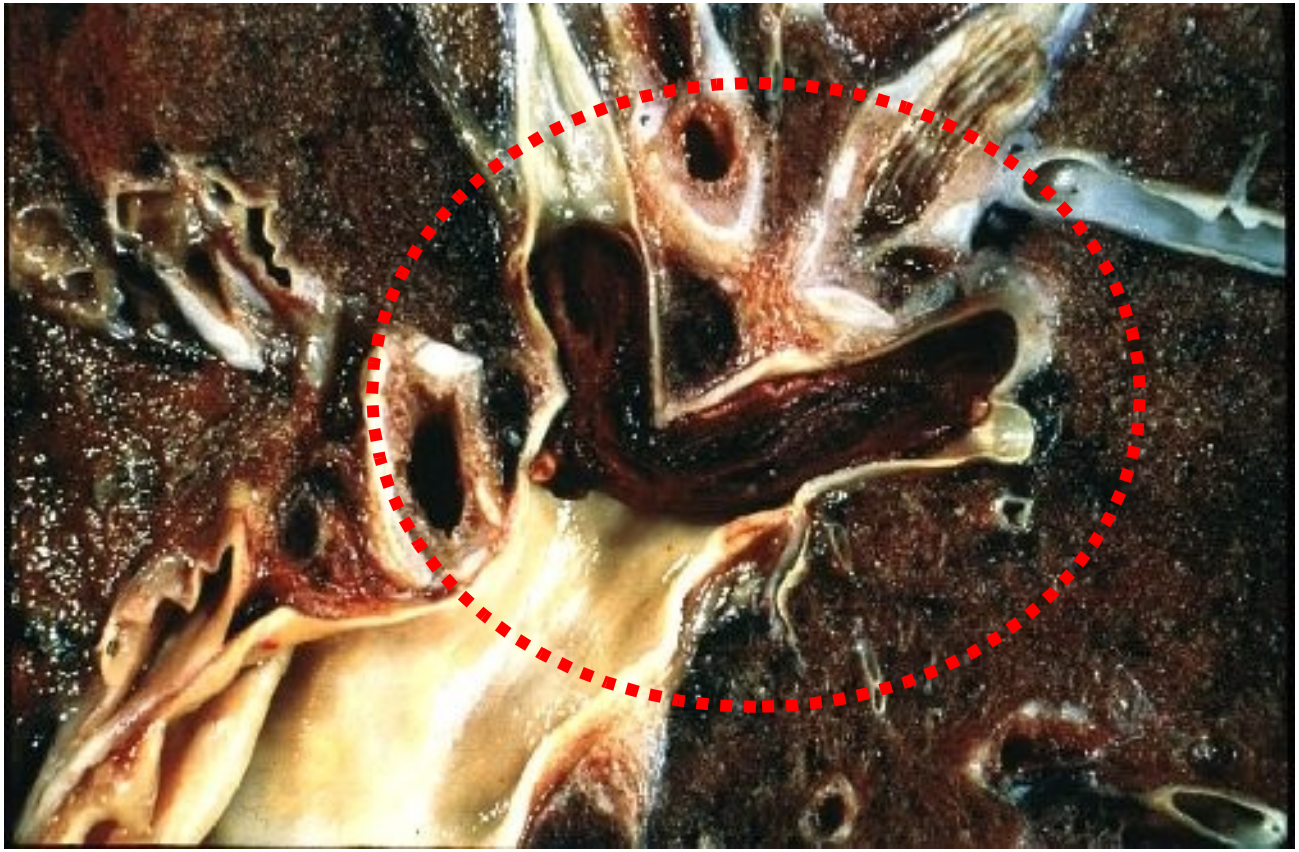
MRA with contrast



MRA Real Time



PULMONARY EMBOLISM

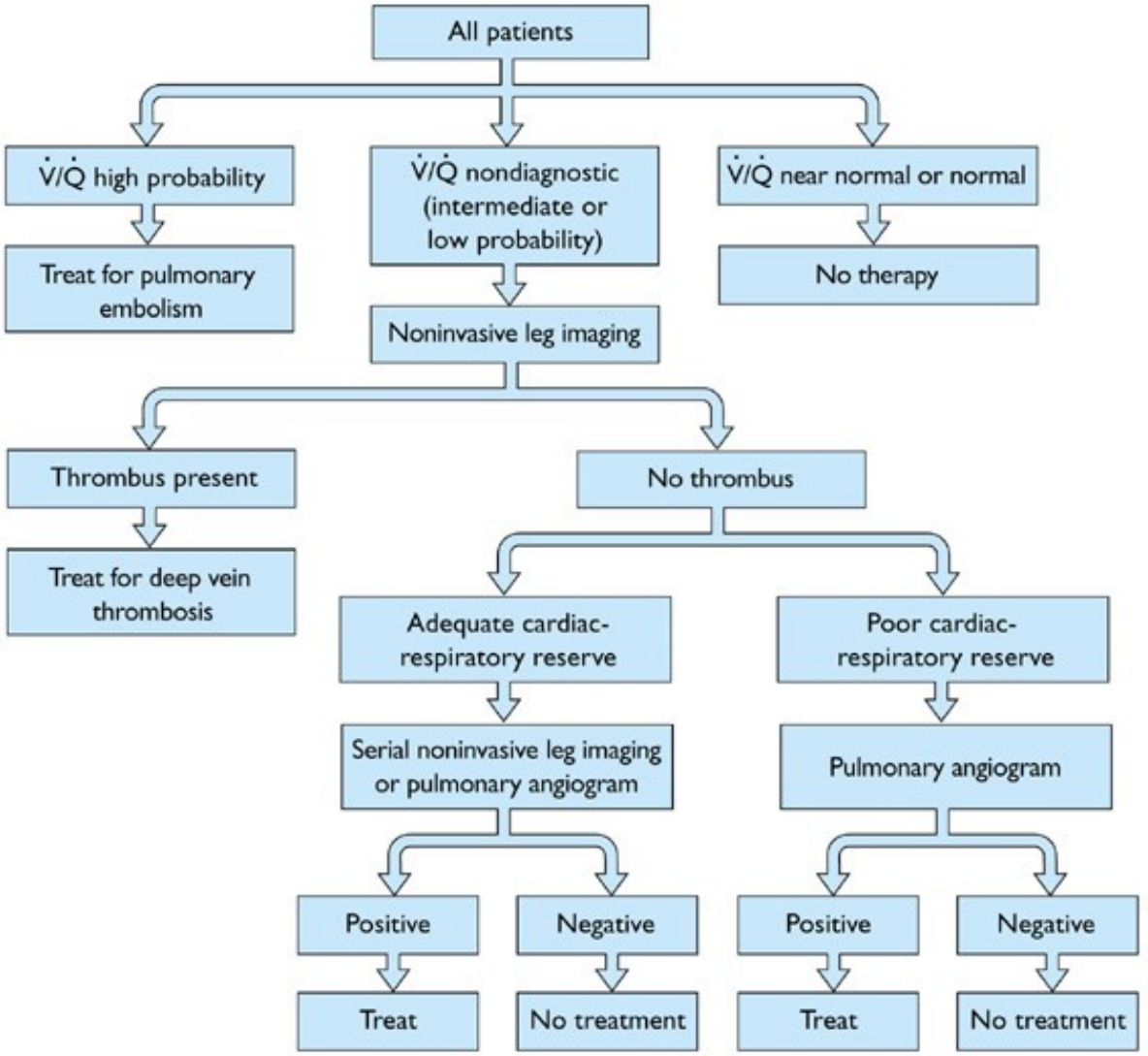


Sensitivity of spiral computed tomography, magnetic resonance angiography, and real-time magnetic resonance angiography, for detecting pulmonary emboli

Reader	CT	MRA	RT-MRA
1	72.1	79.1	97.7
2	69.8	81.4	97.7
Mean	71.0	80.3	97.7
K	0.86	0.84	1

Am J Respir Crit Care Med 2003

Suggested diagnostic strategy for venous thromboembolism



Dosage and monitoring of anticoagulant therapy

Dosage and monitoring of anticoagulant therapy

After initiating heparin therapy, repeat APTT every 6 h for first 24 h and then every 24 h when therapeutic APTT is achieved

Warfarin 5 mg/d can be started on day 1 of therapy; there is no benefit from higher starting doses

Platelet count should be monitored at least every 3 d during initial heparin therapy

Therapeutic APTT should correspond to plasma heparin level of 0.2–0.4 IU/mL

Heparin is usually continued for 5–7 d

Heparin can be stopped after 4–5 d of warfarin therapy when INR is in 2.0–3.0 range

Important drug interactions with warfarin

Drugs that decrease warfarin requirement	Drugs that increase warfarin requirement
Phenylbutazone	Barbiturates
Metronidazole	Carbamazepine
Trimethoprim-sulfamethoxazole	Rifampin
Amiodarone	Penicillin
Second- and third-generation cephalosporins	Griseofulvin
Clofibrate	Cholestyramine
Erythromycin	
Anabolic steroids	
Thyroxine	

Complications of anticoagulation

	Complication	Management
Heparin	Bleeding	Stop heparin infusion. For severe bleeding, the anticoagulant effect of heparin can be reversed with intravenous protamine sulfate 1 mg/100 units of heparin bolus or 0.5 mg for the number of units given by constant infusion over the past hour; provide supportive care including transfusion and clot evacuation from closed body cavities as needed.

Complications of anticoagulation

	Complication	Management
Heparin	Heparin-induced thrombocytopenia and thrombosis	Carefully monitor platelet count during therapy. Stop-heparin for platelet counts <75,000. Replace heparin with direct inhibitors of thrombin-like desirudin if necessary. These agents do not cause heparin-induced thrombocytopenia. Avoid platelet transfusion because of the risk for thrombosis.

Complications of anticoagulation

	Complication	Management
Heparin	Heparin-induced osteoporosis (therapy >1 mo)	LMWHs may have lower propensity to cause osteoporosis as compared with unfractionated heparin; consider LMWH if prolonged heparin therapy is necessary.

Complications of anticoagulation

	Complication	Management
Warfarin	Bleeding	Stop therapy. Administer vitamin K and fresh-frozen plasma for severe bleeding; provide supportive care including transfusion and clot evacuation from closed body cavities as needed
	Skin necrosis (rare)	Supportive care.
	Teratogenicity	Do not use in pregnancy or in patients planning to become pregnant.

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Improved resolution at 2-4 h after onset of therapy			
Angiography	+	-	-
Pulmonary artery pressure	+	-	-
	+	-	-
Echocardiography			
Resolution at 24 h			
Lung scan	+	-	-
Angiography	+	-	-

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Echocardiography	+	-	-
Pulmonary artery pressure	+	-	-
Resolution at 1 wk and 30 d (lung scan)	-	+	-
Rate of confirmed recurrent pulmonary embolism	-	+	-

Risks and benefits of thrombolytics vs heparin therapy for pulmonary embolism

	Thrombolytic therapy	No difference	Heparin
Hospital mortality	-	+	-
Late mortality	-	+	-
Less severe bleeding	-	-	+
Less intracranial hemorrhage	-	-	+
Lower cost	-	-	+

Approved thrombolytics for pulmonary embolism

Approved thrombolytics for pulmonary embolism

Streptokinase

250,000 IU as loading dose over 30 min, followed by 100,000 U/h for 24 h

Urokinase

4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h for 12-24 h

Recombinant tissue-plasminogen activator

100 mg as a continuous peripheral intravenous infusion administered over 2 h

Indications and contraindications for thrombolytic therapy in pulmonary embolism

Indications

Hemodynamic instability

Hypoxia on 100% oxygen

Right ventricular dysfunction by echocardiography

Contraindications

Relative

Recent surgery within last 10 d

Previous arterial punctures within 10 d

Neurosurgery within 6 mo

Bleeding disorder (thrombocytopenia, renal failure, liver failure)

Ophthalmologic surgery within 6 wk

Hypertension >200 mm Hg systolic or 110 mm Hg diastolic

Placement of central venous catheter within 48 h

Hypertensive retinopathy with hemorrhages or exudates

Intracerebral aneurysm or malignancy

Cardiopulmonary resuscitation within 2 wk

Cerebrovascular disease

Major internal bleeding within the last 6 mo

Pregnancy and the 1st 10 d postpartum

Infectious endocarditis

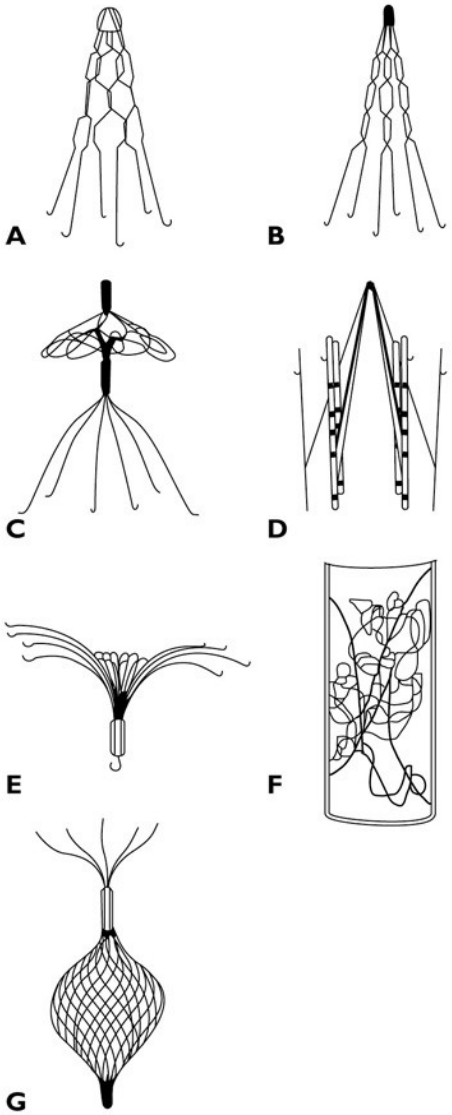
Severe trauma within 2 mo

Pericarditis

Absolute

Active internal bleeding

Various inferior vena caval filters



Indications for inferior vena caval (IVC) filters

Indications for inferior vena caval filter placement

- Anticoagulation contraindicated (eg, patients with multiple trauma, active bleeding)
- Failure of antithrombotic therapy
- Complications from anticoagulant therapy preclude further use
- Prophylaxis against embolism from preexisting deep vein thrombosis in patients with poor cardiopulmonary reserve
- Prophylaxis against embolism in patients at high risk to develop deep vein thrombosis
- Patients with recurrent pulmonary embolism undergoing thromboendarterectomy

Conclusions

- PE is common and under-recognized serious medical problem
- Early diagnosis and treatment is essential for good outcome
- High index of suspicion is needed in high risk patients