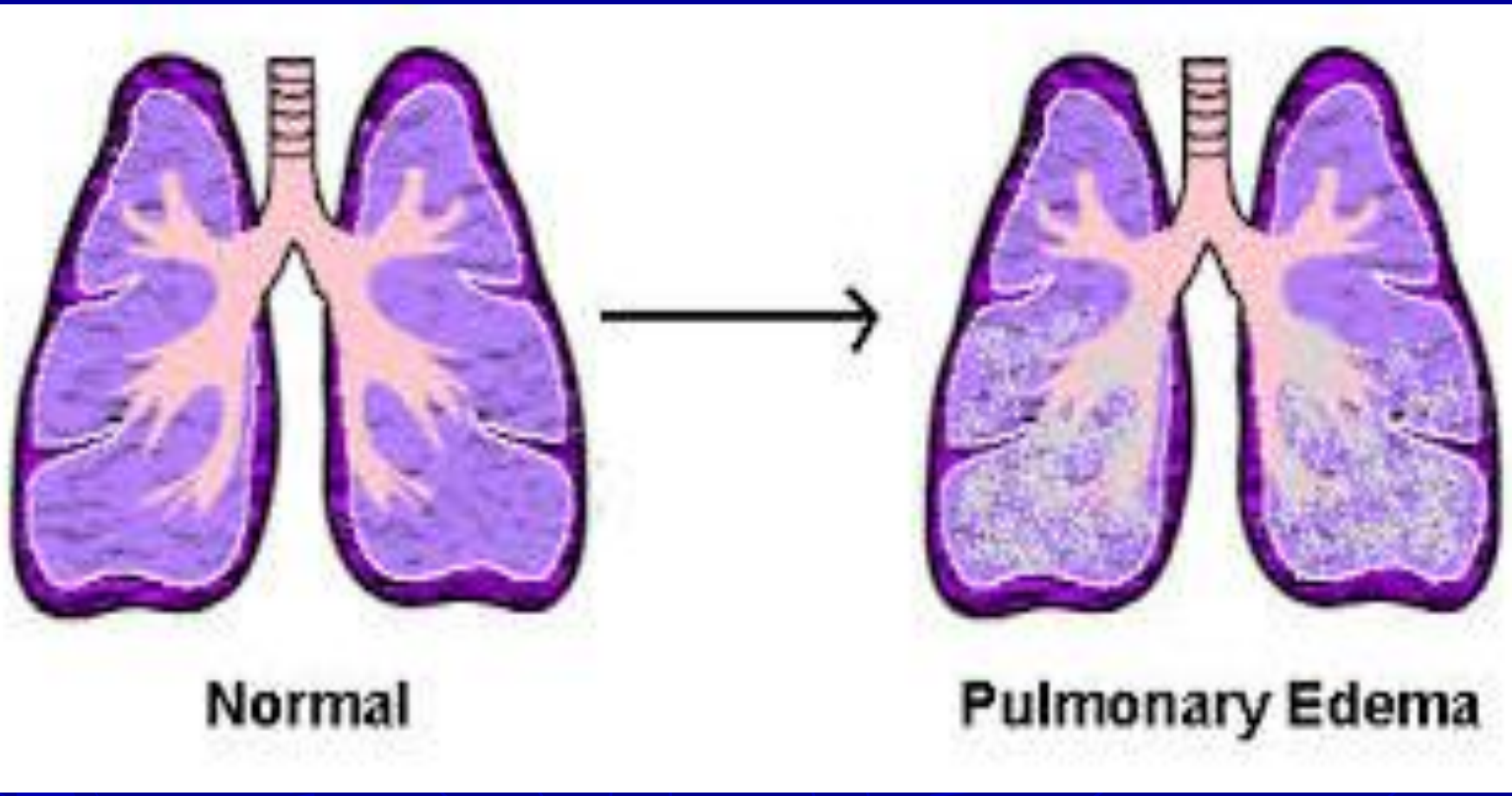
MEDICAL IMAGING FOR EMEGENCY CASES

Oxana Malîga, associated professor, Department of Radiology and Imaging

- Acute pulmonary edema
- Acute respiratory distress syndrome
- Pulmonary embolism
- Radiological contrast agents – adverse reactions



Acute pulmonary edema

excessive accumulation of fluid of vasogenic origin in the pulmonary interstitium or in the alveolar cavity

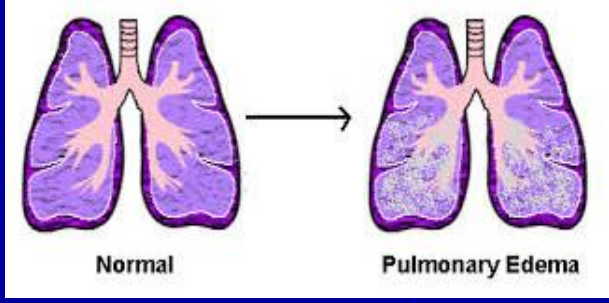
Pulmonary edema

- Increased hydrostatic pressure in the capillaries of the small circulation (congestive factors)
- Increased permeability of vascular wall (membrane factors)
- Increased osmotic pressure in interstitial fluid (osmotic factor)
- Decreased oncotic pressure in the blood plasma (lymphogenic factors)

Pulmonary edema

Can occure in:

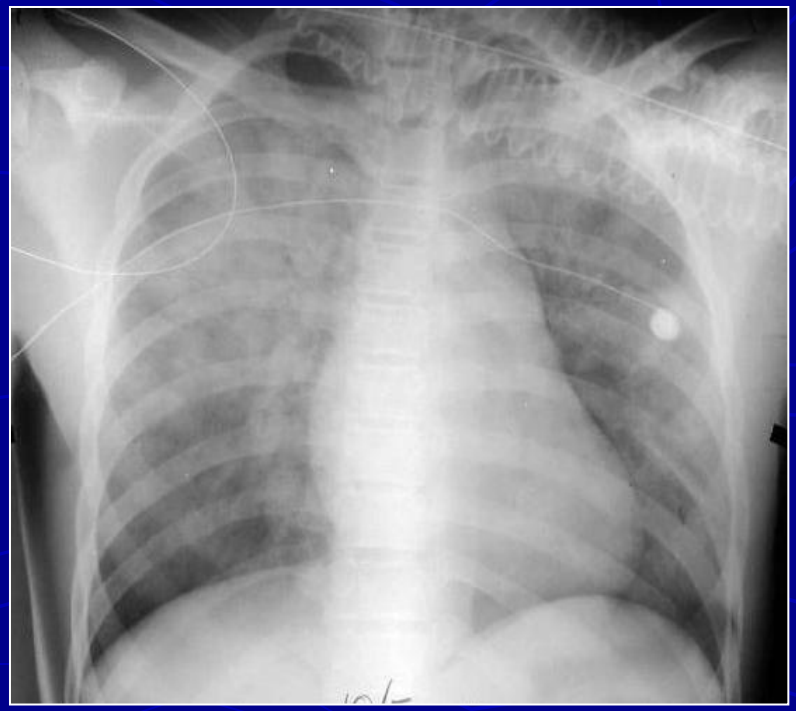
- Systemic arterial hypertension
- Mitral and aortic valve diseases
- Myocardial infarction
- Myocarditis
- Cardiomyopathies
- Cardiac arrhythmias



- Acute pulmonary edema is not a disease but a simptom of a heart disease, the most often, a chronic one.
- Acute pulmonary edema may have a favorable evolution under the treatment, but it's a sign of gravity of the basic pathology and has a bad prediction. Once a patient with a chronic heart disease developped a pulmonaty edema, the probability of death during the next 6 months is about 30%

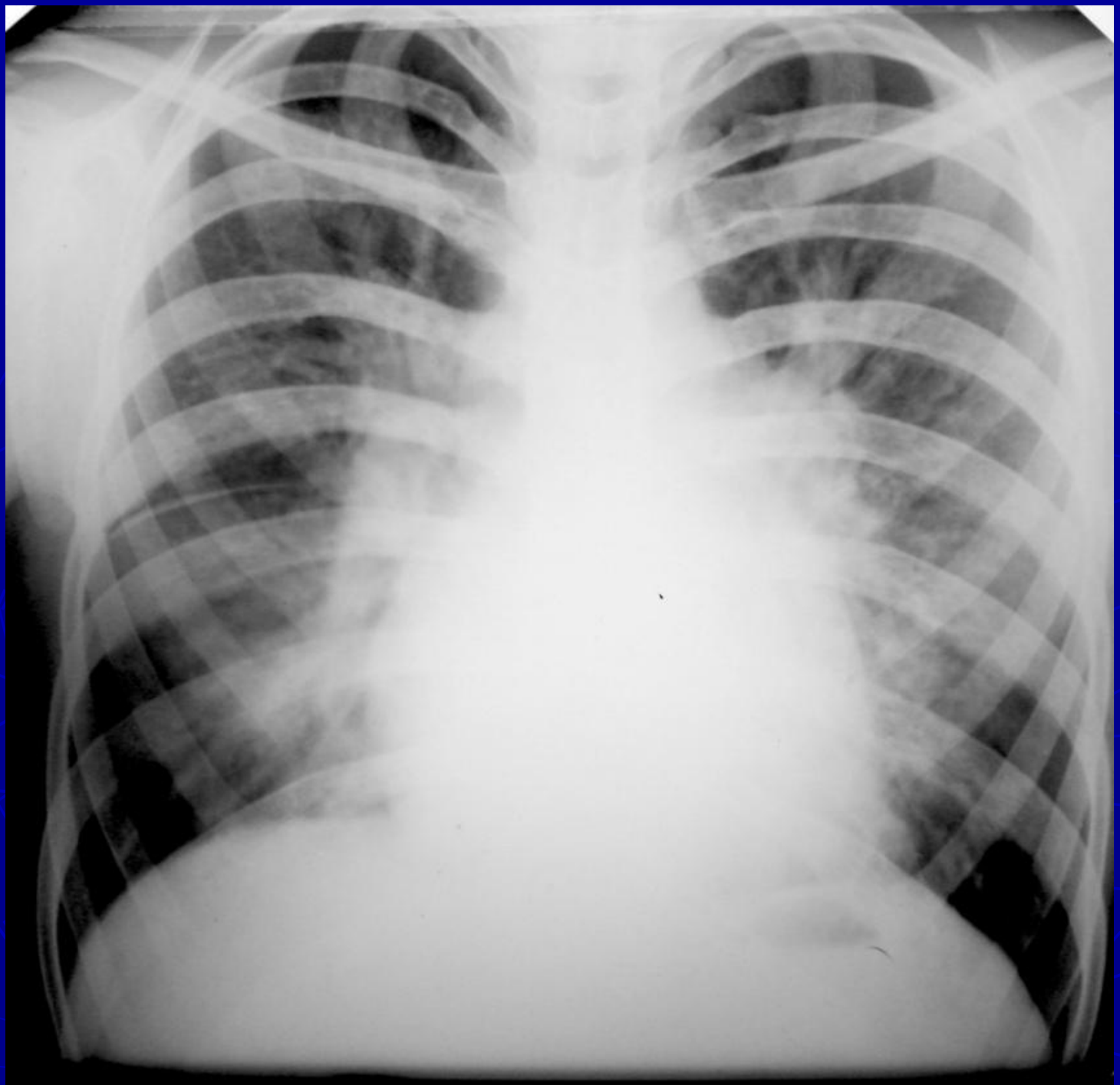
Main imaging methods

- Simple chest radiograph
- Computed tomography of the chest



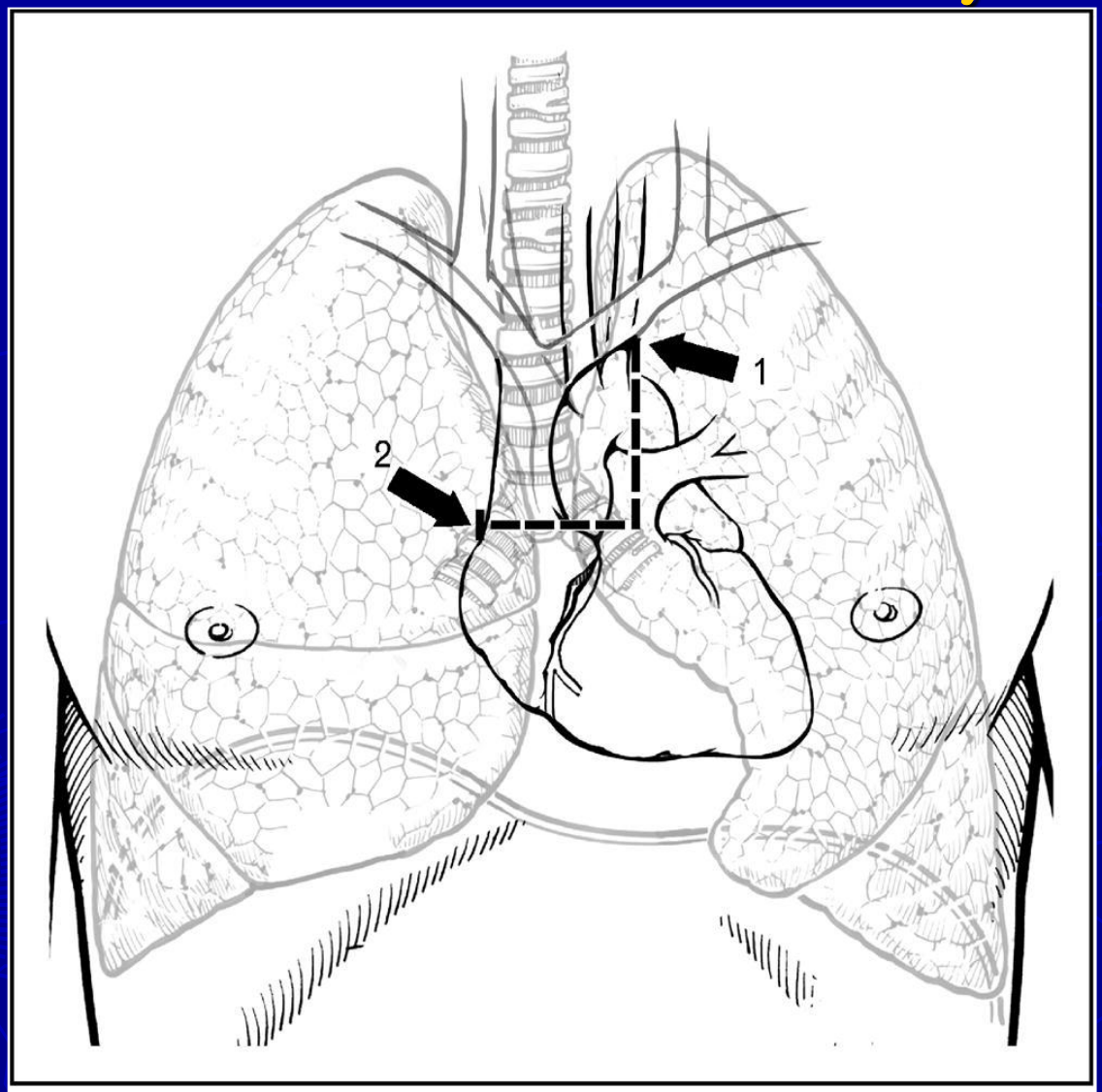
Radiographic signs of pulmonary edema:

- **Stage of pre-edema: venous pulmonary hypertension. Middle capillary blood pressure > 15 mm Hg**
  - Enhanced pulmonary vascular pattern
  - Pulmonary vascular pattern is more marked in the superior pulmonary fields (redistribution)
  - Changes of heart configuration:
    - Dilatation of the heart shadow to the left
    - Dilatation of the shadow of vascular pedicle (fasciculus) (superior mediastinum) comparing to the previous one in the same patient



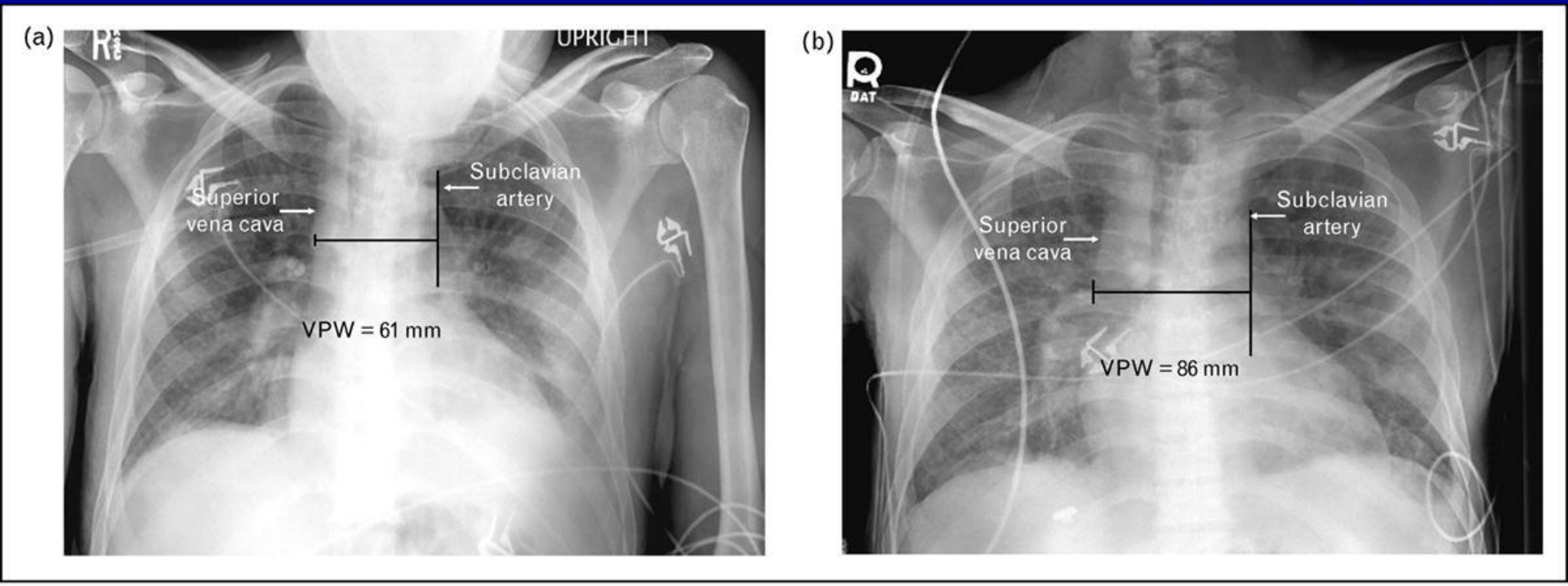
Vascular Pedicle Width in Pulmonary Edema

Criteria of pathology:
VPW ≥ 70 mm
CTR ≥ 0.55



The vascular pedicle width is measured by (1) dropping a perpendicular line from the point at which the left subclavian artery exits the aortic arch and (2) measuring across to the point at which the superior vena cava crosses the right mainstem bronchus.

Vascular pedicle width and fluid status in pulmonary edema

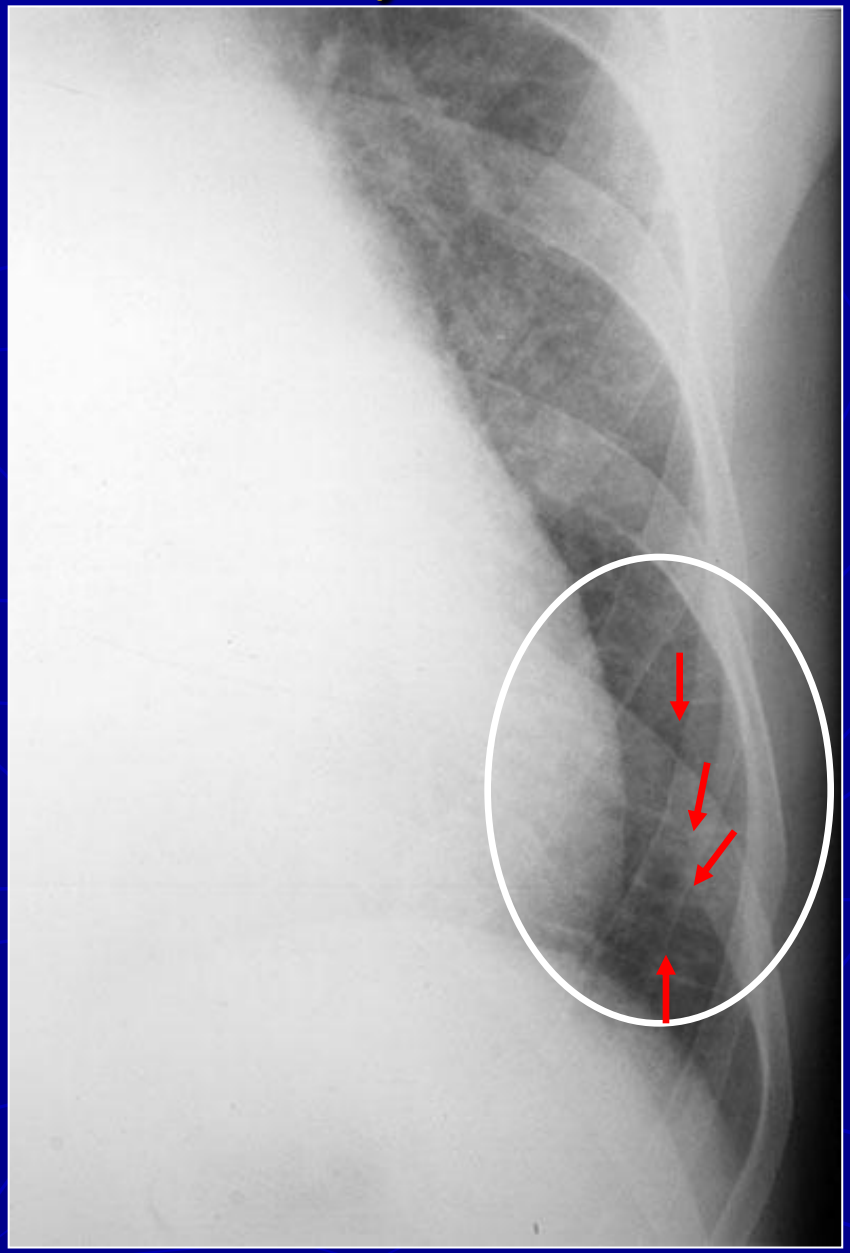


(a) A 53-year-old male with end-stage renal disease (anuria), hypertension, and dementia who was admitted for fever, altered mental status, hypoglycemia, and subsequent hypotension thought to be due to septic shock following hemodialysis with removal of 400 ml volume. This upright, portable, digital chest X-ray showed mild, diffuse interstitial lung disease and a moderate left pleural effusion. Both the cardiothoracic ratio (< 0.55) and vascular pedicle width (VPW; < 0.70 mm) were normal in appearance. His body weight at the time of the chest X-ray was 70.0 kg. (b) The same patient shown in (a) is shown here 12 h later after improvement of hypotension but with worsening oxygenation, requiring 100% non-rebreather. In the interim, he received 10 500 ml of net fluid intake and recorded a marked development of anasarca. His body weight at the time of this chest X-ray was 79 kg. On this semi-erect, portable, digital chest X-ray, the interstitial infiltrates were more prominent and both the cardiothoracic ratio (> 0.55) and the vascular pedicle width (86 mm) had increased markedly.

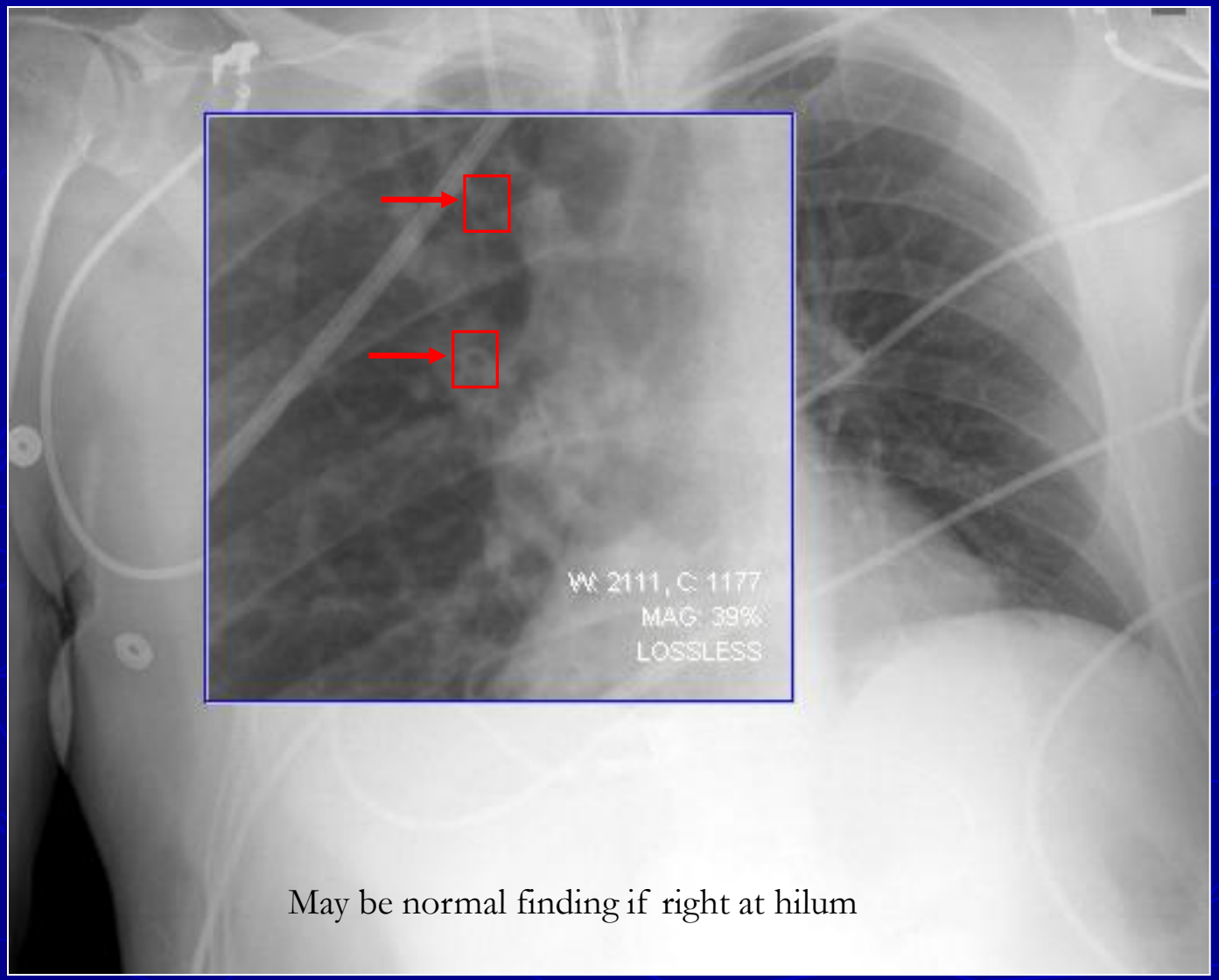
Radiographic signs of pulmonary edema:

- **Interstitial edema. Middle capillary blood pressure > 25 mm Hg**
  - Enhanced pulmonary vascular pattern
  - Deformation of pulmonary pattern, more evident in basal regions
  - Diminuation of pulmonary lucency
  - Peribronhial and perivascular cuffing ("sockets") with blurred contours
  - Dilatation of pulmonary hila
  - Kerley lines (thickening of the interlobular septa, 1-2 cm long, straight, 90º to pleura)
  - Thickening of fissures

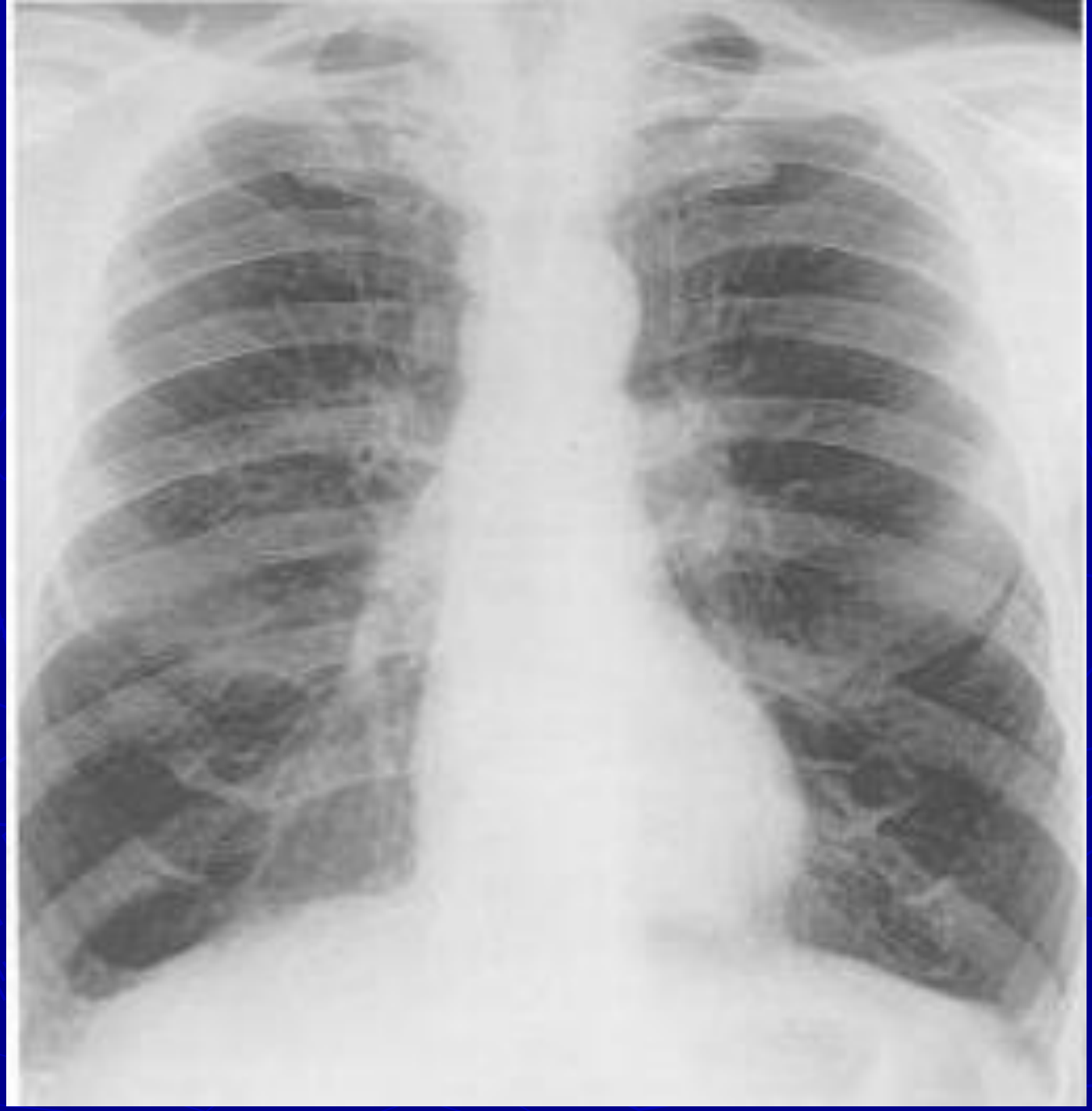
Kerley B lines



Peribronchial Cuffing



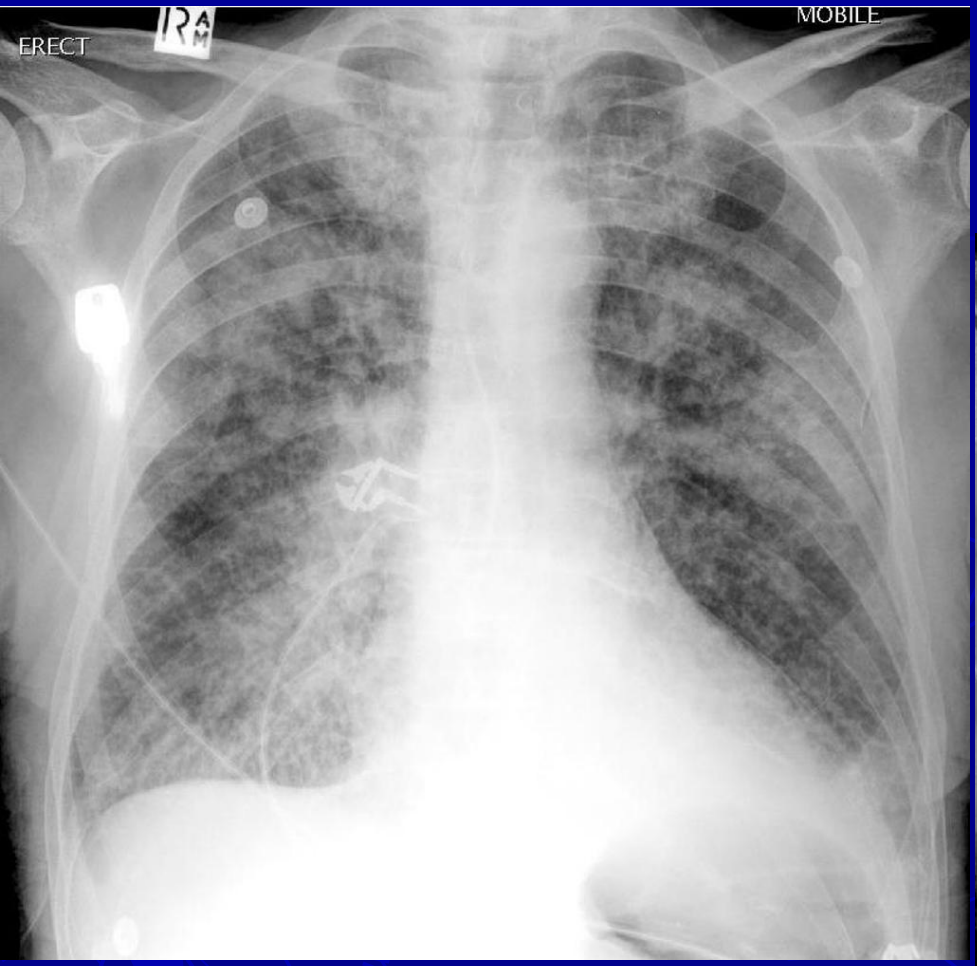
May be normal finding if right at hilum

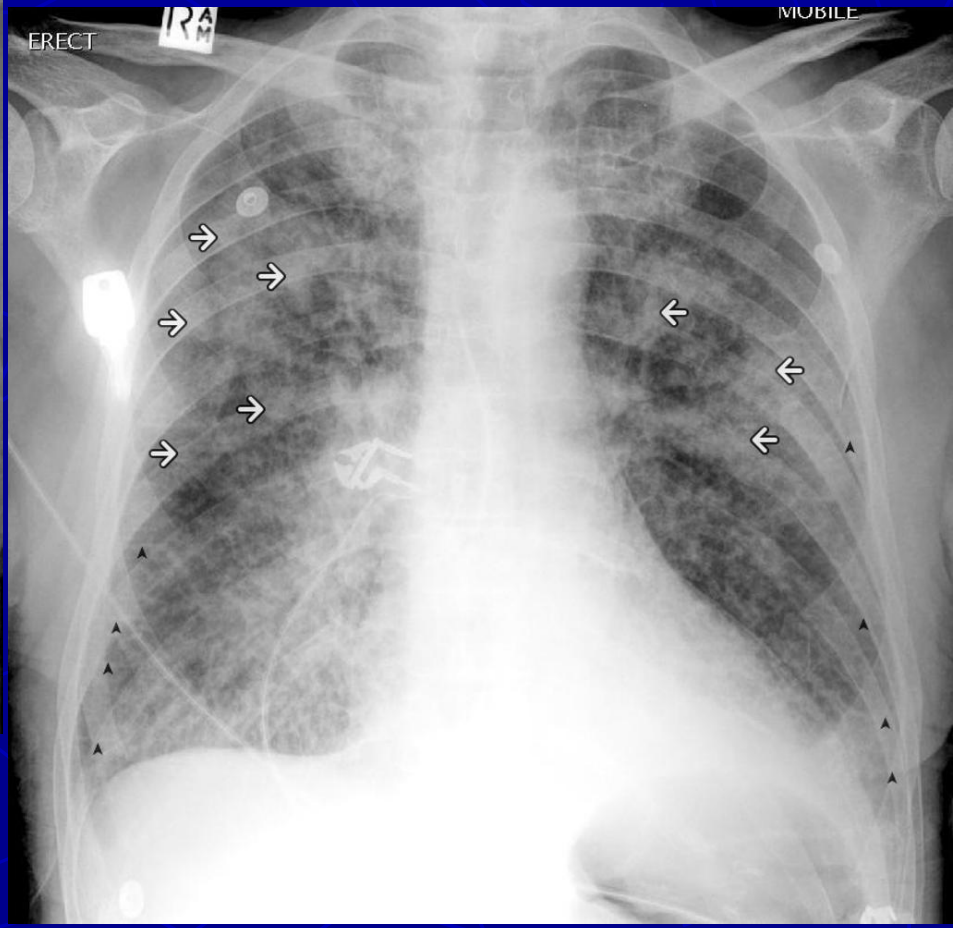


Radiographic signs of pulmonary edema:

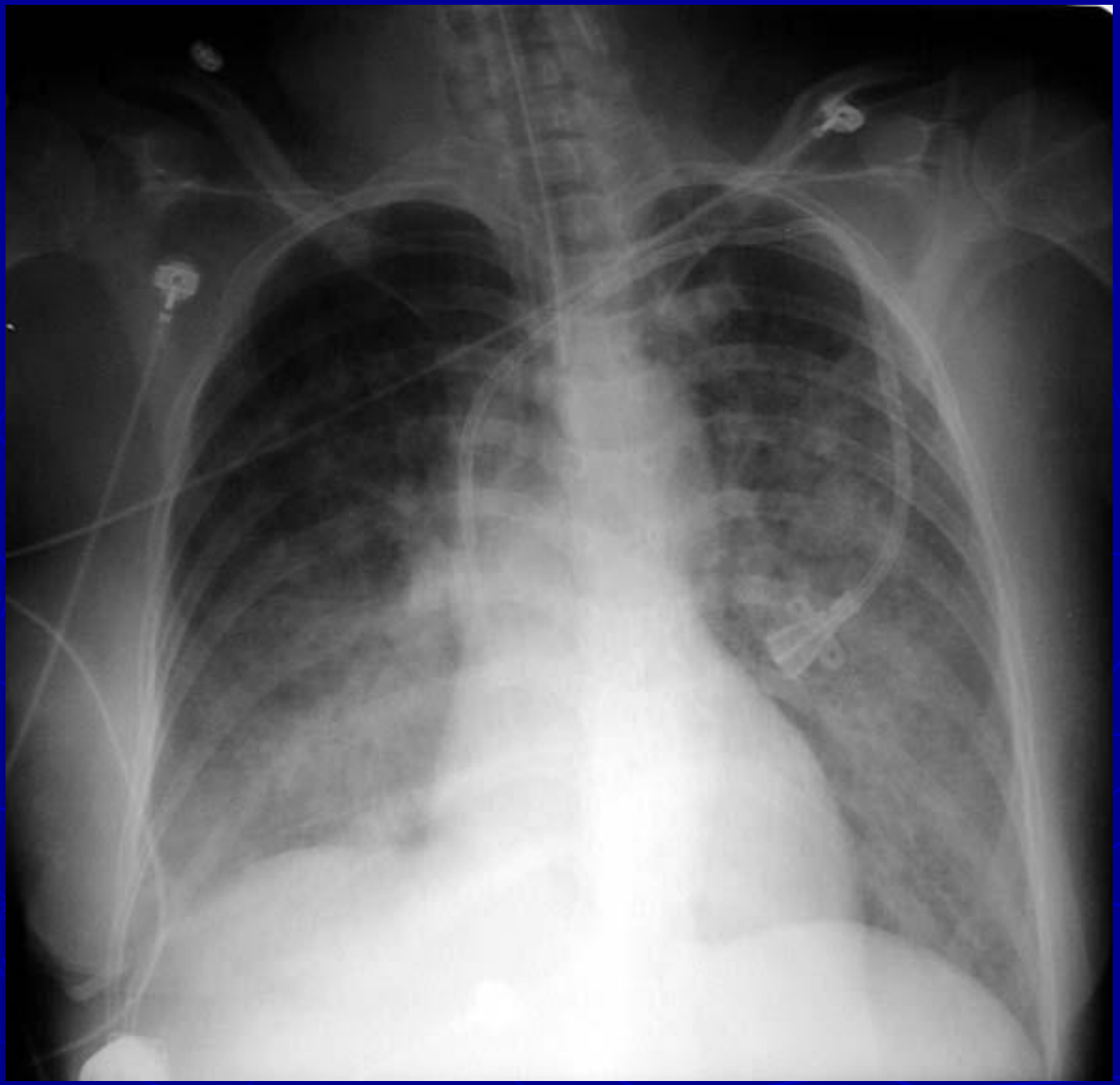
- **Alveolar edema. Middle capillary blood pressure > 35 mm Hg**
- **Opacities appear (infiltration)**
  - Different shape and location
  - Localization do not depend on lobes and segments
- The airless regions are those of posterior and lateral segments (II, VI, IX, X), **but in the PA and lateral chest radiograph opacity first appears in parahilar and inferior regions because of superposition**
- **Sign of "Bat's wings"**
  - Changement in the superior segments of the inferior lobes
  - more frequent in young patients
  - More frequent in acute evolution of pulmonary edema
- Kerley lines (thickening of the interlobular septa)

Acute pulmonary edema

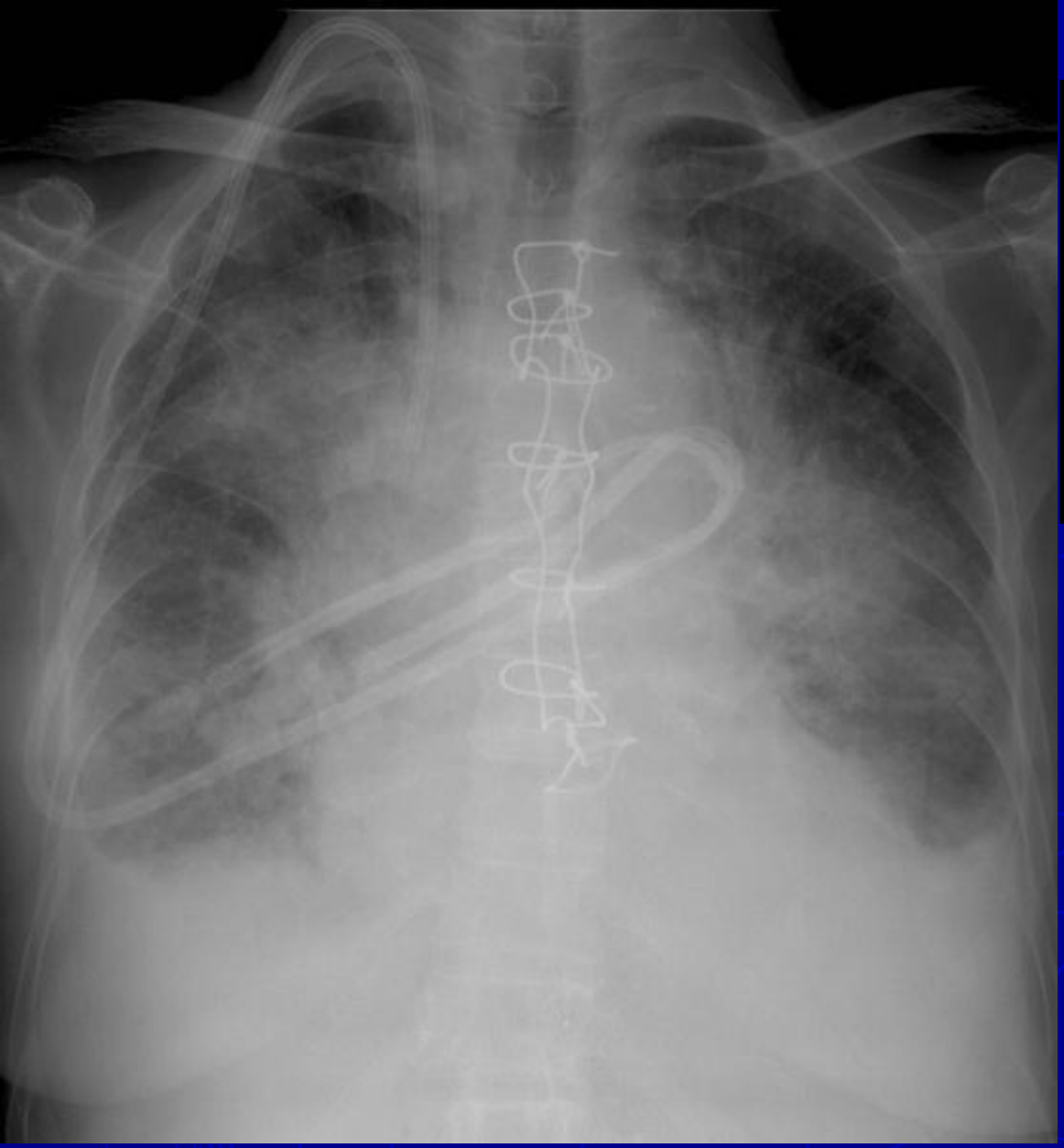


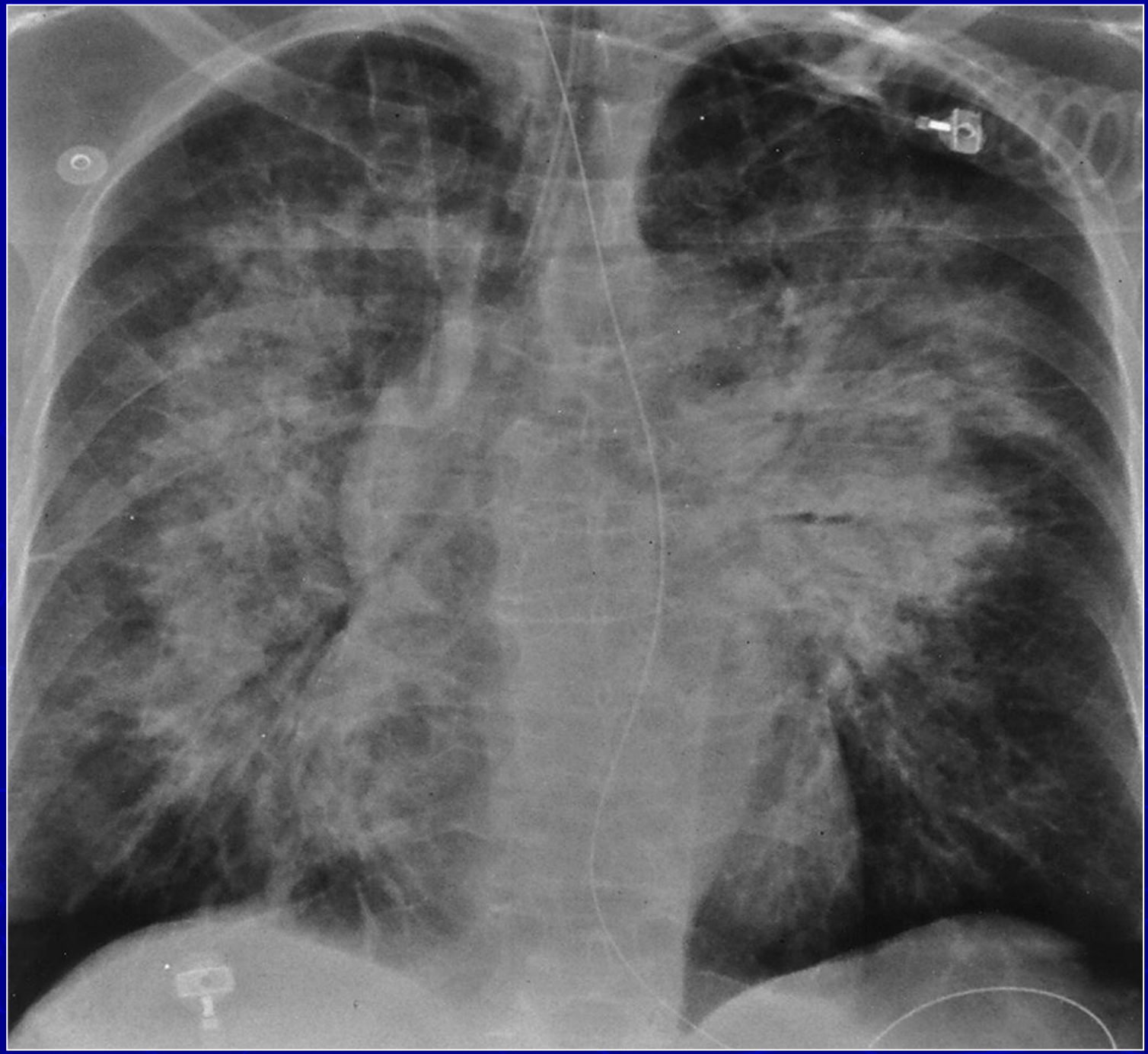


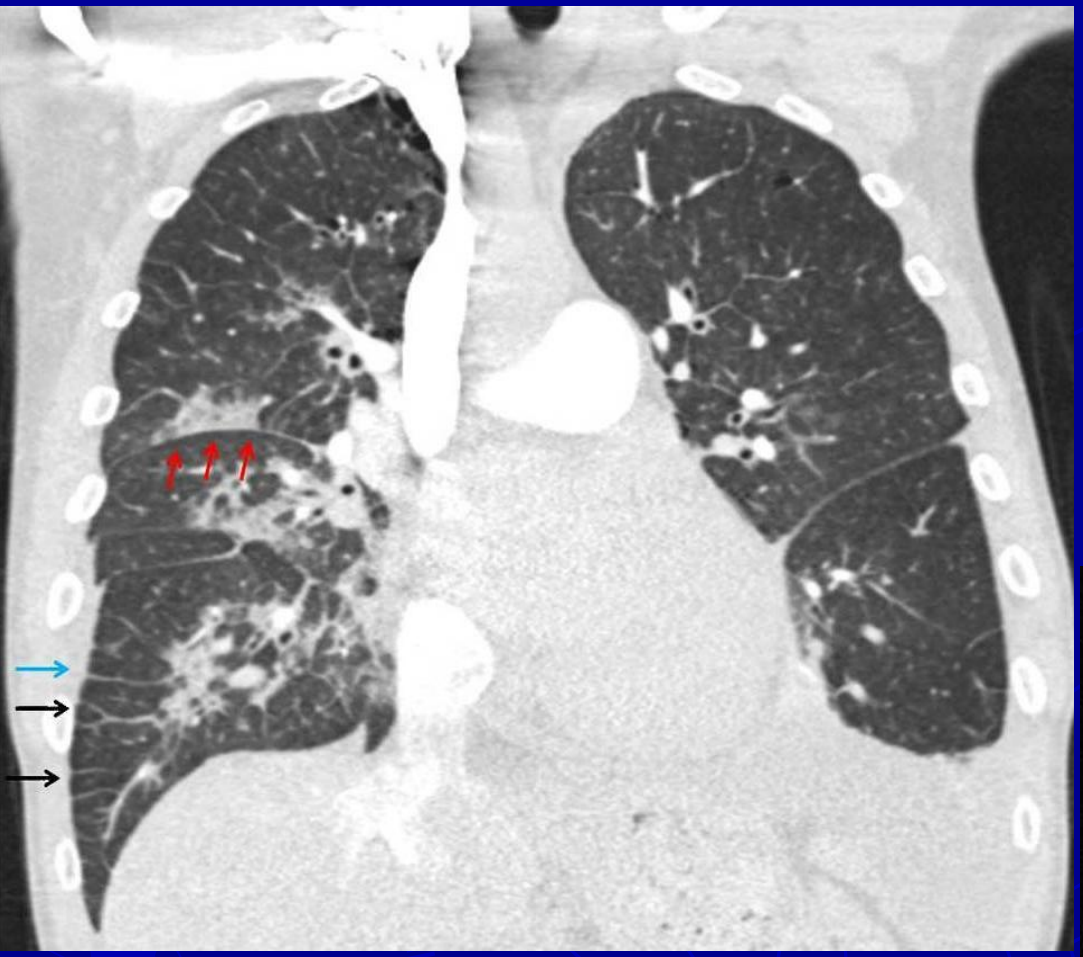
interstitial (black arrowheads) and alveolar (white arrows) pulmonary edema

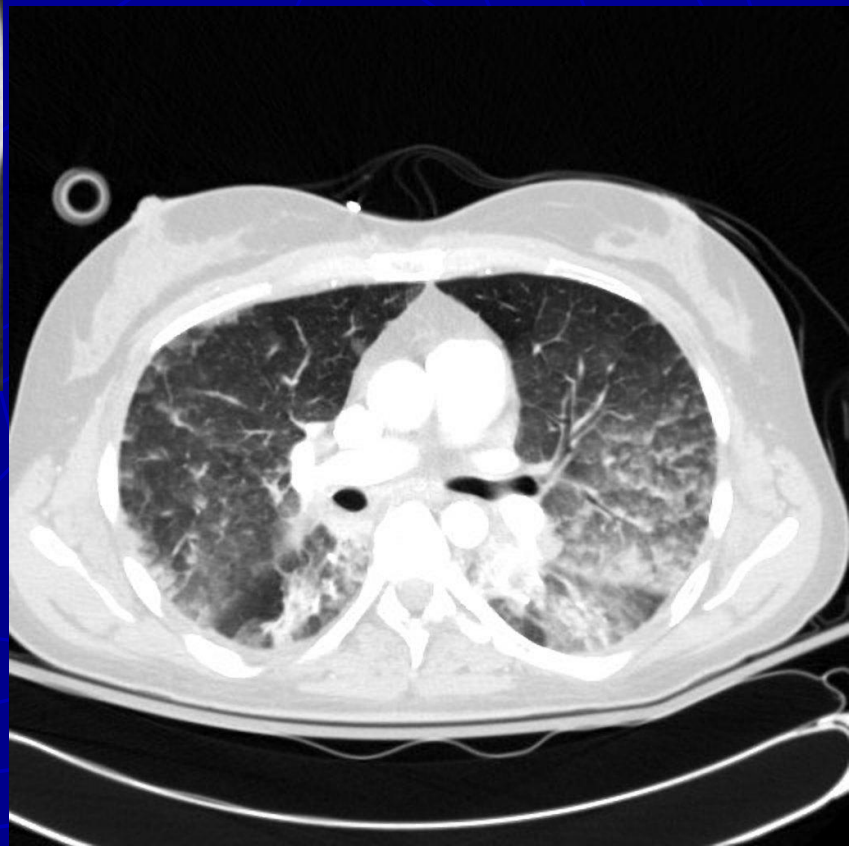


Bat-wings

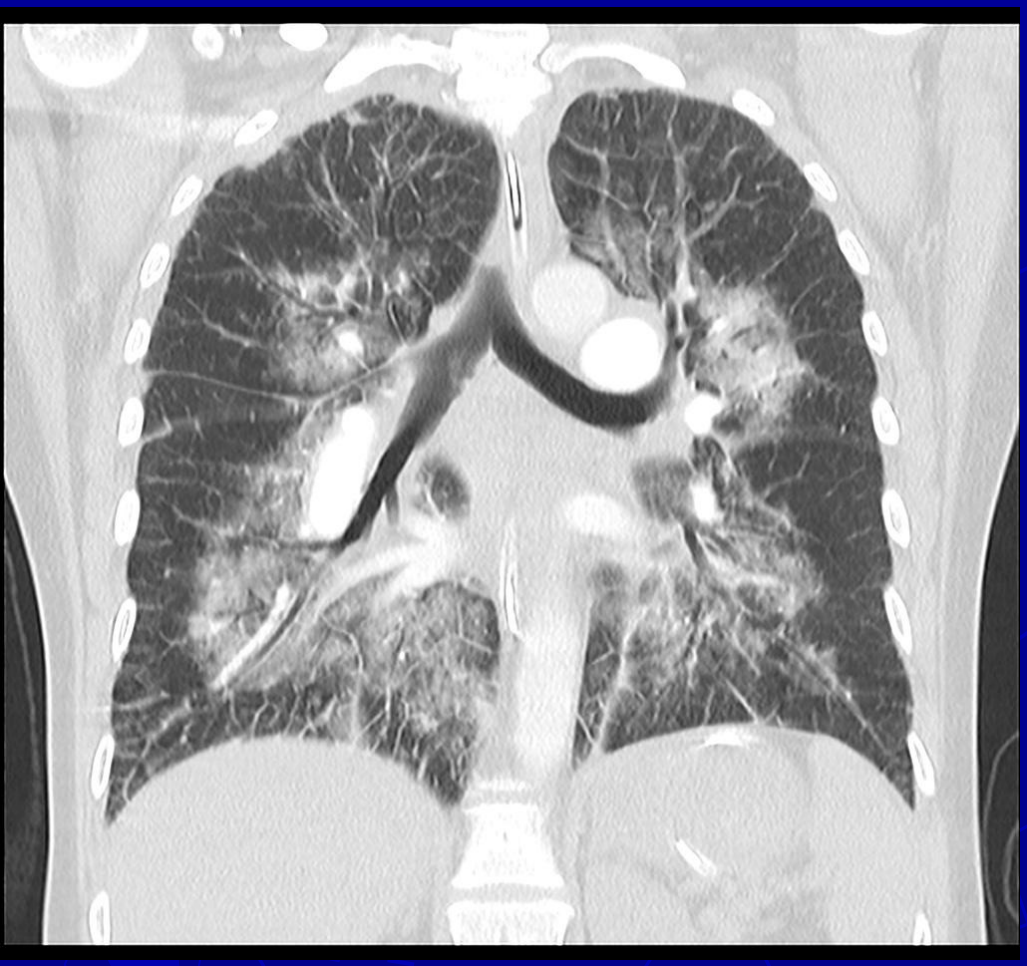


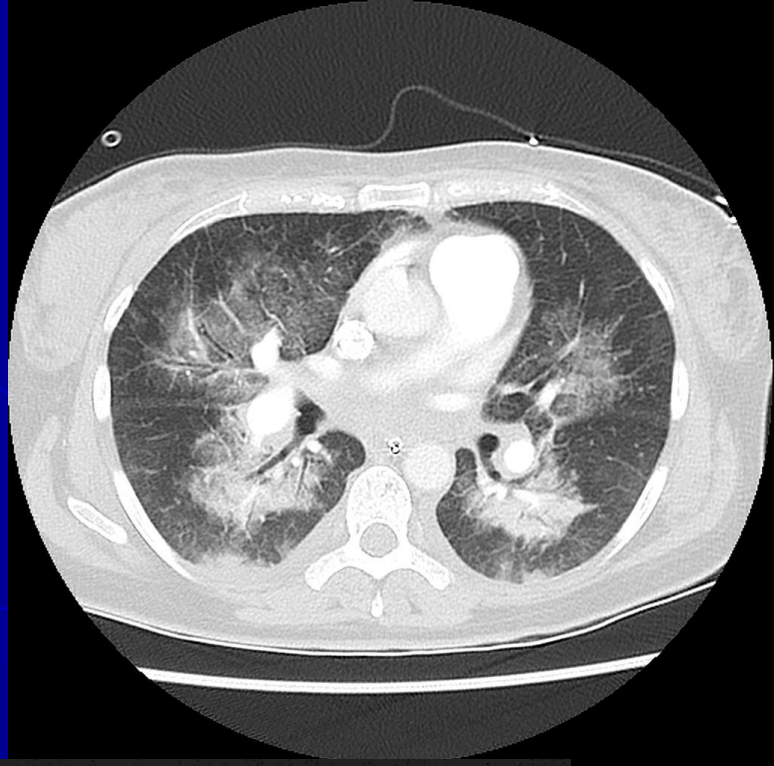


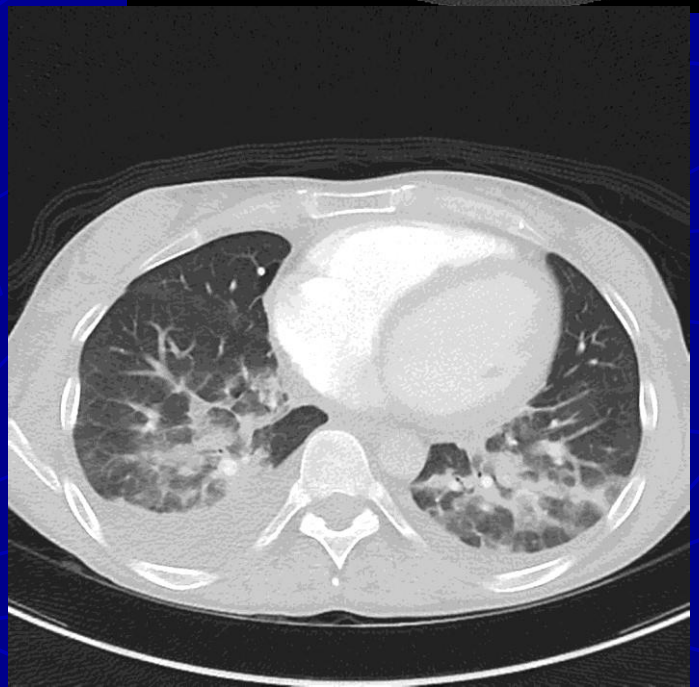




Pulmonary edema. CT





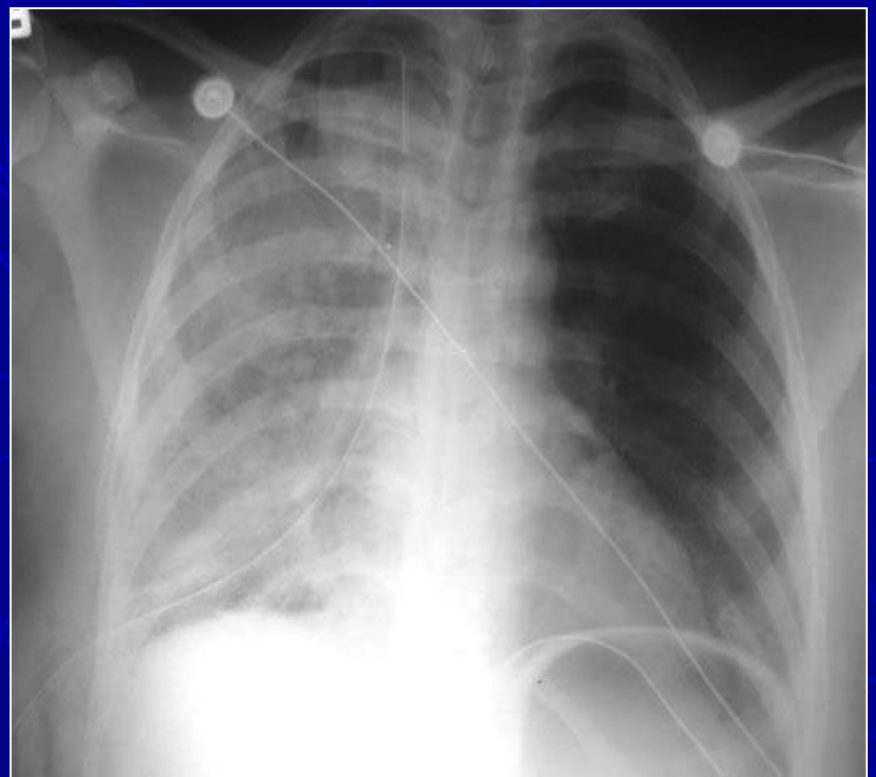


Left atrial pressure & radiographic signs

Normal	5-10 mm Hg
Cephalization	10-15 mm
Kerley B Lines	15-20
Pulmonary Interstitial Edema	20-25
Pulmonary Alveolar Edema	> 25

Asymmetric pulmonary edema

- Chronic lung disease altering vascular flow
- Acute mitral regurgitation - jet to right pulmonary vein - often edema of right upper lobe
- Patient position (gravitational)
- Re-expansion



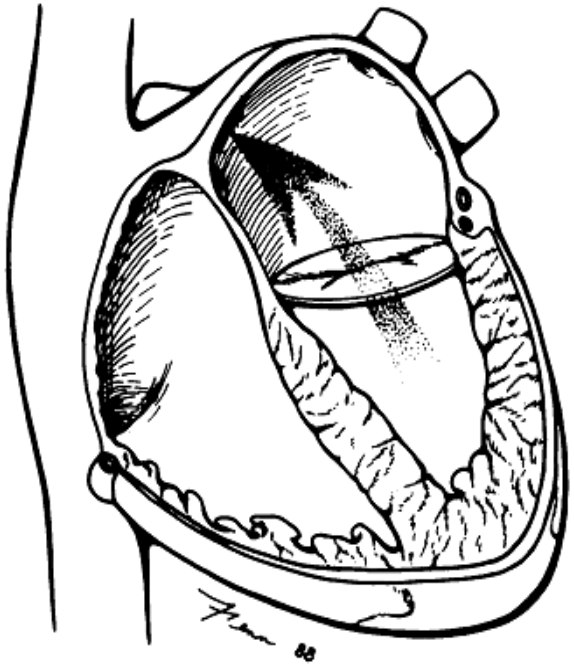
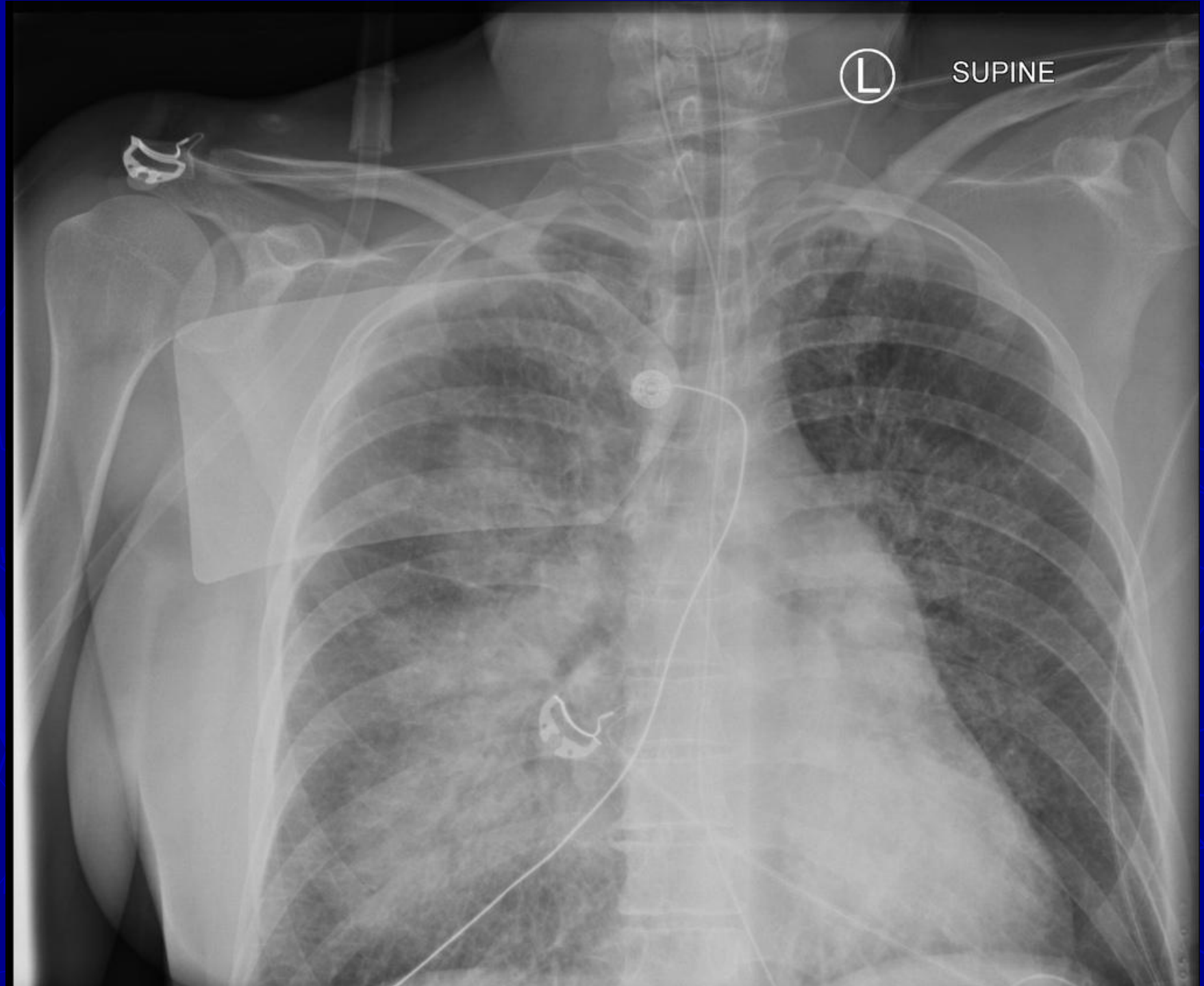


Figure 3. Drawing illustrates how mitral regurgitation might lead to right upper lobe edema. The vector of blood flow from the left ventricle to the left atrium may be directed at the right superior pulmonary vein, accentuating the forces for edema formation in the right upper lobe.


L
SUPINE

Acute respiratory distress synrdrome

ARDS is an acute diffuse, inflammatory lung injury, leading to increased pulmonary vascular permeability, increased lung weight, and loss of aerated lung tissue... [with] hypoxemia and bilateral radiographic opacities, associated with increased venous admixture, increased physiological dead space and decreased lung compliance.

Acute respiratory distress synrdrome

- Asbaugh, Bigelow & Petty described ARDS as: "A syndrome of acute respiratory failure in adults characterized by non-cardiogenic pulmonary edema manifested by severe hypoxemia caused by right to left shunting through collapsed or fluid-filled alveoli."
- The Berlin Definition: "An acute, diffuse, inflammatory lung injury that leads to increased pulmonary vascular permeability, increased lung weight, and a loss of aerated tissue."

(The ARDS Definition Task Force. Acute Respiratory Distress Syndrome: The Berlin Definition.*JAMA* 2012; May 21, 2012:Epub ahead of print.)

Acute respiratory distress syndrome

- Shock lung
- Pump lung
- Traumatic wet lung
- Post traumatic atelectasis
- Adult hyaline membrane disease
- Progressive respiratory distress
- Acute respiratory insufficiency syndrome
- Haemorrhagic atelectasis
- Hypoxic hyperventilation

- Postperfusion lung
- Oxygen toxicity lung
- Wet lung
- White lung
- Transplant lung
- Da Nang lung
- Diffuse alveolar injury
- Acute diffuse lung injury
- Noncardiogenic pulmonary edema.
- Progressive pulmonary consolidation

Acute respiratory distress syndrome

- Injury of pulmonary capillars
- Absence of signs of pulmonary venous hypertension
- Obviously known risk factors
- **Synchronous development of interstitial and alveolar pulmonary edema**

Risk factors

Direct

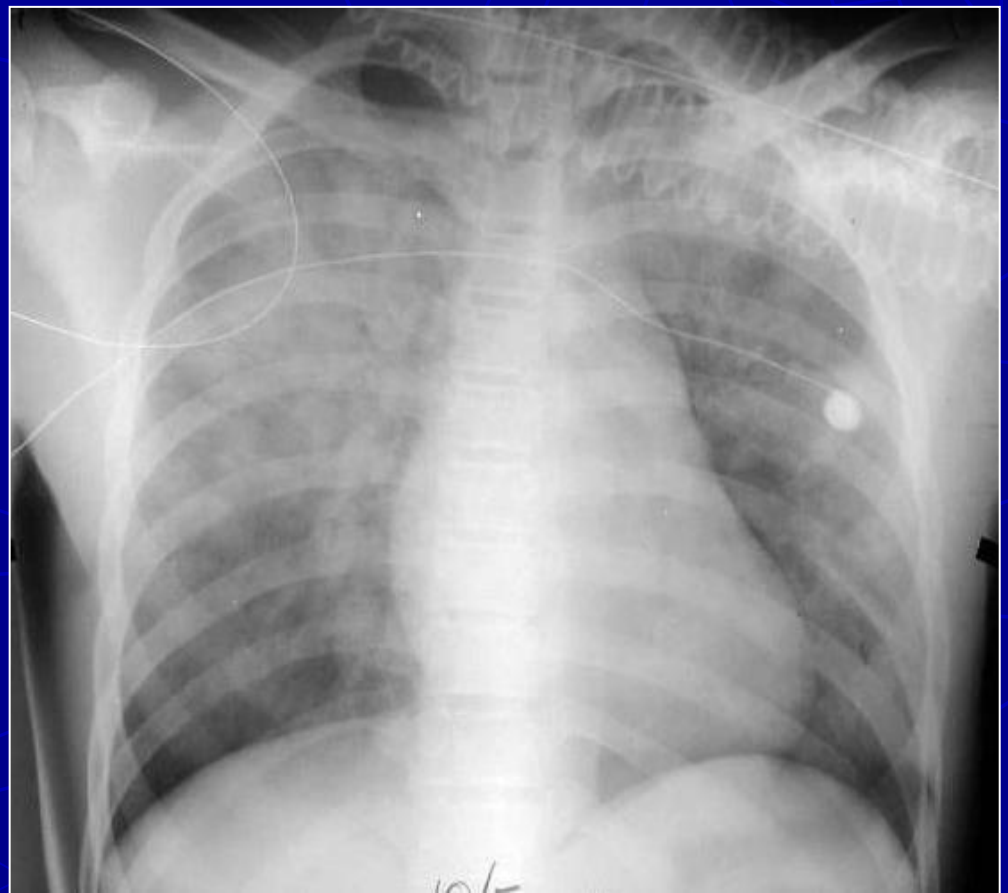
- pneumonia
- aspiration of gastric contents
- lung contusion
- fat embolism
- Amniotic fluid embolism
- near drowning
- inhalational injury
- reperfusion injury

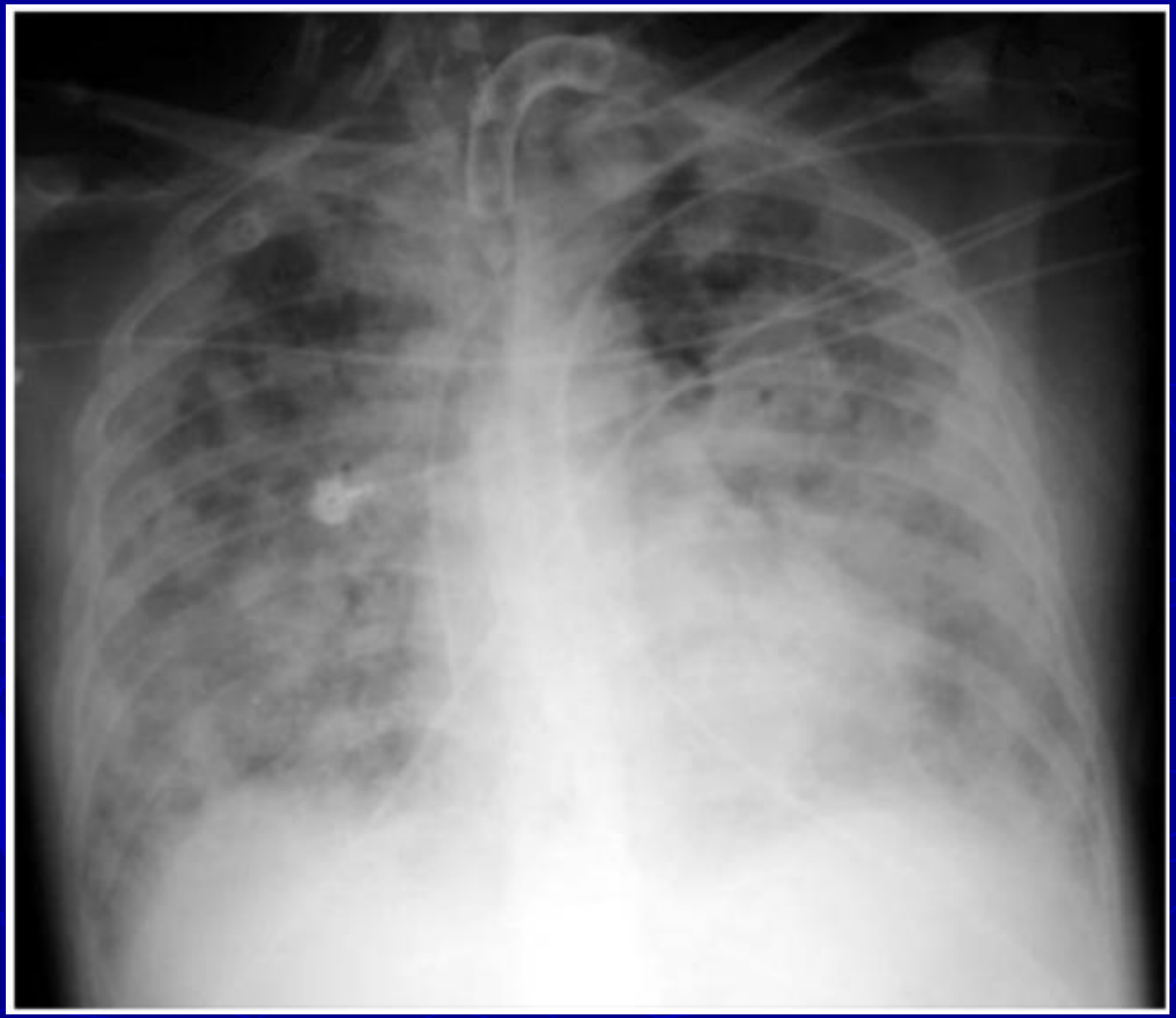
Indirect

- non-pulmonary sepsis
- multiple trauma
- massive transfusion
- pancreatitits
- Salicylate or narcotic overdose
- cardiopulmonary bypass

Chest radiograph

- Bilateral opacities involving at least 3 quadrants that are not fully explained by pleural effusions, atelectasis and nodules





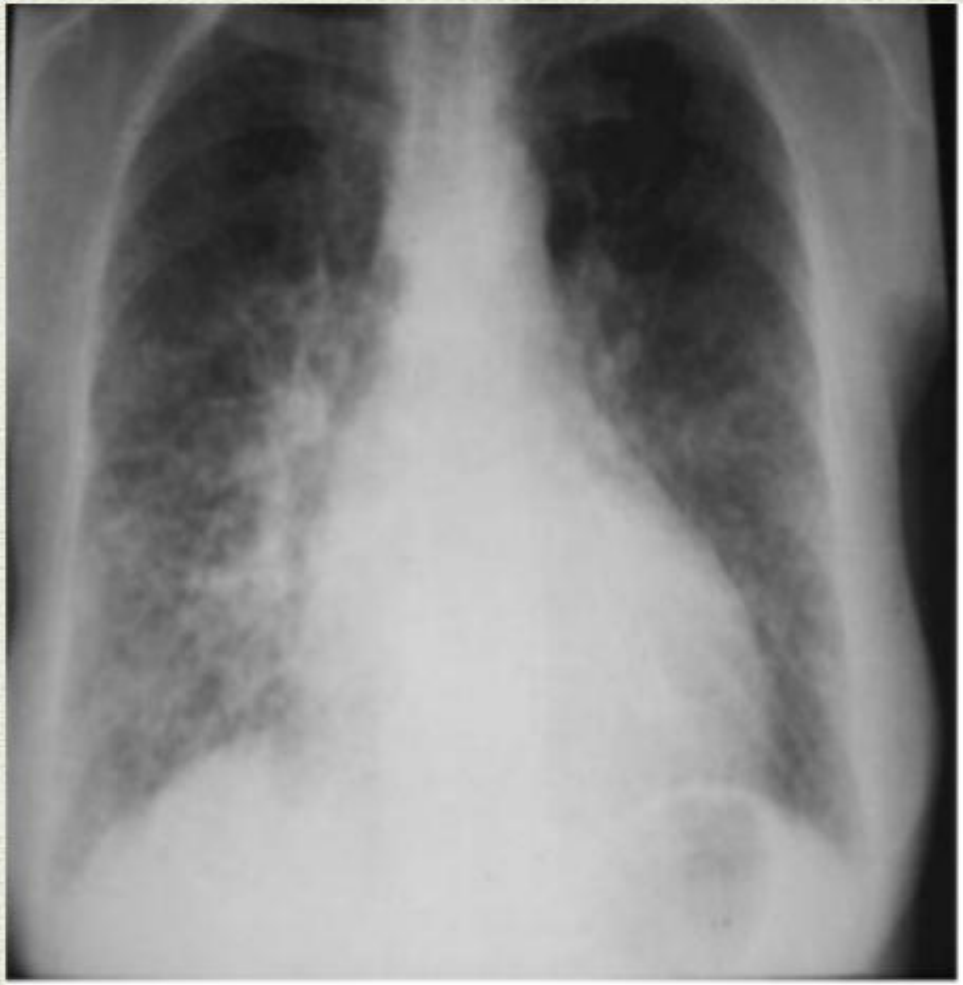
Pulmonary edema

- Patchy infiltrates in bases
- Effusions
- Kerley B lines
- Cardiomegaly
- Pulmonary vascular redistribution
- Excess fluid in alveoli

Acute respiratory distress syndrome

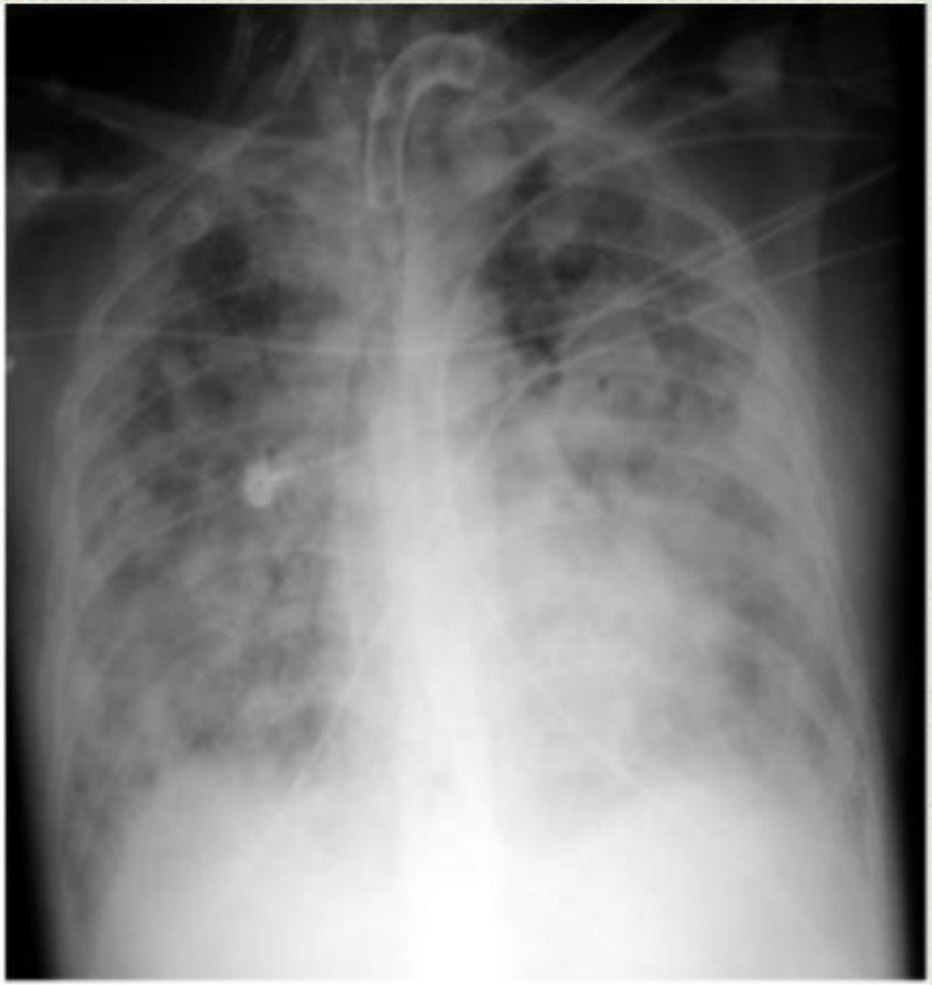
- Homogenous fluffy shadows
- No effusion
- No Kerley B lines
- No cardiomegaly
- No pulmonary vascular redistribution
- Protein, inflammatory cells, fluid

Cardiogenic



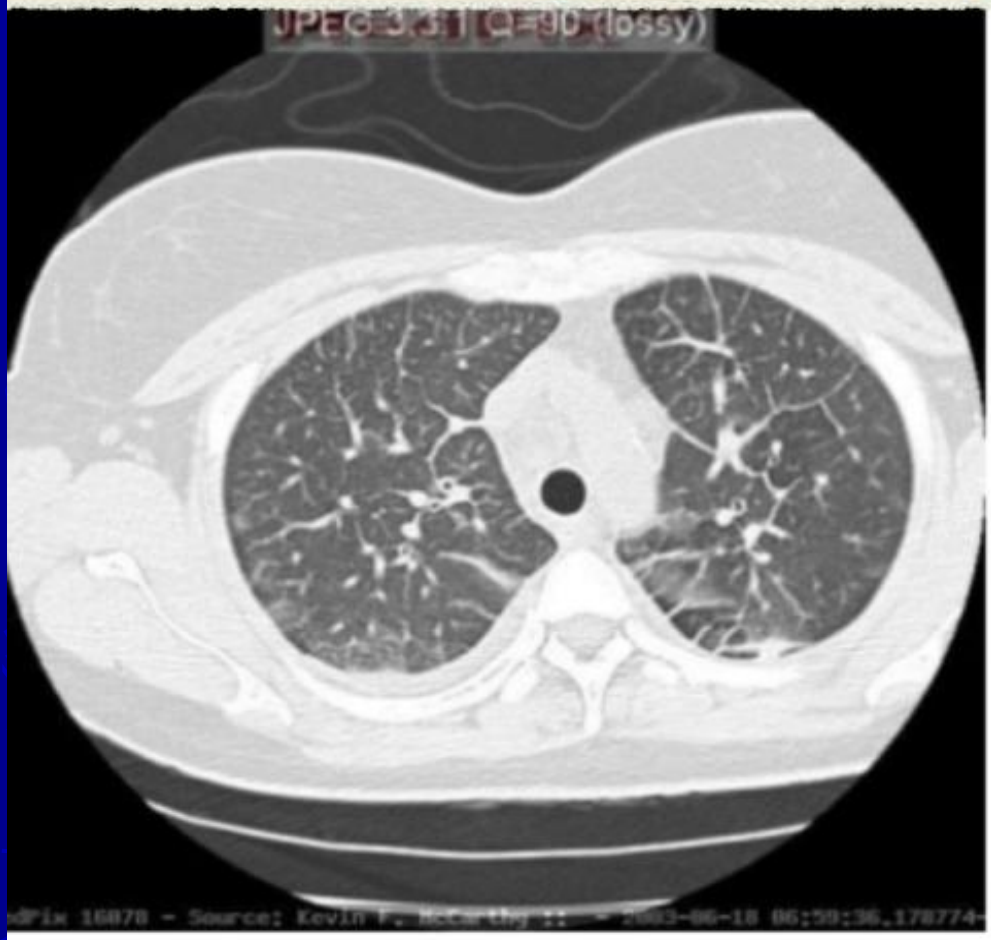
Bilateral infiltrates predominately in lung bases. Kerley B's. Cardiomegaly.

Non-Cardiogenic



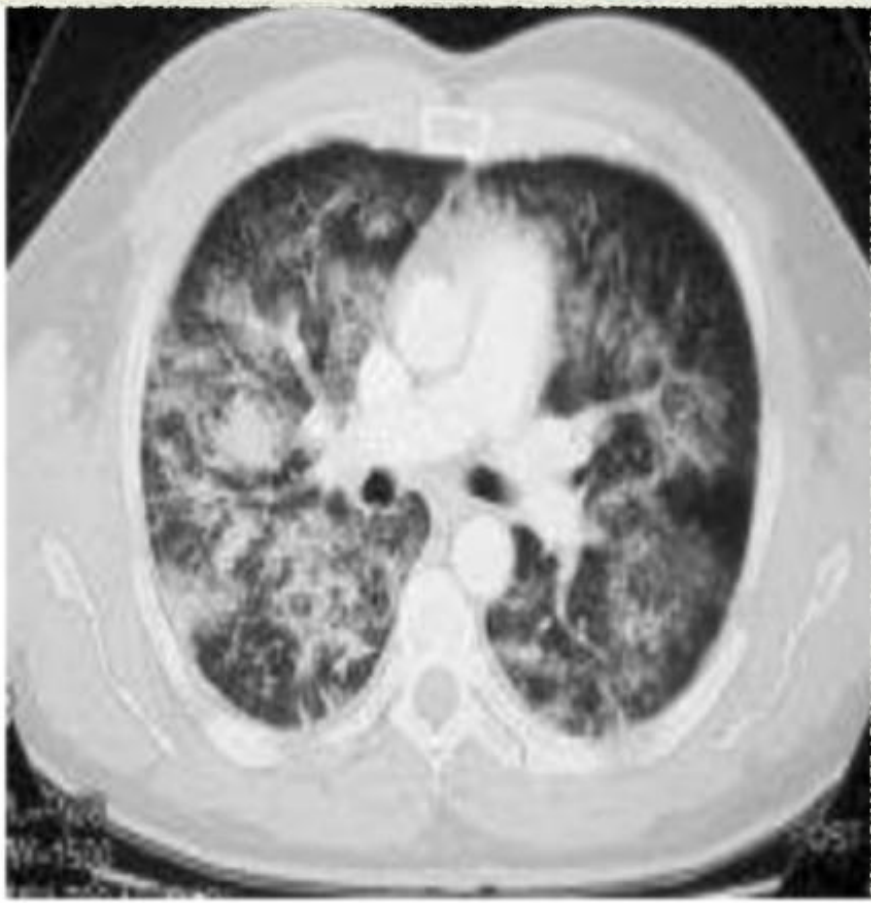
Diffuse Bilateral patchy infiltrates homogenously distributed throughout the lungs. No Kerley B's.

Cardiogenic



Septal thickening. More severe in lung bases.

Non-Cardiogenic



No septal thickening. Diffuse alveolar infiltrates. Atelectasis of dependent lobes usually seen

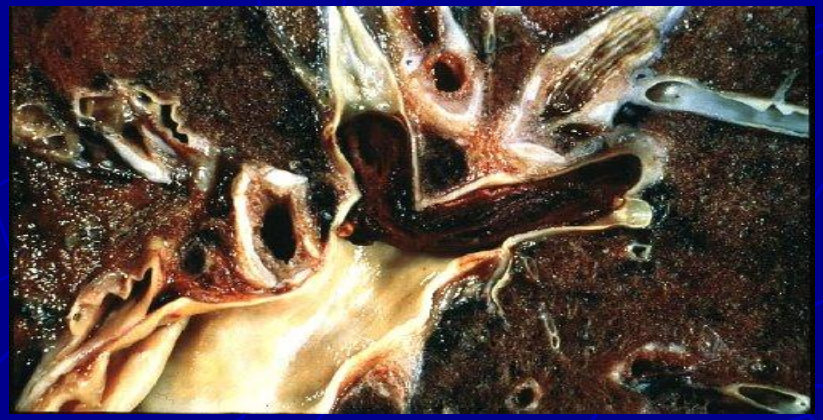
Pulmonary embolism

Disorder caused by total or partial obstruction of the pulmonary artery or of its branches

- **acute**
- **cronic**

Most common acute cardiovascular diseases:

- Myocardial infarction
- Stroke
- **Pulmonary embolism**



Pulmonary embolism results in thousands of deaths each year. It often goes undetected

Pulmonary thrombo-embolism

Source of emboli

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

Risk factors for deep venous thrombosis

- 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

Imaging modalities for acute pulmonary embolism.

- Chest radiograph.
- Ventilation-perfusion lung scan:
- Pulmonary angiography
- Computed tomography

Using Technetium-99m labeled macro aggregated albumin (MAA) for perfusion scans & Xenon-133 for ventilation scans.

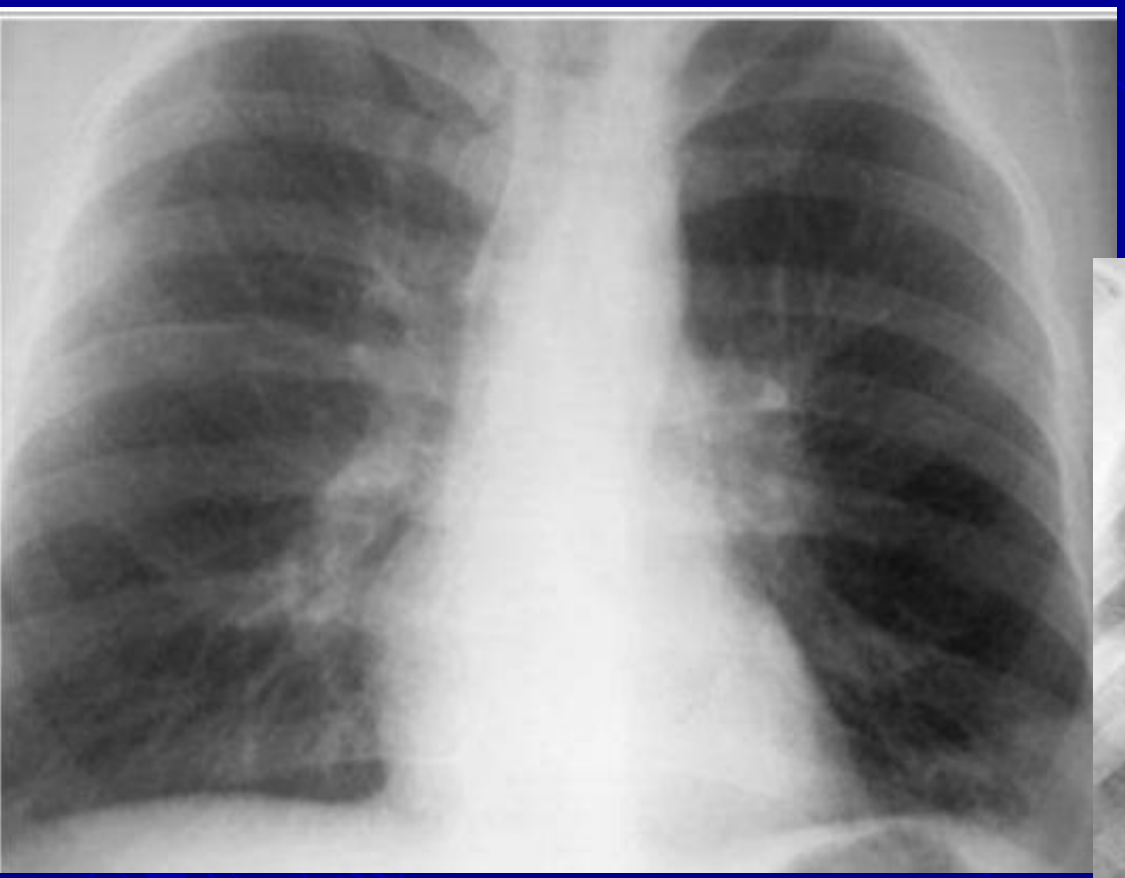
- MRI

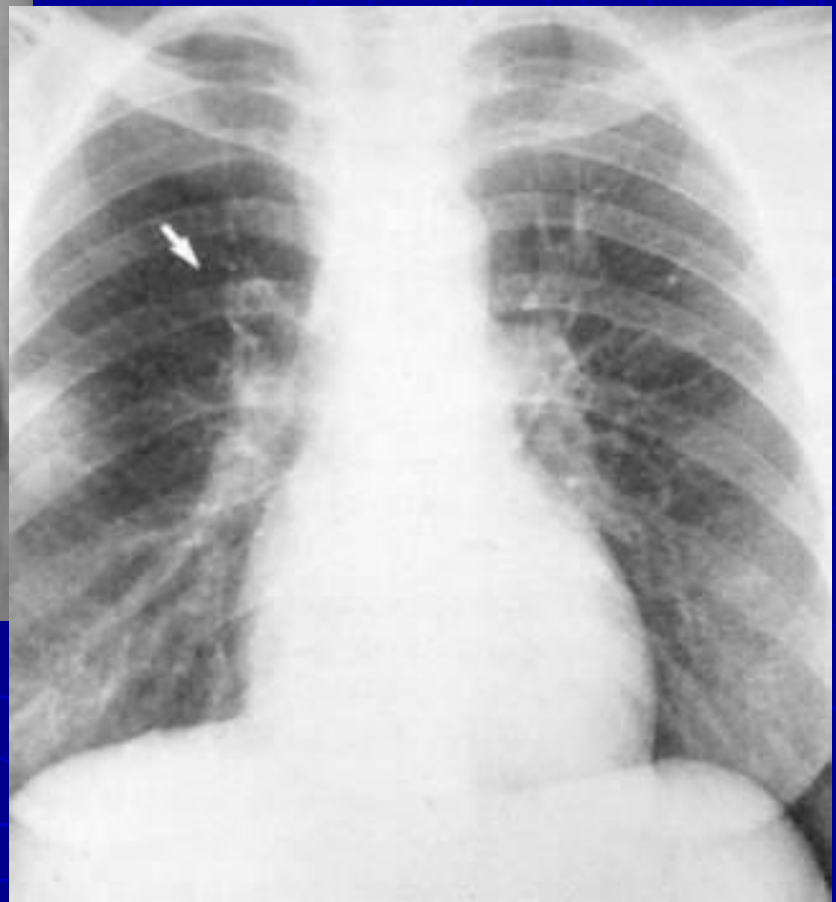
Simple standard chest radiograph

- Focal oligemia (Westermark's sign)
- Hampton's hump
- Sometimes nearly normal radiograph

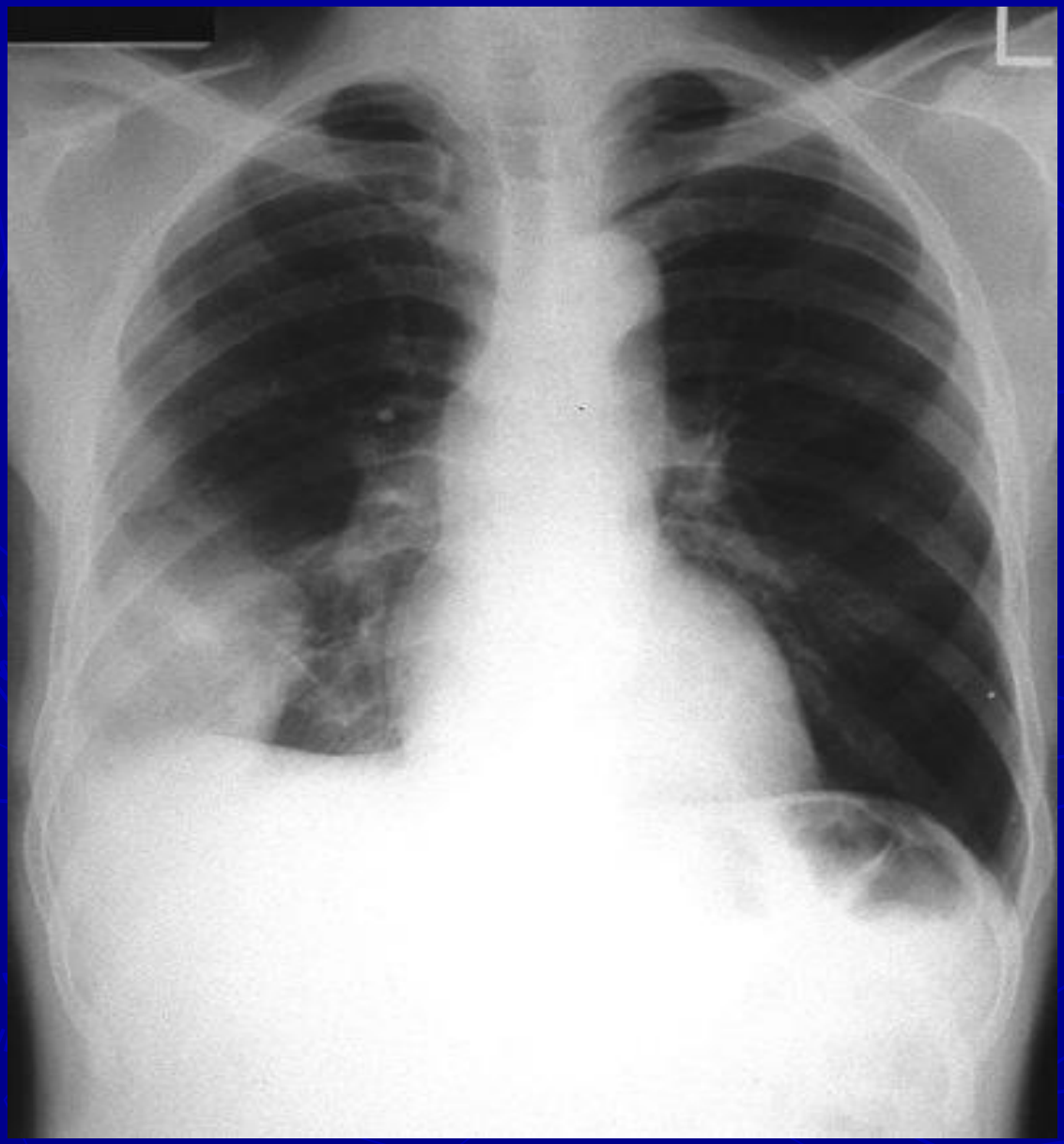
Westermark's sign

Dilatation of pulmonary vessels proximal to embolism along with collapse of distal vessels, often with a sharp cut off.



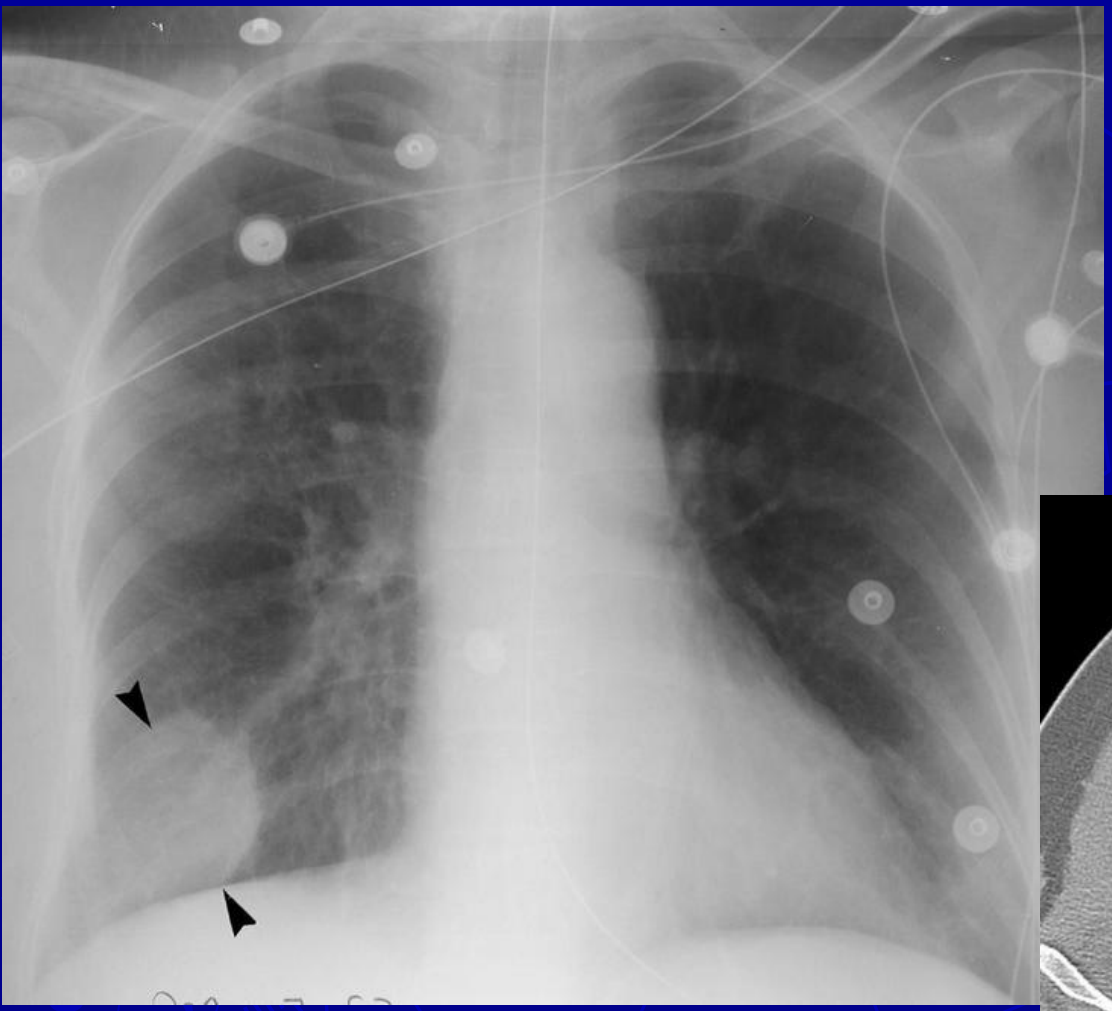


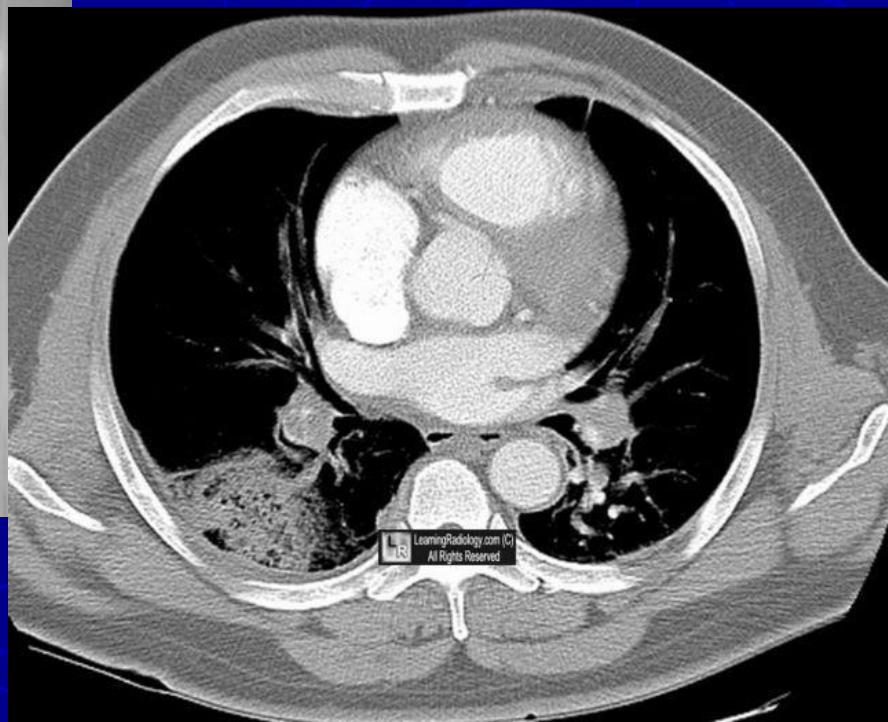
Pulmonary embolism. "Hampton's hump" sign.



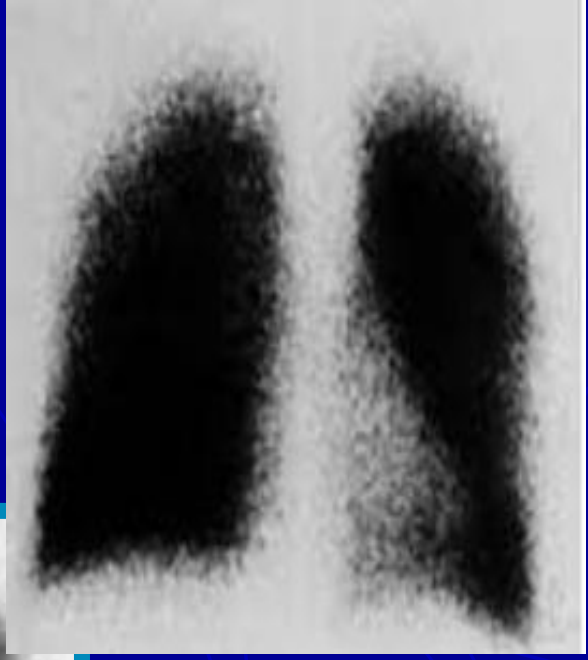
A pleural-based opacity in the lower lobe with the convexity directed towards the hilum signifies pulmonary infarction. This patient had low-grade fever, hemoptysis, and chest pain.

Hampton's hump



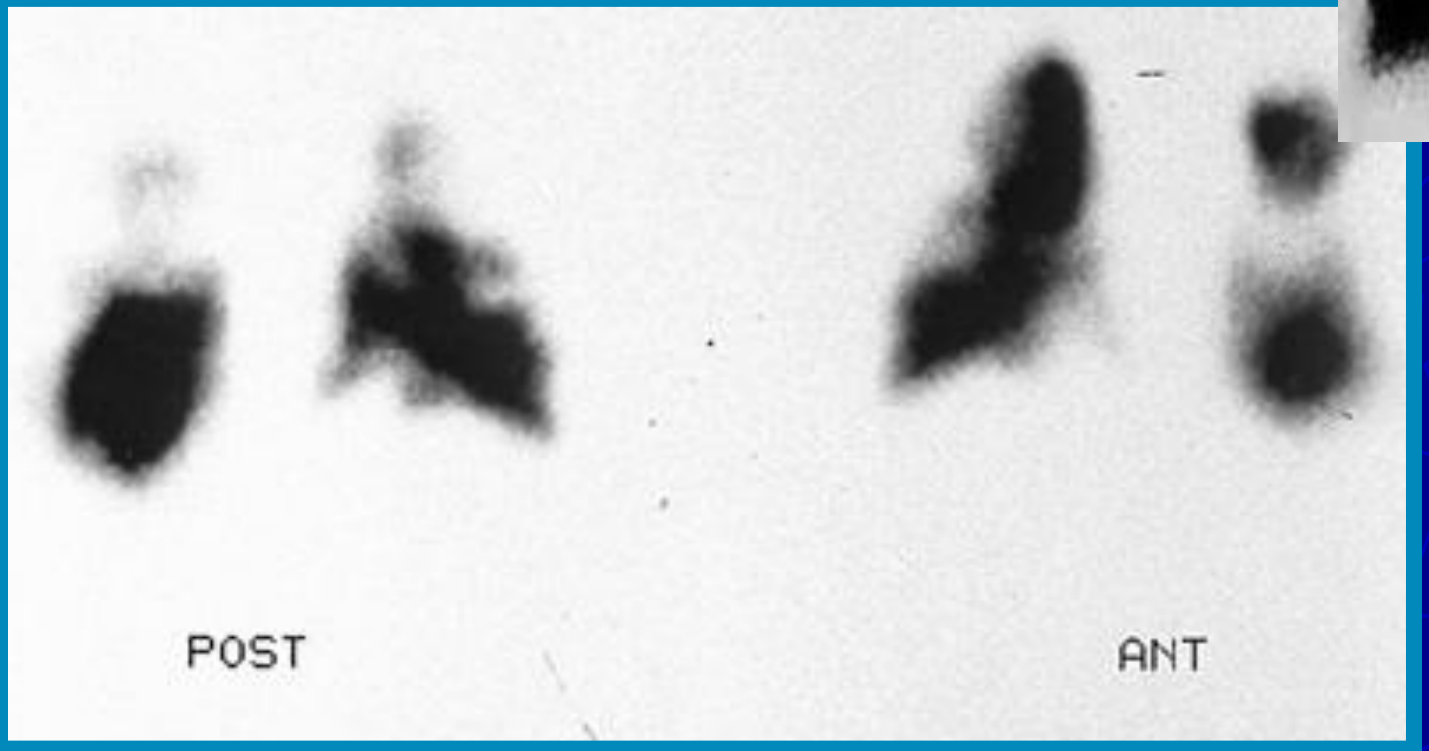


Normal scan



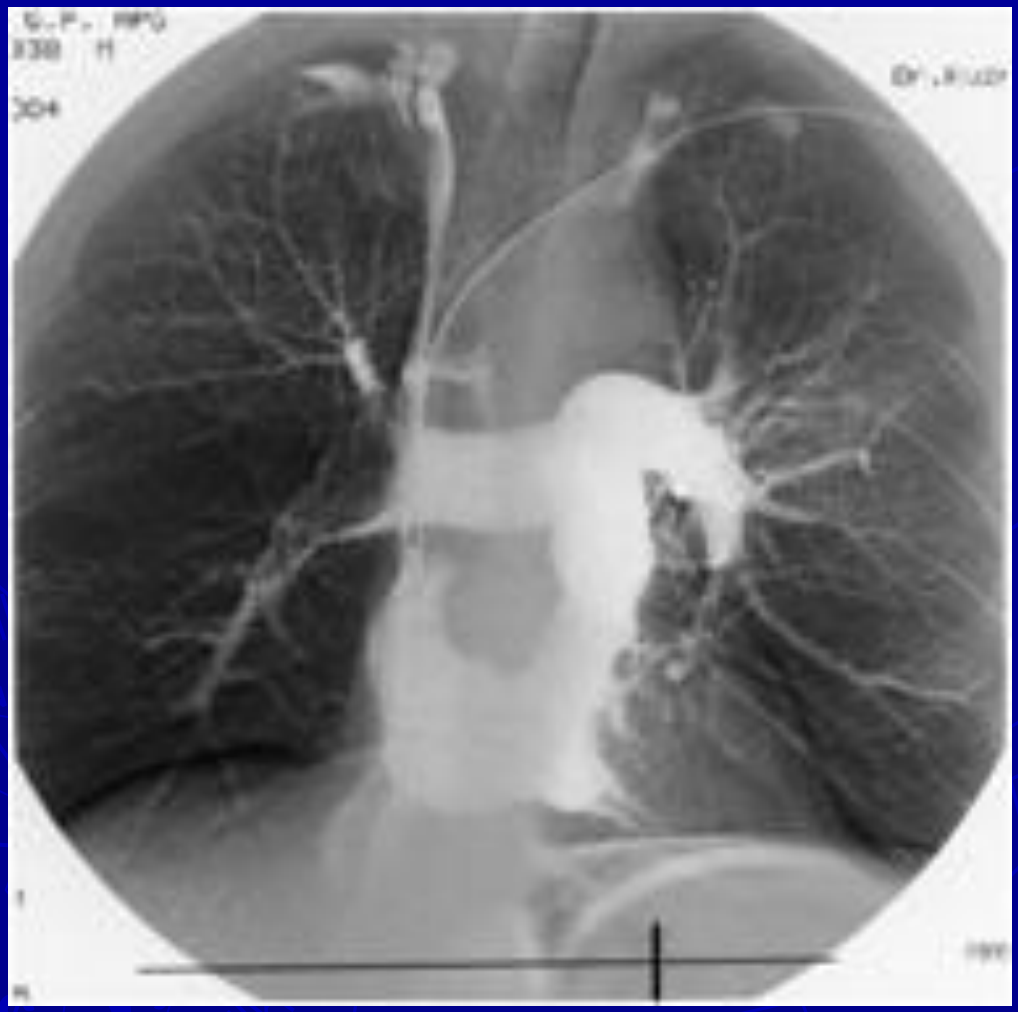
Pulmonary embolism. Scintigraphy.

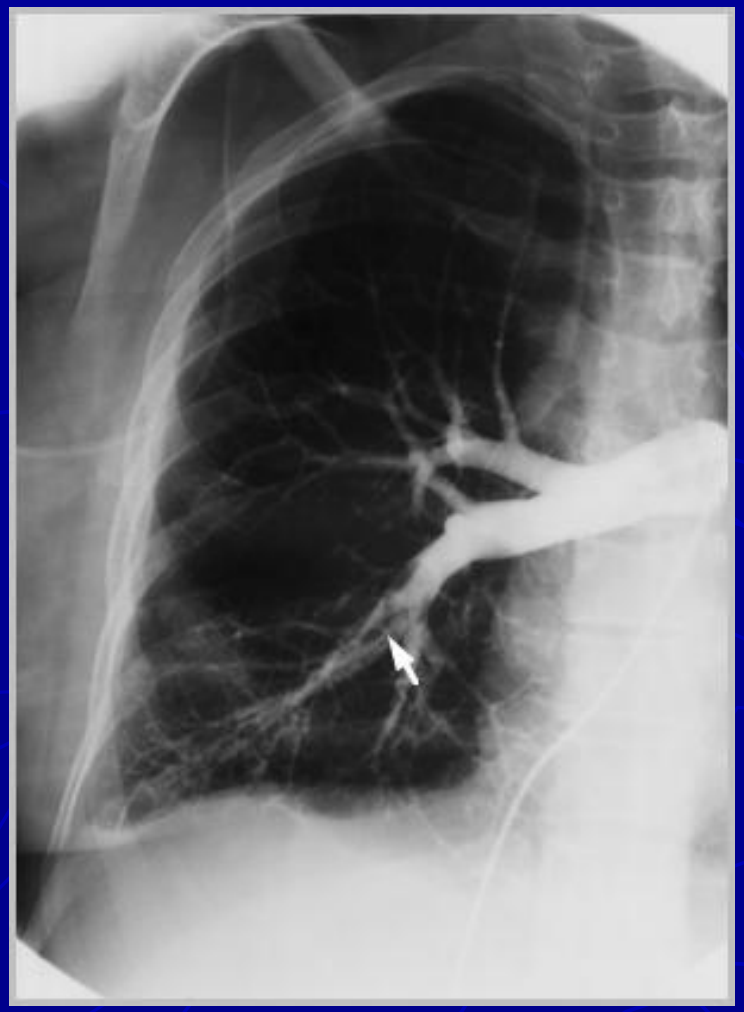
High-probability ventilation-perfusion scan



Multiple large segmental and subsegmental perfusion defects are seen bilaterally. The corresponding ventilation image was normal.

Pulmonary angiogram (angiopulmonography)





The diagnosis can be confirmed by:

- persistent filling defect
- abrupt cut-off of flow.

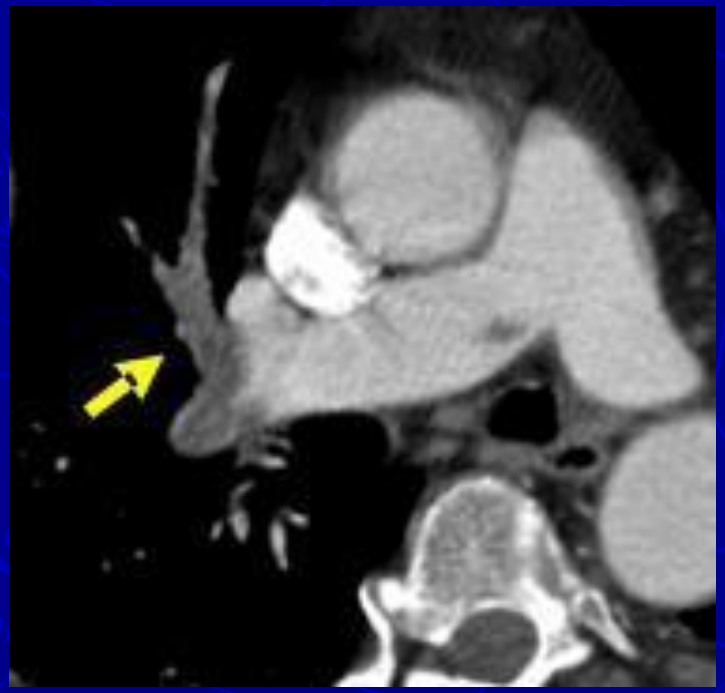
Pulmonary thrombo-embolism: CT signs

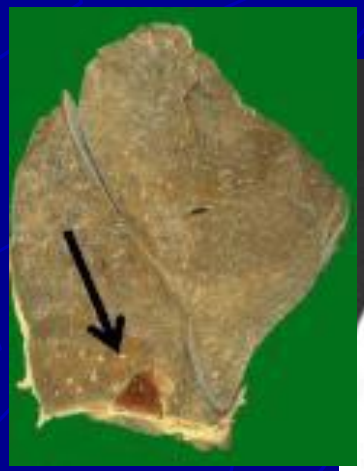
Direct signs:

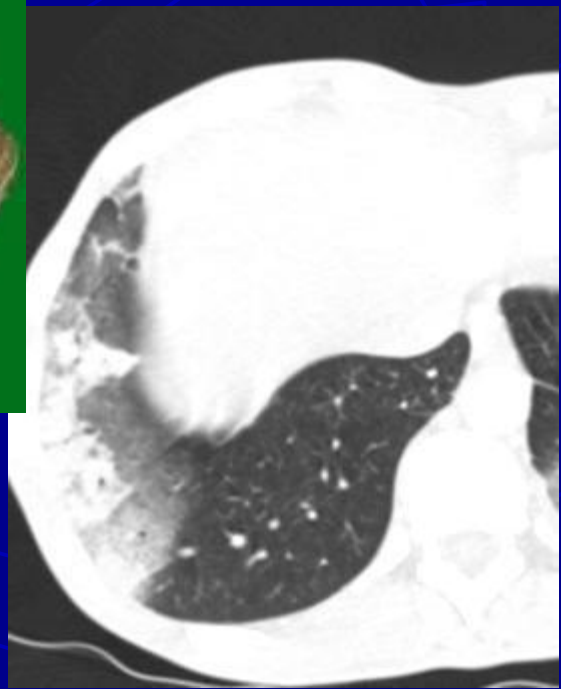
Visualization of the embol

Indirect signs:

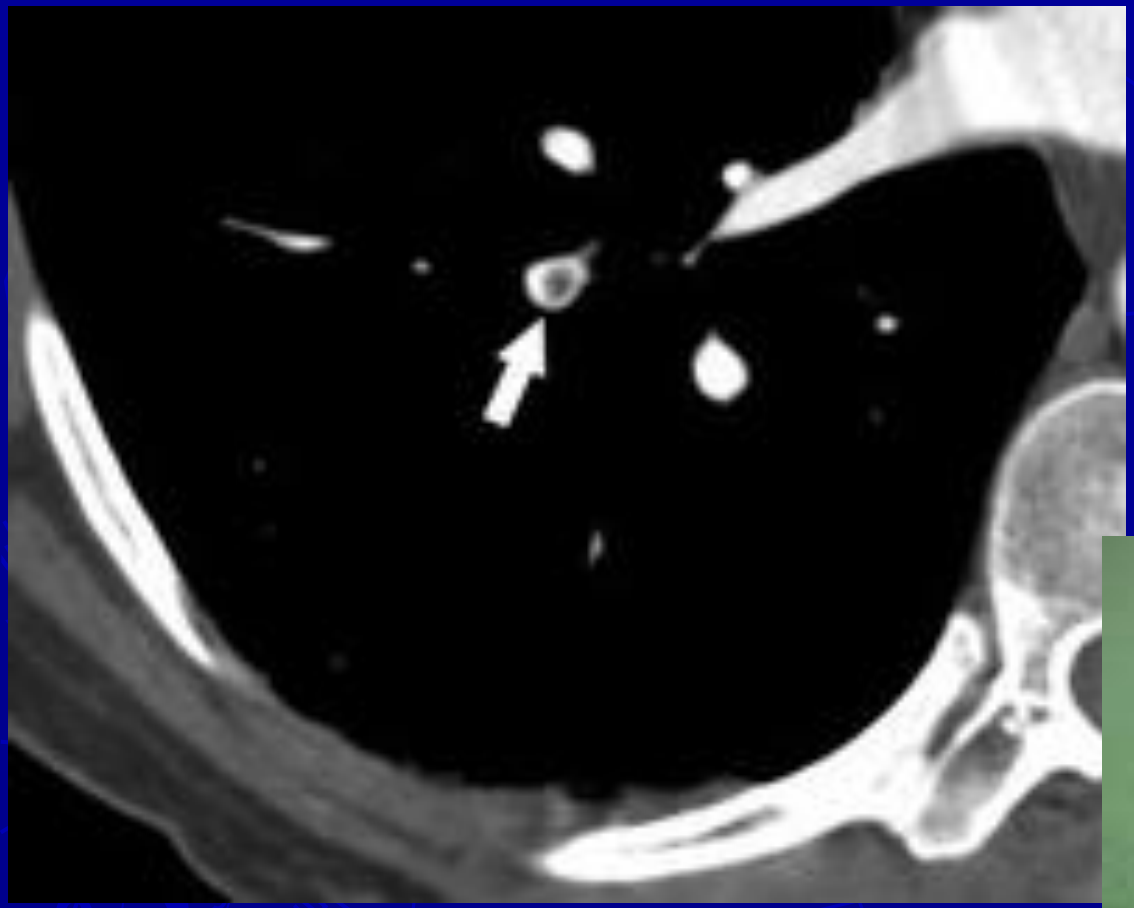
- ✓ Pulmonary infarction
- ✓ Athelectasis
- ✓ Pleural effusion



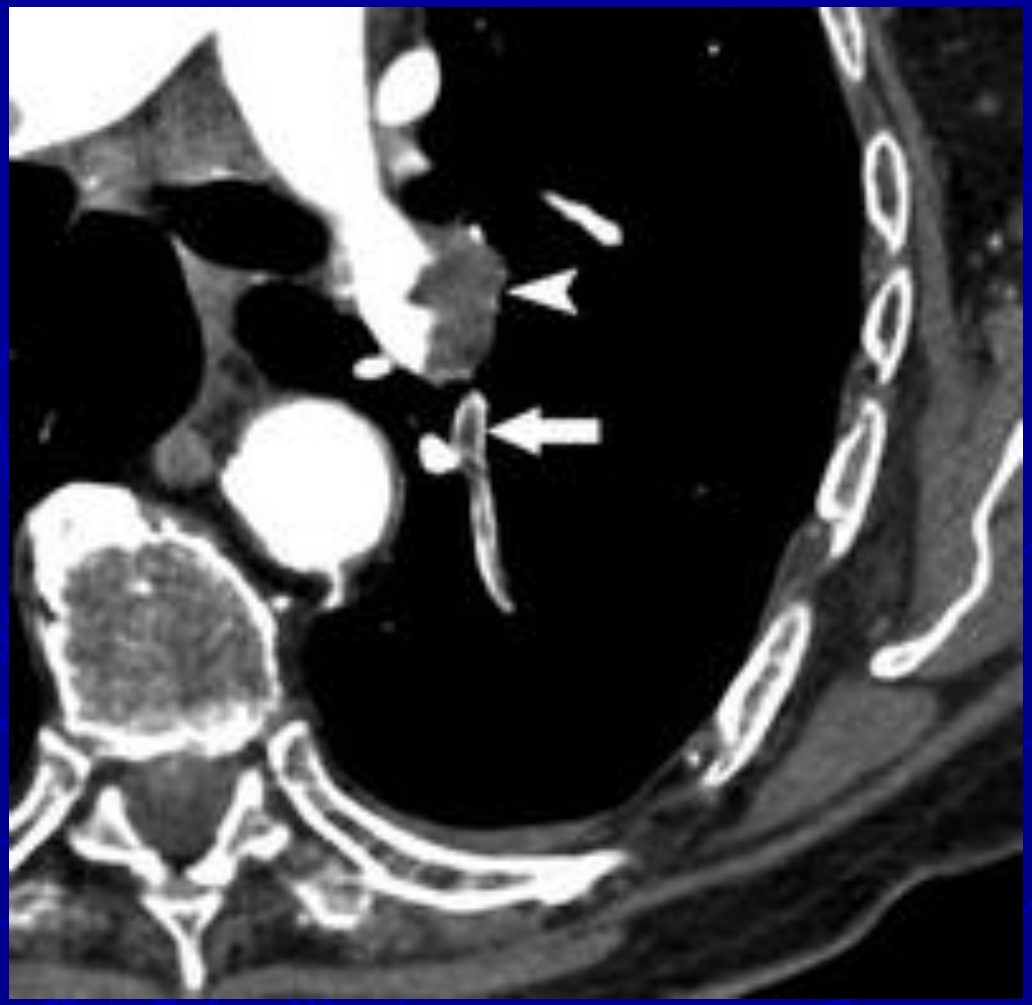


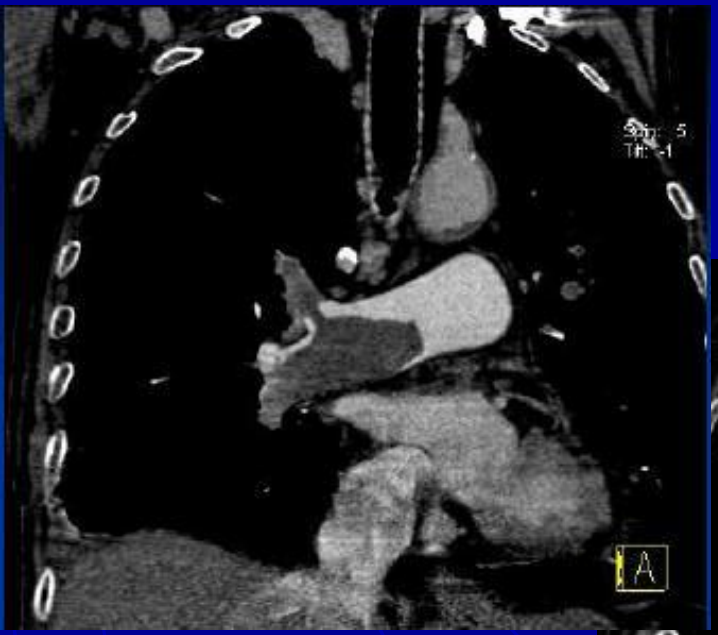


Intraluminal filling defect (polo mint sign)



Intraluminal filling defect (railway track sign)

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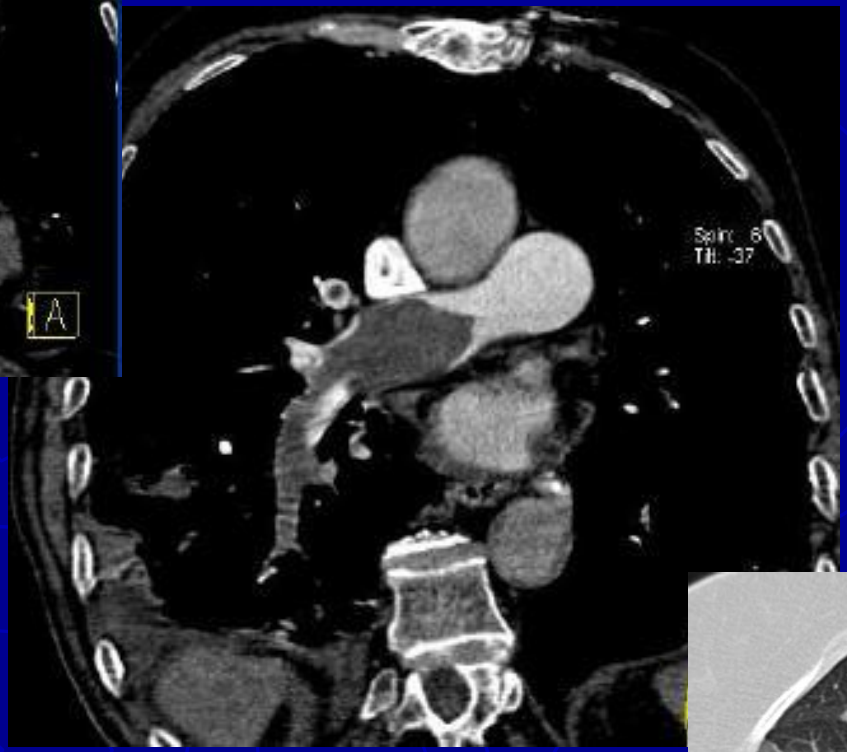
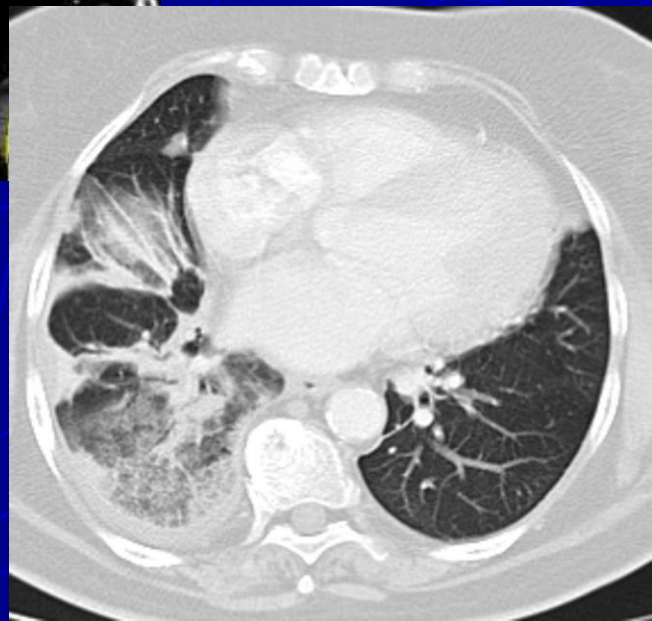
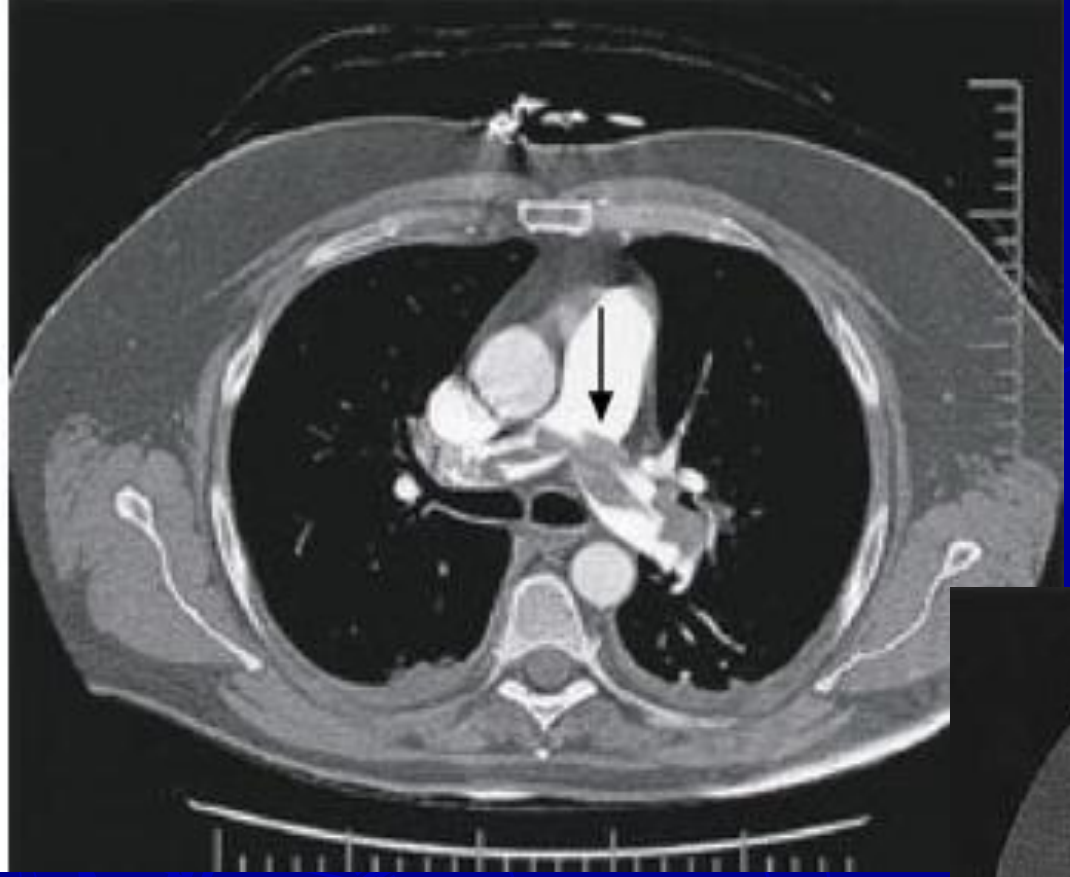
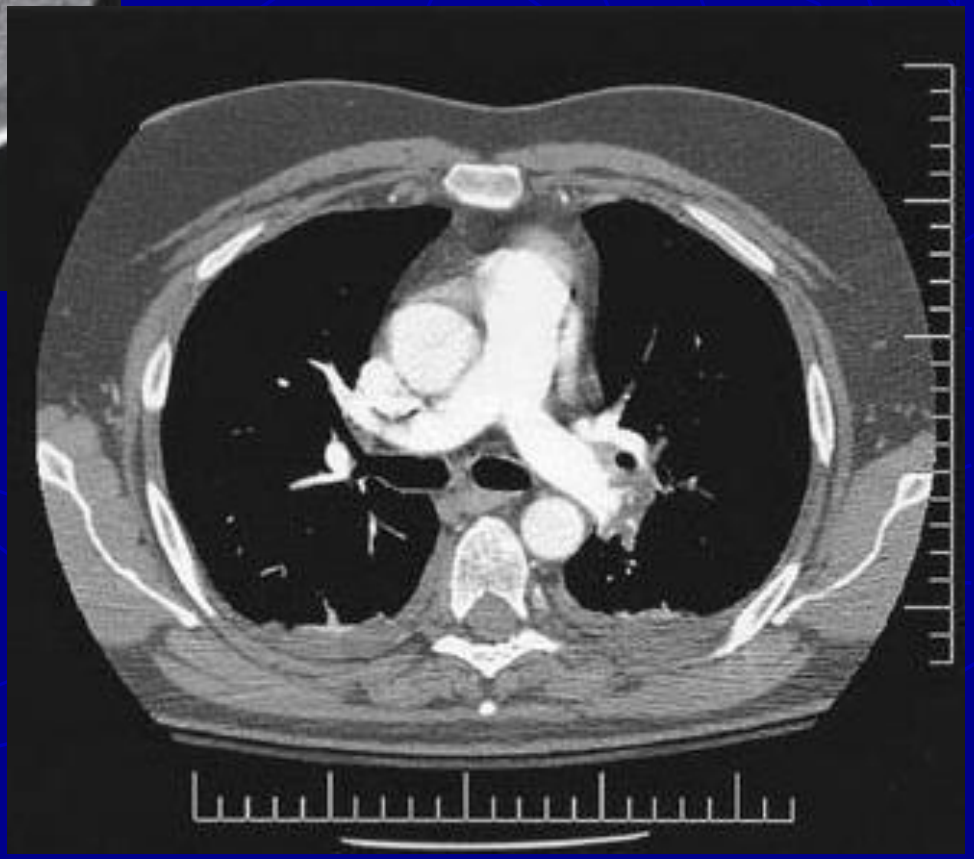
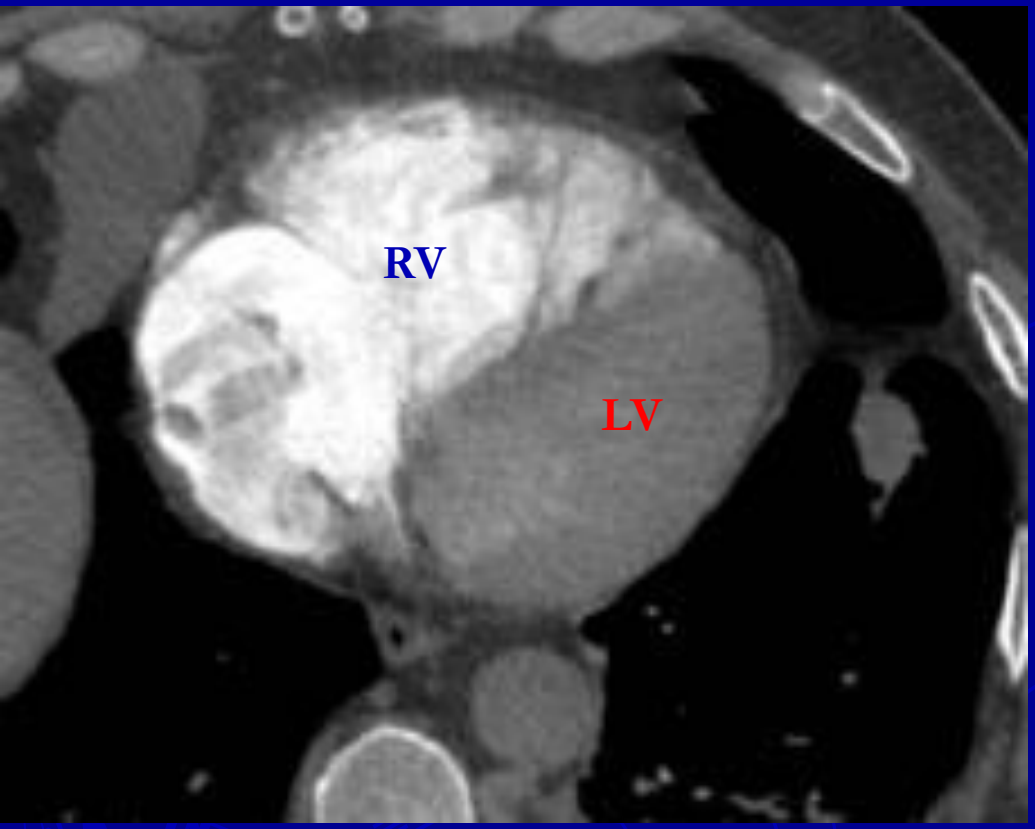
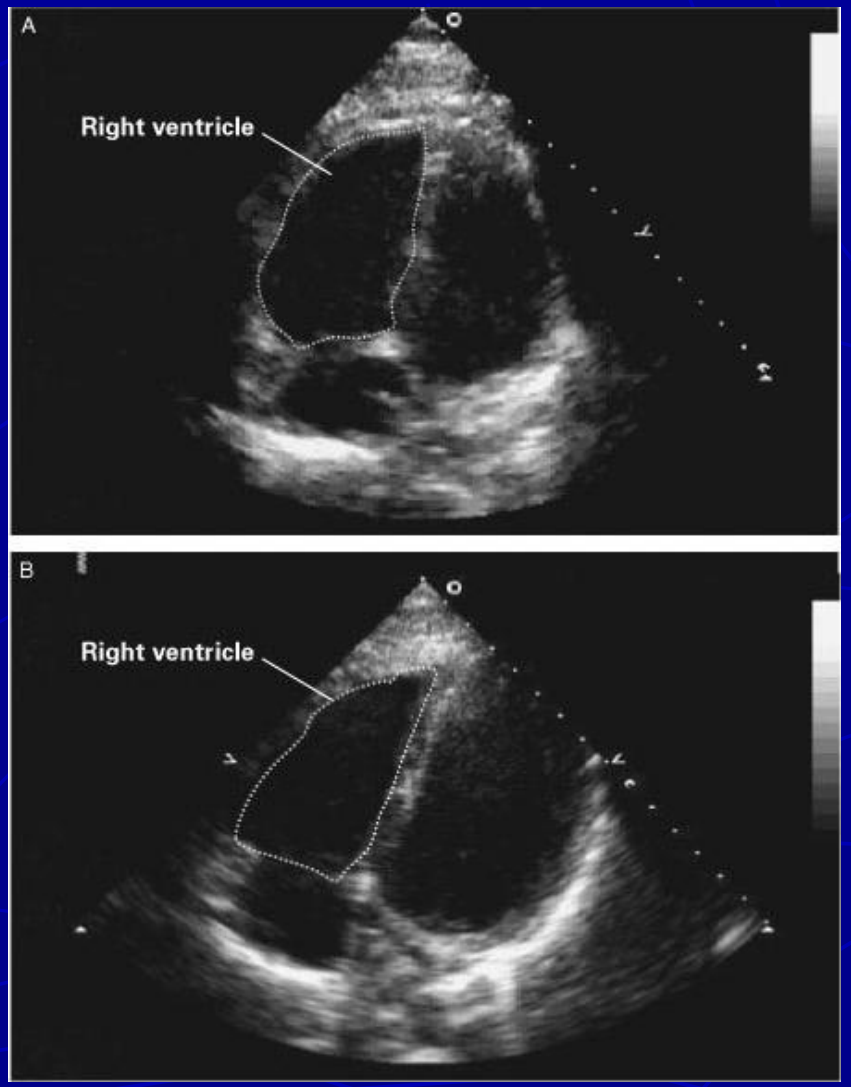








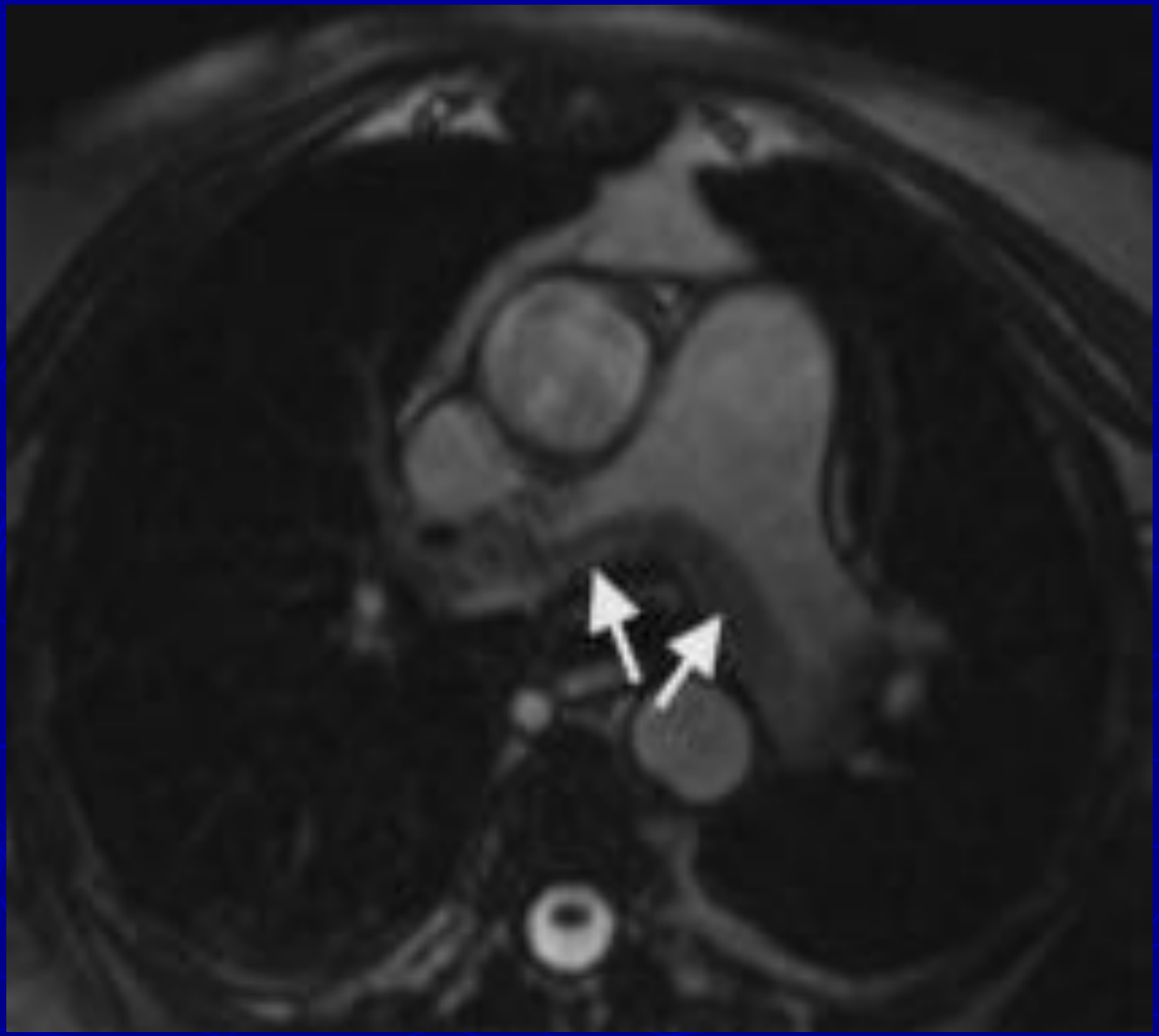




Gravity criteria:

VD/VG>1

MRI. Transversal real-time image showing the saddle thrombus (arrows).



Iodinate contrast agents: what is ideal

- Low viscosity
- Low osmolality
- High water solubility
- Biological inertness
- Safety
- Heat and chemical stability
- Cost effective

Viscosity

- The practical importance of viscosity of a contrast medium relates chiefly to the **force that is required to inject**
- The viscosity can be reduced by lowering the concentration of the contrast medium, but result in **unsatisfactory opacification**. Since viscosity is inversely related to **temperature**, **warming of the contrast medium up to the temperature of human's body will decrease the viscosity and may partly resolve this problem**

Because of their chemical properties, iodinated contrast media usually have greater viscosity and greater osmolality than biological liquids (blood, plasma, cerebrospinal fluid). They can be divided into:

- **Ionic** (a molecule can break up into a positively charged cation and negatively charged anion).
- **Nonionic** (do not have this property = are less osmolar)

Both ionic and nonionic contrast media can be:

- **Monomeric** (a contrast molecule contains only 1 benzene ring and deliver less iodine with each molecule of contrast)
- **Dimeric** (deliver more iodine with each molecule of contrast)

The ratio between the number of iodine atoms and the number of particles in the solution determines the osmotoxicity of contrast agent

TABLE 1
Commonly Used Iodinated Contrast Media

Name	Type	Iodine content (mg/mL)	Osmolality
Ionic			
Diatrizoate (Hypaque 50; GE Healthcare)	Monomer	300	1,550 (high)
Metrizoate Isopaque (Coronar 370; Nycomed A/S)	Monomer	370	2,100 (high)
Ioxaglate (Hexabrix; Mallinckrodt, Inc.)	Dimer	320	580 (low)
Nonionic			
Iopamidol (Isovue-370; Bracco Diagnostics Inc.)	Monomer	370	796 (low)
Iohexol (Omnipaque 350; GE Healthcare)	Monomer	350	884 (low)
Iodixanol (Visipaque 320; GE Healthcare)	Dimer	320	290 (iso)

Osmolality of plasma is 300 mosmol/l

The adverse reactions are commonly related to the following:

1. Physical properties of the contrast media.
2. Iodine concentration of the contrast media.
3. Total dose or volume of the contrast media injected.
4. Rate or speed of the injection.

Types of reactions:

1. Anaphylactoid (idiocyncratic)
2. Nonanaphylactoid (physiochemotoxic or nonidiosyncratic.These reactions are believed to result from the ability of the contrast media to upset the homeostasis of the body, especially the blood circulation)
   - chemotoxic
   - vasovagal
   - idiopatic
3. Combined (1 and 2)

The approach to patients has two general aims:
- to prevent a reaction from occurring and
- to be fully prepared to treat a reaction should one occur

In evaluating a patient for a potential contrast reaction, the followings important immediate assessments should be made:

- How does the patient look?
- Can the patient speak? How does the patient's voice sound?
- How is the patient's breathing?
- What is the patient's pulse strength and rate?
- What is the patient's blood pressure?

The level of consciousness, the appearance of the skin, quality of phonation, lung auscultation, blood pressure and heart rate assessment will allow the responding physician to quickly determine the severity of a reaction.

These findings also allow for the proper diagnosis of the reaction including urticaria, facial or laryngeal edema, bronchospasm, hemodynamic instability, vagal reaction, seizures, and pulmonary edema.

Mild reactions

- Nausea
- Metallic taste
- Warmth
- Facial flashing
- Sneezing
- Dizziness

Approximatelhy in 10% of patients

Disappear if injection stopped and do not appear again when the injection of contrast agents continues

Needn't special treatment. The only measure to be taken is to stop the injection for 20-30 seconds

Moderate reactions

Need a treatment, but not an intensive care

- Urticaria
- Swelling face
- Headache
- Vomiting
- Wheezing
- Bronchospasm
- Cutaneous reactions
- Repeated sneezing
- Tear
- Abdominal pain

- Arterial hypotension
- Tahycardia
- Pallor

This type of reactions can appear additionally to the allergic ones

TREATMENT

- **Administration of oxygen**
- **Administration of adrenalin subcutaneously (0,5 mg, solution 1 mg/ml)**
- **Administration of antihistamines**

Severe reactions

include signs of anaphylactic shock:

- Cardiovascular
- Respiratory
- Neurologic

e.g. laryngeal edema, convulsions, profound hypotension, clinically manifest arrhythmias, unresponsiveness, cardiopulmonary arrest

TABLE 4
General Principles for Managing Contrast Reactions

Principle	Strategy
A	Assessment (severity and category of reaction): blood pressure and pulse monitoring, ECG monitor for evaluation of cardiac rhythm
	Assistance (call for it)
	Airway, oxygen
	Access (venous)—secure/improve intravenous lines
B	Breathing (begin cardiopulmonary resuscitation if necessary, bag-valve mask or mouth mask)
	Beware of paradoxical responses (e.g., β-blockers may prevent tachycardia response)
C	Categorize reaction and patient status
	Circulatory assistance, intravenous fluids
	Call cardiopulmonary arrest response team if necessary
	Cardiac output assessment, decreased venous return
D	Drugs: dose and route, do not delay
	Do monitor, assess, and reassure patients

ECG = electrocardiogram.

TABLE 5
Management of Common Contrast Reactions

Reaction	Etiology	Monitor...	Treatment (*28–30*)
Anaphylactoid			
Urticaria (skin rash)	Anaphylactoid reaction	Initial size with marking and follow	Usually none; diphenhydramine, 25–50 mg orally/intramuscularly/intravenously; epinephrine (1:1,000), 0.1–0.3 mL subcutaneously/intramuscularly
Bronchospasm	Anaphylactoid reaction	Oxygen saturation, pulse, BP	Secure airway; oxygen, 6–10 L/min; metaproterenol/terbutaline inhaler, 2–3 puffs; epinephrine (1:1,000), 0.1–0.3 mL subcutaneously/intramuscularly; epinephrine (1:10,000), 1 mL intravenously (slowly) if hypotensive; call the emergency medical team
Facial or laryngeal edema	Anaphylactoid reaction	Oxygen saturation, pulse, BP	Secure airway; oxygen, 6–10 L/min; call the emergency medical team if severe; epinephrine (1:1000), 0.1–0.3 mL subcutaneously/intramuscularly; epinephrine (1:10,000), 1 mL intravenously (slowly) if hypotensive; call the emergency medical team
Hypotension and tachycardia (fast pulse)	Vasodilation	Oxygen saturation, pulse, BP	Elevate legs 60°; oxygen, 6–10 L/min; rapid intravenous fluids; epinephrine (1:10,000), 1 mL intravenously (slowly); call the emergency medical team
Hypotension and bradycardia (slow pulse)	Vasovagal response	Oxygen saturation, pulse, BP	Elevate legs 60°; oxygen, 6–10 L/min; atropine, 0.6–1 mg intravenously (slowly); repeat to total of 2–3 mg (0.04 mg/kg) if needed; call the emergency medical team
Nonanaphylactoid			
Cardiac arrhythmia	Ionic abnormalities; chemical variations	Oxygen saturation, pulse, BP, ECG	Follow ACLS* protocols; call the emergency medical team
Hypertension	Histamine release of catecholamine	Oxygen saturation, pulse, BP, ECG	Nitroglycerine, 0.4 mg sublingually; nitroglycerine; 2% ointment; phentolamine, 5 mg intravenously for pheochromocytoma; call the emergency medical team
Seizures	Ionic abnormalities; chemical variations	Oxygen saturation, pulse, BP, ECG	Secure airway; oxygen, 6–10 L/min; diazepam, 5 mg intramuscularly/intravenously; midazolam, 0.5–1 mg intravenously; phenytoin infusion, 15–18 mg/kg at 50 mg/min; call the emergency medical team
Pulmonary edema	Osmolar changes, causing large fluid volume shifts	Oxygen saturation, pulse, BP, ECG	Secure airway; oxygen, 6–10 L/min; furosemide, 20–40 mg intravenously (slowly); morphine, 1–3 mg intravenously; call the emergency medical team

All medications are to be administered under physician supervision. BP = blood pressure; ECG = electrocardiogram; ACLS = advanced cardiovascular life support.

TABLE 6
Suggested Supplies and Information to Be Made Available

Category	Specifics
Posted information	Name and contact information for physician on duty, phone number of emergency response team
Support apparatus	Oxygen cylinders, flow valves, tubing, nasal prongs, oxygen masks (adult and pediatric sizes), bag-valve mask, valve masks, endotracheal tubes, laryngoscopes, intravenous fluids (normal saline, Ringer's lactate)
Emergency and monitoring devices	Defibrillator, ECG, blood pressure/ pulse monitor, pulse oximeter
Medications	Epinephrine, 1:10,000, 10-mL preloaded syringe; epinephrine, 1:1,000, 1-mL preloaded syringe; atropine, 1 mg in 10-mL preloaded syringe; β-agonist inhaler; diphenhydramine for intramuscular/intravenous injection; nitroglycerin, 0.4-mg tabs, sublingually

ECG = electrocardiogram.

Prophylaxis

To know the allergic history of the patient (It is most important to target premedication to those who, in the past, have had moderately severe or severe reactions requiring treatment

TABLE 2
Common Factors Predisposing Patient to Contrast Reactions

Factor	Predisposing characteristic
Age	Infants and those older than 60 y
Sex	Females > males
Underlying medical conditions	Asthma, heart disease, dehydration, renal disease, diabetes
Hematologic conditions	Myeloma, sickle cell disease, polycythemia
Medications	NSAIDs, IL-2, β-blockers, biguanides
Contrast-related	>20 mg iodine, faster injection rate, intraarterial, previous contrast reactions

NSAIDs = nonsteroidal antiinflammatory drugs; IL-2 = interleukin-2.

TABLE 3

Premedication Protocols for Patients with Previous or Increased Risk for Contrast Reactions

Premedication	Protocol
Corticosteroids (any of the following)	Prednisone: 50 mg orally, 13, 7, and 1 h before contrast injection
	Hydrocortisone: 200 mg intravenously, 1 h before contrast injection
	Methylprednisone: 32 mg orally, 12 and 2 h before contrast media injection
Antihistamine (optional)	Diphenhydramine: 50 mg intravenously/intramuscularly/orally 1 h before contrast injection