Professor Mike Ardagh

Professor of Emergency Medicine at the University of Otago, and Specialist in Emergency Medicine at Christchurch Hospital
Christchurch

Sudden Shortness of Breath - Concurrent Workshop

Saturday, 30 July 2011 | Start 4:30pm | Duration: 60mins | Skeggs Room



Shortness of breath (respiratory emergencies)

Mike Ardagh
University of Otago, Christchurch
Emergency Dept. Christchurch Hospital.

Three Cases

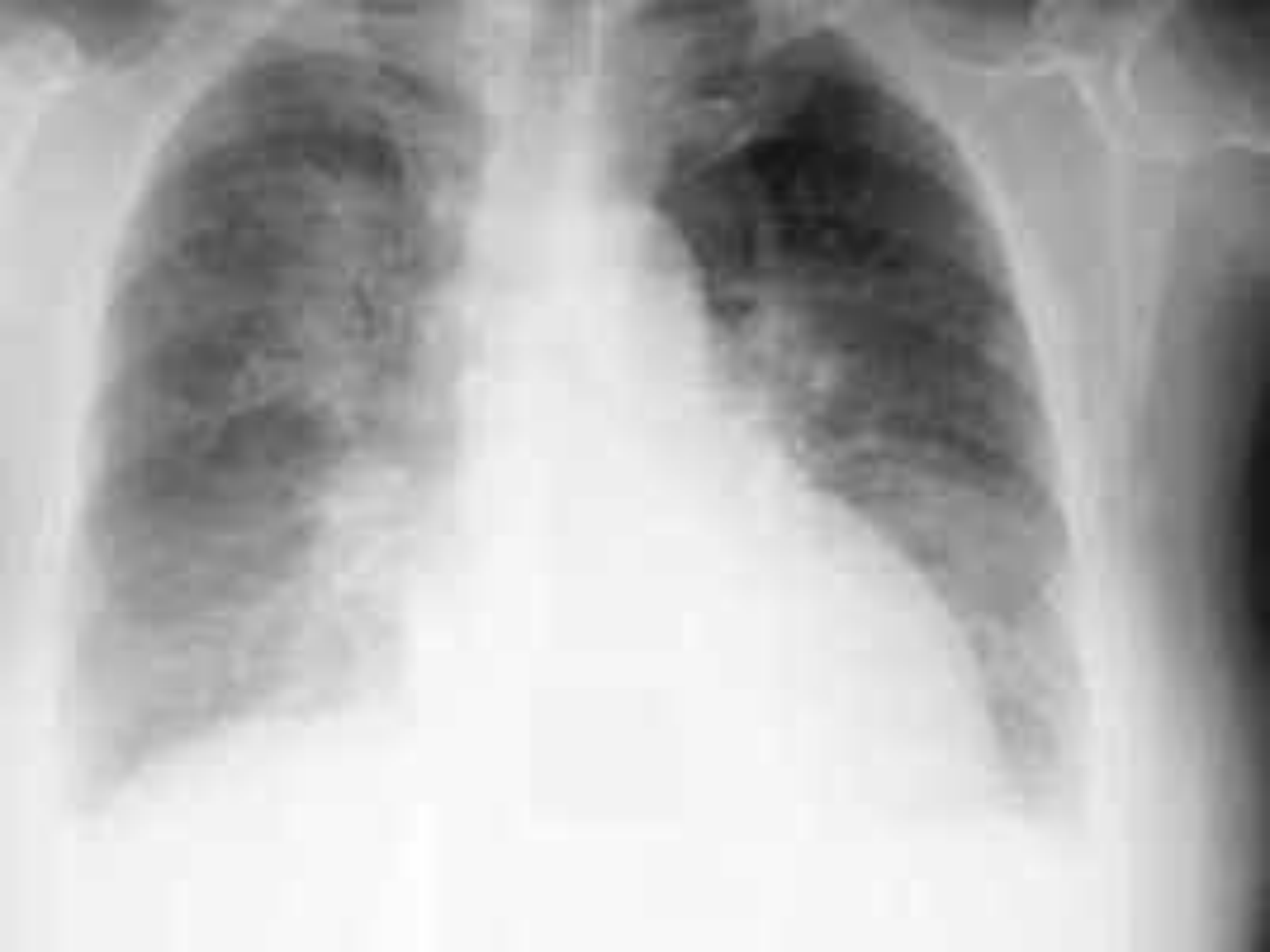
- Acute Pulmonary Oedema
- Airways disease
- Community Acquired Pneumonia
  - With frequent detours into background pathology and physiology

Case 1.

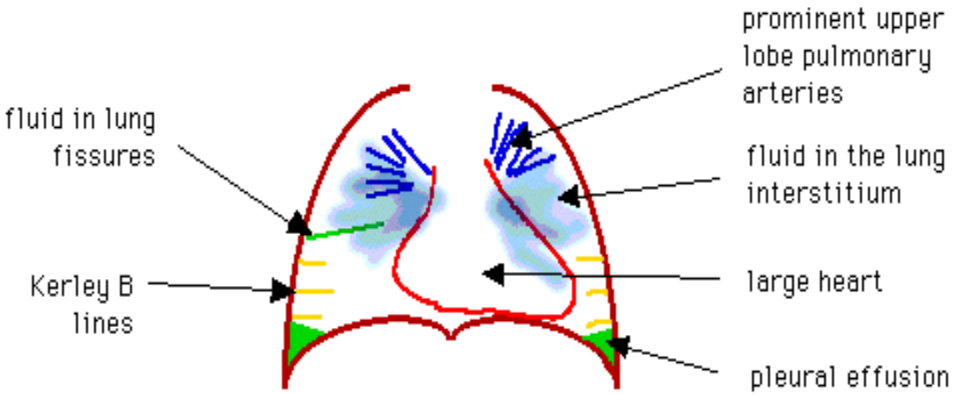
- 64 year old woman, onset of increasing shortness of breath at Phantom of the Opera.
- History of hypertension, ankle swelling and occasional exercise induced chest pain.
- On Frusemide 40mg/day and nitrolingual spray PRN.

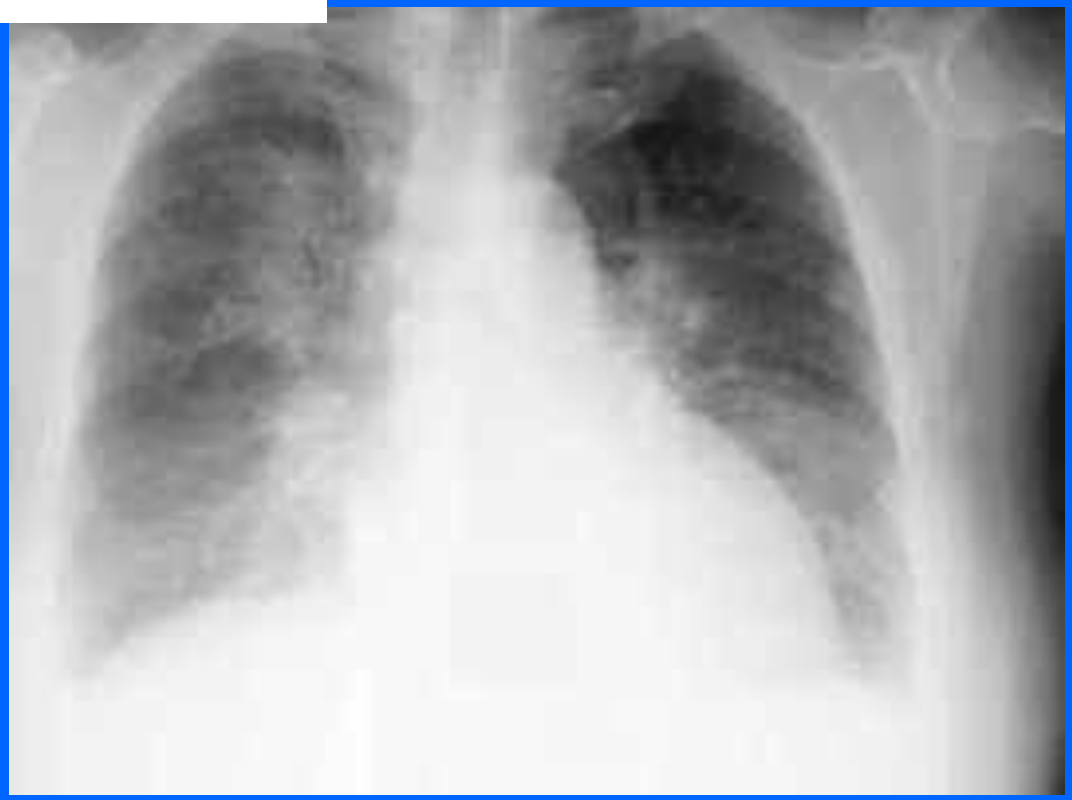
Case 1.

- Very breathless, agitated, confused, with audible moist breath sounds.
- Crackles both sides to midzones.
- SaO2 88% on high flow O2
- Sinus tachy 118/min, BP 210/105
- Mild pitting ankle oedema, JVP +1cm.



The radiological signs of heart failure





Diagnosis

- History and exam
  - Useful
- CXR
  - Very useful
- BNP
  - Possibly useful.

The physiology of noise

- Laminar flow is quiet.
- Turbulent flow makes noise.
- Turbulent flow is inefficient.
- So, noise means inefficient breathing.
- Turbulence is more likely with narrowing of the airway or increased speed of air movement.

The classification of noise

- Stertor – snoring due to oropharyngeal narrowing.
- Stridor – inspiratory noise due to extra-thoracic airway narrowing.
- Wheeze – expiratory noise due to intra-thoracic airway narrowing.
- Crackles – bubbles of air through water.

Case 1

Treatment?

Case 1. A and B

- O2 – titrated to SaO2
  - Air (FiO2 = .21)
  - Nasal prongs (FiO2 = ?)
  - Hudson mask (FiO2 = ?)
  - Hudson mask with reservoir bag (FiO2 = ?)
  - Ambubag-mask negative pressure ventilation (FiO2 = 1 if closed to air).

(they suck it in, and they blow it out)

Case 1. A and B

- CPAP
- Positive pressure ventilation
  - Ambubag-mask positive pressure ventilation
  - Bag to ET tube positive pressure ventilation

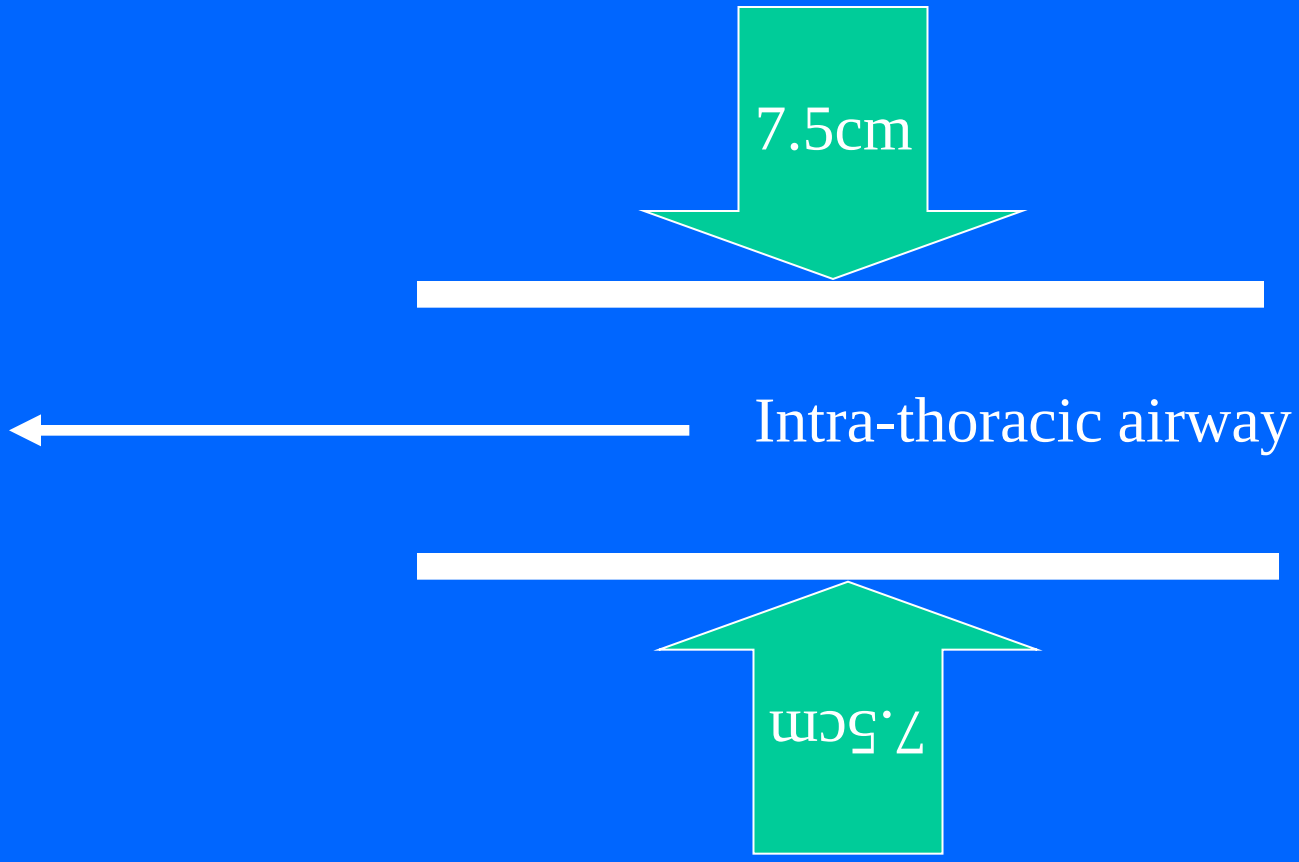
(We blow it in, they blow it out)

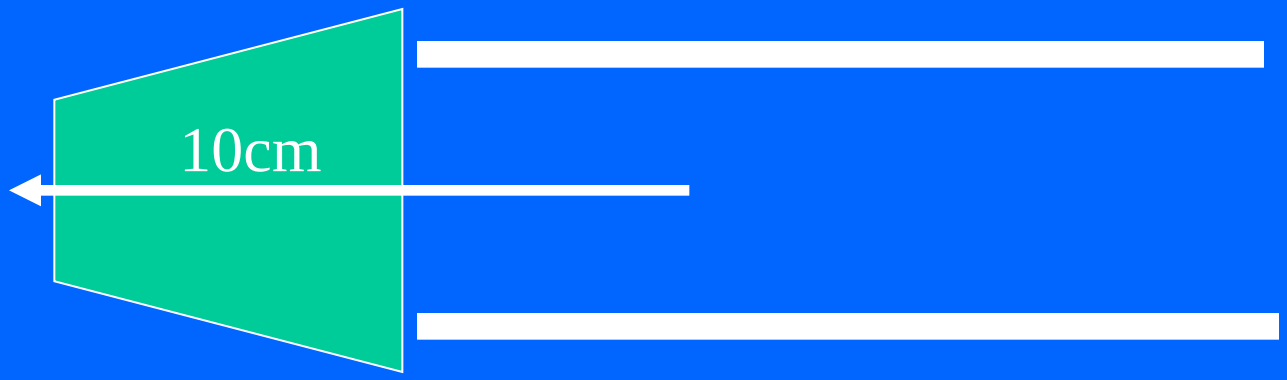
CPAP

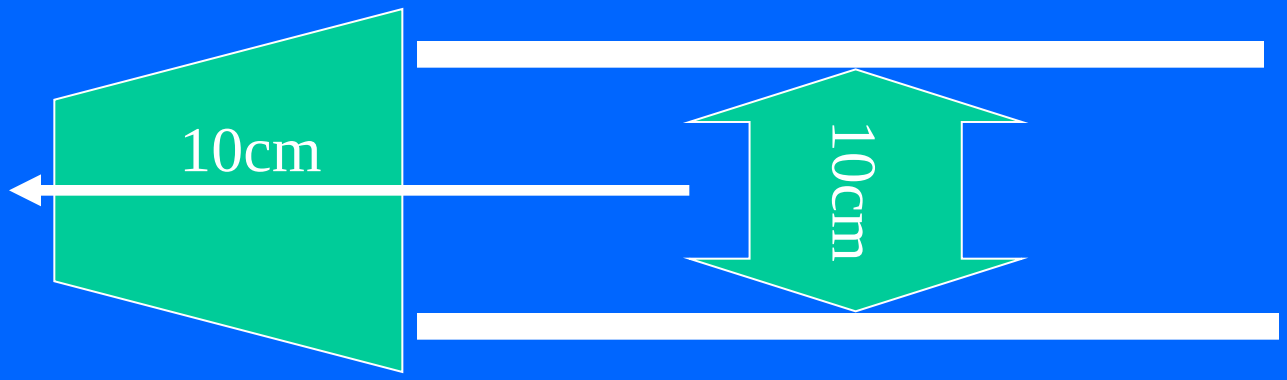
- Tight fitting mask (with high flow O2)
- Breathing out against resistance (CPAP or PEEP valve)
- Pressure ‘splints’ airways open, disperses fluid.
- Raised intra-thoracic pressure drops the BP
- Go up from 5cm water (asthma/COPD - come down from 10cm water)
- No evidence BIPAP is better in APO (but might be in airways disease).

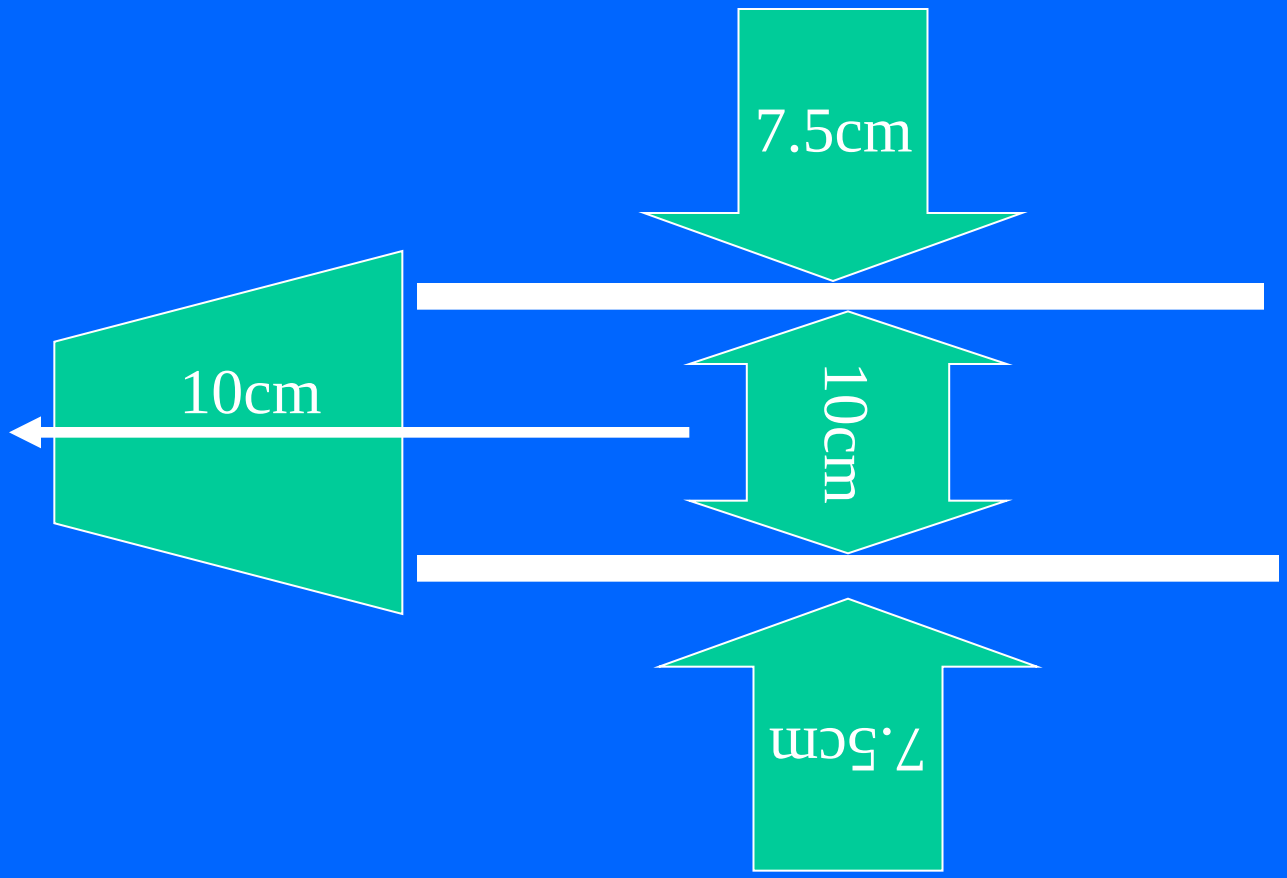
Intra-thoracic airway

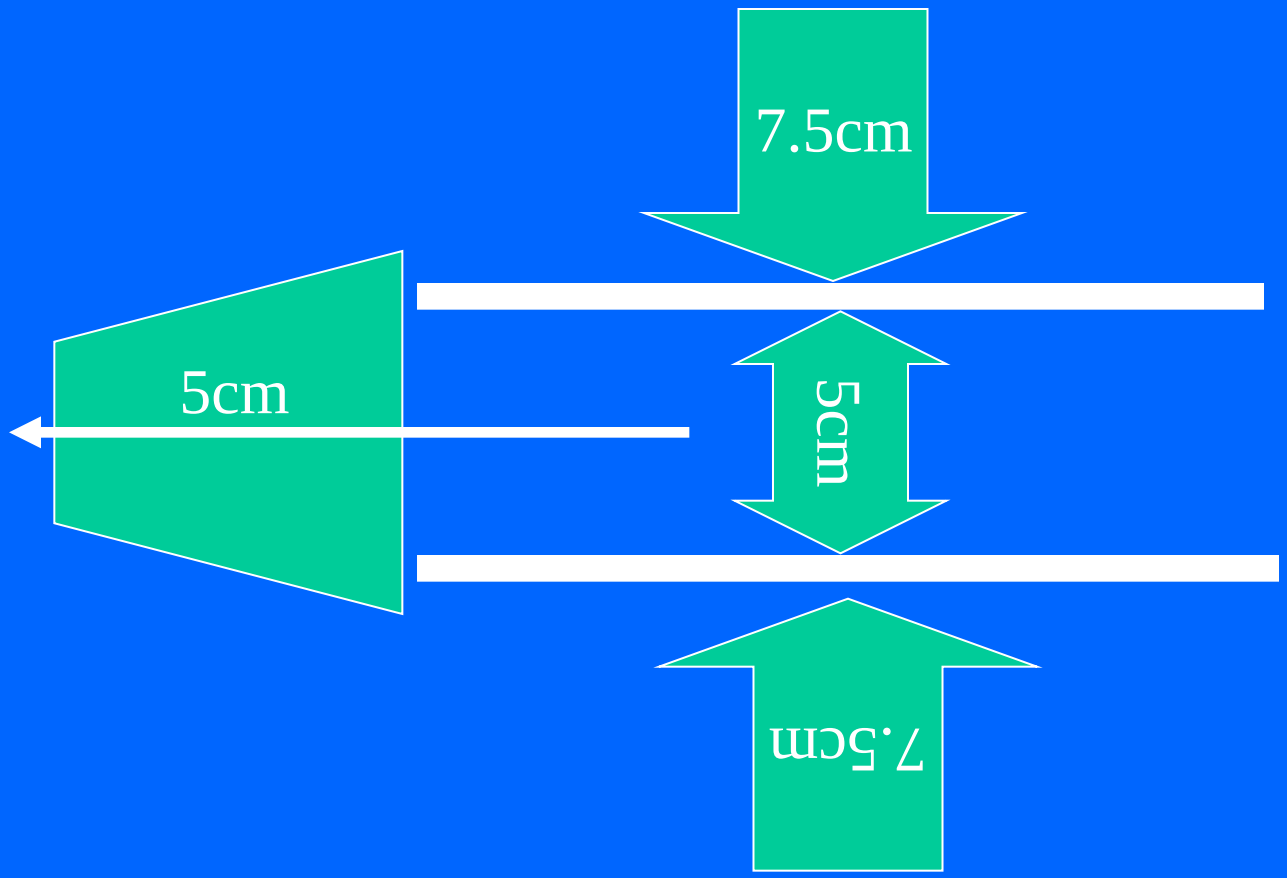

Intra-thoracic airway


7.5cm
Intra-thoracic airway
7.5cm


10cm


10cm
10cm


7.5cm
10cm
10cm
7.5cm


7.5cm
5cm
5cm
7.5cm

APO Treatment

- Proven
  - CPAP
  - Nitrates
    - Increase cGMP in smooth muscle
    - SL or IV (or topical)
    - 5mcg/min, inc by 5 every 5 minutes, until SBP 120
    - Beware Viagra, outflow obstruction and RV infarcts.
- Unproven
  - Frusemide - still advocated.
  - Morphine - small titrated doses only.
  - Nesiritide

The failing heart

- The three contributors
  - Preload – venous return
  - Contractility – ionotropy
  - Afterload – systemic resistance (blood pressure)
- Acute pulmonary oedema (flash flooding) is often a hypertensive (afterload) issue
- CPAP and nitrates lower the BP

What if the BP is low?

- Cardiogenic pulmonary oedema and hypotension is a different disease = cardiogenic shock.
- Outlook is poor and ionotropes are usually needed.
- Low BP due to cardiac cause and dry lungs may respond to fluids (especially if RV infarct or outflow obstruction).

Case 2. Asthma

- 13 year old boy working with his father in the shed.
- Rapid onset (20-30 minutes) of increasing shortness of breath.
- Recently well
- History of asthma and eczema when younger.
- Occasional ventolin use only.

Case 2.

- Assisted in by father, dragged in a panic.
- Severe respiratory distress, agitated, pale.
- Cannot talk, significant accessory use, sternal, intercostal and subcostal recesssion.
- A few high pitched wheezes in chest, but generally very little to hear.

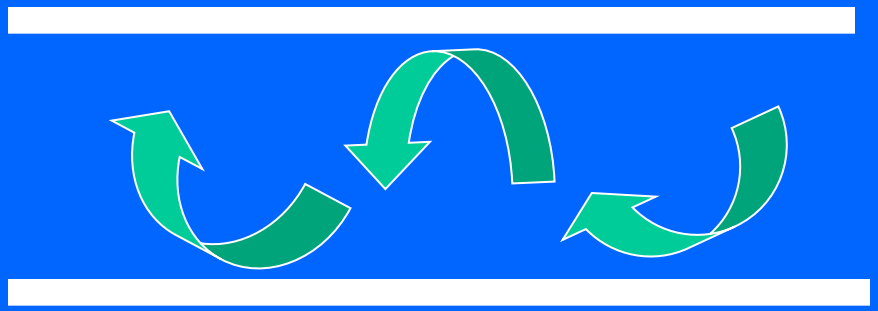
Case 2.

What is the most important intervention in severe asthma?

Calmness

Laminar flow

Turbulent flow – caused by speed of breathing



Case 2. A and B

- O2 – titrated to SaO2
  - Air (FiO2 = .21)
  - Nasal prongs (FiO2 = ?)
  - Hudson mask (FiO2 = ?)
  - Hudson mask with reservoir bag (FiO2 = ?)
  - Ambubag-mask negative pressure ventilation (FiO2 = 1 if closed to air).

(they suck it in, and they blow it out)

A note about CO2 retainers

- Some chronic airways disease patients run on an ‘hypoxic drive’ (rather than a hypercapnic drive like us).
- Overdoing the O2 can (rarely) cause hypoventilation and progressive CO2 retention (causing acidocis and hypercapnic narcosis).
- SaO2 of high 80s or 90 is OK in these patients

Case 2. A and B

- CPAP or BIPAP
- Positive pressure ventilation
  - Ambubag-mask positive pressure ventilation
  - Bag to ET tube positive pressure ventilation

(We blow it in, they blow it out)

BIPAP

- Proven benefit in airways disease.
- PEEP (or CPAP) to splint airways open.
- Inspiratory push (triggered by the patient's negative pressure) decreases work of breathing.
- However, not up-front – takes a bit of set up and can be frightening for the naïve asthmatic.

Positive pressure ventilation in an asthmatic

- We blow it in but they struggle to blow it out – causing air trapping, barotrauma and hypotension
- Avoid intubating an asthmatic if possible.
- If you must allow very long expiratory times.

Case 2. Drug treatment.

- β2 agonists (salbutamol) still top of the list.
- Spacers are good in the mild/moderate.
- Nebulisers (salbutamol 5mg) commonly used in severe cases
  - Continuous, keep it topped up
  - 8l/min of O2 to get good droplets
  - Give them space if they’ve got the flu

Case 2. Drug treatment.

- Add anti-muscarinic agent to nebuliser (ipratropium 0.5mg) if you want (but keep the β2 agonist going).
- If very poor insp effort, or need more O2, then IV β2 agonist an option (salbutamol 250 to 500mcg slow bolus +/- infusion at 5mcg/min).

Case 2. Drug treatment.

What about adrenaline?

Airways obstruction

- Pathology
  - Bronchospasm
  - Mucosal swelling
- Mechanism
  - Type 1 hypersensitivity
  - Infection
  - Chronic airways disease
- History
  - Precipitous
  - Grumbling

Case 2. Drug treatment.

- Adrenaline can be very useful especially if
  - Precipitous (anaphylactic) history
  - Peri – respiratory arrest.
- 0.5mg (0.5ml of 1:1000) IM (deltoid).
- Repeat if necessary.
- IV uncommon if not in cardiac arrest
  - 0.01-0.05mg increments (0.1 – 0.5ml of 1:10,000)
  - Or, 1mg in 1000ml of Normal Saline titrated.

Case 2. Drug treatment.

- Corticosteroids
  - Prednisone 40mg po or Hydrocortisone 200mg IV
- Aminophylline
  - Uncommon use.
  - Some patients and some doctors like it.
  - Possibly its action is CNS stimulation

Case 2. Drug treatment.

- Magnesium
  - A drug looking for a disease
  - Evidence of benefit in severe cases
  - Don’t underdo the β2 agonists in your enthusiasm
  - Don’t worry if you forget it.
- Ketamine
  - Bronchodilating dissociative anaesthetic agent.
  - Good choice for RSI if contemplated.

Case 2. Other treatment.

- Fluids
  - Yes, patient often dry.
- Heliox
  - Lighter than air
  - Decreases work of breathing
  - Forget it.

Case 2. Other treatment.

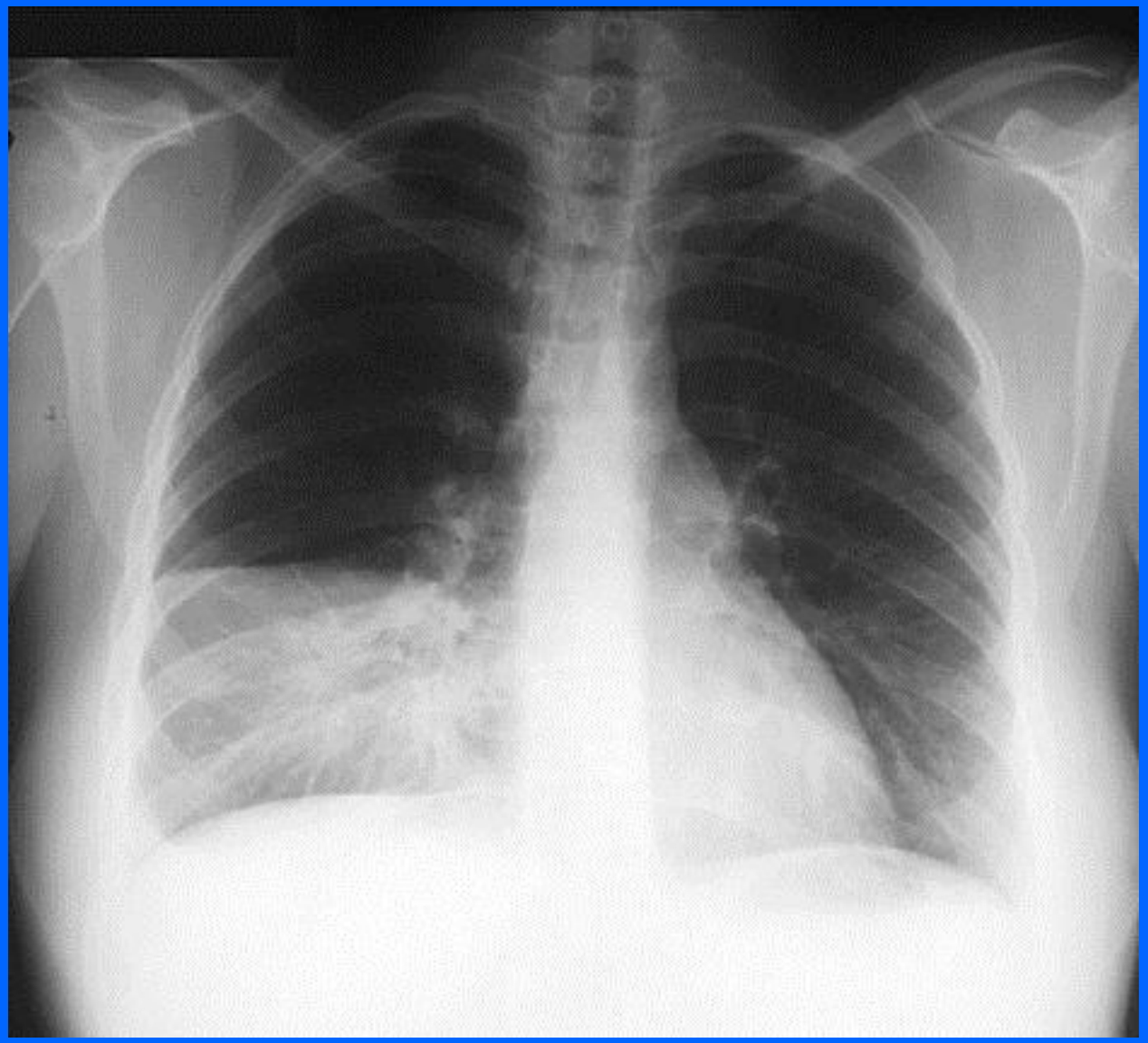
- Antibiotics
  - Sometimes
- Drainage of pneumothorax
  - Severe asthmatic, peri-arrest, with signs of possible tension → needle thoracocentesis
  - Possible pneumothorax clinically → CXR

Case 3. Pneumonia

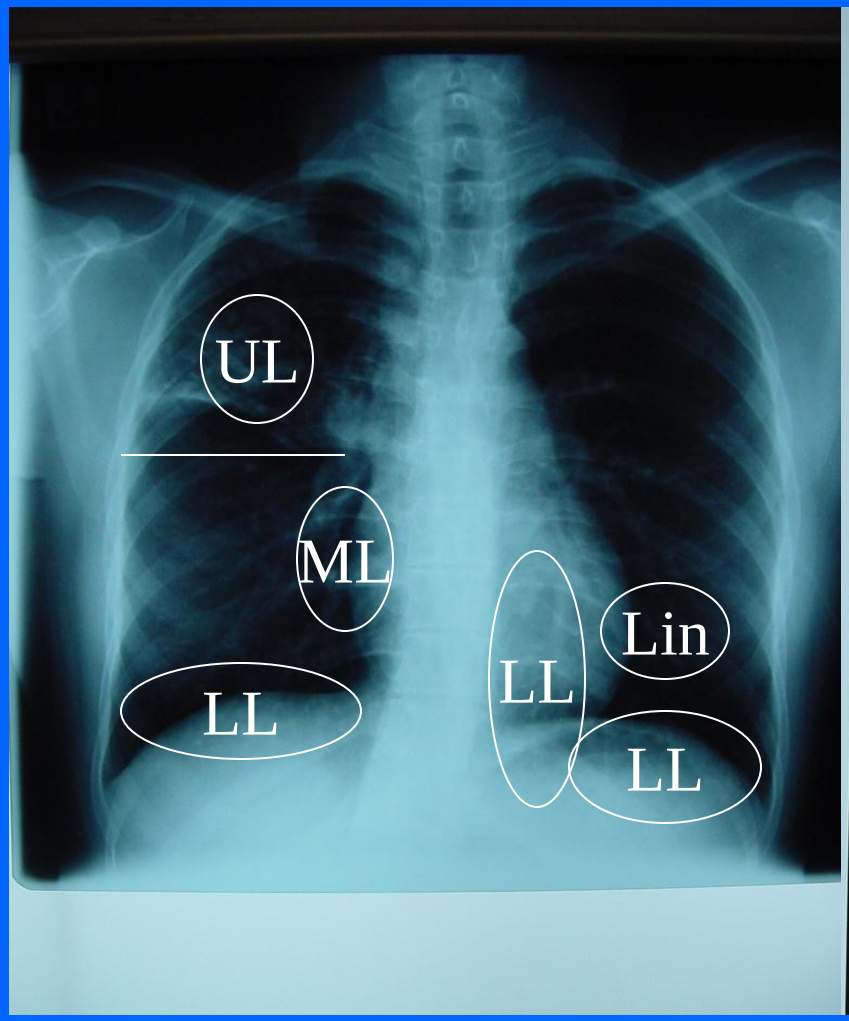
- 30 year old man
- Flu like illness with cough and progressive shortness of breath.
- Now high fever and delerium.
- Normally well smoker.
- No allergies.

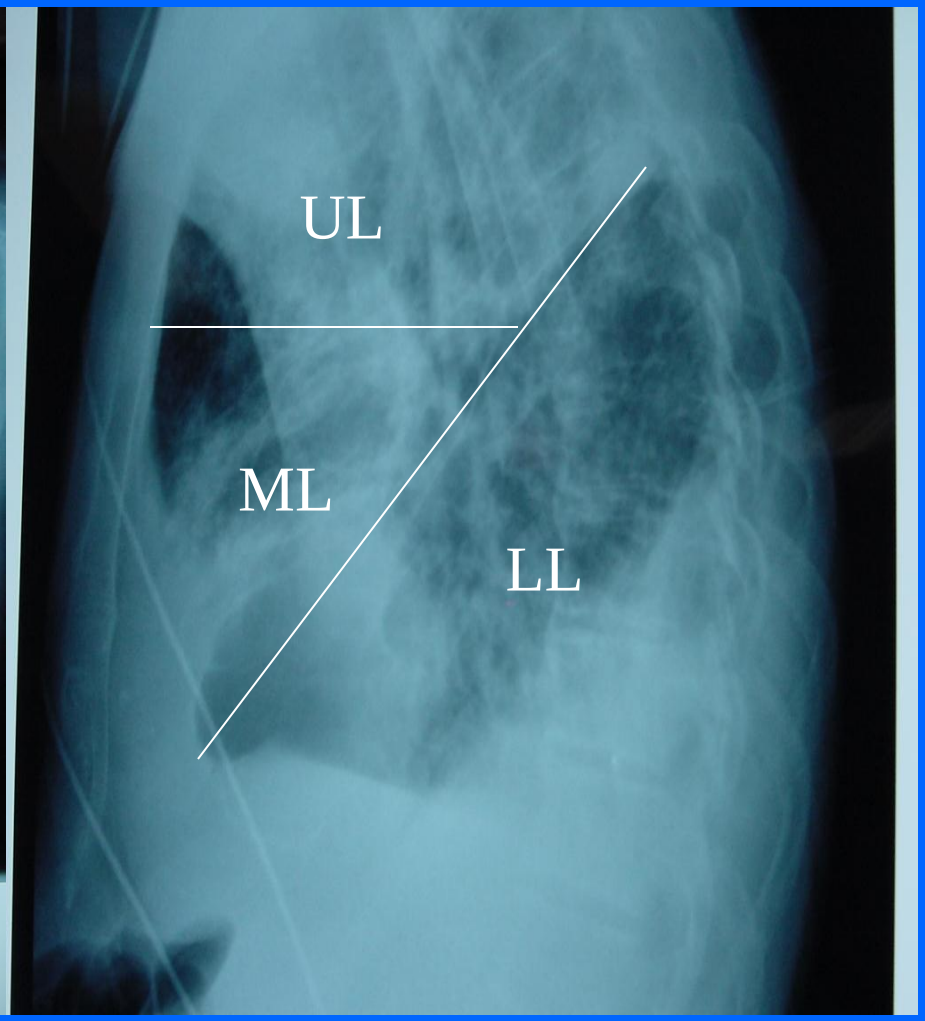
Case 3. Pneumonia

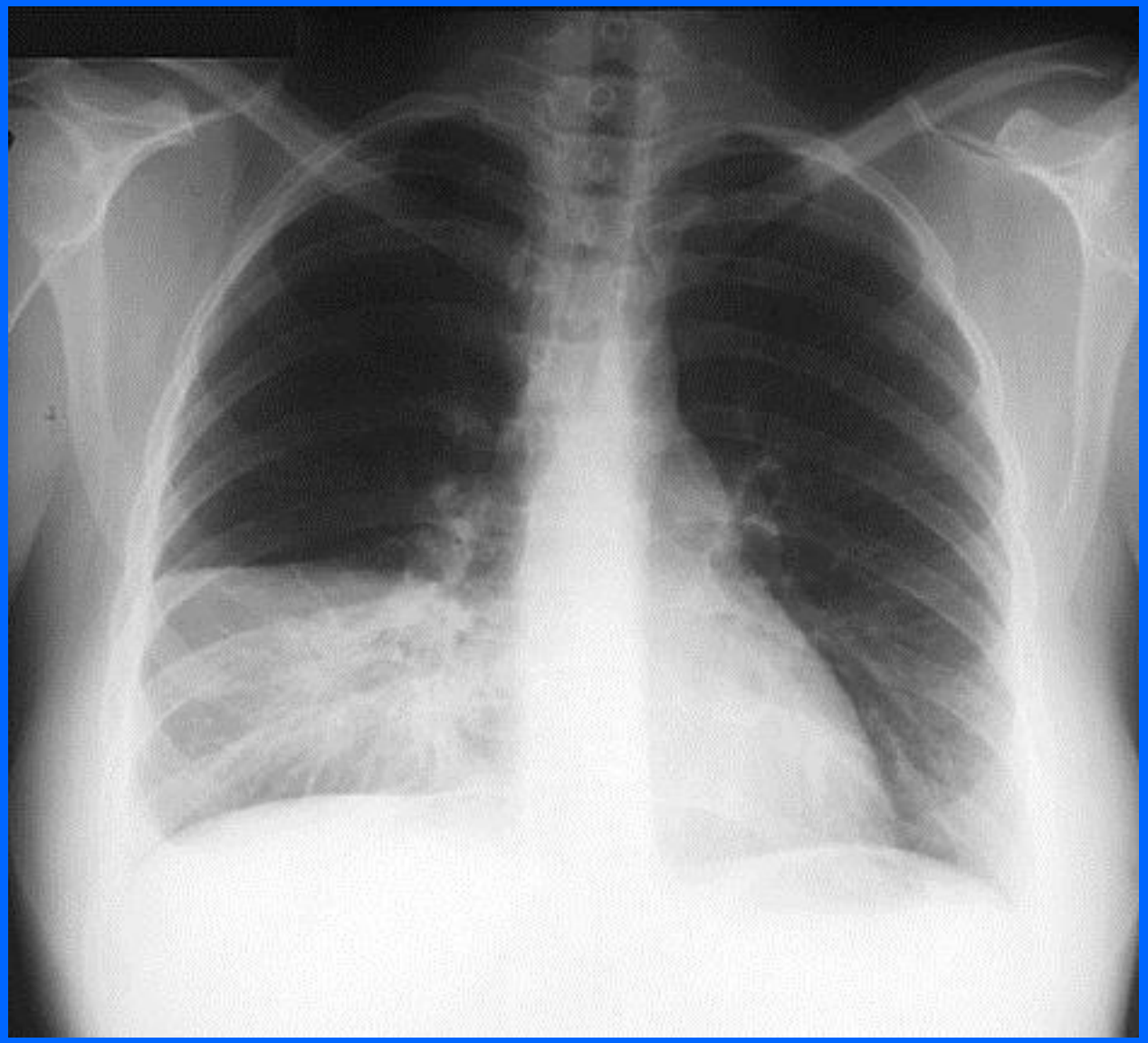
- Looks unwell – delerious, flushed, ‘hyperdynamic’, T=39.5
- RR = 40, with increased work of breathing.
- HR = 120/min, BP =110/50.
- Dullness right base with crackles above (increased vocal fremitus and resonance).

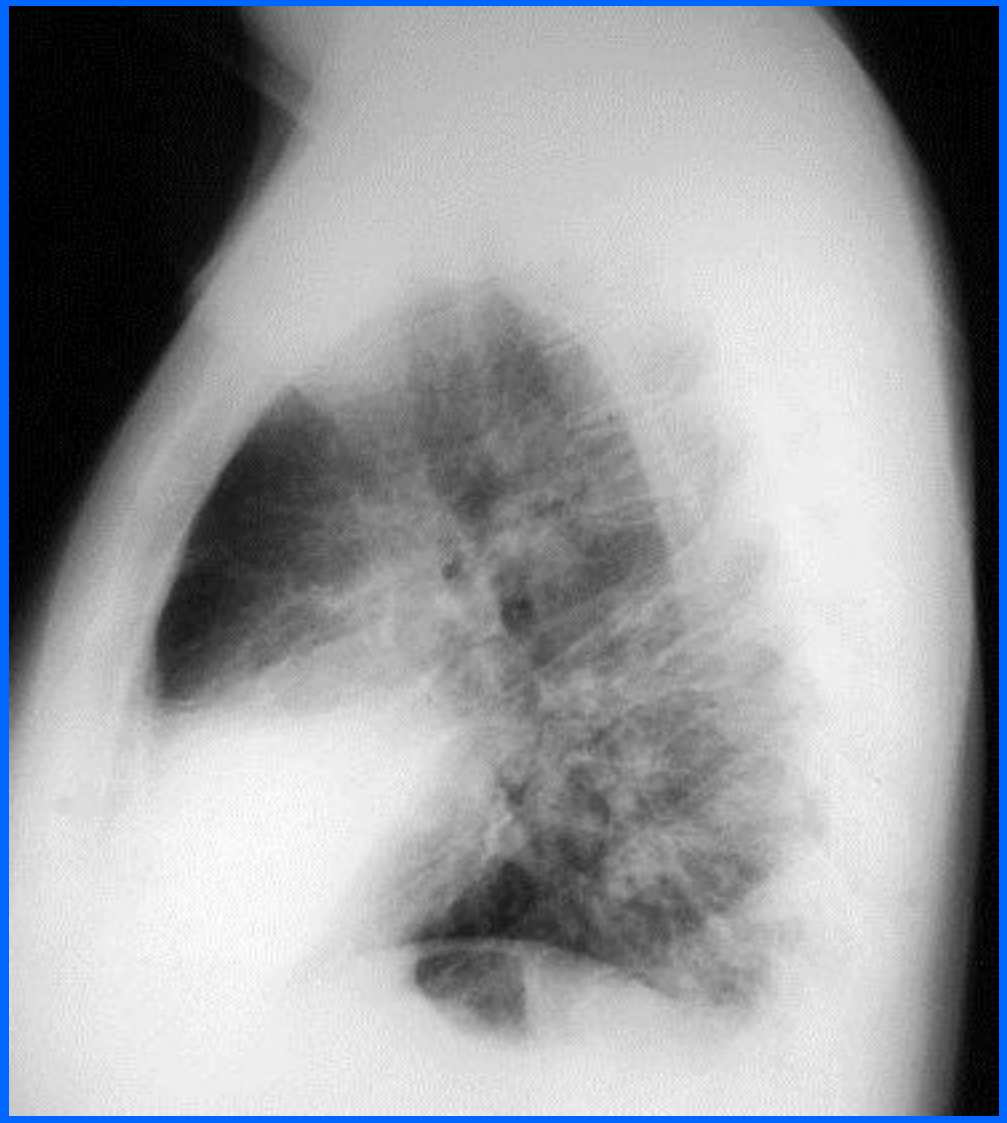


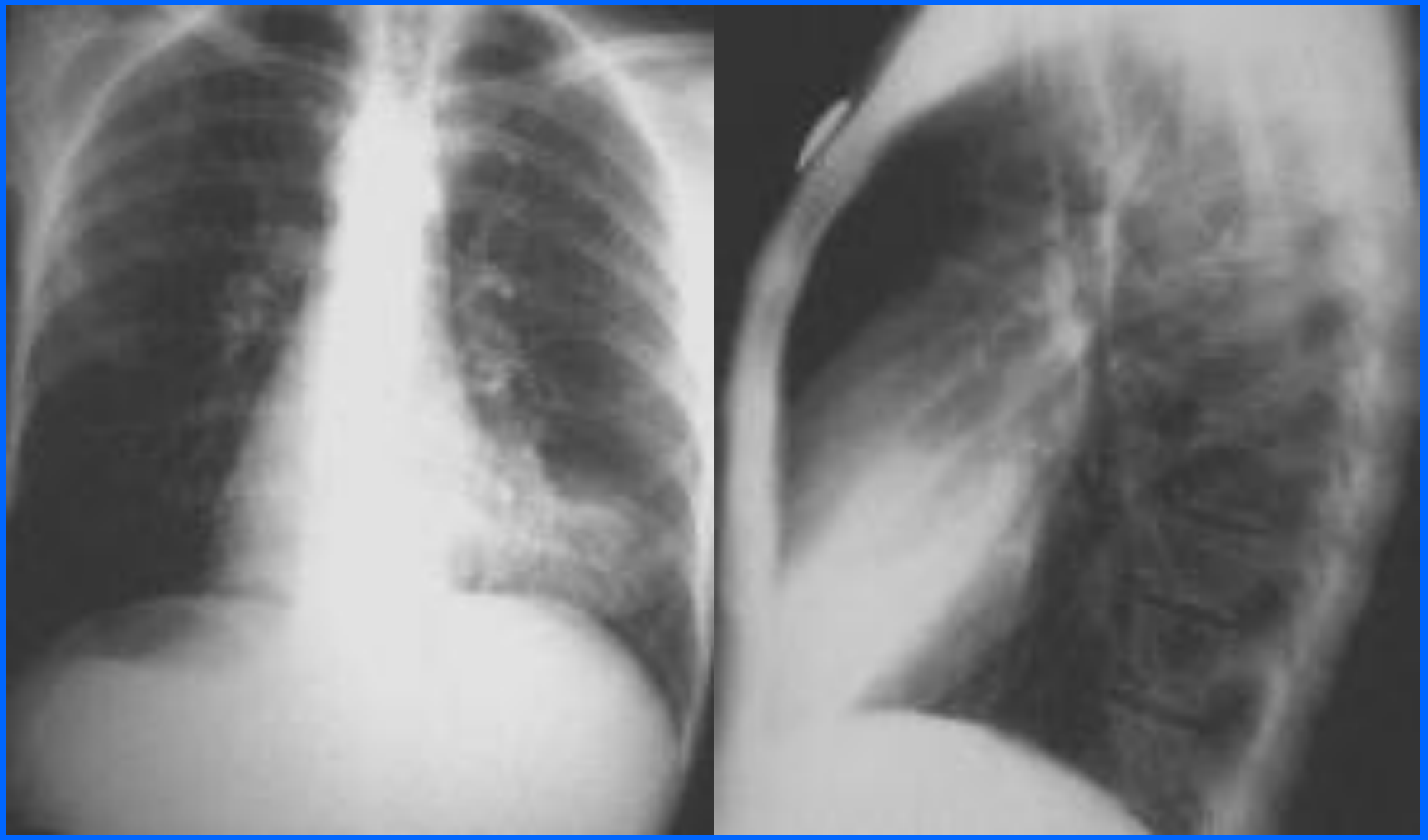
Pneumonia and the CXR

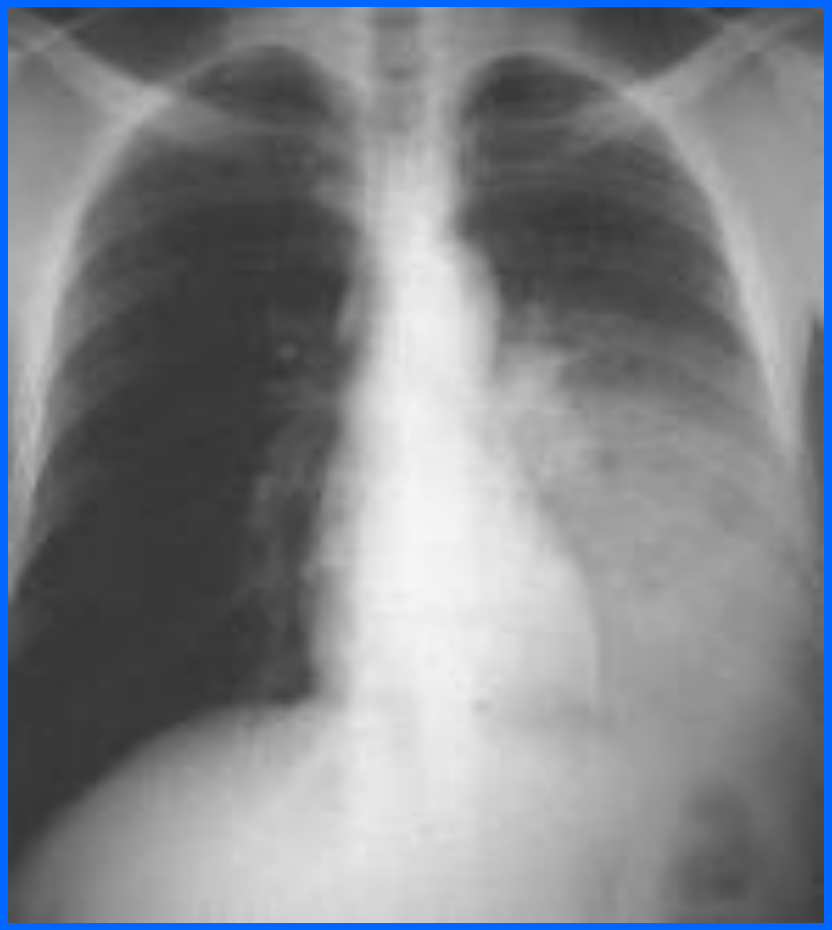


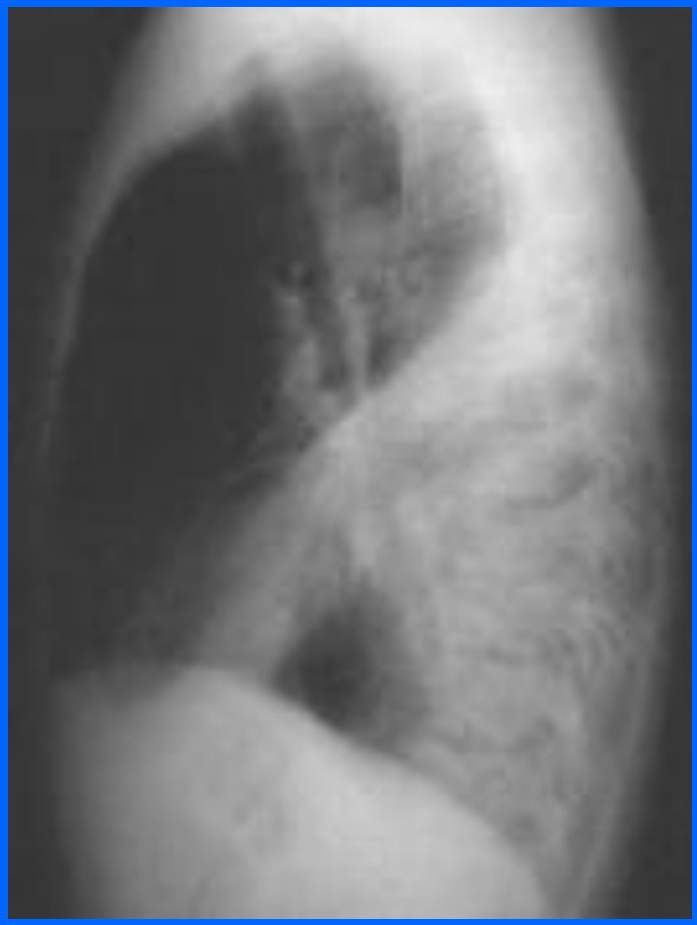


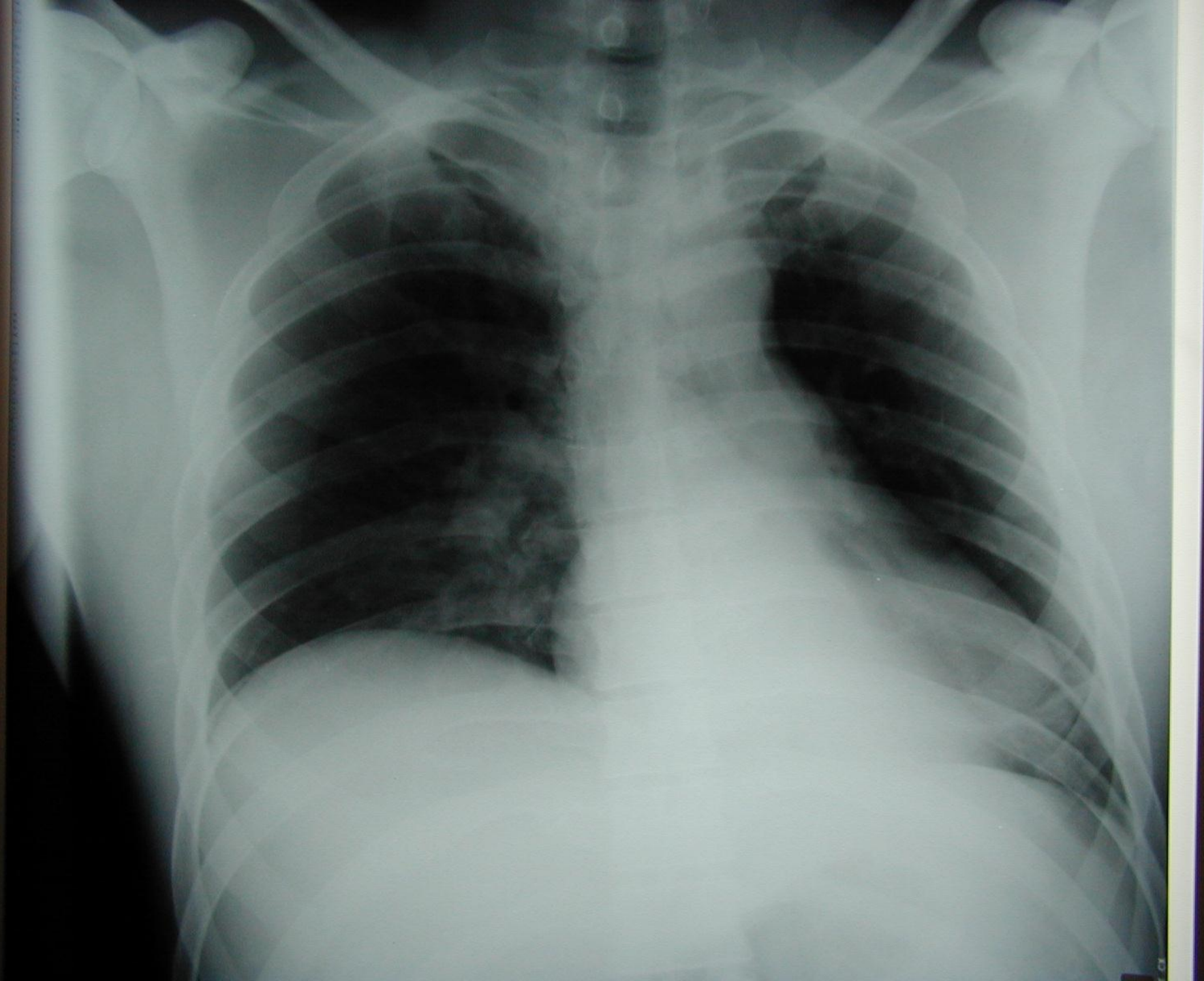


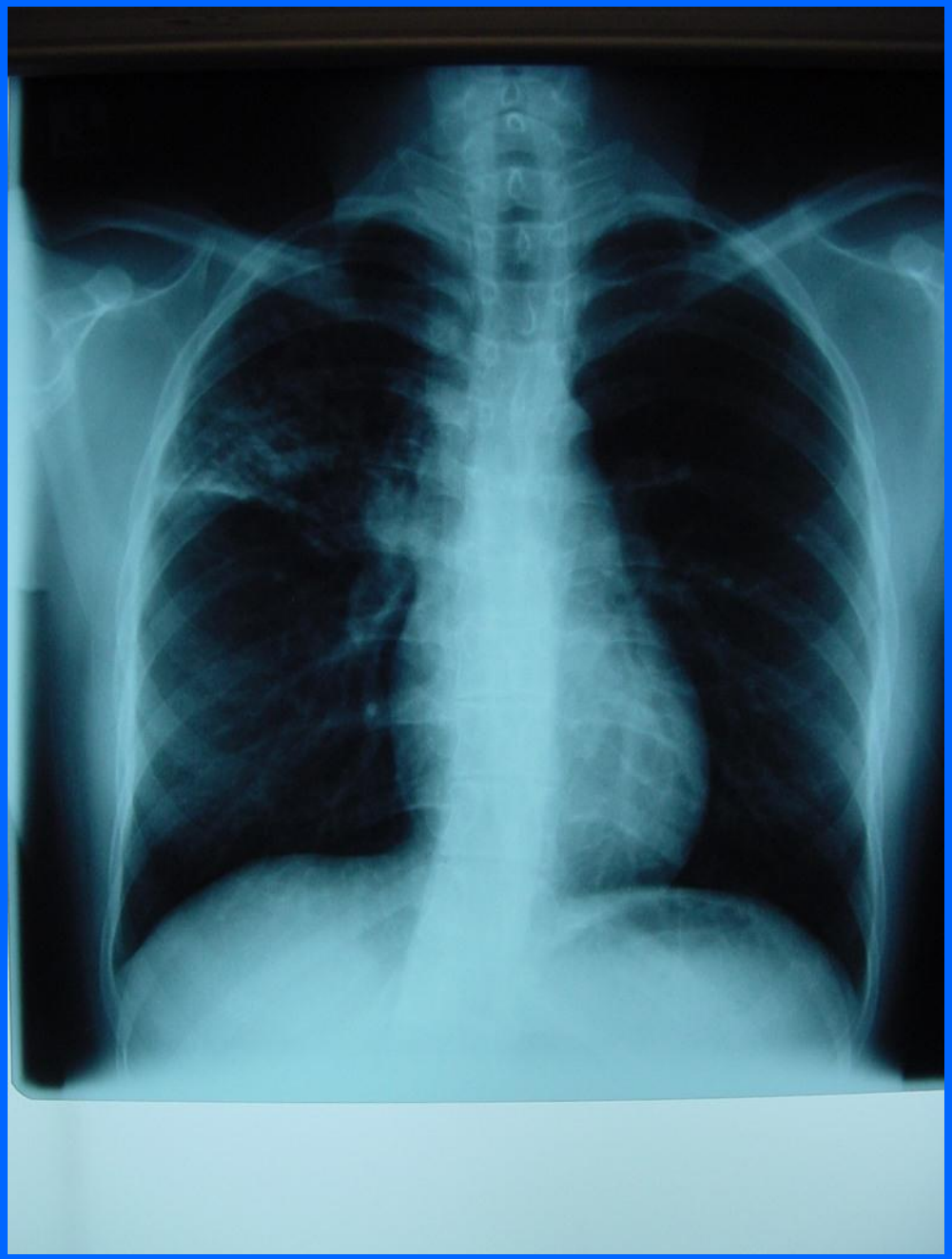


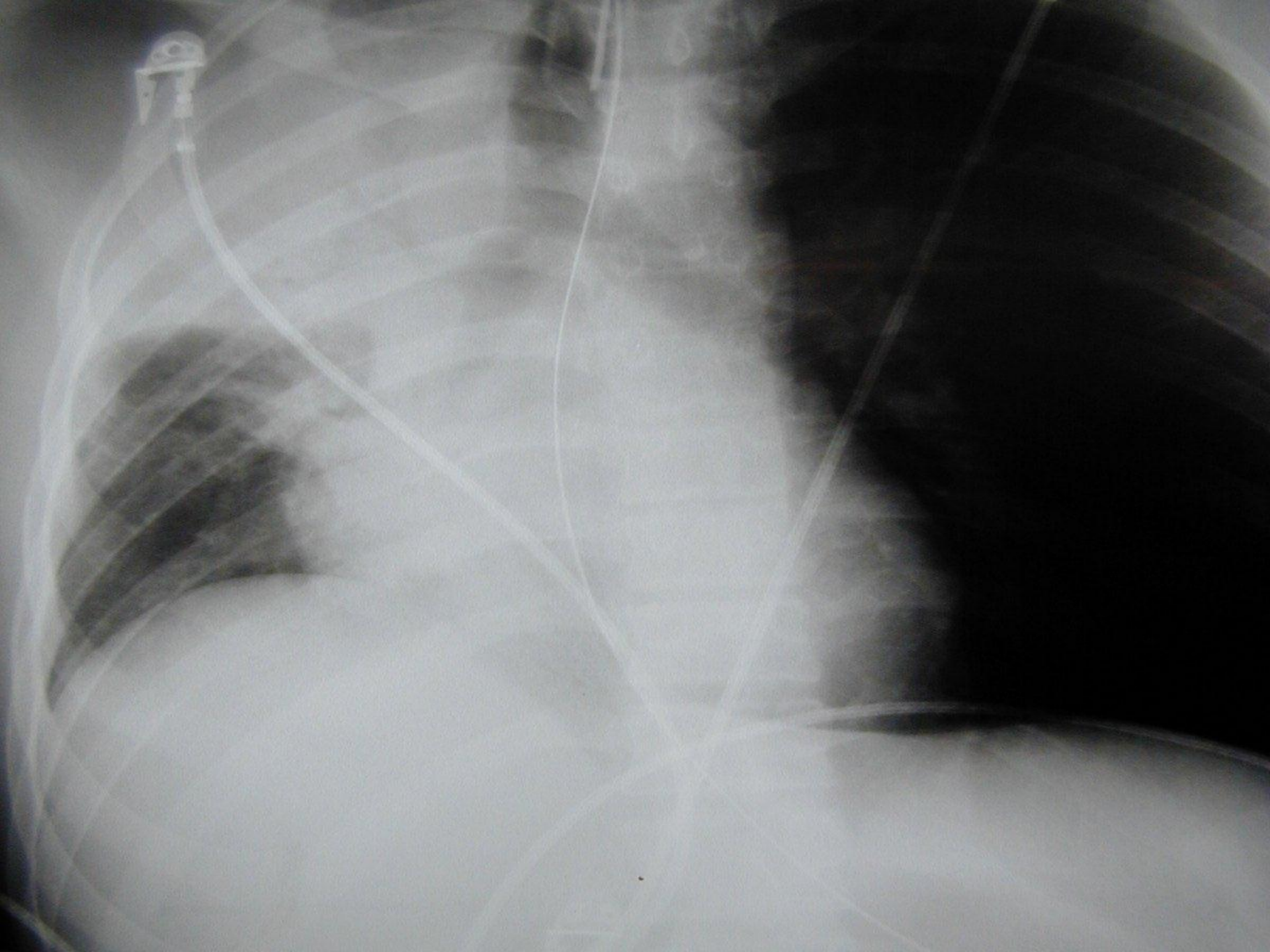












Pneumonia Severity Assessment.

- CURB + Age Score
  - Confusion (MSQ < 8)
  - Urea (>7mmol/l)
  - Resp rate (>30/min)
  - BP (diastolic < 60mmHg or systolic < 90)
  - Age (>65).

Pneumonia Severity Assessment.

- CURB + Age
  - 0-1; mortality < 2%, oral ABs and home
  - 2; mortality 5-10%, IV Abs
  - 3-4; mortality 10-50%, Aggressive treatment

Case 3. Treatment

- Fluids and antibiotics
  - Prognosis is related to time to these treatments.
- Fluids
  - Titrated, but probably a couple of litres of normal saline.
- Antibiotics
  - IV penicillin or augmentin
  - Plus a macrolide
  - Or broader cover if very unwell (Ceftiraxone/gentamicin/clarithromycin)

Respiratory Emergencies

- APO
  - O2, CPAP, Nitrates.
- Asthma
  - O2, β2 agonists, ?adrenaline, ?other stuff.
- Pneumonia
  - CURB + Age score
  - Severe pneumonia treated aggressively.