Respiratory Insufficiency
Respiratory Failure

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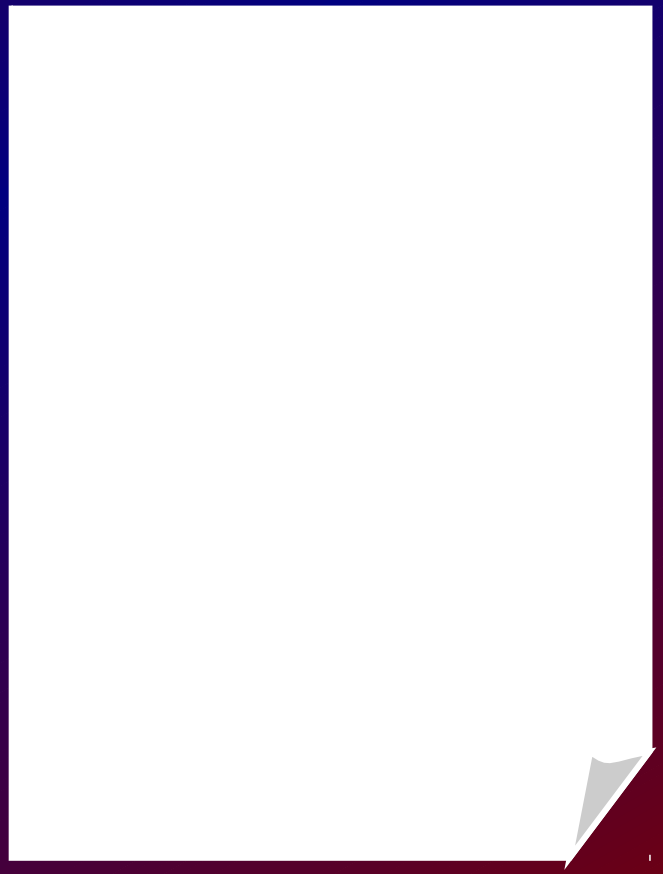
inspired by
www.aic.cuhk.edu.hk

https://emedicine.medscape.com/article/167981-overview

Interpretation of arterial blood gases

- Oxygenation
- Ventilation
- Acid base status



Oxygenation

- What is the P_aO_2?
- Is this is adequate for the amount of inspired oxygen?
- Does the ABG result agree with the saturation probe?

pH

PaCO2

PaO2

HCO3-

Base excess

Saturation

Oxygenation

- Normal P_aO_2 breathing air (FiO_2 = 21%) is 12-13.3 kPa ; small reduction with age
- Lower values constitute hypoxaemia
- P_aO_2 <6.7 kPa on room air = respiratory failure
- P_aO_2 should go up with increasing FiO_2
- A P_aO_2 of 13.3 kPa breathing 60% O_2 is not normal
- You need to know the FiO_2 to interpret the ABG

Oxygenation

- Correlate the ABG result with the saturation probe result
- If there is a discrepancy:
  - Is there a problem with the probe (poor perfusion? etc)
  - Is there a problem with the blood gas (is it a venous sample?)

Oxygenation

- Is the PaO_2 is lower than expected?
- Calculate the A-a gradient to assess if the low PaO_2 is due to:
  - Low alveolar PAO_2
  - Structural lung problems causing failure of oxygen transfer

Oxygenation

The alveolar gas equation:

$$p_AO_2 = F_IO_2(P_{ATM} - pH_2O) - \frac{p_aCO_2(1 - F_IO_2[1 - RQ])}{RQ}$$

$P_AO_2 = [94.8 \times F_IO_2] - [P_aCO_2 \times 1.25]$

The alveolar-arterial oxygen difference

$(A\text{-}a)\ PO_2 = P_AO_2 - P_aO_2$

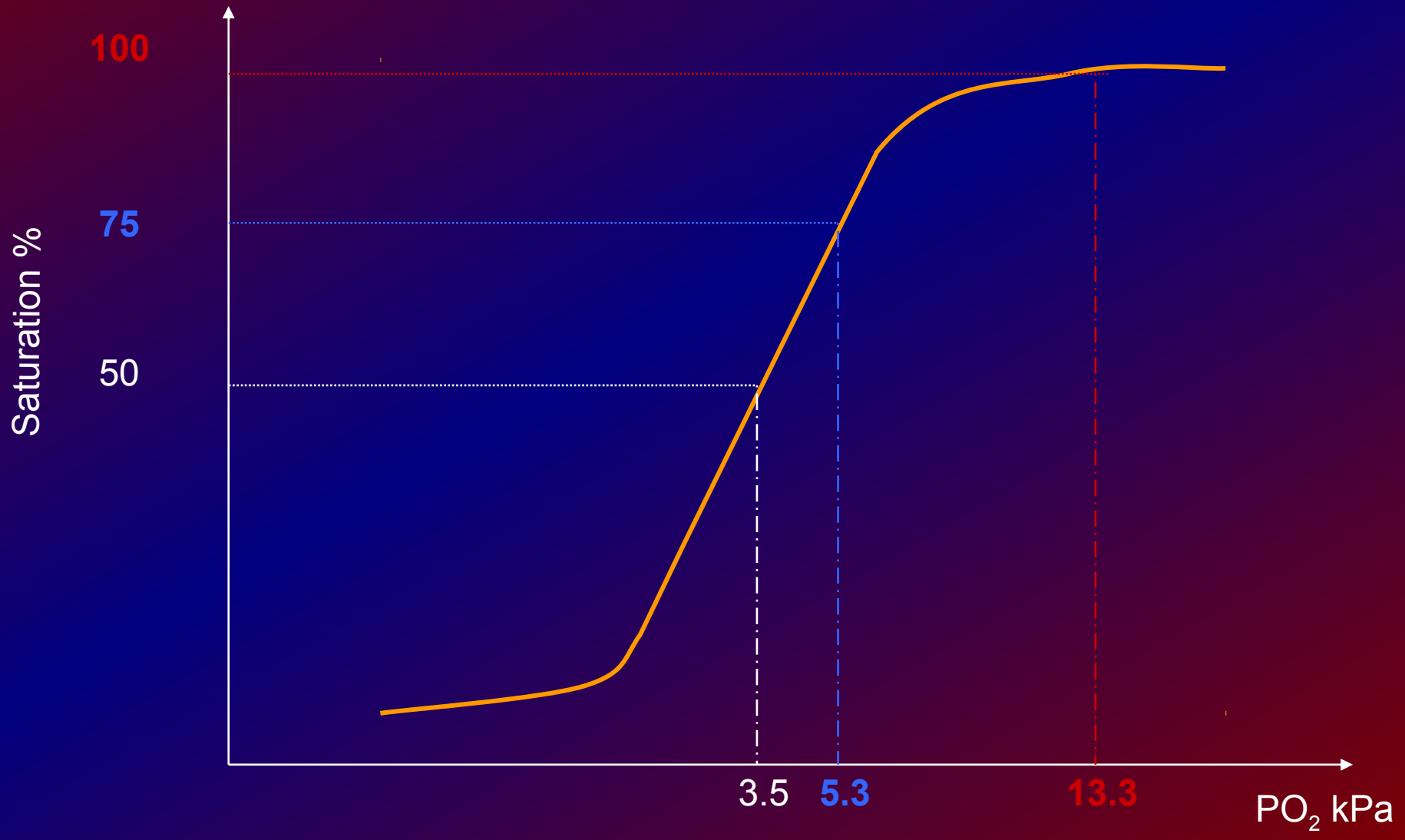
Aa Gradient = [FiO2*(Patm-PH2O)-(PaCO2/0.8)] - PaO2

Aa Gradient = (713 x FiO2) – (pCO2 / 0.8) – (paO2) [mmHg]

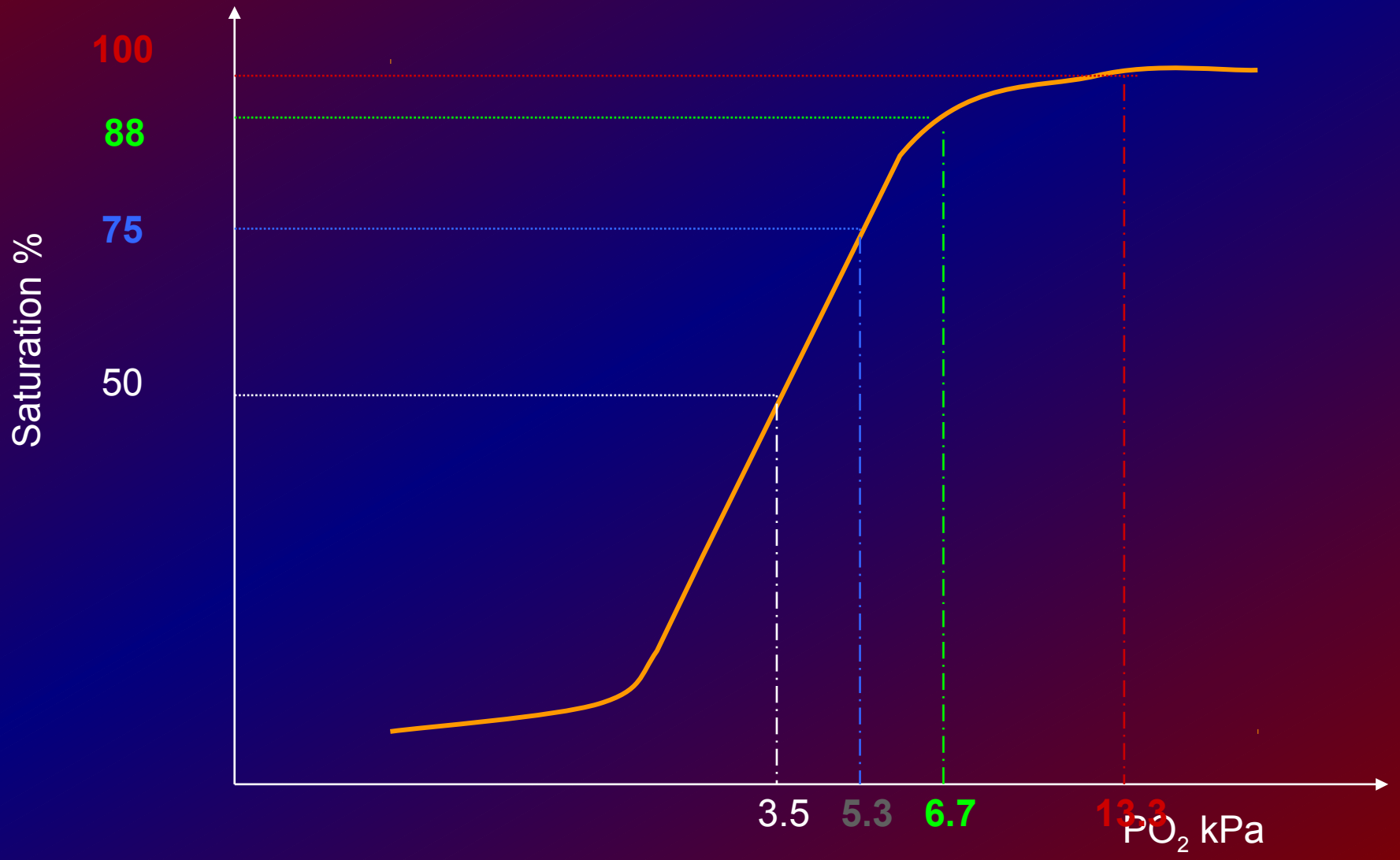
A-a difference

- is less than 10 (15) mmHg. 1,3kPa (2kPa)
- Normally, the A-a gradient increases with age. For every decade a person has lived, their A-a gradient is expected to increase by 1 mmHg.

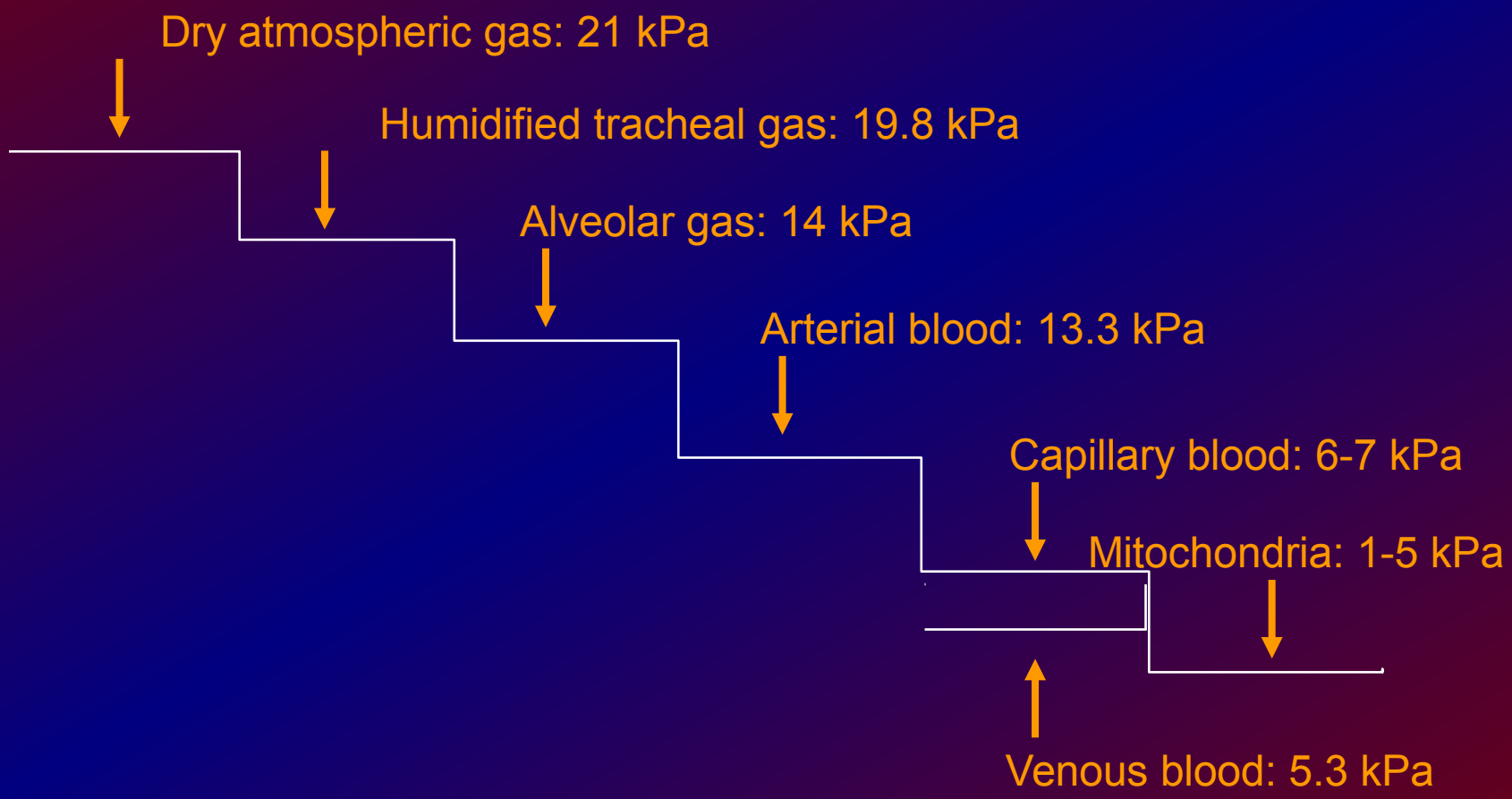
Oxygen dissociation curve



Oxygen dissociation curve



Oxygen cascade



PaCO2 5,3 kPa

- The arterial pCO2 is normally maintained at a level of about 40 mmHg by a balance between production of CO2 by the body and its removal by **alveolar ventilation**. If the inspired gas contains no CO2 then this relationship can be expressed by:
- paCO2 is proportional to VCO2 / VA

where:

- VCO2 is CO2 production by the body
- VA is Alveolar ventilation

An increase in arterial pCO_2

can occur by one of three possible mechanisms:

- Presence of excess CO_2 in the inspired gas
- Decreased alveolar ventilation
- Increased production of CO_2 by the body

Respiratory Insuf. = Resp.Failure Definition

- The condition in which the lungs cannot take in sufficient oxygen and/or expell sufficient carbon dioxide to meet the needs of the cells of the body.
- Also called pulmonary insufficiency, respiratory failure.
- In practice, respiratory failure is defined as a PaO2 value of less than 60 mmHg (8kPa) while breathing air or a PaCO2 of more than 50 mmHg (6.6kPa).
- Need of arterial blood gasses

RF Classification

- Acute / Chronic
- hypoxemic or hypercapnic
- Acute hypercapnic respiratory failure develops over minutes to hours; therefore, pH is less than 7.3.
  Chronic respiratory failure develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH usually is only slightly decreased.
- The distinction between acute and chronic hypoxemic respiratory failure cannot readily be made on the basis of arterial blood gases. The clinical markers of chronic hypoxemia, such as polycythemia or cor pulmonale, suggest a long-standing disorder.

ARF

- **ARF is not a specific disease but a reaction to an underlying condition**, e.g. trauma, sepsis or pneumonia.
- Due to different definitions, the incidence and mortality rates for ARF vary across studies.
- In addition, the underlying condition strongly influences prognosis.

Respiratory failure

- Acute respiratory failure (ARF) is a common and important indication for critical care with a substantial mortality.
- It is defined as all acute lung conditions with the exception of chornic obstructive lung disease that require active therapy.

Pump failure or lung failure ?

- The respiratory system can be modelled as a gas exchanger (the lungs) ventilated by a pump. The dysfunction of each of the two parts, pump or lungs, may cause respiratory failure defined as inability to maintain adequate blood gases while breathing ambient air.

Pump failure

- Pump failure primarily results in alveolar hypoventilation, hypercapnia and respiratory acidosis.

Pump failure

- Insufficient alveolar ventilation may result from a number of causes intrinsically affecting one or more of the elements of the complex chain that starts from:
  - the **respiratory centres** (pump controller)
  - **central and peripheral nervous ways**
  - **chest wall**, the latter including both the respiratory muscles and all the passive parts that couple the muscles with the lungs.

Pump failure

- Insufficient alveolar ventilation may even take place in the absence of any intrinsic problem of the pump, when a high ventilation load overcomes the natural capacity of the pump.
- Excessive load can be caused by airway obstruction, respiratory system stiffening or high ventilation requirement, and finally results in intrinsic pump dysfunction due to respiratory muscle fatigue.

Lung failure

- Lung failure results from any damage of the natural gas exchanger: alveoli, airways and vessels.
- Lung failure involves impaired oxygenation and impaired CO2 elimination depending on a variable combination of:
  - True intrapulmonary shunt
  - Increased alveolar dead space
- Lung damage also involves increased ventilation requirement and mechanical dysfunctions resulting in high impedance to ventilation.

Cause

- intoxikation, cerbral insult
- sy Guillain Barré, trauma, poliomyelitis
- myastenia, neuritis, tetanus, botulism
- PNO, haemothorax
- upper airway obstruction
- asthma, COPD, bronchiolitis, fibrosis, ARDS, aspiration
- cardiology

Pathophysiologic causes of acute respiratory failure

- Hypoventilation,
- V/Q mismatch,
- shunt

Hypoventilation

- usually occurs from depression of the CNS from drugs or neuromuscular diseases affecting respiratory muscles. Hypoventilation is characterized by hypercapnia and hypoxemia.

V/Q mismatch

- most common cause of hypoxemia. V/Q units may vary from low to high ratios in the presence of a disease process.
- The low V/Q units contribute to hypoxemia and hypercapnia
- in contrast to high V/Q units, which waste ventilation but do not affect gas exchange unless quite severe.
- The low V/Q ratio may occur either from a decrease in ventilation secondary to airway or interstitial lung disease or from overperfusion in the presence of normal ventilation. The overperfusion may occur in case of pulmonary embolism, where the blood is diverted to normally ventilated units from regions of lungs that have blood flow obstruction secondary to embolism. **Administration of 100% oxygen eliminates all** of the low V/Q units, thus leading to correction of hypoxemia. Hypoxemia increases minute ventilation by chemoreceptor stimulation, but the $PaCO_2$ level generally is not affected.
- PEEP is less toxic than 100% O_2

Shunt

- is defined as the persistence of hypoxemia despite 100% oxygen inhalation. The deoxygenated blood (mixed venous blood) bypasses the ventilated alveoli and mixes with oxygenated blood that has flowed through the ventilated alveoli, consequently leading to a reduction in arterial blood content. The shunt is calculated by the following equation:
- $QS/QT = (CCO2 – CaO2)/CCO2 – CvO2)$

Symptoms:

- Fatigue ... Exercise intolerance
- Dyspnea = an uncomfortable sensation of breathing, shortness of breath
- Cyanosis, a bluish color of skin and mucous membranes, indicates hypoxemia. Visible cyanosis typically is present when the concentration of deoxygenated hemoglobin in the capillaries or tissues is at least 5 g/dL.
- Heavy breathing
- Rapid / slow breathing
- confusion and somnolence .. coma

Imaging Studies:

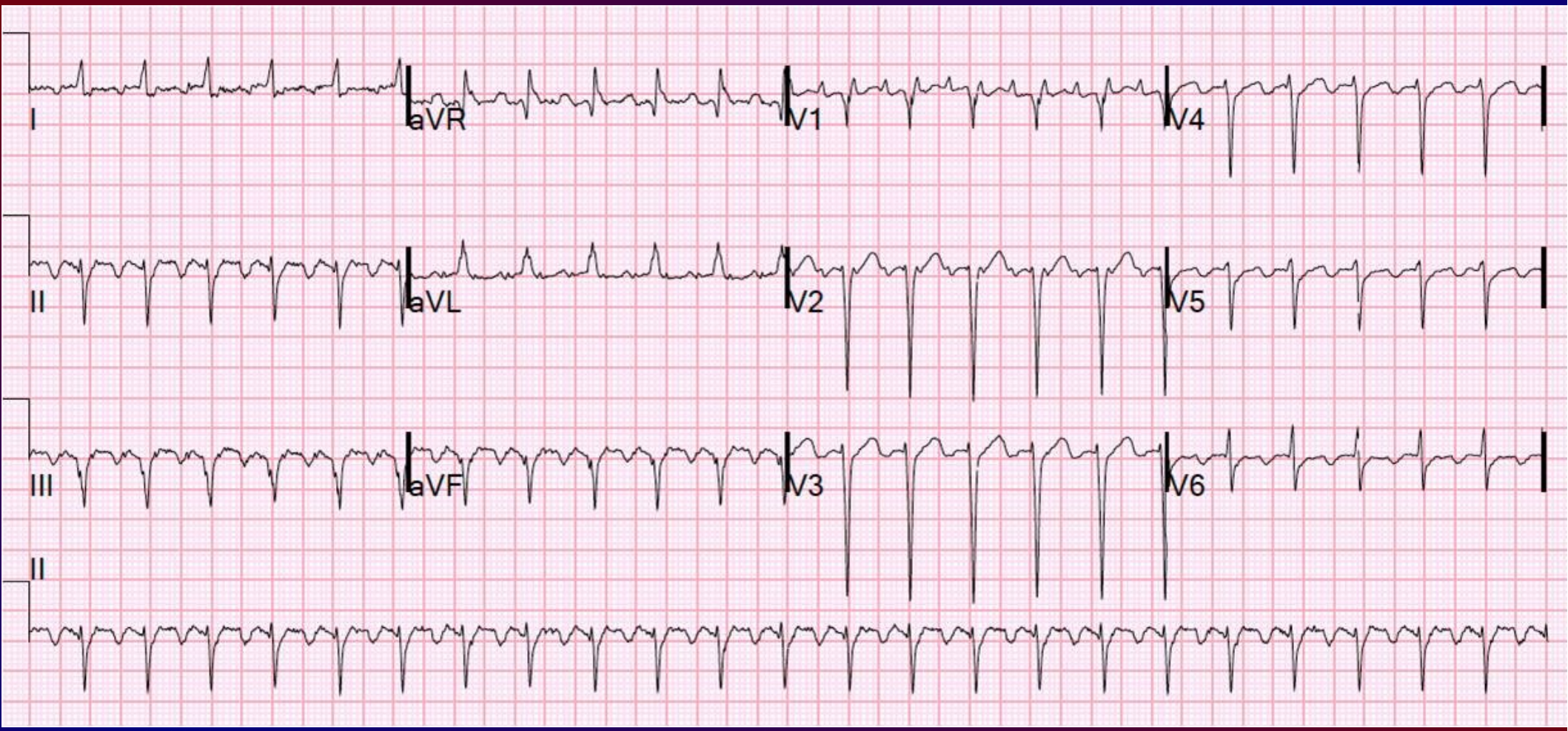
- Chest radiograph

Chest radiography is essential because it frequently reveals the cause of respiratory failure. However, distinguishing between cardiogenic and noncardiogenic **pulmonary edema** often is difficult.

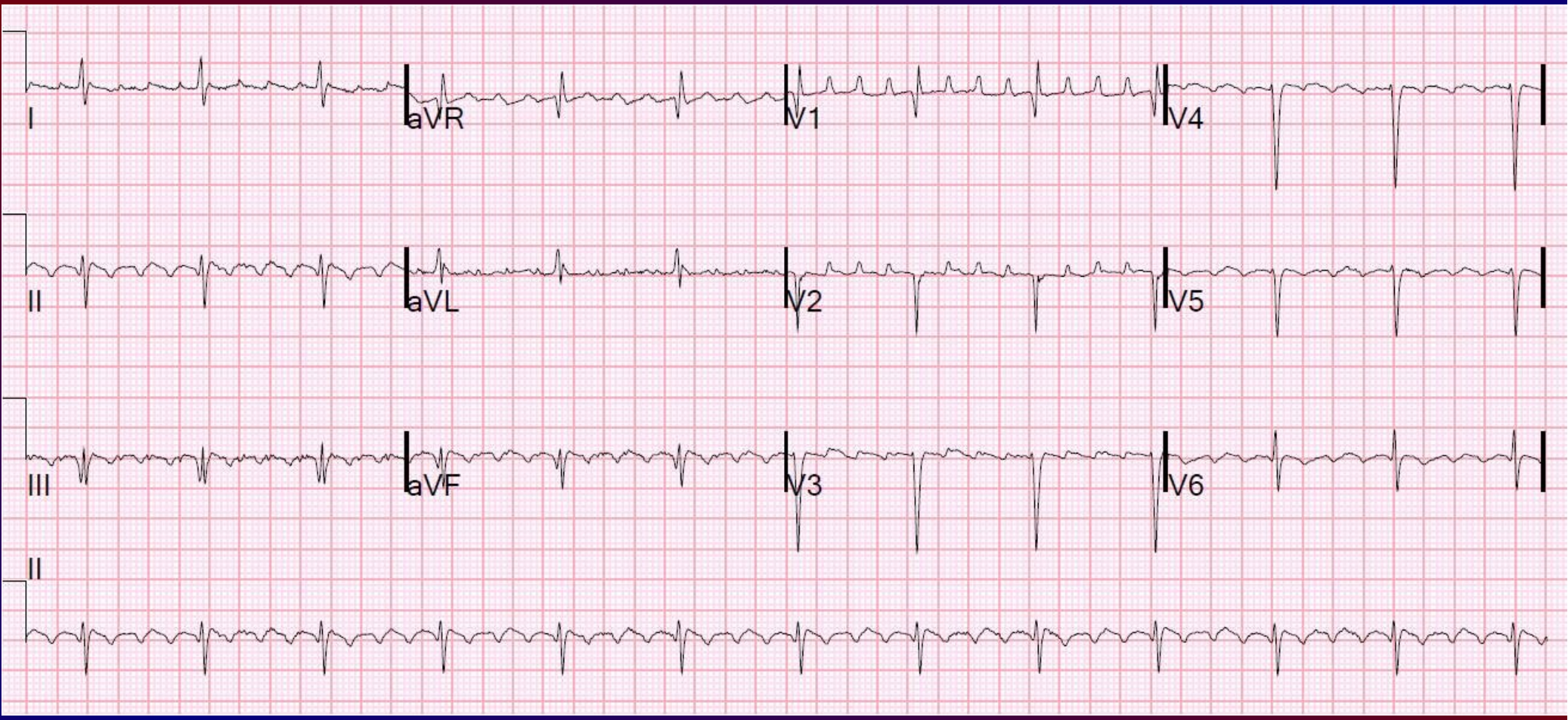
heart size, vascular redistribution, peribronchial cuffing, pleural effusions, septal lines, and perihilar bat-wing distribution of infiltrates suggest hydrostatic edema; the lack of these findings suggests acute respiratory distress syndrome (ARDS).

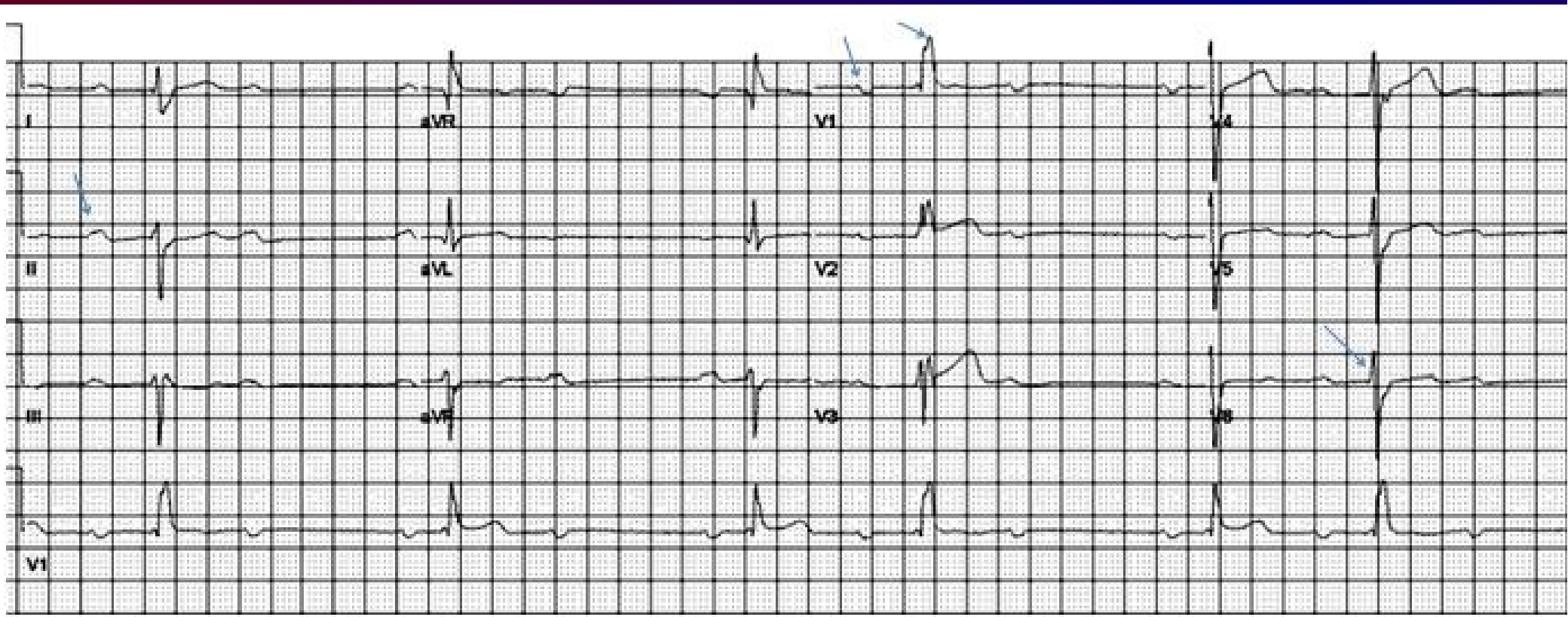
ECG

- supra ventricular tachycardia
- MI
- Cor Pulmonale


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

after diltiazem




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

Cor Pulmonale

- right ventricular hypertrophy (RVH)
- Right axis deviation
- R/S amplitude ratio in V1 greater than 1 (an increase in anteriorly directed forces may be a sign of posterior infarction)
- R/S amplitude ratio in V6 less than 1
- P-pulmonale pattern (an increase in P wave amplitude in leads 2, 3, and aVF)
- S1 Q3 T3 pattern and incomplete (or complete) right bundle branch block, especially if pulmonary embolism is the underlying etiology
- Low-voltage QRS because of underlying COPD with hyperinflation

Echocardiography

- is a useful test when a cardiac cause of acute respiratory failure is suspected.
- (The findings of left ventricular dilatation, regional or global wall motion abnormalities, or severe mitral regurgitation support the diagnosis of cardiogenic pulmonary edema.)

Pulmonary Function Tests

Normal values of forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) suggest a disturbance in respiratory control.

- A decrease in FEV1 -to-FVC ratio indicates airflow obstruction, whereas a reduction in both the FEV1 and FVC and maintenance of the FEV1 -to-FVC ratio suggest restrictive lung disease.
- Respiratory failure is uncommon in obstructive diseases when the FEV1 is greater than 1 L and in restrictive diseases when the FVC is more than 1 L.

Therapy:

The goal:

- PaO2 of 60 mmHg or an arterial oxygen saturation (SaO2) of greater than 90%.
- Experts believe that hypercapnia should be tolerated until the arterial blood pH falls below 7.2

O2: [nasal, mask, m+ reservoar] fiO2 up to 40%

Arteficial Ventilation

- NonInvasive / Invasive

ECMO

Case #1:

- 70 y.o. male has been treated for chronic obstructive pulmonary disease (COPD) for 14 years.
- Now he is being treated for exacerbation at internal department. Despite full therapy, his status is worsening progressively: he is in increasing respiratory distress and his consciousness worsens.

#1 COPD acute exacerbation

- You find the patient on a standard ward, hardly breathing O2 via facemask (15l/min, FiO2 1,0), unresponsible, on painful stimulus he distracts arm and opens his eyes. BP 170/70, TF 130/min, sat 99%, RR 12/min.

What next??

- laboratory??

#1 COPD acute exacerbation

Arterial blood gases: pH=7.11, pO_2=16kPa, pCO_2=14 kPa, BE=+16 mM

Determine the type of acid-base disorder, incl. compenstation if present.

What is the cause of hypercapnia?

Describe the regulation of minute ventilation during both normal status and chronic hypercapnia.

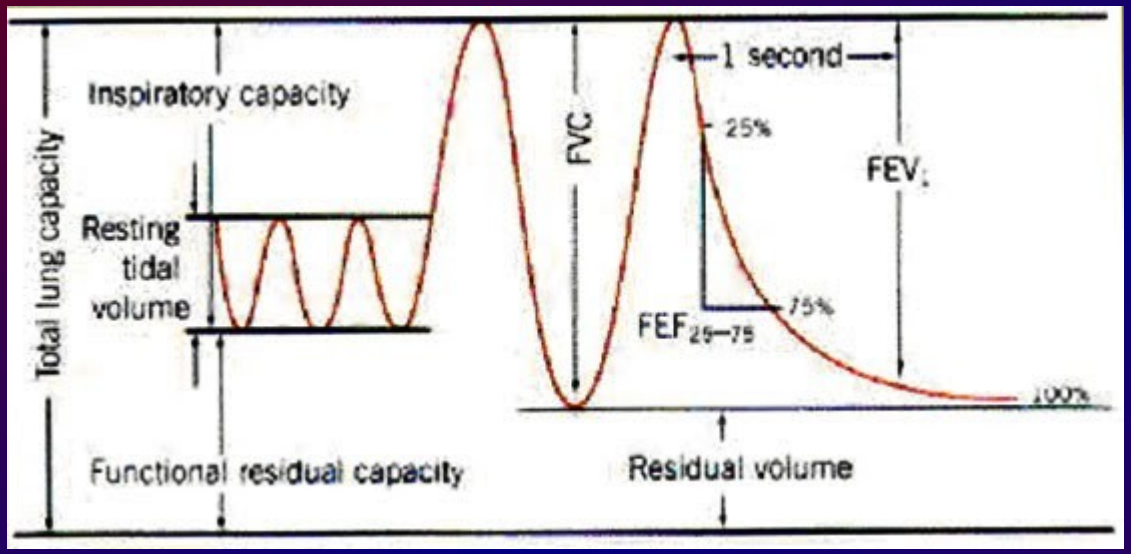
What was wrong in the treatment of this patient?

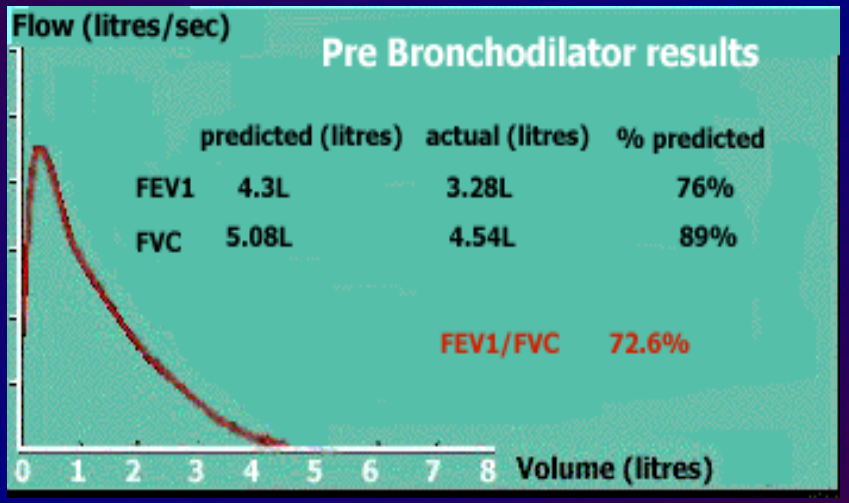
#1 COPD acute exacerbation

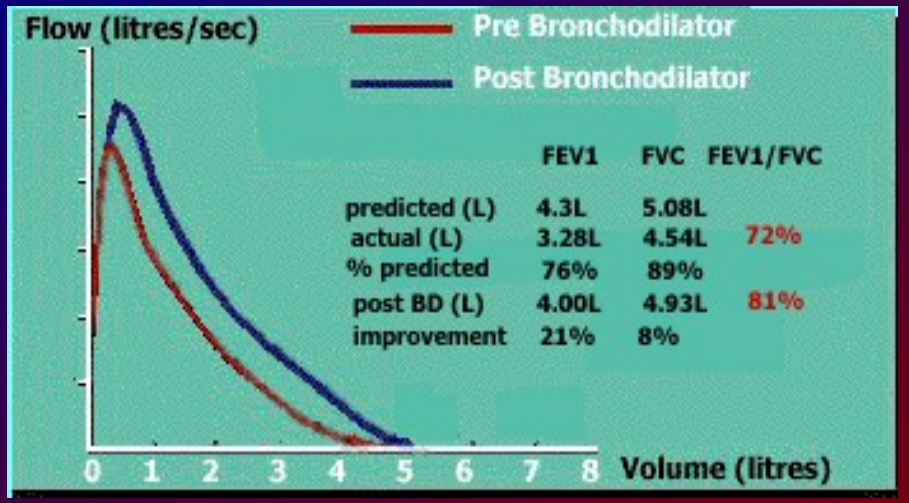
Arterial blood gases: pH=7.11, pO_2=16kPa, pCO_2=14 kPa, BE=+16 mM

Describe typical physical findings on a chest of patient with bronchial obstruction.

Describe also typical spirometry finding in bronchial obstruction. What is FEV1/FVC?







http://www.worldcme.com/webpages/members_only/Asthma/case5/c5post.htm

OTI ??

Should this patient be intubated and mechanically ventilated?

Determine Glasgow Coma Score of this patient.

Ethical aspects

- If this patient had expressed the wish not to be intubated before this episode, would you accept it and provide sympthomatic treatment only (i.e. morphin, fluids)?
- Explain terms witholding and withdrawing treatment.

Pathophysiology: obstructive and restrictive disorders of ventilation, incl. spirometric findings

Pathology: COPD, chronic bronchitis, emphysema

Example 2.

A 33 male patient with ARDS has a saturation of 91% on Fi02 0.4

- Is he hypoxic?
- Is there an acid base or ventilation problem?

pH	7.43
PaCO2	4.76
PaO2	8.1
HCO3-	23
Base excess	-0.6
Saturation	90%

Is he hypoxic?

YES.

The SpO2 and calculated saturation agree

pH	7.43
PaCO2	4.76
PaO2	8.1
HCO3-	23
Base excess	-0.6
Saturation	90%

Is he hypoxic?

YES.

(A-a) PO2 = 23.9 kPa

There is major problem with oxygen transfer into the lung

pH	7.43
PaCO2	4.76
PaO2	8.1
HCO3-	23
Base excess	-0.6
Saturation	90%

Is there an acid base or ventilation problem?

pH	7.43
PaCO2	4.76
PaO2	8.1
PaCO2	23
Base excess	-0.6
Saturation	90%

Is there an acid base or ventilation problem?

NO.

pH, PaCO2 and PaCO2 are normal

This is pure hypoxaemic respiratory failure

pH	7.43
PaCO2	4.76
PaO2	8.1
PaCO2	23
Base excess	-0.6
Saturation	90%

Example 9.

Is he hypoxic?

ppH	7.23
PPaCO2	3.3
PPaO2	10.6
HHCO3-	8
BBase excess	-10
SSaturation	96%

Example 9.

Is he hypoxic?

NO. This is a normal PaO2 for a patient this age breathing room air

ppH	7.23
PPaCO2	3.3
PPaO2	10.6
HHCO3-	8
BBase excess	-10
SSaturation	96%

Emergency department :

- consciousness,
- sitting, haevy breathing, sweating.
- What to do?

Examination>

Airway

Breathing

Circulation

Disability

Electrolytes

Fluids

Gut

Hematology

Infection

Lines

Med, Nutrition, ...

A

B

C

D

...

Problem:

Plan:

Physical Examination>

Airway

Breathing

Ciruculation

Disability

Electrolytes

Fluids

Gut

Hematology

Infection

… today topic

A open, clear, cough / upper airway obstruction, OTI, TS
O2 mask

B Lung Auscultation – vesicular / wheezes tubular / gurgling...

SpO2, Ventilation

...

Problem: Respiratory Failure

Plan: O2 mask / NIV / OTI

treat the cause

Questions: Essential:

- Problem>
- Patient history

monitoring:

- SpO2, ECG, NIBP

O2 mask

Asthma

A unable to converse, accessory muscles of respiration are used, sitting position

B tachypneic, Loud expiratory wheezing, SpO2 92 on air

C s.r. 100/min, pulsus paradoxus

D ...

Problem:

Plan:

Asthma

A unable to converse, accessory muscles of respiration are used, sitting position

B tachypneic, Loud expiratory wheezing, SpO2 92 on air

C s.r. 100/min, pulsus paradoxus

D ...

Problem: Moderate asthma

Plan:

Asthma

A unable to converse, accessory muscles of respiration are used, sitting position

B tachypneic, Loud expiratory wheezing, SpO2 92 on air

C s.r. 100/min, pulsus paradoxus

Problem: Moderate asthma

Plan:

SABA á 20min, á 3h + corticoid (p.os)

Asthma 2

A sitting, suprasternal retractions;

B breathless during rest; Loud biphasic (expiratory and inspiratory) wheezing SpO2 85 on Air

C s.r. 130/min, hypertension, sweating

D confused...

Problem:

Plan:

Asthma 2

A sitting, suprasternal retractions;

B breathless during rest; Loud biphasic (expiratory and inspiratory) wheezing SpO2 85 on Air

C s.r. 130/min, hypertension, sweating

D confused...

Problem: Life-Threatening Asthma

Plan:

Astma 2

A sitting, suprasternal retractions;

B breathless during rest; Loud biphasic (expiratory and inspiratory) wheezing SpO2 85 on Air

C s.r. 130/min, hypertension, diaphoresis = sweating

D confused...

Problem: Life-Threatening Asthma

Plan: OTI + sedation, vent.

- SABA á 20min, á 3h + corticoid i.v.

Asthma

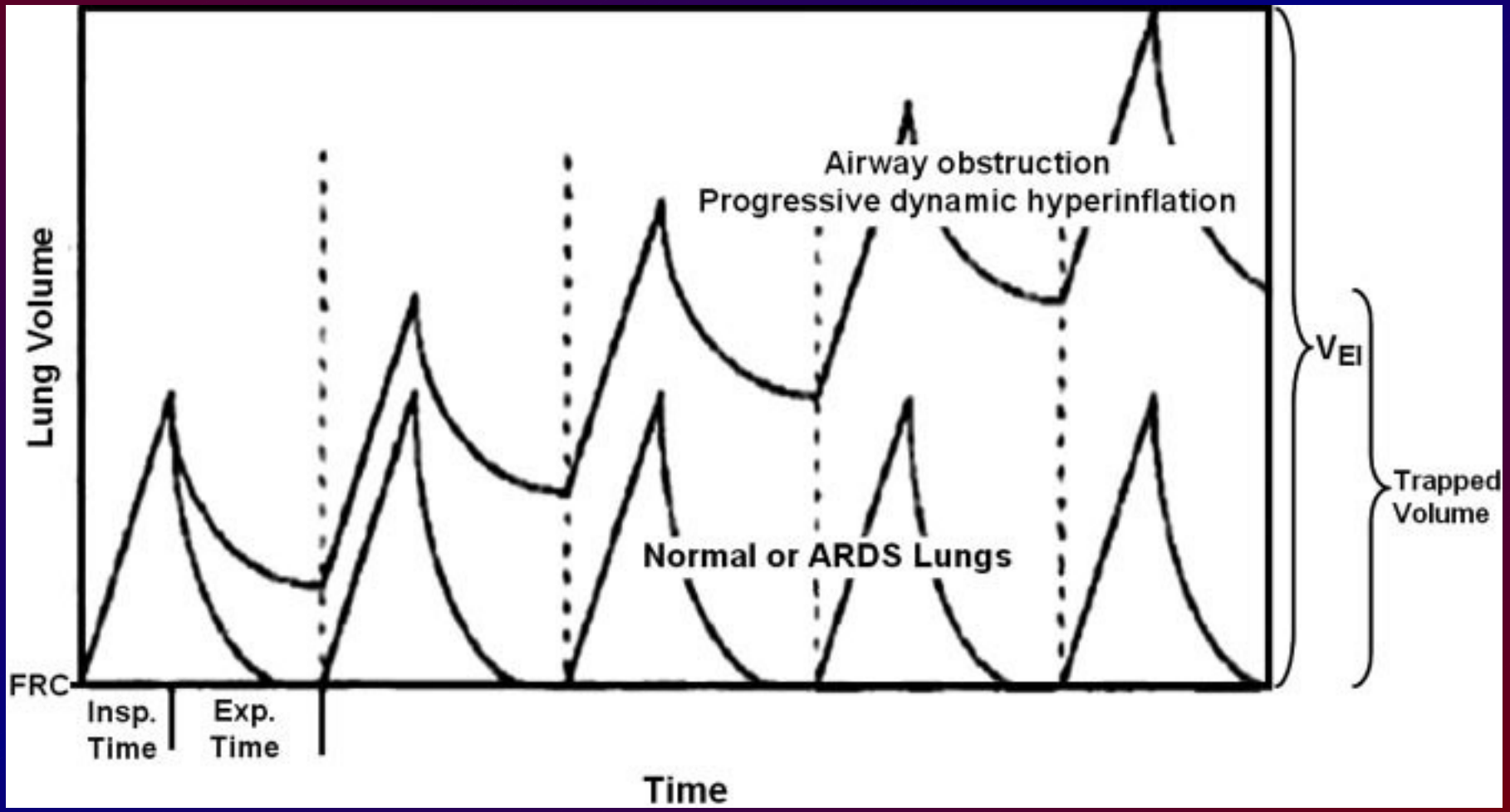
- Do NOT administer metylxantins (tachykardia)

Table 2. Drug Dosages for Severe Acute Asthma

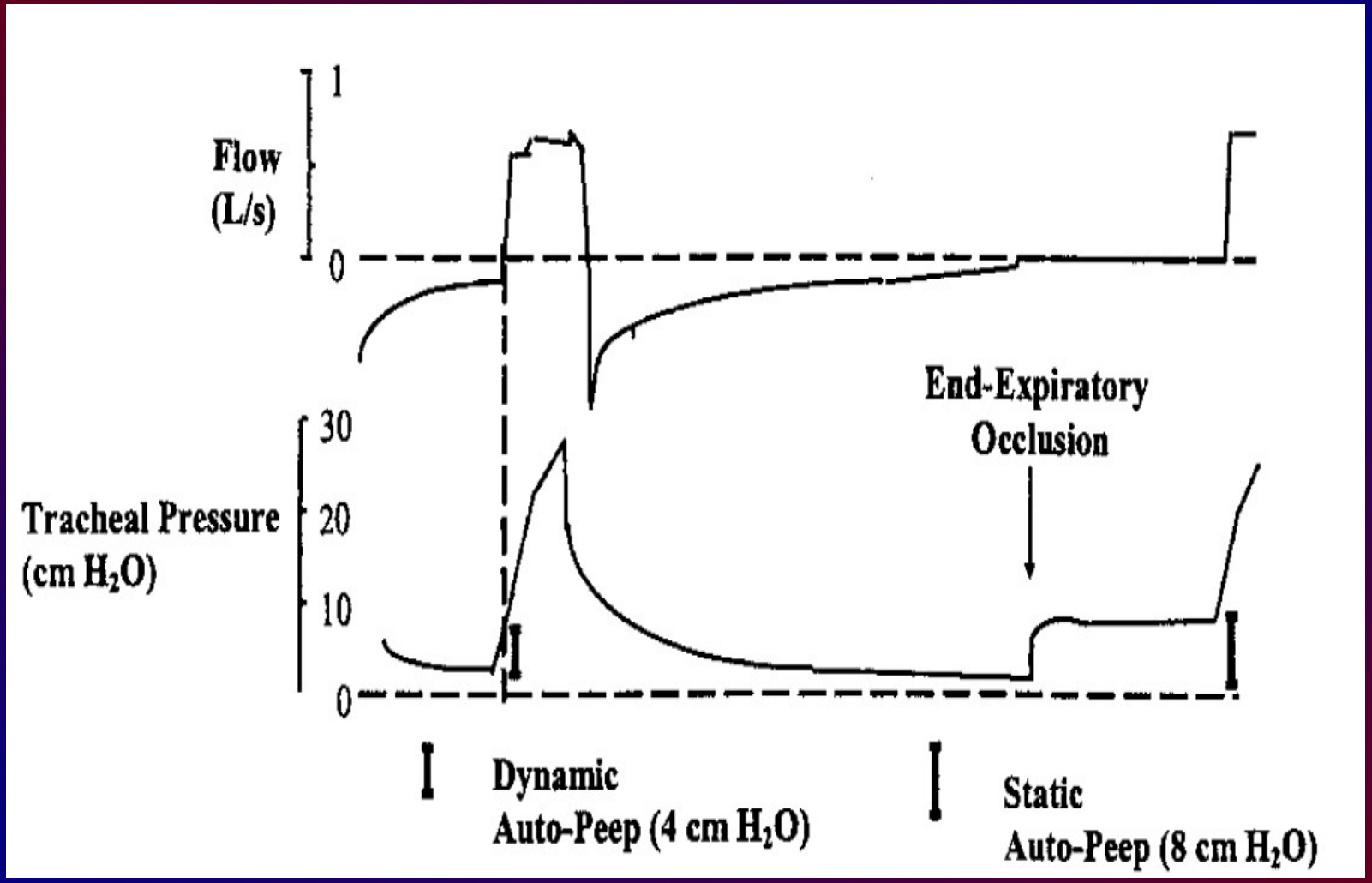
Drug	Dosage
Albuterol via nebulizer	2.5–5.0 mg every 20 min for 3 doses, then 2.5–10 mg every 1–4 h as needed, or 10–15 mg/h continuously
Albuterol via MDI	4–8 puffs every 20 min, up to 4 h, then every 4 h as needed
Ipratropium via nebulizer	0.5 mg every 20 min for 3 doses, then as needed (may be mixed with albuterol)
Ipratropium via MDI	8 puffs every 20 min as needed up to 3 h
Prednisone/methylprednisolone	40–80 mg/d in 1–2 divided doses until peak expiratory flow reaches 70% of predicted

MDI = metered-dose inhaler
(Adapted from Reference 24.)

Asthma = obstruction



http://www.rtjournalonline.com/Life%20threatening%20Asthma%20Physiology%20and%20Management.pdf



MET call

A spont. vent., O2 mask (40% O2)

B 30/min, SpO2 85%

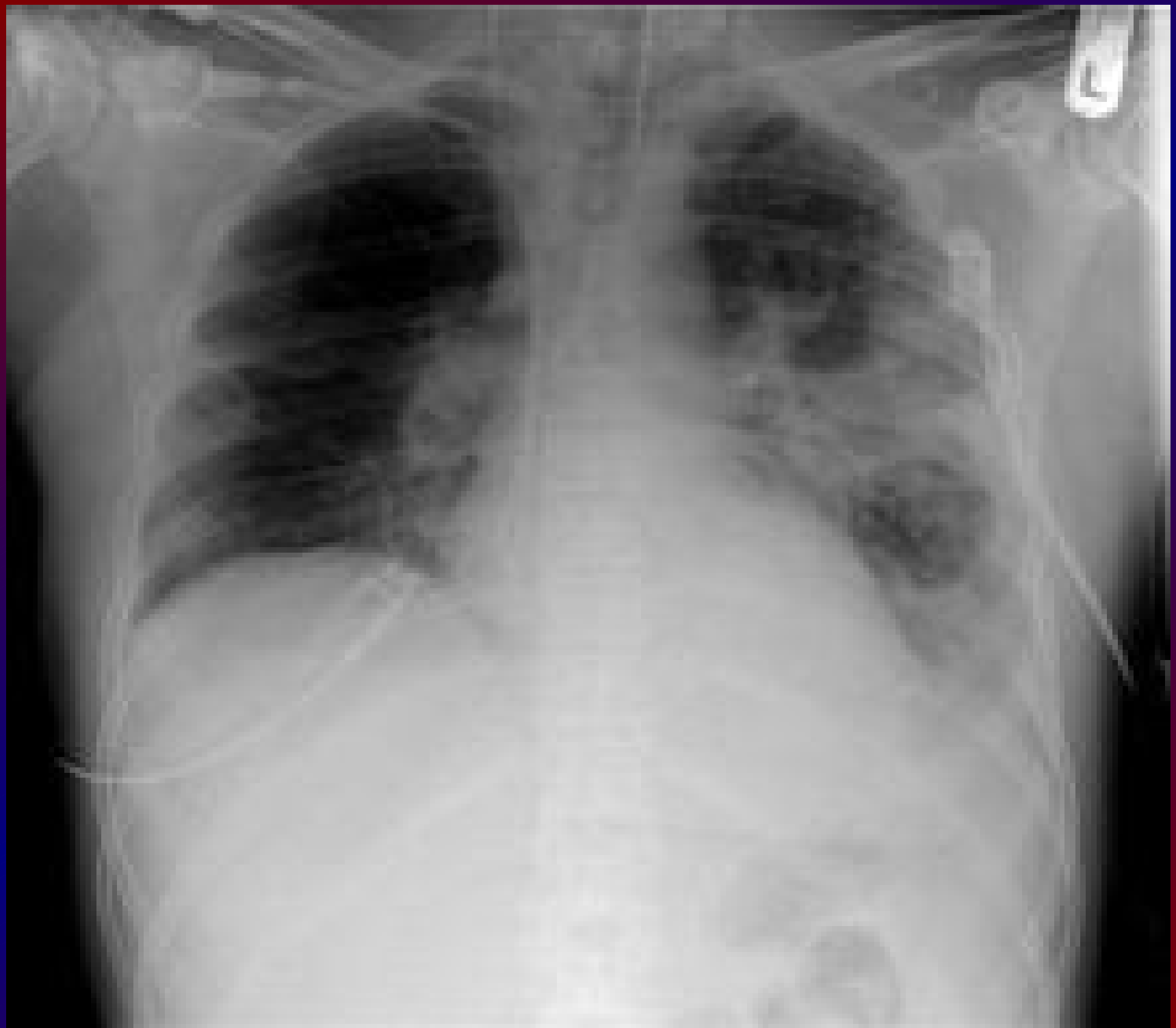
C 110/min, 80/40mmHg

D somnolence … sopor

I: 39°C, 2D stand.wards. Augmentin iv.

Problem:

Plan:



MET call

A

B

C 110/min, 80/40mmHg

D somnolence … sopor

I: 39°C, 2D stand.wards. Augmentin iv.

Problem: bronchopneumonia

Plan: ATB, OTI, Vent.

MET call

A O2 mask

B rapid, shallow breading, SpO2 90%, crackles

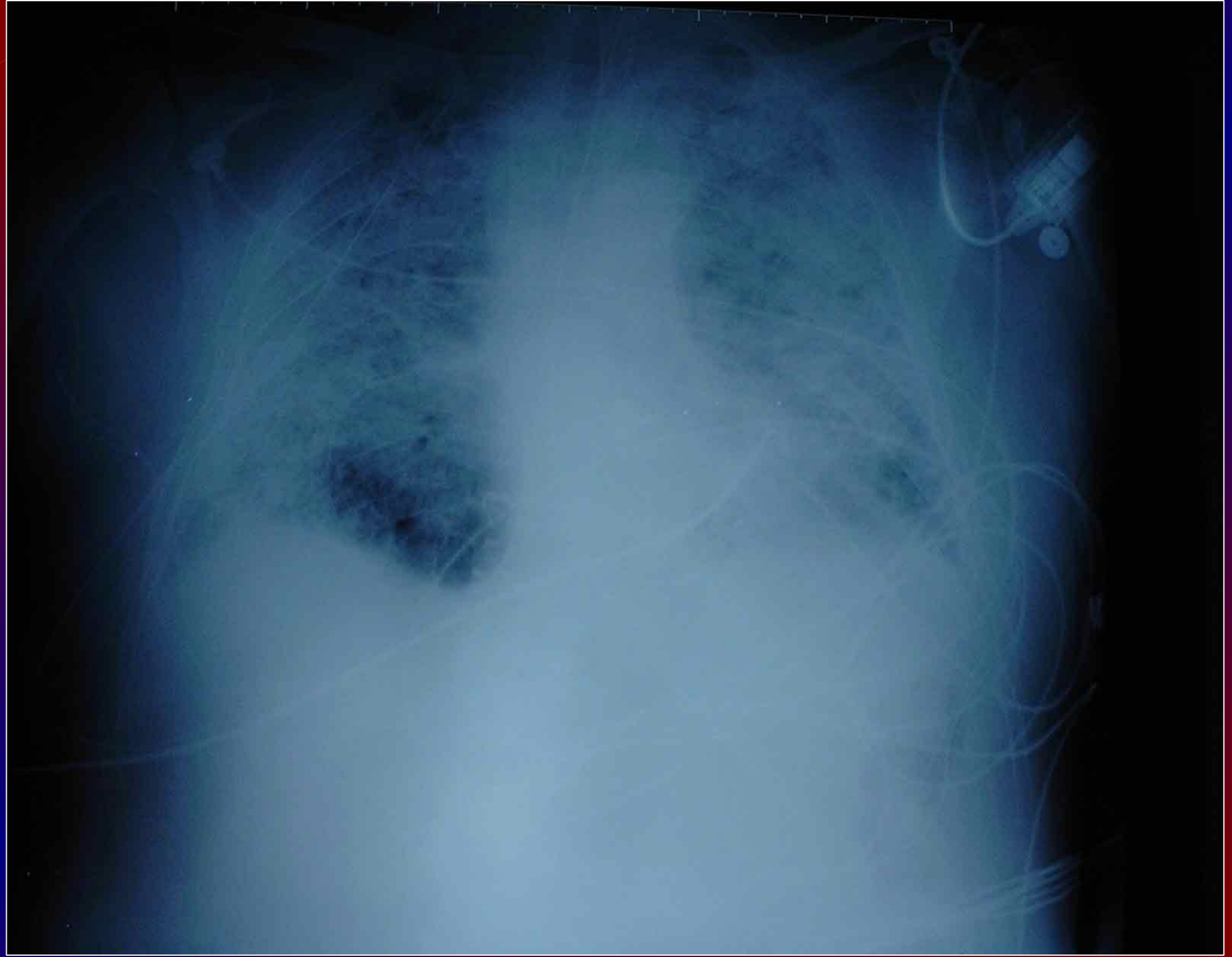
C s.r. + Norepinephrine

D somnolence

...

Problem: pancreatitis

Plan:



ARDS = acute respiratory distress syndrome

- Clinical presentation - Tachypnea and dyspnea; crackles upon auscultation
- Clinical setting - Direct insult (aspiration) or systemic process causing lung injury (sepsis)
- Radiologic appearance - Three-quadrant or 4-quadrant alveolar flooding
- Lung mechanics - Diminished compliance (< 40 mL/cm water)
- Gas exchange - Severe hypoxia refractory to oxygen therapy ($PaO_2/FIO_2 < 200$)
- Normal pulmonary vascular properties -

End.

COPD Signs

- barrel chest,
- pursed-lip breathing,
- productive cough,
- cyanosis.

Barrel Chest

- lungs become enlarged, the diaphragm is displaced downward and is unable to contract efficiently. Furthermore, the chest wall is enlarged, making accessory breathing muscles (muscles in the neck, upper chest, and between the ribs) less efficient as well. These changes contribute to shortness of breath.