UMF
UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ

Acute Respiratory Failure

Sanda-Maria Copotoiu

Respiratory failure

- **Definition**
- **Classification**
- **Physiology recall**
- **Diagnosis**
- **Respiratory monitoring**
- **Management**

Aims

1. Diagnose – rapid & accurate
2. Manage in due time the syndrome
3. Understand

Respiratory failure

UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ

Definition

Acute or chronic impairment of respiratory system function to maintain normal O_2 and CO_2 values when breathing room air.

Oxygenation failure

- **paO_2 < normal predicted values for age & altitude**
- **↓ Inspired O_2 concentration**
- **V/P mismatch**

Respiratory failure

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Ventilatory failure

↓ **CO_2 elimination→↑$paCO_2$ >45mmHg**

Most common causes:

- **Exacerbation of COPD**
- **Asthma**
- **Neuromuscular fatigue**

Dyspnoea, tachypnoea, tachycardia, accessory muscles of ventilation, altered counsciousness

$PaCO_2$ variations

Increased PCO_2	Decreased PCO_2
Fever	Pulmonary embolism
Sepsis	Cardiac arrest
Malignant hyperthermia	Hypothermia
Hypoventilation	Hyperventilation
Bicarbonate bolus	Hypometabolic states
Venous carbon dioxide embolism	Hypotension
Increased cardiac output	Decreased cardiac output
Restoration of pulse with cardiopulmonary resusciation	Esophageal intubation
Chronic obstructive lung disease	Disconnection from ventilator
	Extubation

Classification criteria

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Pathophysiology

Time

Etiology

Classification criteria

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Pathophysiology

Hypoxemic = type I

PaO2 ≤ 60mmHg

PaCO2 ≤ 40mmHg

Hypercapnic = type II

PaCO2 ≥ 50mmHg

PaO2 ≤ 60mmH

Classification criteria

UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ

Pathophysiology

Hypoxemic = type I

$PaO_2 \leq 60$ mmHg

$PaCO_2 \leq 40$ mmHg

Hypercapnic = type II

$PaCO_2 \geq 50$ mmHg

$PaO_2 \leq 60$ mmHg

↑CO_2 production

↑metabolism

↓CO_2 exhalation

Classification criteria

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Pathophysiology

- Hypoxemic = type I
  - $PaO_2 \leq 60mmHg$
  - $PaCO_2 \leq 40mmHg$
- Hypercapnic = type II
  - $PaCO_2 \geq 50mmHg$
  - $PaO_2 \leq 60mmHg$

Time

- **Acute – min, hrs**
- **Chronic - years**

Etiology

Classification criteria

UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ

Etiology

- CNS
- Spinal cord
- Neuromuscular system
- Chest wall
- Airways – upper, lower
- Lung parenchyma
- CV system

Pathophysiology

Air PA = P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2

$P_AO_{2\ =}$ FiO_2 x (BP – PH_2O) – P_ACO_2/R

R = respiratory exchange ratio ≈ 0.8 at rest

Pathophysiology

Air PA = $P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$

$P_AO_2 = FiO_2 \times (BP - PH_2O) - P_ACO_2/R$

R = respirat[illegible]ange ratio ≈ 0.8 at rest

P_ACO_2
Alveolar pressure
FiO2
Ventilation

Oxygen cascade

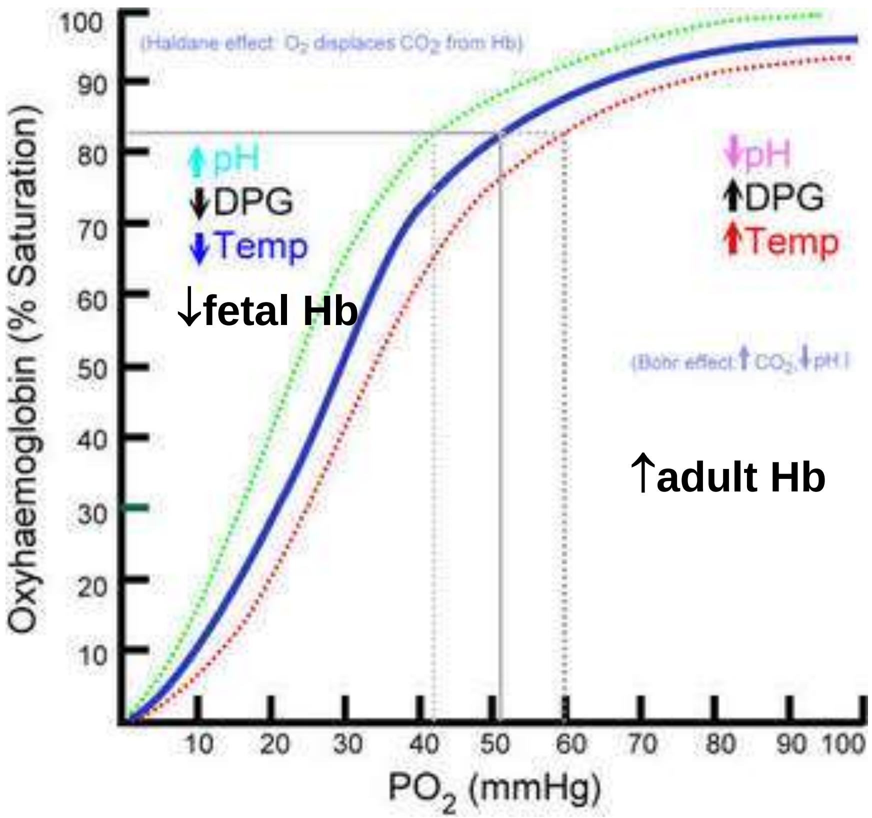
Oxygen transport

UNIVERSITATEA DE
MEDICINĂ ȘI FARMACIE
TÎRGU MUREȘ

- **Bound**
  - **20mlO_2/100ml arterial blood**
  - **15mlO_2/100ml venous blood**
- **Free – dissolved in blood**
  - **0.023ml/kPa/100ml blood**

Oxyhemoglobin dissociation curve

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ



Oxygen transport

- **Bound**
  - **20mlO_2/100ml arterial blood**
  - **15mlO_2/100ml venous blood**

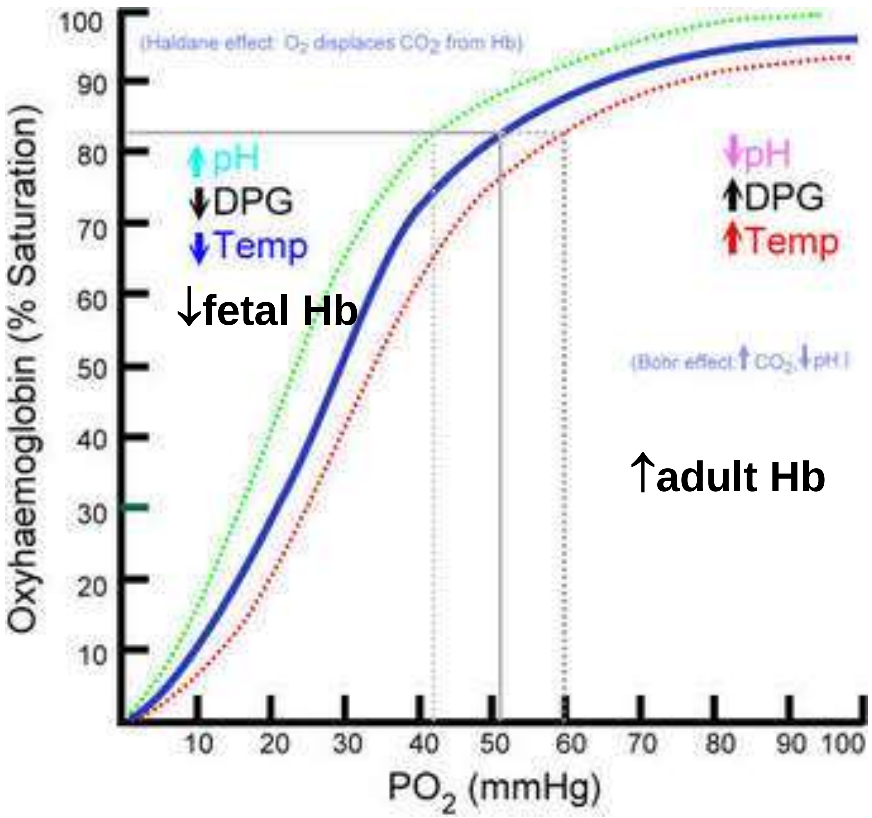
- **Free – dissolved in blood**
  - **0.023ml/kPa/100ml blood**

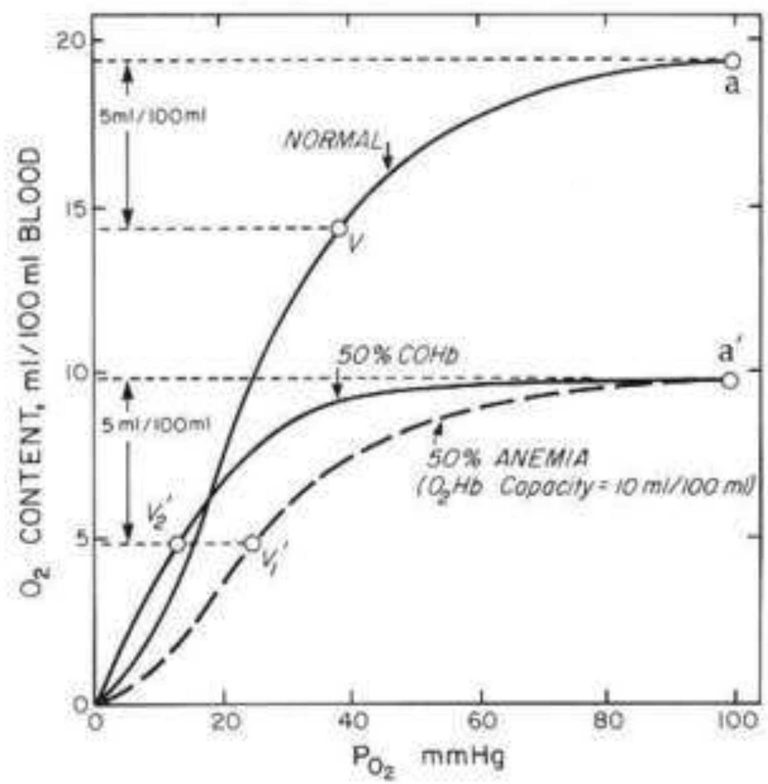
$$CaO_2 = Hb \times SaO_2 \times 1.4 + 0.003 \times PaO_2 \text{ 100ml blood}$$

UNIVERSITATEA DE MEDICINĂ ȘI FARMACIE TÎRGU MUREȘ

Oxyhemoglobin dissociation curve

UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ





Oxygen content of the blood

Substance	Arterial Blood	Arterial Blood	Venous blood
Oxygen	HbO2	18-20ml%	?
Oxygen	Dissolved	0,3ml%	0,11-0,18ml%
CO_2	$HbCO_2$	5%of CO2	4-8ml% 30% of CO2
CO_2	Dizolvat	5%dinCO2	2,7ml% 10% of CO2
Bicarbonate	?	90%din CO2	50ml 60% of CO2

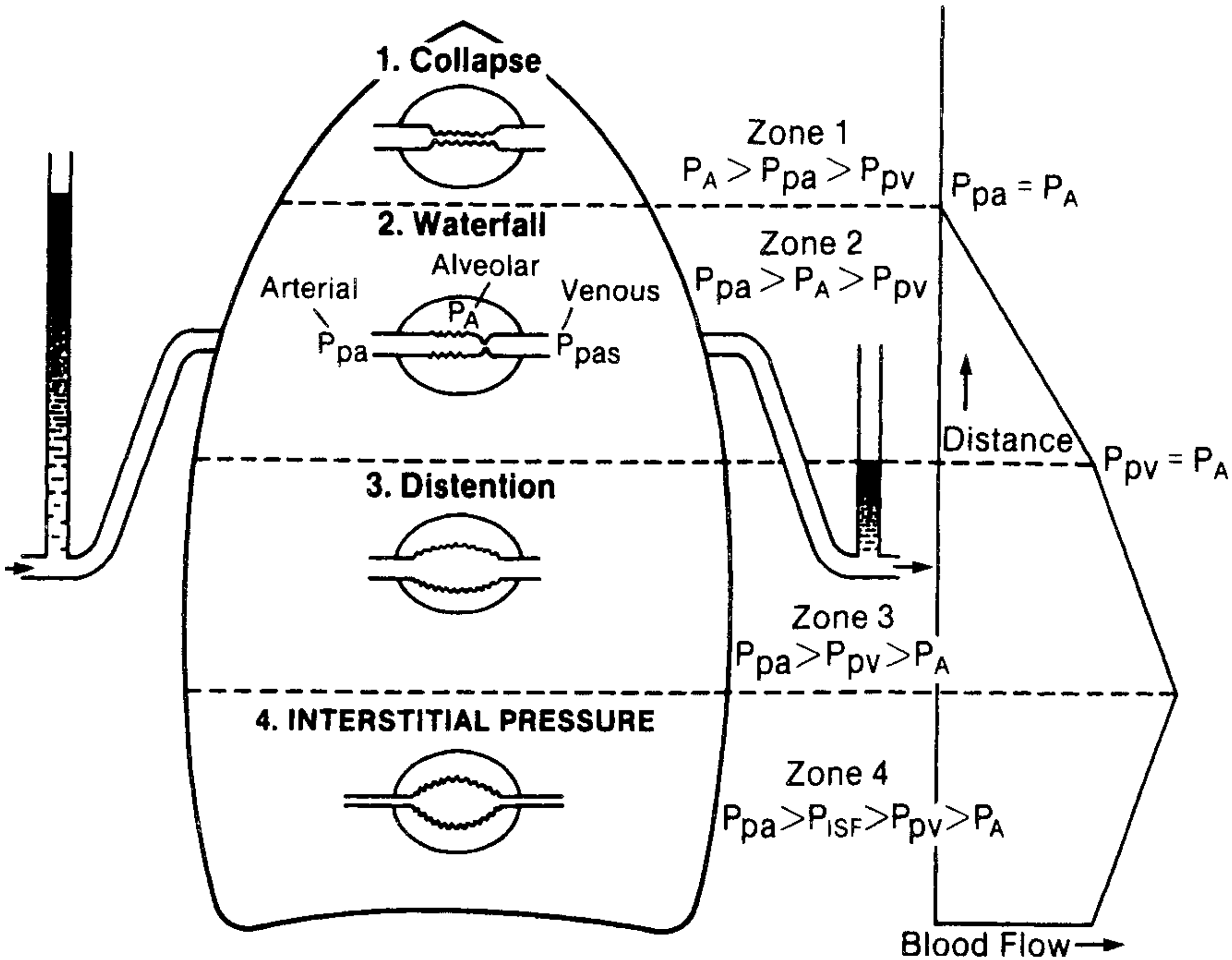
Gas exchange

Lung units: alveoli + capillaries

Diffusion abnormalities

The West zones

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ



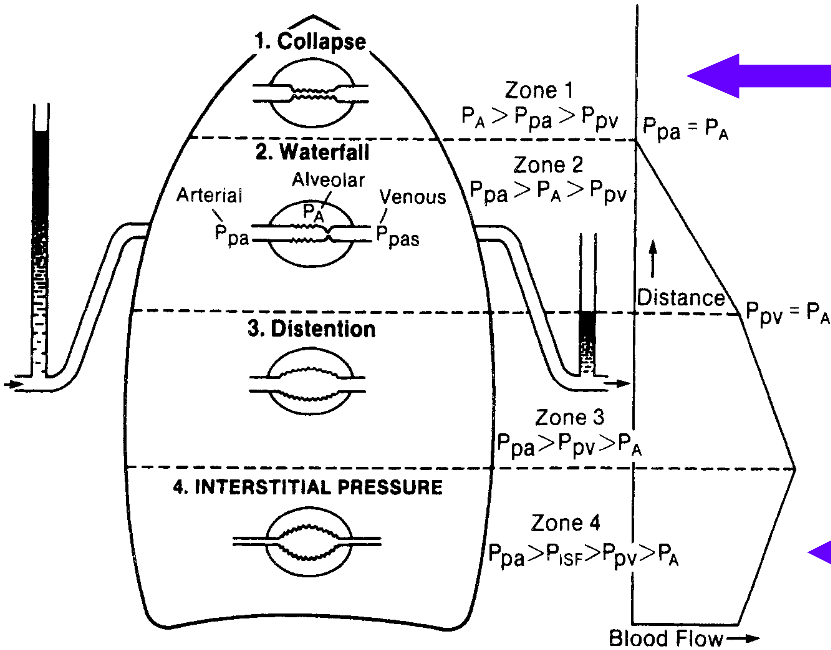
V/P

V/P = 1 ideal unit

V/P < 1 underventilated, normally perfused

V/P > 1 overventilation, underperfusion

V/Q


1. Collapse
Zone 1
PA > Ppa > Ppv
Ppa = PA
2. Waterfall
Alveolar
PA
Arterial
Ppa
Venous
Ppas
Zone 2
Ppa > PA > Ppv
Distance
Ppv = PA
3. Distention
Zone 3
Ppa>Ppv>PA
4. INTERSTITIAL PRESSURE
Zone 4
Ppa>PISF>Ppv>PA
Blood Flow

V/Q = 3.3

V/Q = 0.6

Intrapulmonary shunt

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

- True V/Q = 0

- Shunt fraction; **Qs** 1 > V/Q > 0

Pulmonary vasoconstriction

Intrapulmonary shunt

- True V/Q = 0

- Shunt fraction; **Qs** 1 > V/Q > 0

Pulmonary vasoconstriction

Causes

- Pneumonia
- Lung edema
- Atelectasis
- Collapse
- Pulmonary hemorrhage
- Lung contusion

A-a gradient = difference

diagnosis

- **a shunt**
- **a diffusion abnormality**

Alveolar gas equation

$P_A = FiO_2 \times (BP – H_2O) – P_ACO_2/R$

5 mmHg (0.5-1kPa) – 15mmHg

>15-20mmHg = lung disease

Dead space ventilation

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

a. Air reaching only the conducting airways = anatomic dead space

b. Air to the alveoli inert as to gas exchange with the capillaries

A + b = physiologic dead space

dead space ventilation = 20-30% of V_T

V_D/V_T = 0.2-0.3

Dead space ventilation

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

- **↓↓↓ CO**
- **↑↑ intra-alveolar pressure → stretching the alveolar capillaries**

Alveolar hypoventilation

- ↑$paCO_2$ ↓O_2

- **Brainstem**
  - **Trauma, haemorrhage, infarction, hypoxia, infection.**
  - **Metabolic encephalopathy**
  - **Depressant drugs**
- **Spinal cord**
  - **Trauma, tumor, transverse myelitis**
- **Nerve root injury**

Alveolar hypoventilation cont

- **Nerve**
  - **Trauma**
  - **Neuropathy**
  - **Motor neuron disease**
- **Nm junction**
  - **Myastenia gravis**
  - **Nm blocade**
- **Respiratory muscle fatigue**
  - **Disuse atrophy**
  - **Myopathy**
  - **Malnutrition**
- **Respiratory system**
  - **Airway obstruction (upper or lower)**
  - **Decreased lung, pleural or chest wall compliance**

Lung compliance

UNIVERSITATEA DE MEDICINĂ ȘI FARMACIE TÎRGU MUREȘ

- **Volume change/unit pressure =ΔV/ ΔP**
- **Lung compliance 200ml/cmH_2O**
- **Chest compliance 100ml/H_2O**

We are not the same and we tend to change position in space

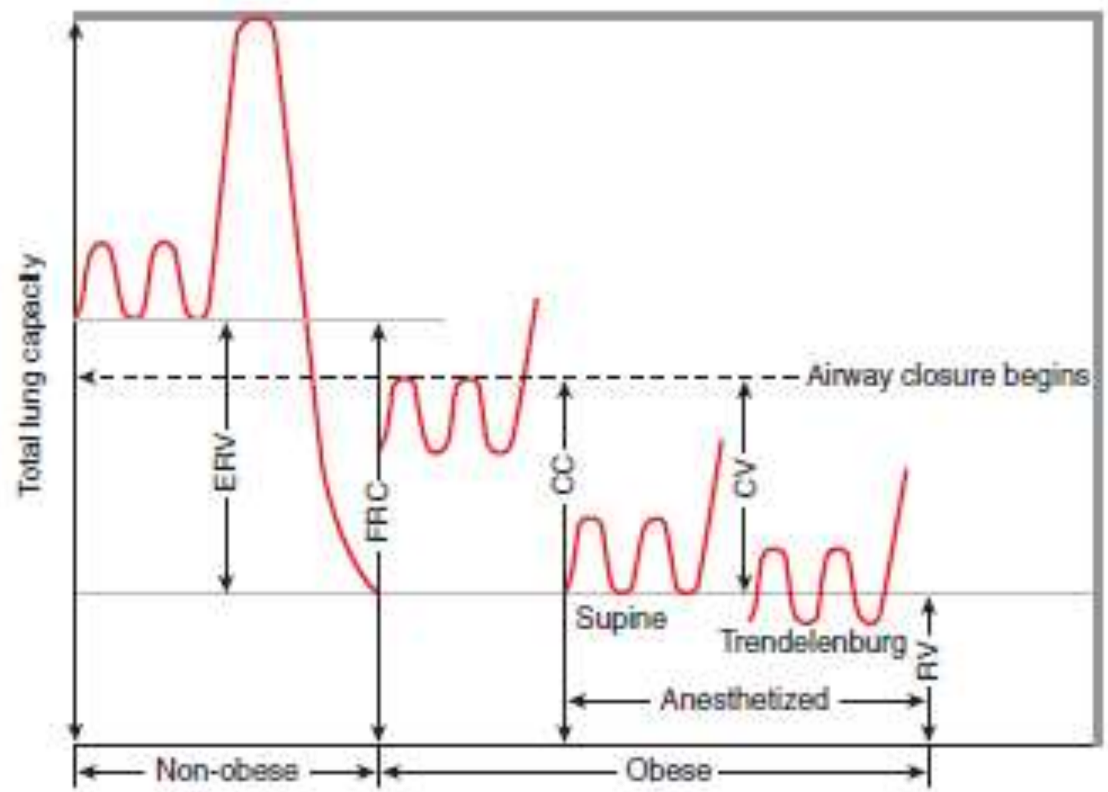
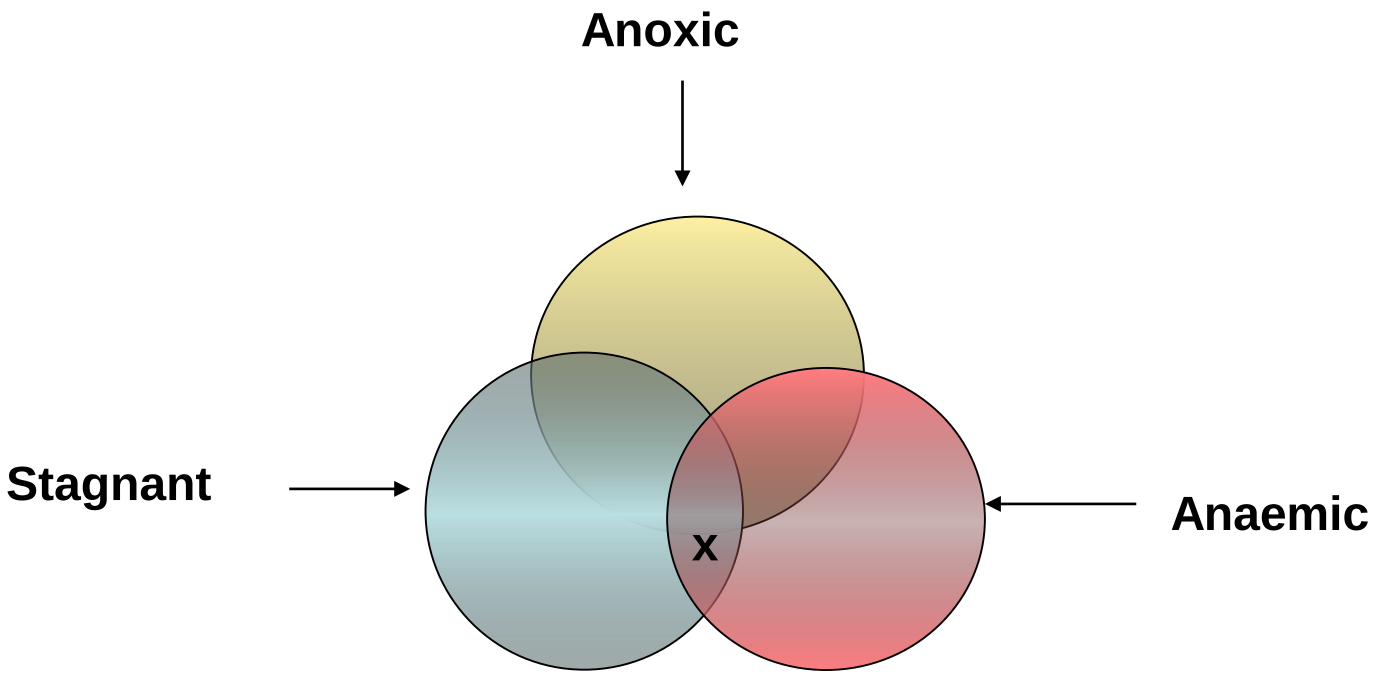


FIGURE 22–1. Effects of obesity, positioning, and anesthesia on lung volumes. CC, closing capacity; CV, closing volume; ERV, expiratory reserve volume; FRC, functional residual capacity; IRV, inspiratory reserve volume; RV, residual volume. (Modified from Ogunnaike and Whitten[37] with permission.)

Cost of breathing

2-3% of oxygen delivery

Hypoxia



= histotoxic

Hypoxia

UNIVERSITATEA DE MEDICINĂ ȘI FARMACIE TÎRGU MUREȘ

Fulminant $PaO_2 < 20mmHg$

- **Pressure loss at 30 000m**
- **Unconsciousness 15-20 sec**
- **Brain death 4-5 min**

Acute $25mmHg < PaO_2 < 40mmHg$

- **18 000 – 25 000m**
- **Inebriation**
- **Coma**
- **Death / min-hrs**

Hypoxia

Chronic 40mmHg< PaO_2 < 60mmHg

- **10 000-18 000m extended periods of time**
- **fatigue: dyspnoea, shortness of breath, respiratory arrhythmias**

Cyanosis if HHb/ml capillary blood; anemia!!!

& polycitemia

Tachycardia

Tachypnea

Hyperoxia

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

- **$FiO_2 > 0.6$**
- **Acute**
- **Chronic oxygen toxicity**

Hypercapnia

$PaCO_2$ > 45mmHg

↑ CO_2 production due to ↑metabolism

- **Sepsis**
- **Burns**
- **Overfeeding**

↓ CO_2 excretion

- **↓ Ventilation**

Effects

- **Stimulation of ventilation**
- **Cerebral vasodilation**
- **Simpatetic stimulation**
- **Perifferal vasodilation by direct effect on vessels**
- **Central depression – lethargy, coma**

Hypercapnia

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

$PaCO_2$ > 45mmHg

↑ CO_2 production due to ↑metabolism

- Sepsis
- Burns
- Overfeeding

↓ CO_2 excretion

- ↓ Ventilation

Effects

- **Stimulation of ventilation**
- **Cerebral vasodilation**
- **Simpatetic stimulation**
- **Perifferal vasodilation by direct effect on vessels**
- **Central depression – lethargy, coma**

Hypercapnia

PaCO2 > 45mmHg

↑ CO2 production due to ↑metabolism
- Sepsis
- Burns
- Overfeeding

↓ CO2 excretion
- ↓ Ventilation

Effects
- Stimulation of ventilation
- Cerebral vasodilation
- Simpatetic stimulation
- Perifferal vasodilation by direct effect on vessels
- Central depression – lethargy, coma

Permissive hypercapnia

↑CO due to ↑sympathetic activity

↑splanchnic & renal blood flow

Hypocapnia

UNIVERSITATEA DE
MEDICINĂ ȘI FARMACIE
TÎRGU MUREȘ

$PaCO_2 \leq$ 35mmHg

- **Cerebral vasoconstriction**
  - **$\downarrow$Ca pl$\rightarrow$ $\uparrow$muscle excitability**
- **Alcalosis**

Hypocapnia

$PaCO_2 \leq 35$mmHg

- **Cerebral vasoconstriction**
  - **↓Ca pl→ ↑muscle excitability**
- **Alcalosis**

Address the cause!

Respiratory monitoring

- **Clinical exam**
- **Pulse oxymetry**
- **Capnography**
- **Ultrasound scanning**
- **Arterial blood gases ABG**
- **∅ respiratory function tests**

Pulseoxymetry

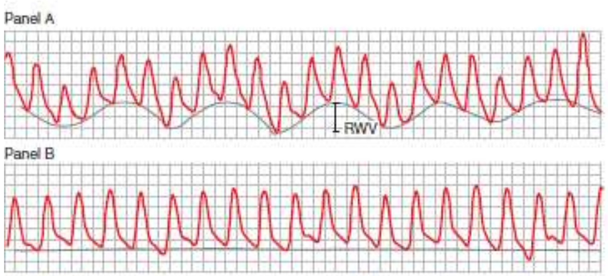


FIGURE 31–5. Pulse oximeter tracings from a 60-year-old woman with exacerbation of chronic obstructive pulmonary disease who was admitted to the ICU in ventilatory failure. **A.** Patient's pulse oximetry tracing at the time of admission reveals respiratory variability in the pulse oximeter plethysmography tracing. Measured pulsus paradoxus at this time was 16 mm Hg. **B.** Patient's pulse oximetry tracing after 12 hours of aggressive therapy. Pulsus paradoxus at this time was 8 mm Hg. Note the absence of respiratory waveform variation (RWV) in the baseline of the oximeter tracing after clinical improvement in airflow and resolution of elevated pulsus paradoxus. (From Hartert et al.[49] with permission.)

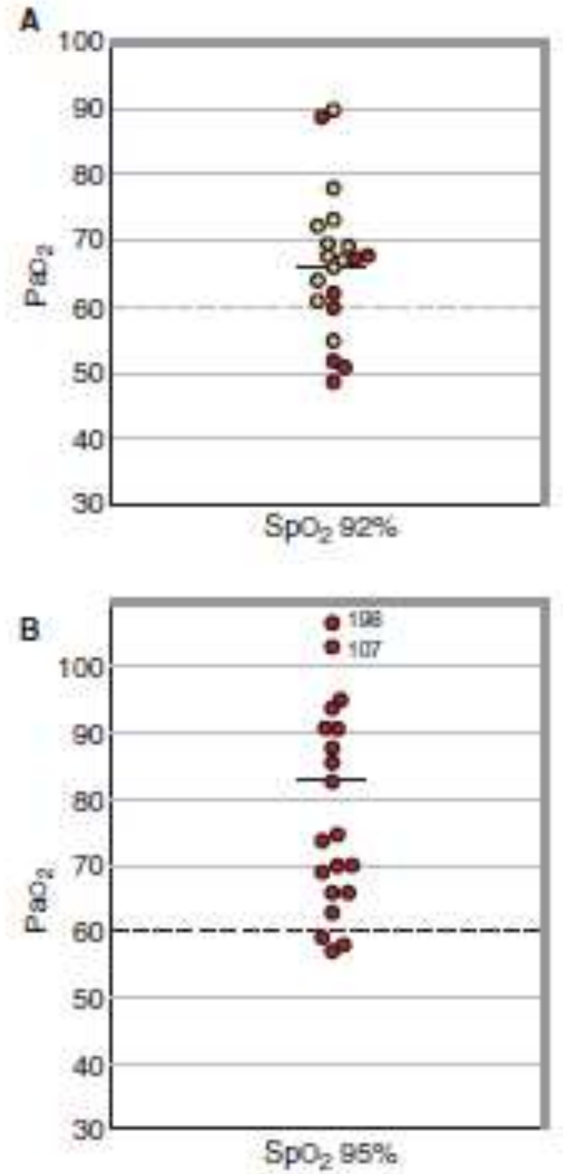
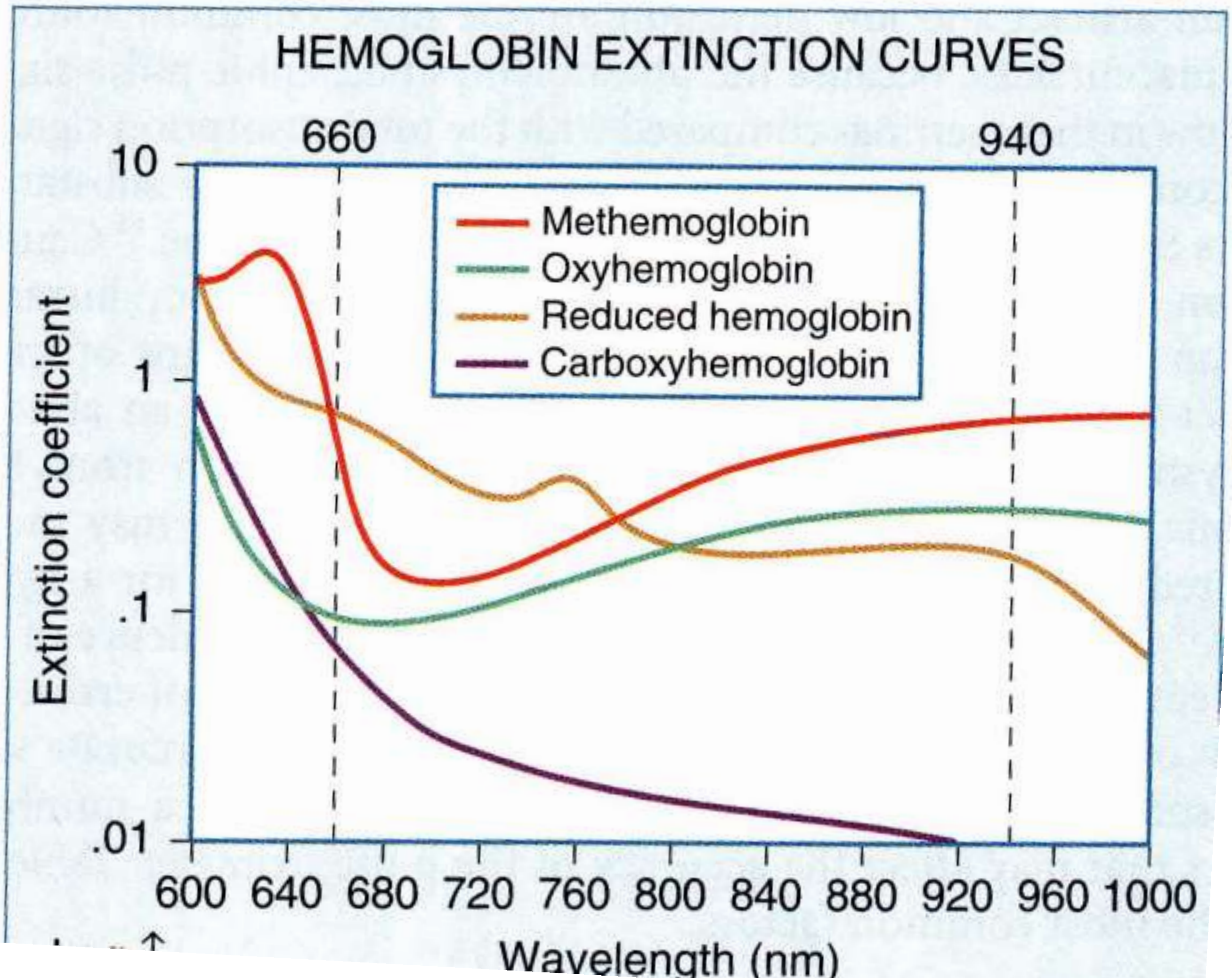


FIGURE 31–4. Top. In white patients (open circles), $SpO_2 \geq 92\%$ is reliable in predicting $PaO_2 \geq 60$ mm Hg. **Bottom.** In black patients, $SpO_2 \geq 95\%$ was required to reliably predict $PaO_2 \geq 60$ mm Hg.


HEMOGLOBIN EXTINCTION CURVES
660
940
Methemoglobin
Oxyhemoglobin
Reduced hemoglobin
Carboxyhemoglobin
Extinction coefficient
10
1
.1
.01
600 640 680 720 760 800 840 880 920 960 1000
Wavelength (nm)

Capnometry $ETCO_{2,}$ $P_{ET}CO_2$

Measurement of expired CO2 and numeric display of expired CO2 at the patients' airway opening

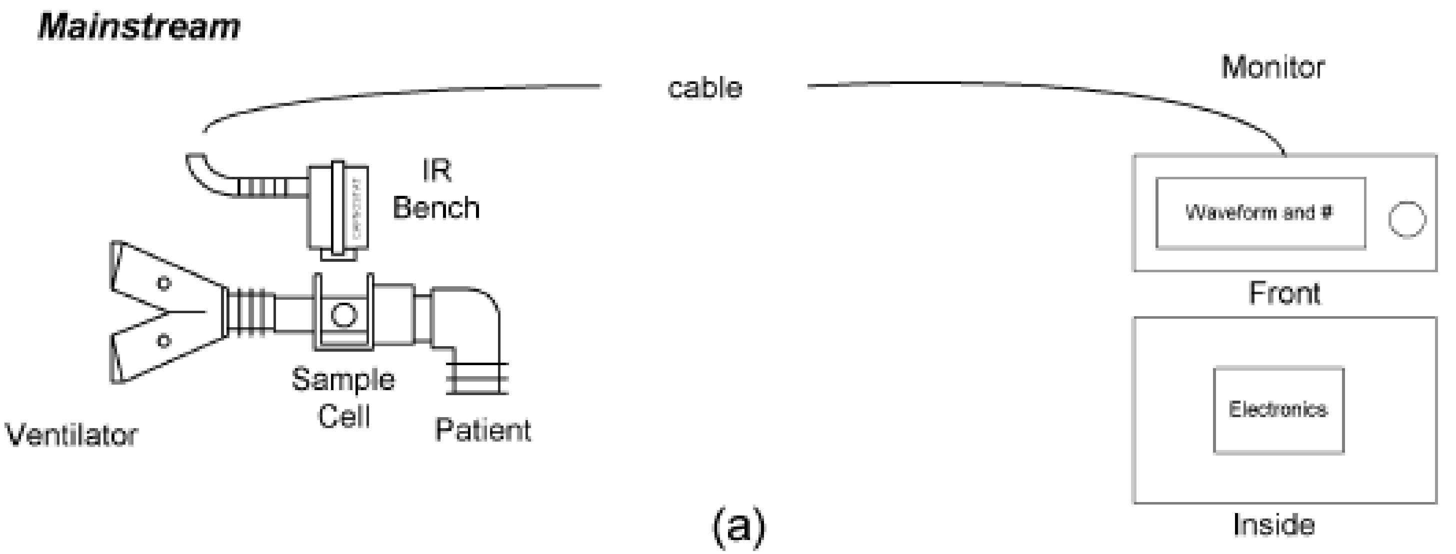
+ waveform plotting CO2 against time or volume = capnography, capnogram

Sensor: passing infrared light through a sample chamber to a detector on the opposite side

CO2 peak wavelenght of absorbtion 4.27µ

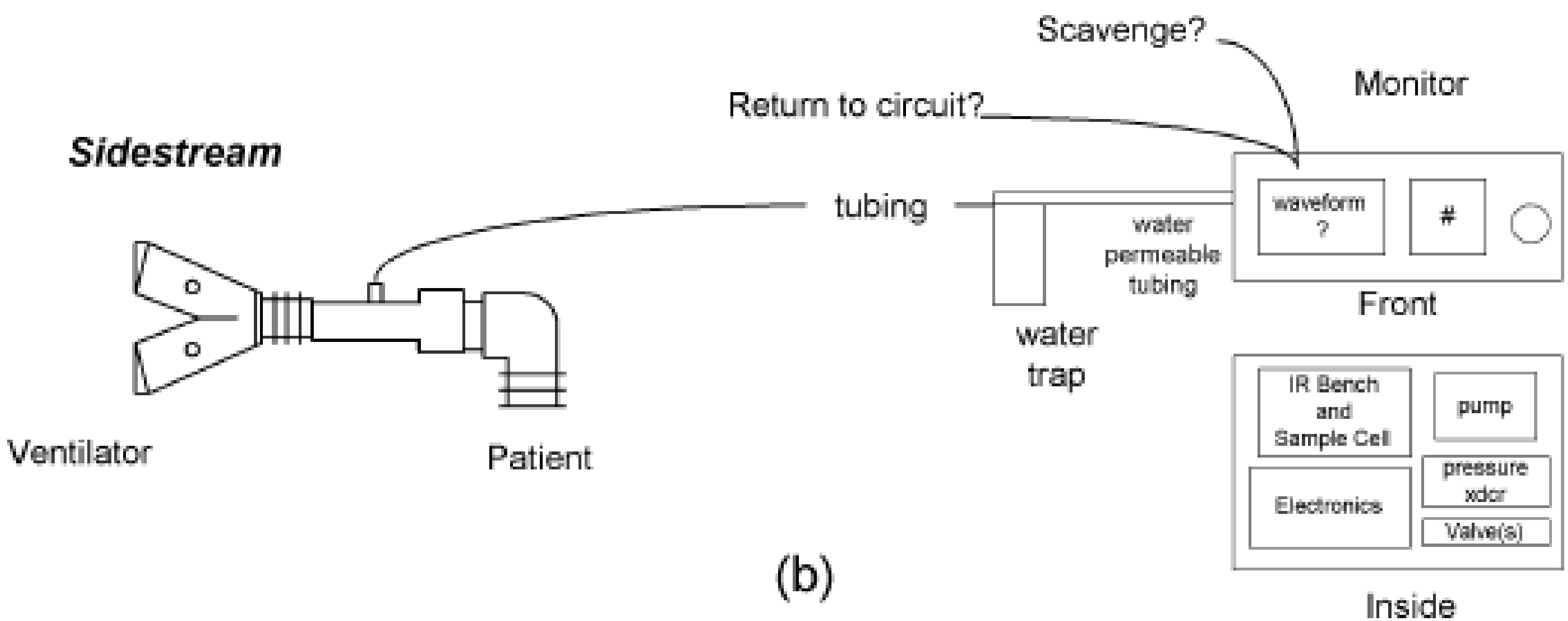
- **Sidestream**
- **mainstream**

Capnography

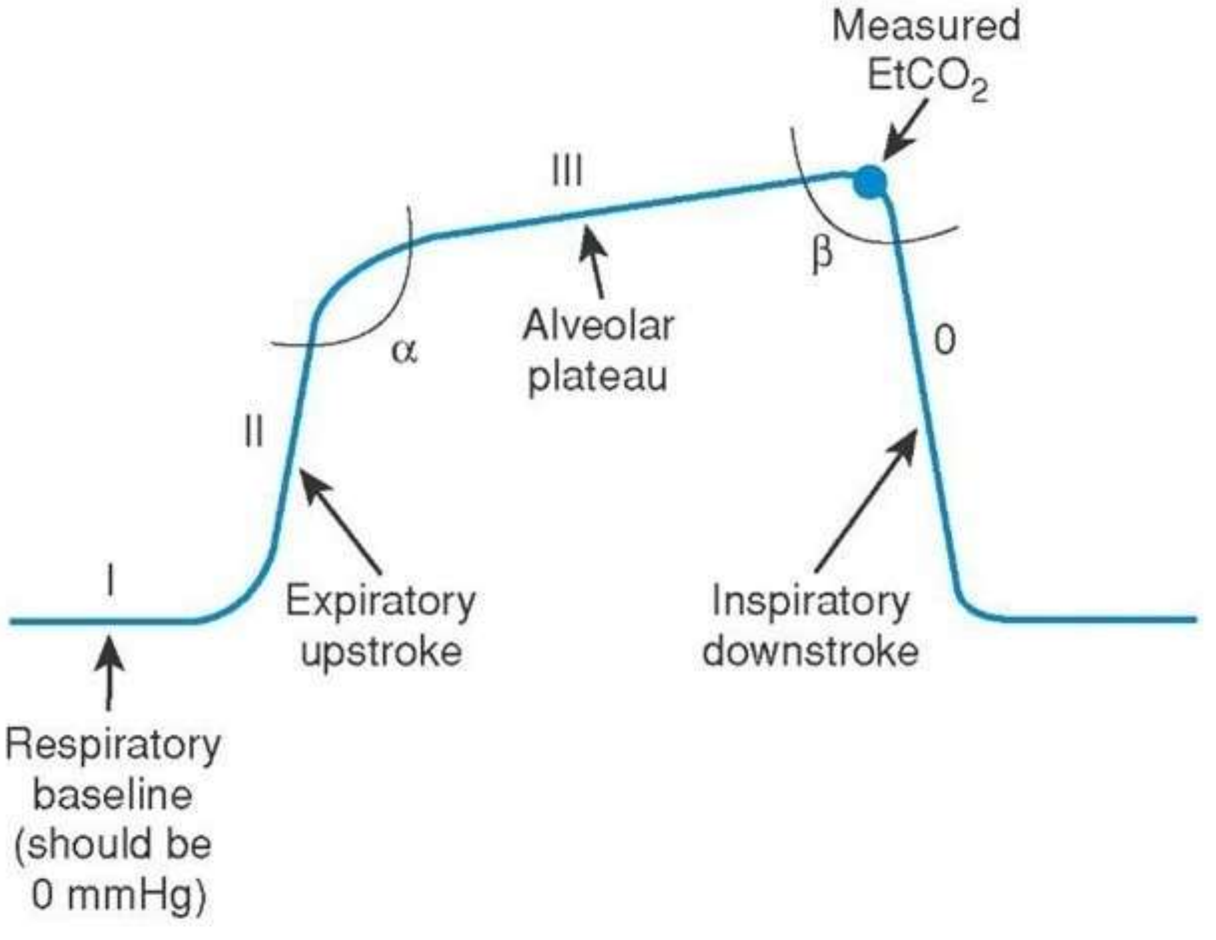


(a)

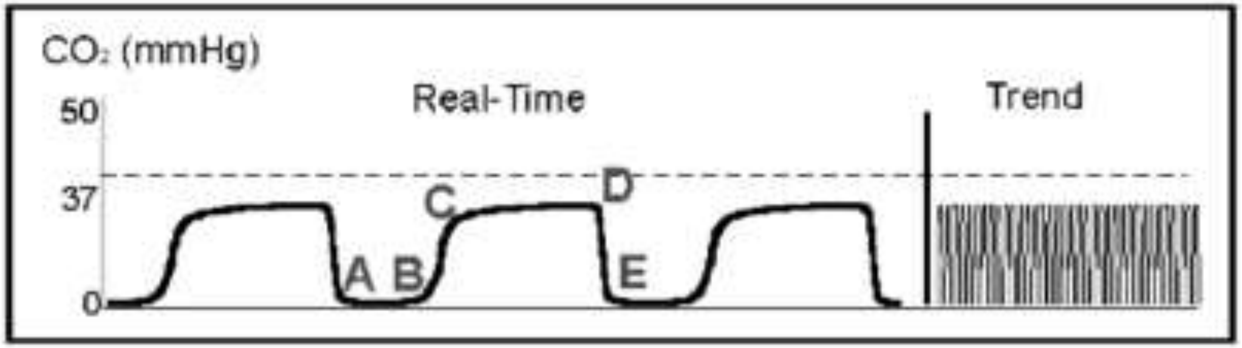
Capnography



Capnogram


Measured
$EtCO_2$
III
β
α
Alveolar
plateau
0
II
I
Expiratory
upstroke
Inspiratory
downstroke
Respiratory
baseline
(should be
0 mmHg)

Capnogram



- A – B Baseline
- B – C Expiratory Upstroke
- C – D Expiratory Plateau
- D $ETCO_2$ value
- D – E Inspiration Begins

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Capnography indications

- Diagnosis of pulmonary embolism
- Assessing lung recruitment response to PEEP
- Detection of intrinsic PEEP
- Evaluation of weaning
- Indirect marker of elevated dead space ventilation
- Assessment of CP resuscitation
- Indirect CO measuring by CO_2 rebreathing
- Verification of endotracheal cannulation
- Detection of airway accidents
- Determination of feeding tube placement

$PaCO_2$ – $PETCO_2$ gradient

- **normal 4-5mmHg**
- **↑↑Critically ill pts**
- **Eg: COPD 7-16mmHg**
- **ALI, cardiogenic PE: 4-12mmHg**

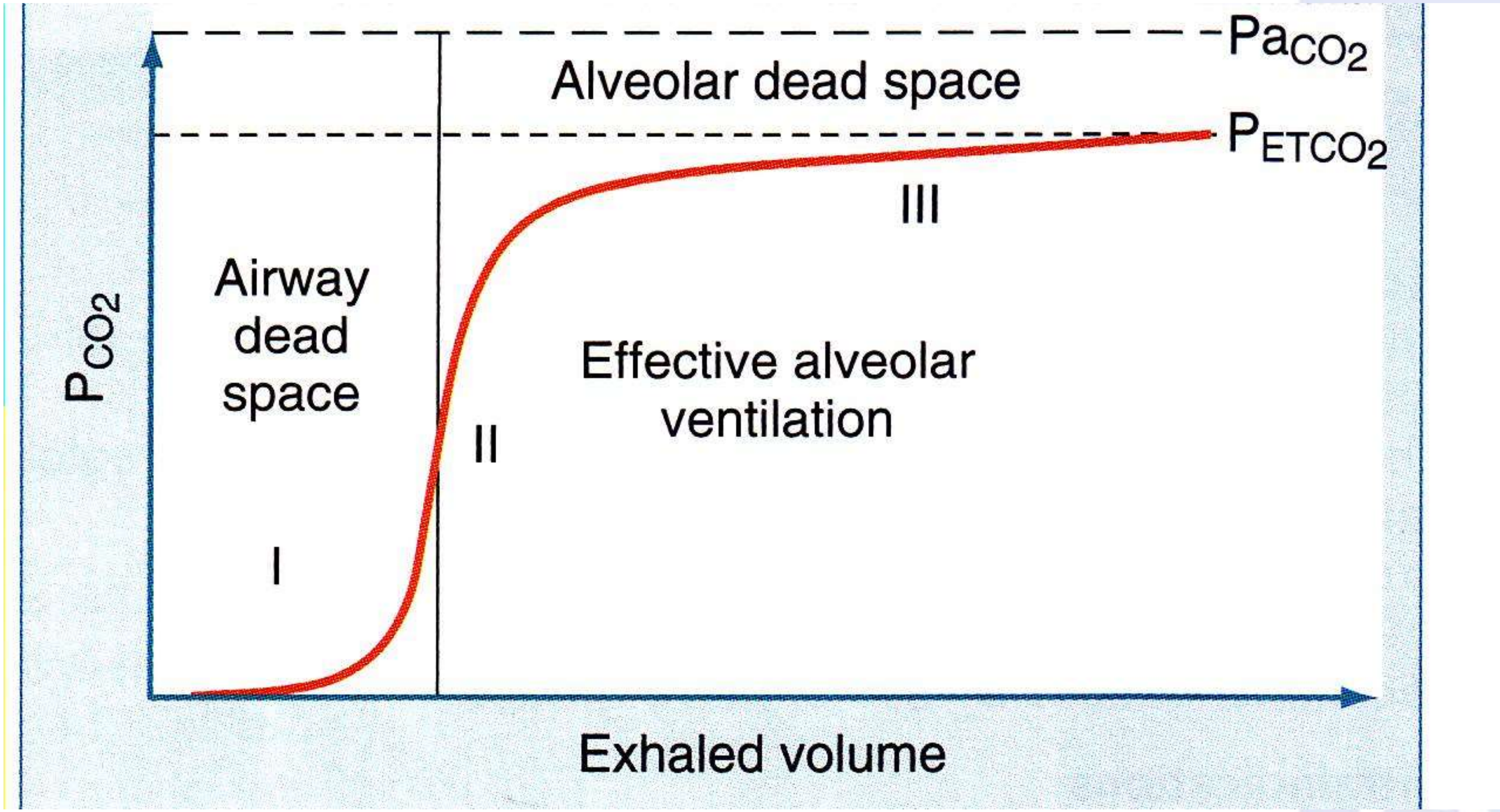
Caused by:

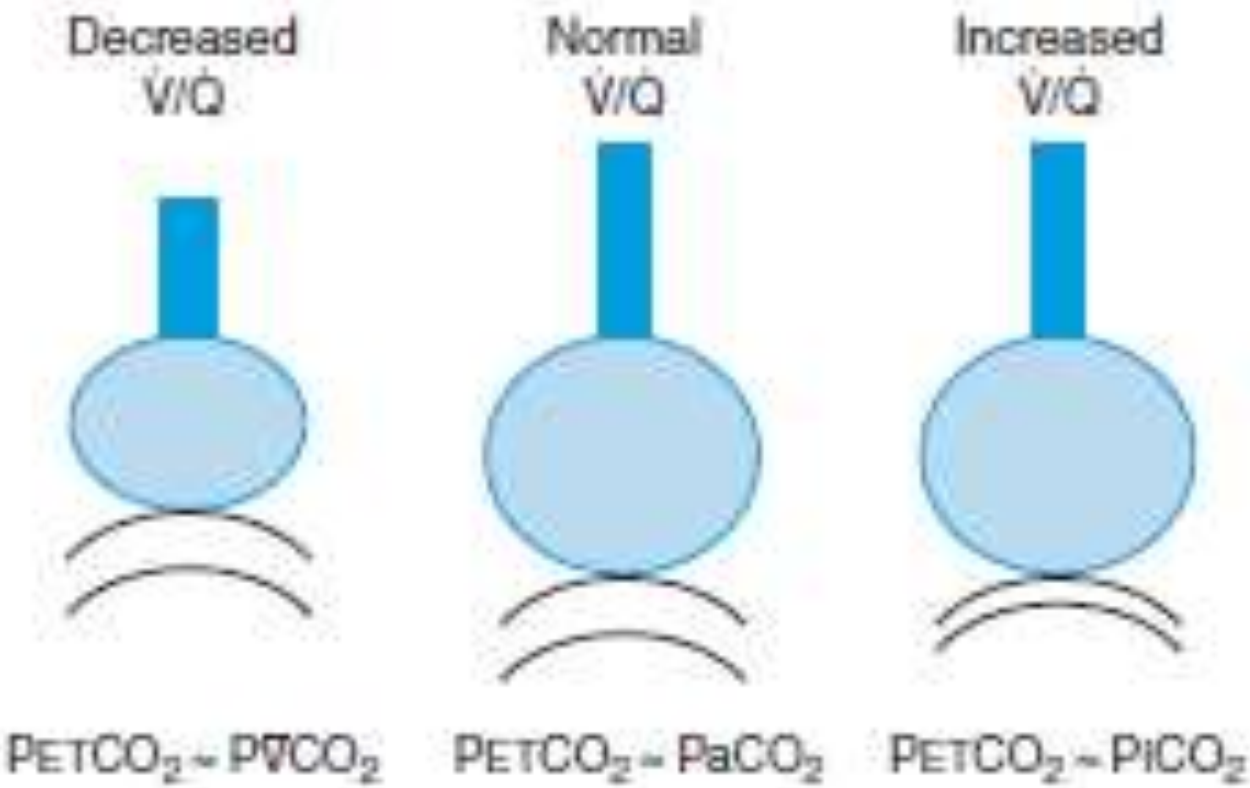
paCO2 reflects mean PCO2 in alveolar gas

PETCO2 approximates peak PaCO2

Single breath CO_2 waveform
CO_2 elimination as a function of the volume of the exhaled gas

UMF
UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ





$P_{ET}CO_2$ with low $\dot{V}/\dot{Q}$, normal $\dot{V}/\dot{Q}$, and high $\dot{V}/\dot{Q}$.

Management of ARF

a. Diagnosis & treatment of the underlying condition

b. Respiratory support $\rightarrow$ adequate oxygenation to the tissues since hypoxaemia is deleterious and rapid reversal critical.

Respiratory support

- **Oxygen supplementation**
- **Mechanical ventilation**
- **Physical therapy**

Why do we need MV?

a. ARF or imminent despita maximal treatment

Why do we need MV?

a. **ARF or imminent despita maximal treatment**
b. **Following major surgery in GA**

Why do we need MV?

a. ARF or imminent despita maximal treatment
b. Following major surgery in GA
c. Cardiogenic shock – to reduce the oxygen cost of ventilation when CO↓

Aspirations vs reality

Ideally:

MV would replicate the mechanics and physiology of spontaneous respiration reaching adequate oxygenation and ventilation.

Aspirations vs reality

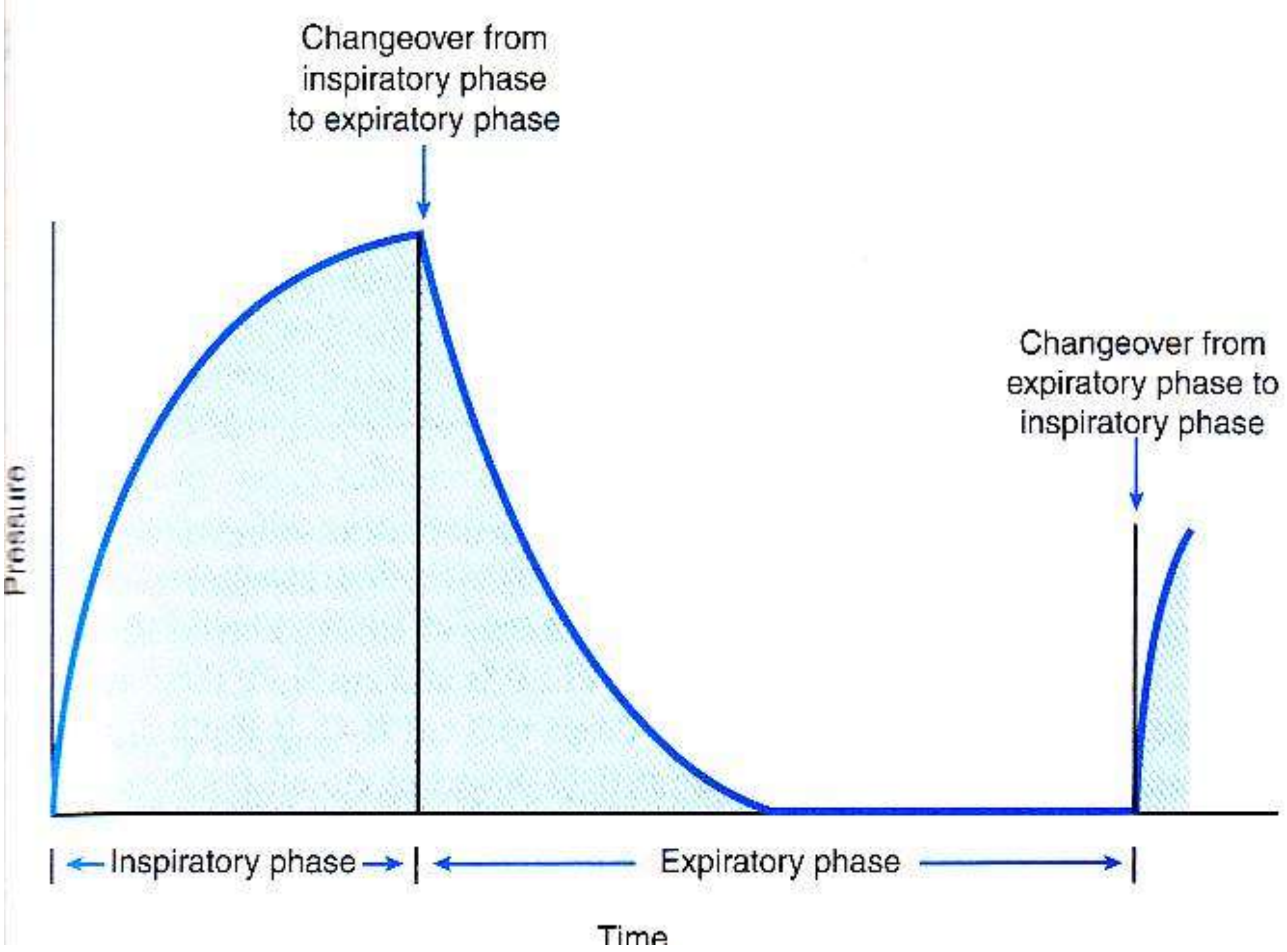
Ideally:

MV would replicate the mechanics and physiology of spontaneous respiration reaching adequate oxygenation and ventilation.

Reality

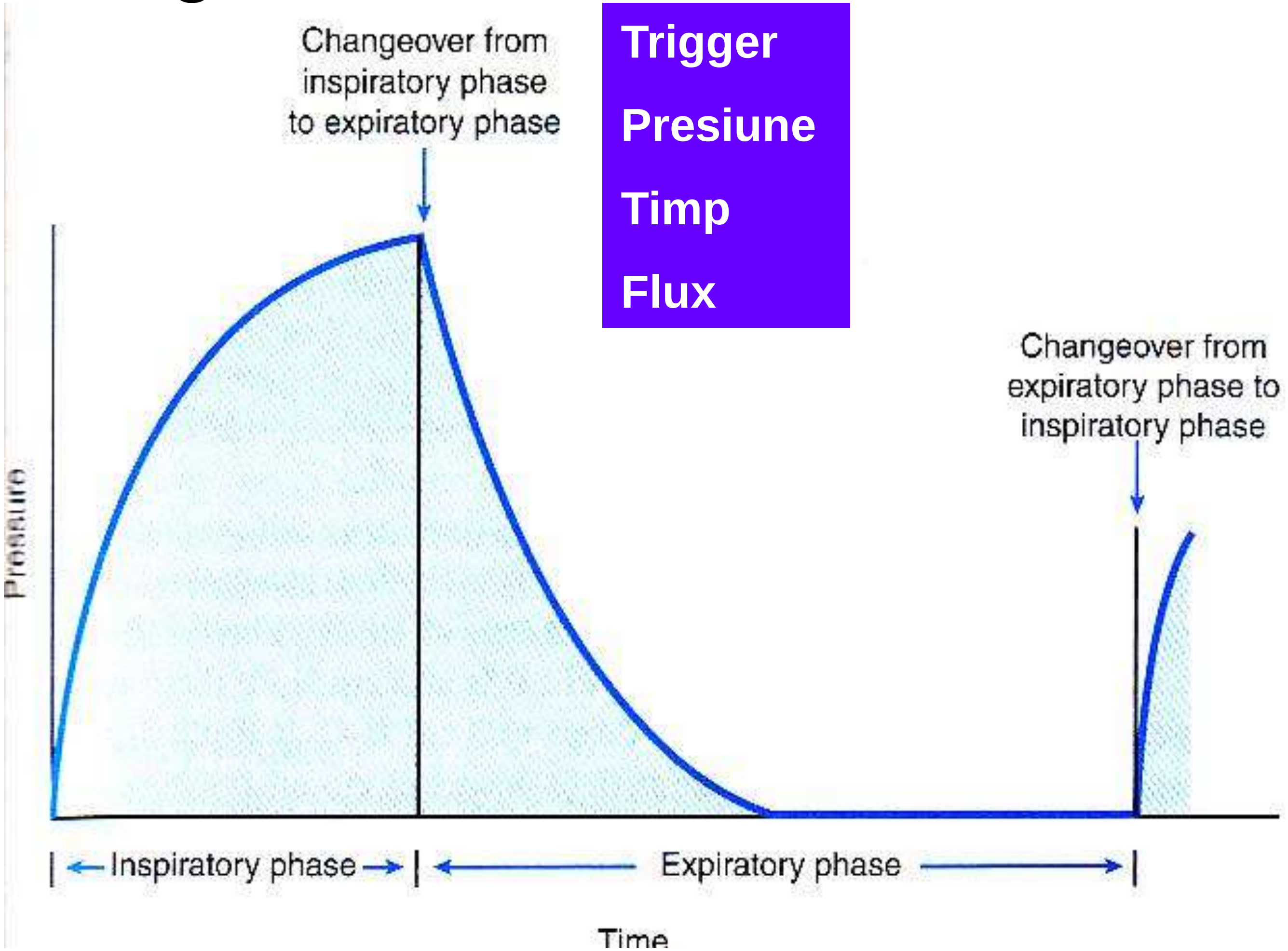
MV works with positive pressure during inspiration, exhaling to athmospheric pressure or to a preset PEEP.

The 4 phases of the respiratory cicle during MV



Pierce LNB, 2007

The 4 phases of the respiratory cicle during MV



Trigger

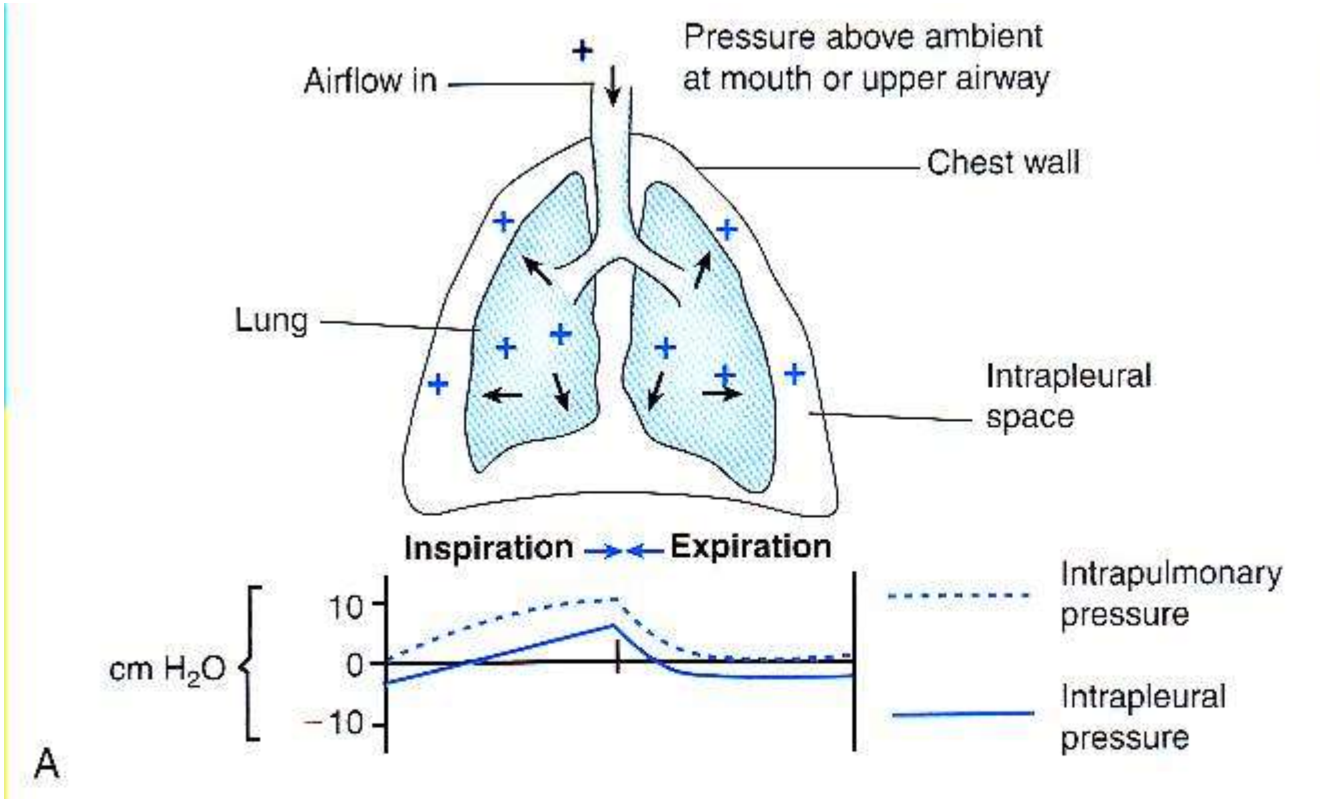
Presiune

Timp

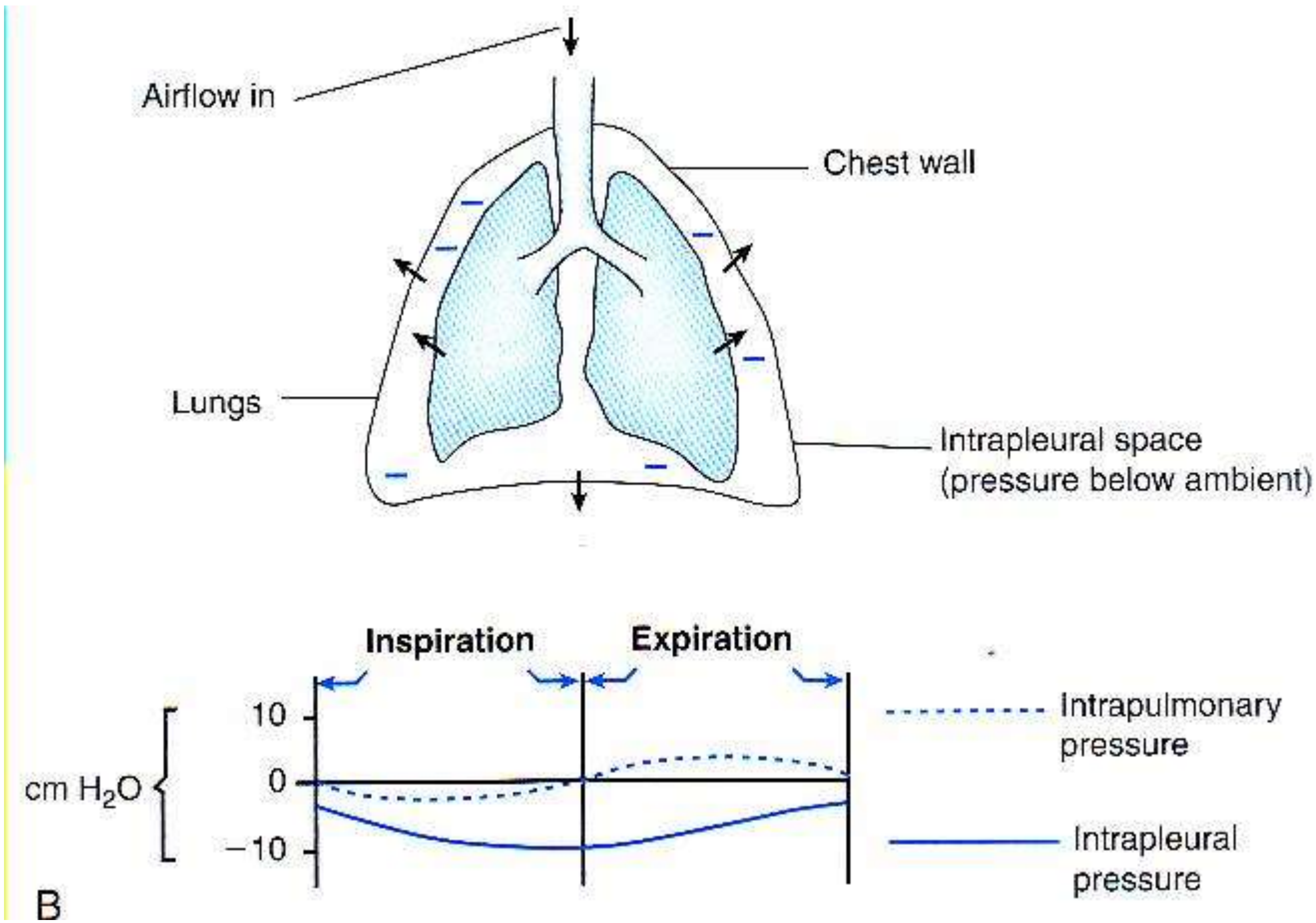
Flux

Pierce LNB, 2007

Intrathoracic pressures during IPPV

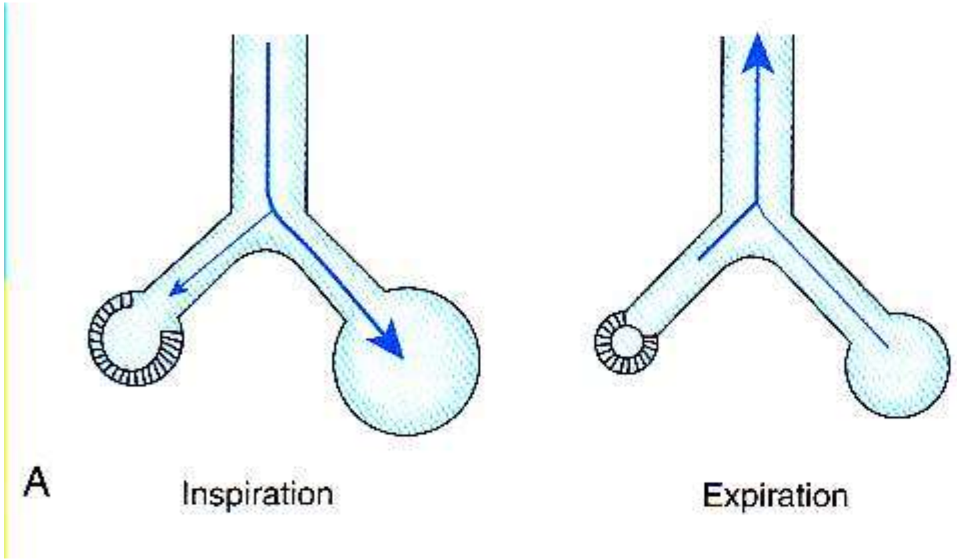


Intrathoracic pressures during spontaneous ventilation

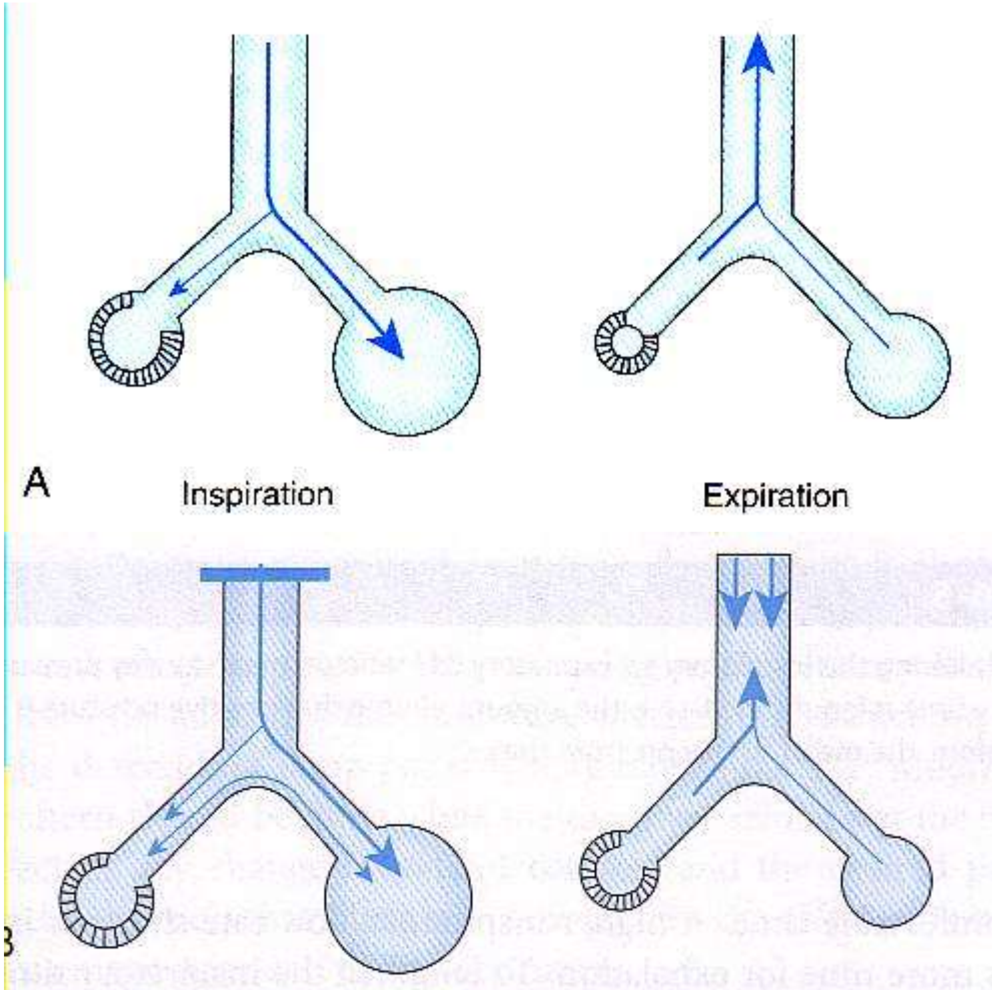


LNB Prierce, 2007

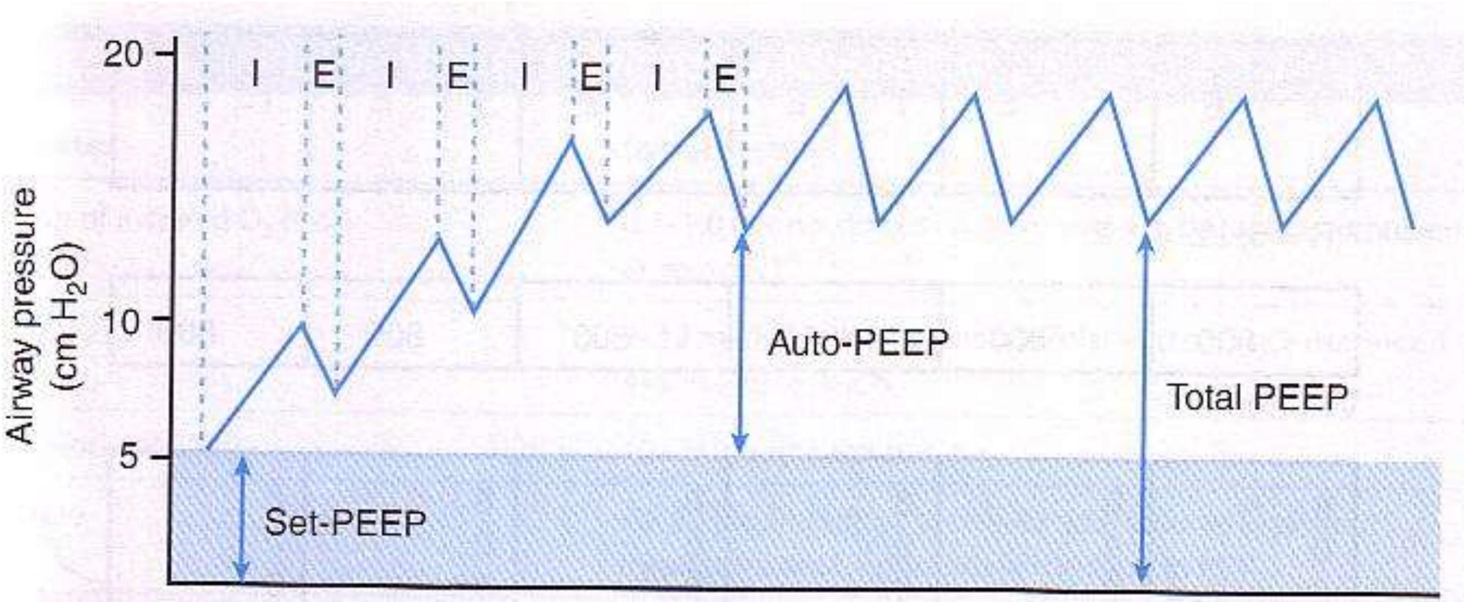
Standard ventilation and IRV's effects on gas distribution in pulmonary units with variable time constants



Efectele ciclului respirator în ventilaţia standard şi IRV asupra distribuţiei gazelor în unităţile pulmonare cu constante de timp variabile



PEEP and auto PEEP associated to IRV



Adverse effects associated to MV

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

PEEP

Hemodynamics

↓CO rapidly→↓DO2 due to ↑P intrathoracic

Adverse effects associated to MV

UNIVERSITATEA DE
MEDICINĂ ŞI FARMACIE
TÎRGU MUREŞ

PEEP

Hemodynamics

↓CO rapidly→↓DO2 due to ↑P intrathoracic

↓CO due to ↓venous return as a consequence of an increase in transmural pressure

↑ PVR due to the transmition of positive pressure to the alveoli →↑ RV afterload →↓ Rvemptying

As RV ESV↑→ displacement of the interventricular septum→↓ diastolic filling of the LV→↓ CO

Adverse effects associated to MV cont

Treatment

Intravascular volume replenishment + inotropics and vasoactive drugs

!!! For a PEEP $\geq$ 10cmH_2O use a Swan Ganz catheter

Adverse effects associated to MV

Hemodiynamics cont

↑intrathoracic pressure may improve LV function by effectively ↓afterload

Sudden disconnection from MV may speed up the flash pulmonary oedema by an acute of afterload + ↑ venous return

↑ Ventilated zones vs perfused due to an ↑ intrathoracic pressure + supine ↑ V/Q >1 ↑dead space

↑ Dyspnoea, anxiety, discomfort due to inadequate support → stress related cathecolamine release →↑ myocardial oxygen demand, ↑arhythmia risk

Adverse effects associated to MVcont

UNIVERSITATEA DE
MEDICINĂ ȘI FARMACIE
TÎRGU MUREȘ

Physical effectsh– mecanical

Barotrauma – overdistention, ↑peak inspiratory pressure. Incid 7-25%

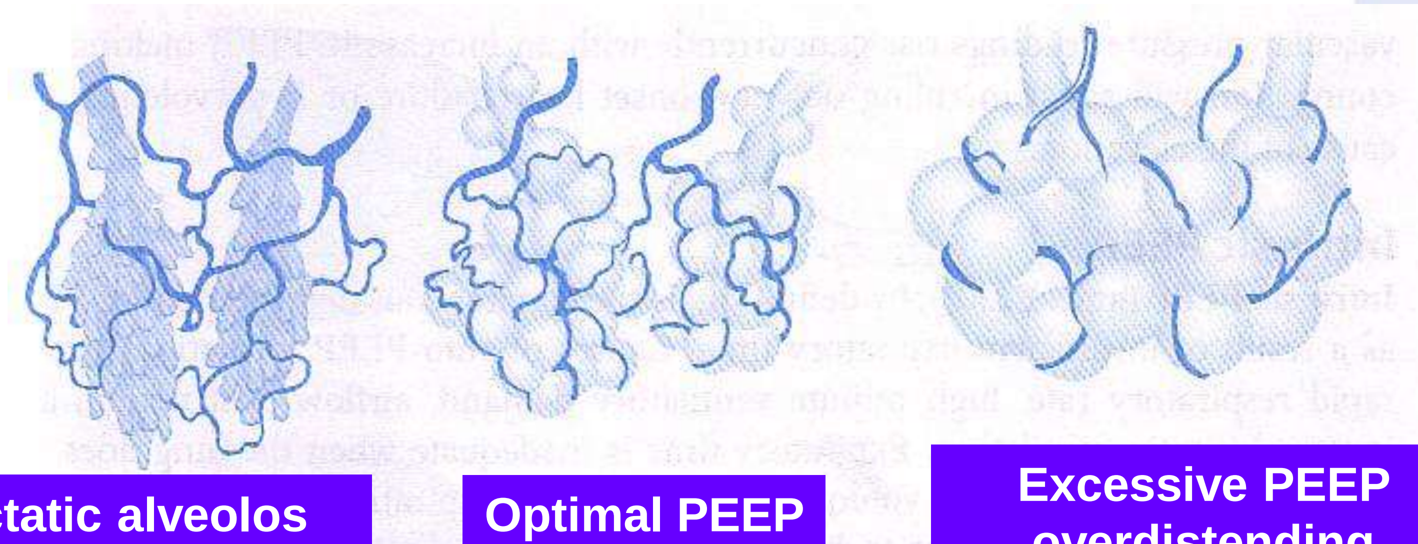
Incid of pneumothorax identical for HFJV and standard MV

Carbon GC et al, Chest 1983; 84;551

Gluch HE et al , Chest 1993; 103: 1413

Adverse effects associated to MVcont

Inhomogenous ventilation due to PEEP



Procesele necrotizante →↑incidenţei barotraumei

Adverse effects associated to MVcont

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

VILI ventilation induced lung injury

Cause: excessive distention

- **Alveoli rupture**
  - **Pneumpmediastinum**
  - **Pneumopericardium**
  - **Subcutaneous emphysema**
  - **Pneumothorax**
  - **Gazeous emboli**

Adverse effects associated to MV cont

UNIVERSITATEA DE MEDICINĂ ȘI FARMACIE TÎRGU MUREȘ

- **parenchimal injury for transpulmonary distension pressures >30-35cmH2O = diffuse alveolar injuries , ↑↑cytokines, bacterial translocation**
- **Friction forces – repetitive opening/closure of the alveoli (collapse)**
- **Acceleration of the initial rapid flows in the lungs**
- **The concept of "protective ventilation" with values < normal**

cont

Desincronizarea pacient/ventilator

3 faze ale ciclului respirator asistat: trigger, target, cycle

Oricare în contratimp→oboseală musculară

↓

Lupta cu ventilatorul

↓

Sedare excesivă

↓

↑suportului ventilator

Efectele adverse asociate VM cont

UNIVERSITATEA DE
MEDICINĂ ȘI FARMACIE
TÎRGU MUREȘ

Desincronizarea pacient/ventilator

3 faze ale ciclului respirator asistat: trigger, target, cycle

Oricare în contratimp→oboseală musculară

Efectele adverse asociate VM cont

Desincronizarea pacient/ventilator

3 faze ale ciclului respirator asistat: trigger, target, cycle

Oricare în contratimp→oboseală musculară

↓

Lupta cu ventilatorul

Adverse effects associated to MV cont

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ

Patient/ventilator dysynchrony

3 phases of the assited respiratory cycle : trigger, target, cycle

Any couter time→muscular fatigue

↓

Fighting the ventilator

↓

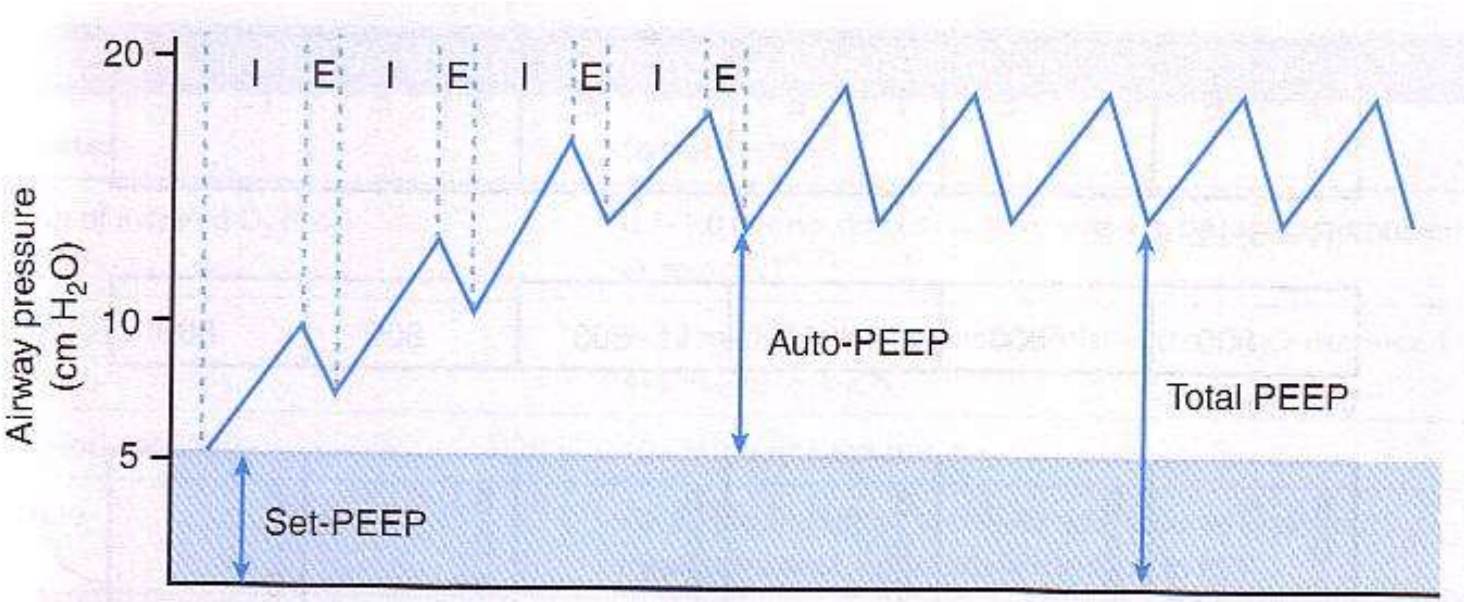
Excessive sedation

↓

↑ventilatory support

PEEP and auto PEEP associated IRV

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MUREŞ



Total PEEP total = auto PEEP + preset PEEP

Adverse effects associated to MVcont

UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE TÎRGU MURES

Hyperoxygenation injury

Health related infections

- Protective mechanism of glotus eliminated →continuous flow of the oropharyngeal secretions into the trachea.
- Atracheal cannula triggers cough reflex = pathogens entry site – circuit contamination
- Parenchimal injury responsible for MV and treatment of complications opens the way to infections
- ICU – antibiotics heavily, severe patients APACHE 2> 26, SOFA > 4
- VAP

Theretically and virtually , all pts with a TC are colonnized with the prevalent germ within 48 hours MacInyre, Textbook of Crit Care, 6th Ed, JL Vincent et al, Elsevier, Saunders, 2011

VAP prevention

Antibiotic strategies

Manipulating the circuits – change only if visible contamination

Subglottic continuous drainage

BAL – inaccurate, confounding

Gastrointestinal hemorrhage

Why VM?

Why VM?