




Noninvasive and invasive ventilation in Obstructive Pulmonary Diseases


Dorel Sandesc
"V. Babes" University of Medicine and Pharmacology
Timisoara


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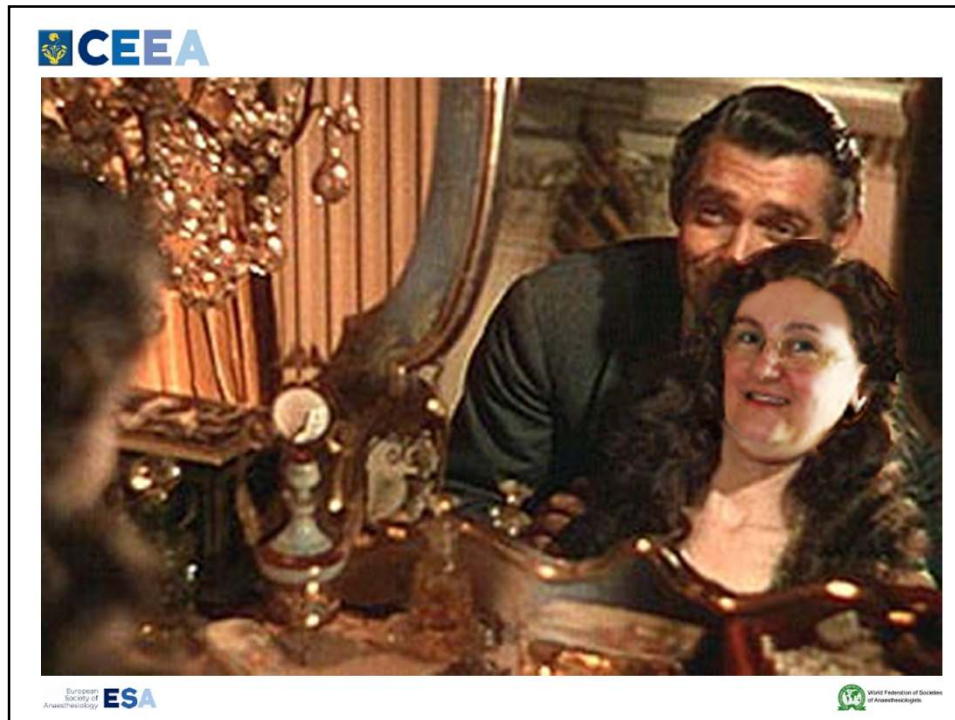
 World Federation of Societies of Anaesthesiologists





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CEEA

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Pathophysiology

Non-Invasive Ventilation(NIV)

Mechanical Invasive Ventilation


European Society of Anaesthesiology ESA

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Are You At Risk?

The World Health Organization (WHO) projects that by the year 2020, COPD will be the 4th leading cause of death or disability.



COPD

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is a lung disease in which the lung is damaged, making it hard to breathe. COPD can be diagnosed through a Computerized Pulmonary Lung Function Test available at Lorma's Pulmonary Unit.


For more information, see your doctor or contact Lorma's Pulmonary Unit at:


072-700-0000


LORMA

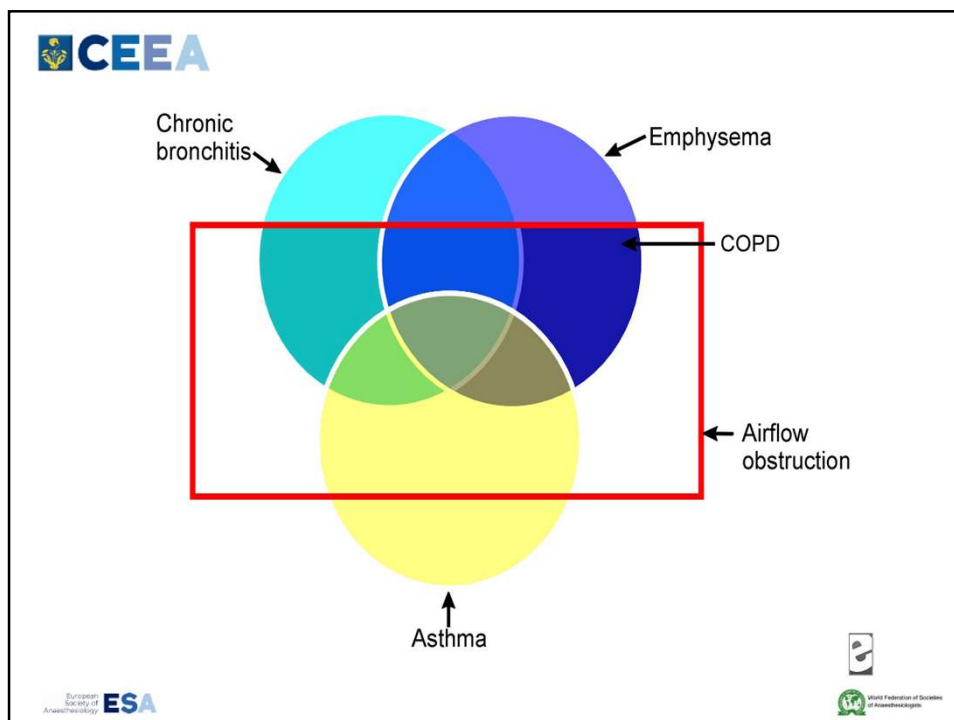
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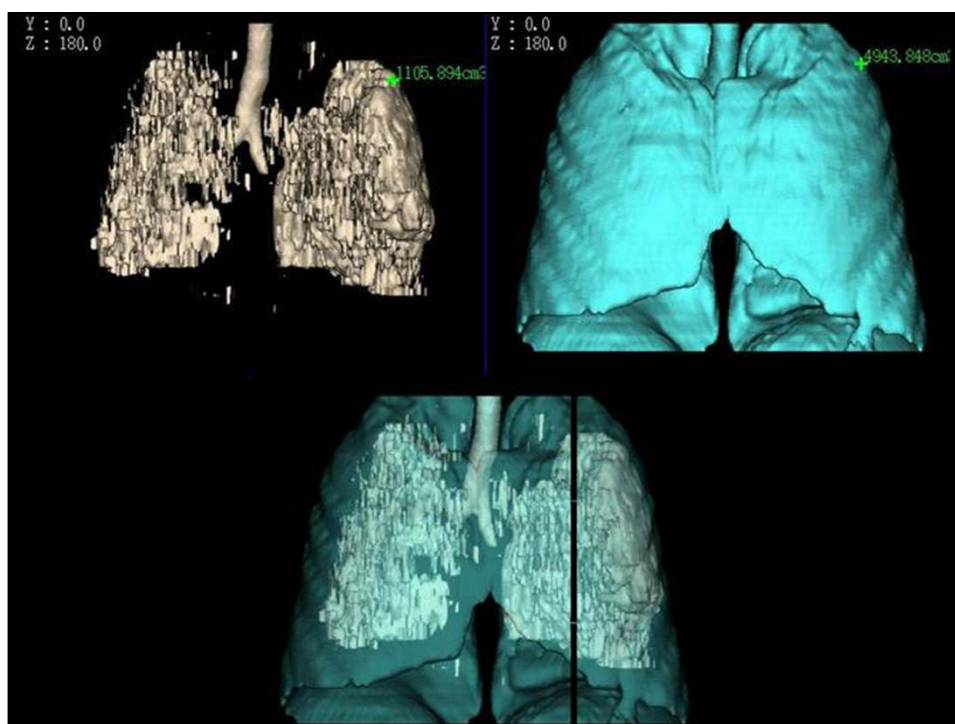
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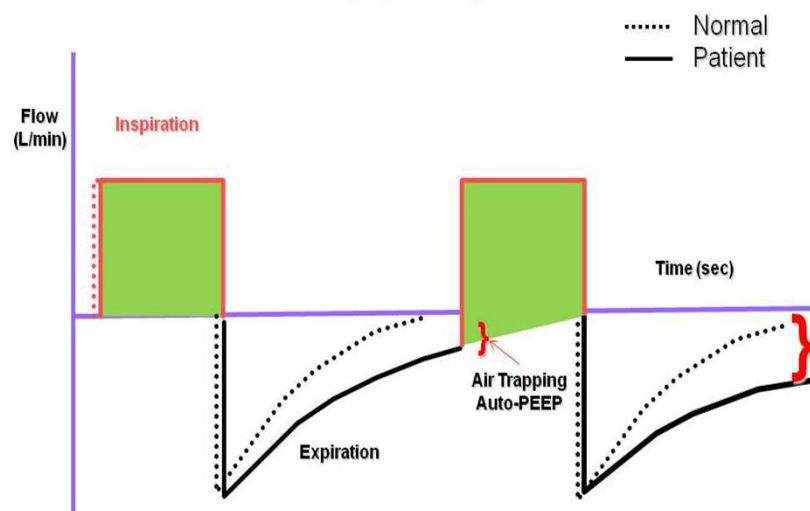
Auto-PEEP/ Intrinsic PEEP/Air trapping/Dynamic Hyperinflation

What is Auto-PEEP/DHI?

- Normally, at end expiration, the lung volume is equal to the FRC
- When PEEPi occurs, the lung volume at end expiration is greater than the FRC



Air Trapping

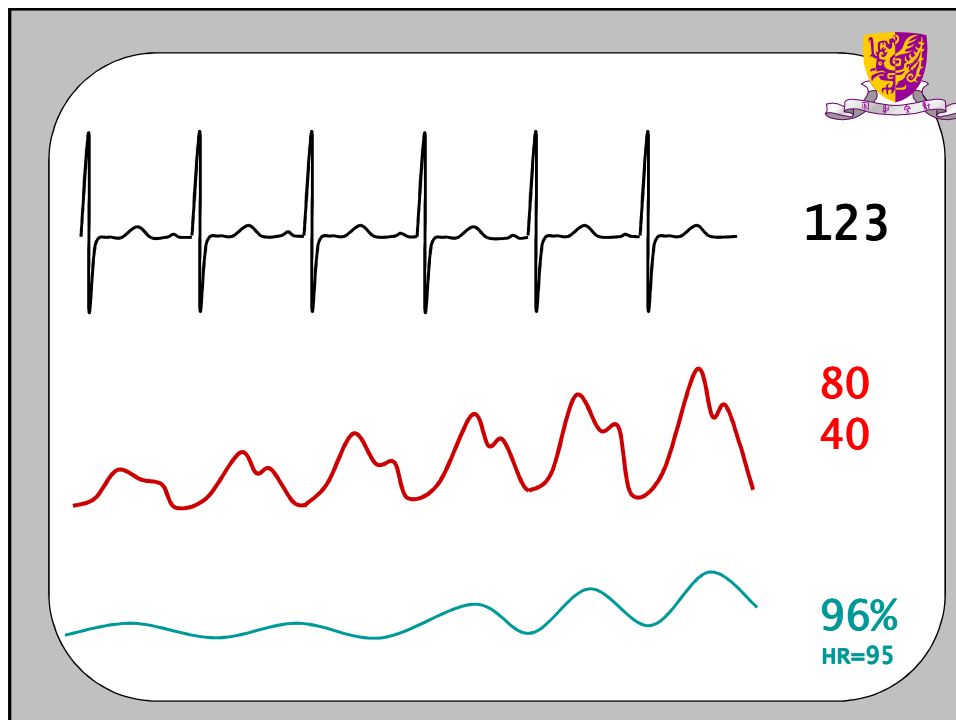




Clinical consequences of DHI

Table 2. IMPORTANT CLINICAL CONSEQUENCES OF iPEEP AND DHI

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- Respiratory effects
- ↓ effectiveness of respiratory muscles
 - Ineffective triggering of ventilator
 - ↑ workload and respiratory muscle fatigue
 - ↑ intrapulmonary pressures and risk of barotrauma
- Cardiovascular side-effects
- ↓ venous return
 - ↓ right and left ventricular preload
 - ↑ right ventricular afterload
 - ↓ left ventricular compliance
 - electromechanical dissociation
- Incorrect physiological measurements
- underestimates airways
 - overestimates compliance
-





COPD and cardiovascular mortality

Forced expiratory volume in one second (FEV_1):

- second to smoking
- above blood pressure and cholesterol

as a predictor of all-cause and cardiovascular mortality !!!



COPD-increased risk of cardiovascular disease and death: pathogenetic models

Inflammation model

Pulmonary Hypertension model

Lung hyperinflation model

Shared genetics model





The Pulmonary Hypertension Model

Factors affecting PH in COPD

Airflow limitation

Emphysema

Alveolar hypoxia

Hypercapnic acidosis

Polycythaemia

Lung/systemic inflammation

Impact on pulmonary vessels

Pressure swings

Reduction of vascular bed

Vasoconstriction/remodeling

Vasoconstriction

Hyperviscosity

Remodelling, including fibrosis

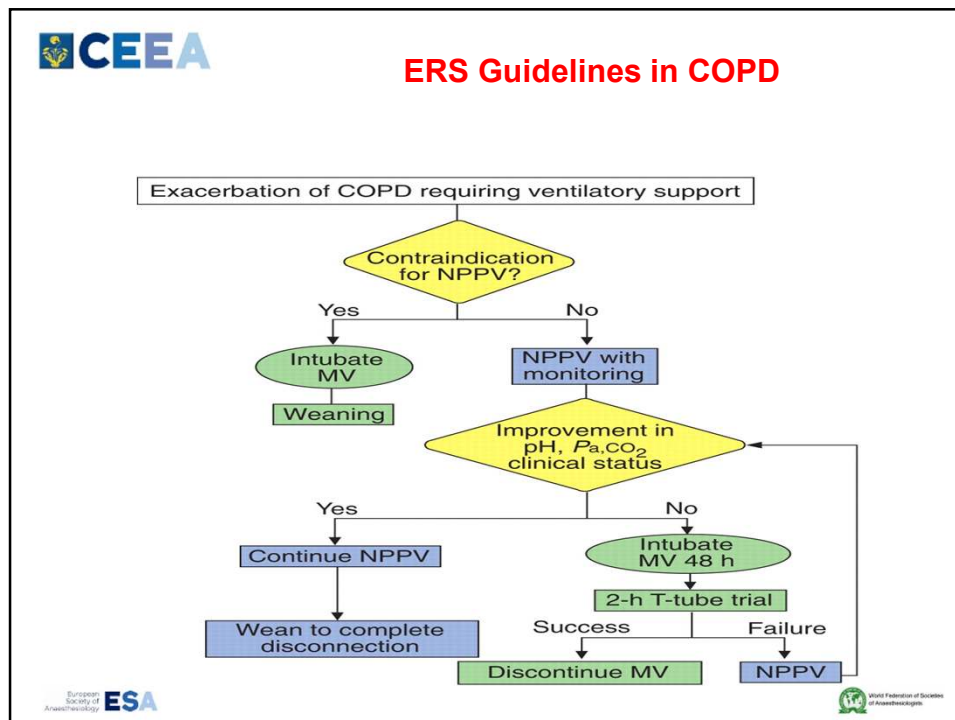
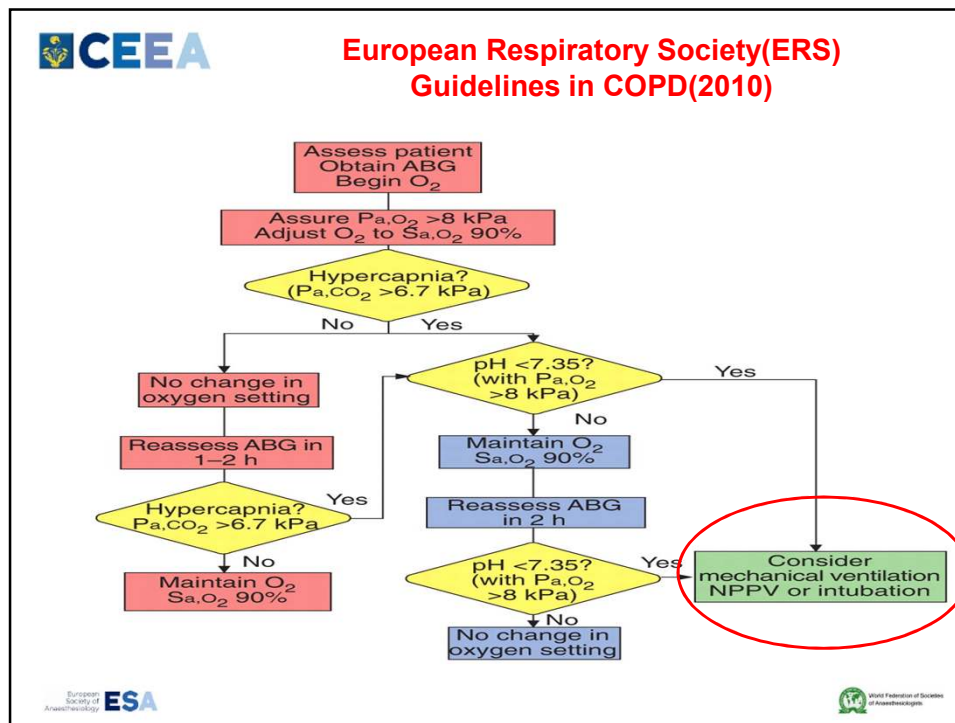


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Non-invasive ventilation in chronic obstructive pulmonary disease: British National Guidelines 2008



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British National Guidelines 2008 NIV: Inclusion criteria

COPD in whom a respiratory acidosis persists despite maximum standard medical treatment, which includes:

- controlled oxygen to maintain SaO₂ 88–92%
- nebulised salbutamol 2.5–5 mg
- nebulised ipratropium 500 µg
- prednisolone 30 mg
- antibiotic agent (when indicated), ETC.

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British National Guidelines 2008 NIV: Exclusion criteria

Life-threatening hypoxaemia
 Severe co-morbidity
 Confusion/agitation/severe cognitive impairment
 Facial burns/trauma/recent facial or upper airway surgery
 Vomiting
 Fixed upper airway obstruction
 Undrained pneumothorax
 Upper gastrointestinal surgery
 Inability to protect the airway
 Copious respiratory secretions
 Haemodynamically unstable requiring inotropes/pressors (*unless in a critical care unit*)
 Patient moribund
 Bowel obstruction



British National Guidelines 2008 NIV: Set-up

- The patient in a sitting or semi-recumbent position
- A full-face mask should be used for the first 24 hours, followed by switching to a nasal mask if preferred by the patient (C)
 - An initial inspiratory positive airway pressure (IPAP) of 10 cm H₂O and expiratory positive airway pressure (EPAP) of 4–5 cm H₂O should be used (A)
 - IPAP should be increased by 2–5 cm increments at a rate of approximately 5 cm H₂O every 10 minutes, with a usual pressure target of 20 cm H₂O or until a therapeutic response is achieved or patient tolerability has been reached (A)



British National Guidelines 2008 NIV: Set-up

- Oxygen, when required, should be entrained into the circuit and the flow adjusted to achieve the target saturation, usually 88–92%.¹⁹ (B)
- Bronchodilators, although preferably administered off NIV, should as necessary be entrained between the expiration port and face mask. (Delivery of both oxygen and nebulised solutions is affected by NIV pressure settings (A)
- If a nasogastric tube is in place, a fine bore tube is preferred to minimise mask leakage



British National Guidelines 2008 NIV: Monitoring

Clinical observations including:

- respiratory rate, heart rate
- level of consciousness, patient comfort
- chest wall movement, ventilator synchrony, accessory muscle use

Frequent clinical monitoring of acutely ill patients:

- every 15 minutes in the first hour
- every 30 minutes in the 1- to 4-hour period
- hourly in the 4- to 12-hour period



British National Guidelines 2008 NIV: Monitoring

Baseline observations:

- arterial blood gas (ABG)
- respiratory rate
- heart rate

Continuous pulse oximetry and electrocardiogram (ECG)

Repeat ABGs:

- after 1 hour of NIV therapy and 1 hour after every subsequent change in settings
- after 4 hours, or earlier in patients who are not improving clinically



British National Guidelines 2008 NIV: Monitoring

Patient comfort and enhanced compliance are key factors in determining outcome(A) !!!

- Synchrony of ventilation should be checked frequently
- A clinical assessment of mask fit to include skin condition and degree of leak should be performed at the same time.



British National Guidelines 2008 NIV: Weaning

- Initially weaning should be during the day
- Extended periods off the ventilator for meals, physiotherapy, nebulised therapy etc.
- After successfully weaning during the day, many patients will require an additional night on NIV (C)
- The weaning strategy should be documented in the medical and nursing records (C)



British National Guidelines 2008 NIV: Weaning

The following is recommended (A):

- continue NIV for 16 hours on day 2
- continue NIV for 12 hours on day 3 including 6–8 hours overnight use
- discontinue NIV on day 4, unless continuation is clinically indicated.

Note that some patients may:

- *show at an earlier stage that they no longer require NIV and self-wean*
- *improve rapidly, prompting a clinical decision to wean early*
- *require long-term nocturnal support, indicated following assessment by the respiratory team.*



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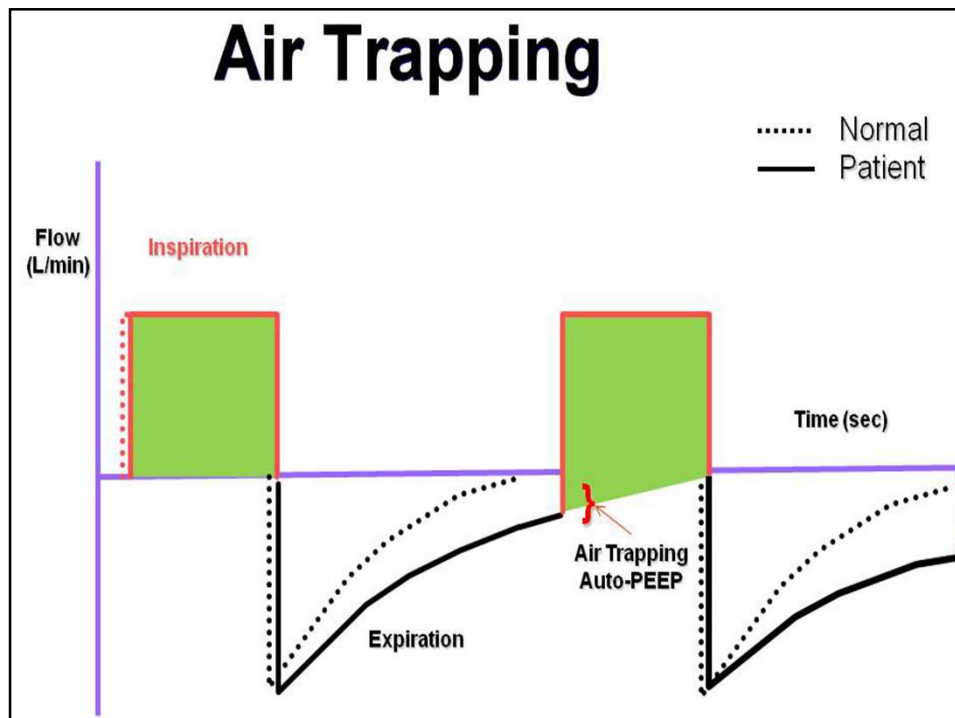
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-



Ventilation in COPD and Asthma

Goals:

1. Diminish dynamic hyperinflation
2. Diminish work of breathing
3. Controlled hypoventilation (permissive hypercapnia)



Which mode of ventilation ?

- Volume controlled ?
- Pressure controlled ?

In terms of barotrauma and cardiovascular effects it is alveolar pressure that is important, not airway pressure !



Which mode of ventilation ?

Volume control

- Lung compliance relatively normal =alveolar pressure unlikely to be high.
- Airway pressure possible high- but does not matter.

Pressure control

- Tidal volume will be low because of high resistance in airways
- If pressure set very high: risk of barotrauma and volutrauma when asthma “breaks”.



Assist control-options

Longer inspiratory time ?

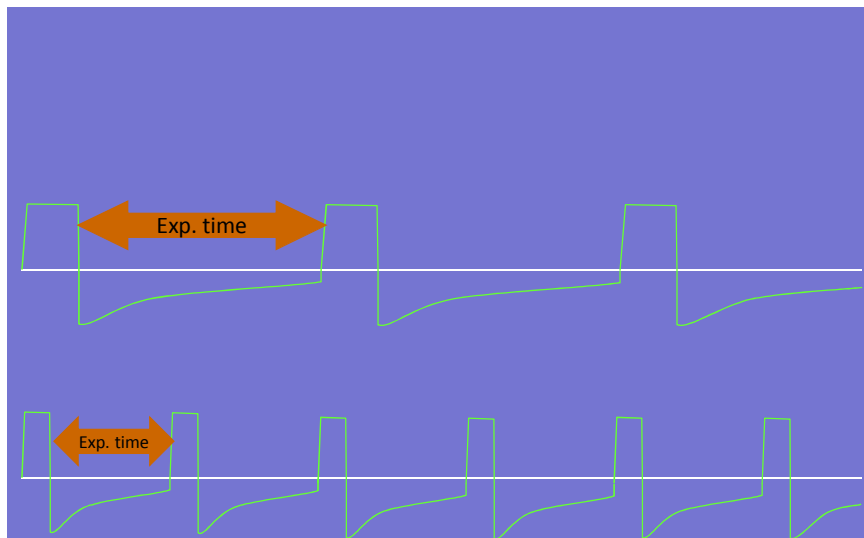
- Improved oxygenation
 - Higher mean airway pressure
 - Re-distribution
- Lower peak airway pressure
 - More time available to deliver set tidal volume

Shorter inspiratory time ?

- Longer expiratory time
- Less risk of gas trapping
- Less effect on cardiovascular system



Assist control-options: Respiratory Rate





Assist control-options

Inspiratory pause ?

- Lungs held in inspiration
- More even distribution of ventilation
- Improves oxygenation
- Additional to set inspiratory time
- BUT reduces the expiratory time !



Ventilation goals1: Diminish DHI

How?

- Low V_t (6-8 cc/kg)
- Low RR (8-10 b/min)
- Maximize expiratory time (low I:E ratio)

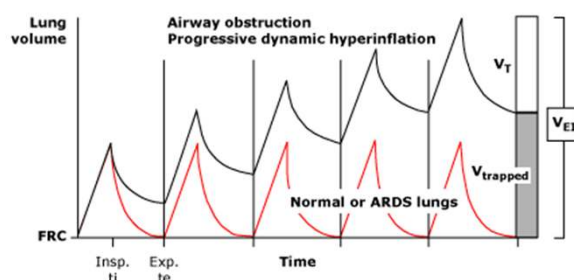
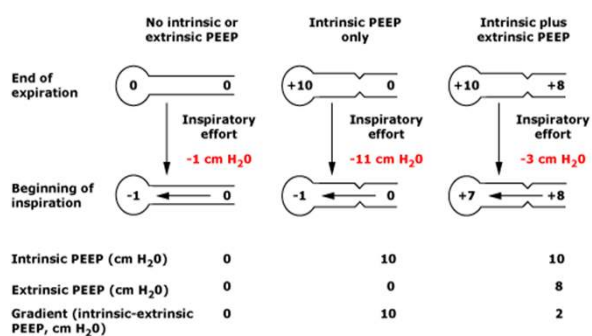


Ventilation goals 2: Diminish work of breathing

To PEEP or not to PEEP ?



Vent Settings Asthma





Ventilation goals 2: Diminish work of breathing

How:

- Add PEEP (about 85% of PEEPi)
- Applicable in COPD and Asthma.



3. Controlled hypercapnia

Why?

- Limit high airway pressures and thus diminish the risk of complications





Controlled hypercapnia

How?

- Control the ventilation to keep adequate pressures up to a PH > 7.20 and/or a PaCO₂ of 80 mmHg



Controlled hypercapnia

CI:

- Head pathologies
- Severe HTN
- Severe metabolic acidosis
- Hypovolemia
- Severe refractory hypoxia
- Severe pulmonary HTN
- Coronary disease





Vent Settings Asthma

Respiratory rate 8- 10 breaths/min
 (allows more time for exhalation)
 Low I:E ratio(>1:2)
 Tidal volume less than 8 mL/kg
 Inspiratory flow of 80 to 100 L/min
 Extrinsic positive end-expiratory pressure *less than* 80
 percent of the intrinsic PEEP
 Continue inhaled medications and steroids



Cursul de Ghiduri si Protocoale in ATI si Medicina de Urgenta, editia X Timisoara, 4-6 Oct 2012

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Thank you, bye !

