# **Respiratory failure**

#### (Respiratory insuficiency)

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## **Respiratory failure** – definition

 Failure of ability to secure the metabolic needs of organism i.e. proper <u>oxygenation</u> and <u>excretion of CO2</u> Clinical signs of RF are **not always presen**, it is necassary the examination of **blood gases** 

For diagnosis is necessary: to folow development of clinical signs and blood gas analysis

# Sings and symptoms

- Dyspnea
- Tachypnea
- Hypoxemia

**Result of non adequate gasses exchange are acid base disturbances.** 

Presence of acidosis is not main criterion for setting diagnosis, but acid base shift (movement) alows to define duration, cause and folowed treatment of RF.

We should evaluate : respiratory and metabolic part, level of compensation, if the disorder is acute or chronic Partial respiratory failure (hypoxemia) – Type 1
 PaO<sub>2</sub> < 55 – 60 torr</li>
 PaCO<sub>2</sub>< 40 torr</li>
 PA-aO<sub>2</sub> increased (over 10 torr)

• <u>Global RF</u> (hypoxemia + hypercapnia) – Type 2 hypoxemia + retention of CO<sub>2</sub>)  $PaO_2 < 55 - 60$  torr  $PaCO_2 > 45$  torr  $PA-aO_2$  normal or increassed pH decreased

#### Acute /Astma bronchiale, /ARDS

- begin within minutes and days
- hypoxemia
- respiratory alkalosis or acidosis
- immediatly life threatening

#### Chronic /COPD/

- begin within days or months/years
- hypoxemia
- hypercapnia and metabolic compensation
- pulmonary hypertension
- potentionnaly life threatening

## Based on this evaluation we have 3 types of resp. acidosis:

- Acute: resp. acidosis non compesated
- Acute mixed: (respiratory+metabolic) non compensated
- **Chronic:** respiratory acidosis partialy or fully compensated

## From clinical view:

- Partial (hypoxemic RF, hypoxemia) Type 1
- Global (hypoxemia + hyperkapnia) Type 2

## Etiology of RF

Lung pathology **Obstructive diseases** Upper airways (Laryngitis, Epiglotitis) Lower airways (Astma bronchiale, COPD,) Restrictive diseases (ARDS, Pneumonia, Cystic fibrosis, Emphysema) Trauma of lung (contusion, PNO, Hemo, Fluido) Outside lung pathology Cardiac dysfunction (Pulm.edema: Arrhytmia, Congestive heart failure, Valve pathology) Neurological disease (Cerebrovascular Accident, CNS, stem, perif.nn.) muscles or chest diseases wall Intoxic. with drugs (Mo, diazepins) that supress resp.

#### **Arterial hypoxemia**

**Definition: decreased partial preassure of oxygen in blood less than 60mmHg /8,0kPa or causing Hb O2 saturation of less than 90%** 

Causes

- a) low concentration in inspired gas (FiO<sub>2</sub>)
- b) alveolar hypoventilation
- c) impaired oxygen diffusion
- d) ventilation perfussion inequality
- e) shunt
- f) desaturation of mixed venous blood

#### **Hypoxemie – etiology**

atelectasis

diffuse lung infitrations

oedema

ARDS

unilateral lung diseases

#### Shunt (Qs/Qt) =

# % of venous blood with no contant with fully healty alveolo-capillary membrane

Normal values 3 – 5 %

Need of mechanical ventilation - about 30%

Hypoxemic index PaO<sub>2</sub> / FiO<sub>2</sub>

normal values above 400 need of MV below 200

shows, how is the function of lung impaired regardless of etiology of lung desease

### Therapy

**T1 respiratory failure:** oxygen therapy

<u>T2 respiratory failure:</u> (oxygen therapy and also need of elimination of CO2) = mechanical ventilation

## Oxygen therapy goals:

- Concentration of oxygen
  flow v. rebreathing
  (n. canula, f. mask, rebreathin mask, CPAP mask)
- Warm
- Wet nebulisation

   (humidification in mechanical ventilation also use for drug administration: broncholytics, mucolytics

## Toxicity of oxygen

- Emergency situations no problem
- Chronic aplications: over hours (14 hours?)
   danger concentr. > 50% retrolental fibroplasia brochopulmonal dysplasia lungs fibrosis

#### **Mechanical ventilation**

CMV – control /countinous/ mechanical ventilation

IPPV - intermitent positive pressure ventilation

Total ventilatory support
 Partial ventilatory support

#### CMV

- Volume control ventilation VCV
- Pressure control ventilation PCV

- Intermitent mandatory ventilation IMV
- Synchr. interm. mand. ventilation sIMV PsIMV VsIMV

## **Objectives**

 ® Understand how ventilators control breath delivery - phase, type and control variables.
 ® Understand the basic adjuncts and modes of ventilation.

# Phase Variables

® Trigger (start)- begins inspiratory flow
 ® Cycling (end)- ends inspiratory flow
 ® Limiting (continue)- places a maximum value on a "control variable"

pressure

volume

flow

time

## Breath Type... Only Two (for now)!

- <u>Mandatory</u>
  - Ventilator does the work
  - Ventilator controls start and stop
- ®Spontaneous
  - Patient takes on work
  - Patient controls start and stop

# Control

®Delivery of a mandatory breath at a set time interval time is the trigger to start the breath



### **Volume Control Breath Types**



# Pressure Control Ventilation -PCV

®The ventilator delivers a set pressure limit over a set inspiratory time



# Volume vs... Pressure Control Ventilation Volume Ventilation Pressure Ventilation

- ® Volume delivery constant
- Inspiratory pressure varies
- Inspiratory flow constant
- Inspiratory time determined by set flow and V<sub>T</sub>

- Nolume delivery varies
- Inspiratory pressure constant
- Inspiratory flow varies
- Inspiratory time set by clinician

# Assist, Assist Control

® Patient is able to trigger the start of inspiration



# Synchronize Intermittent Mandatory Ventilation - SIMV

® A minimum mandatory breath rate is set with spontaneous breathing supported between the mandatory cycles



# Pressure Support Ventilation -PSV

The ventilator delivers a set pressure limit with end inspiration driven by the patient



# Safety Issue - PSV

® PSV is a spontaneous mode of ventilation, therefore the patient must demonstrate they can trigger the ventilator and that volumes are appropriate

<sup>®</sup> High and low rate, apnea, and high and low tidal volume alarms need to be assessed

# Potential Complications of MV

 Ventilator malfunction
 Manually ventilate patient ® Barotrauma Alveolar rupture due to overdistention Monitor PIP, breath sounds Pulmonary Oxygen toxicity ® goal: FIO<sub>2</sub>  $\leq$  .50 and PaO<sub>2</sub>  $\geq$  70 Cardiovascular compromise/arrhythmias R Monitor vital signs

# Potential Complications of MV

#### Infection

- ET tube bypasses natural airway defense mechanisms
  - Nosocomial pneumonia, aspiration pneumonia
- Good handwashing, provide mouth and tube care

#### Psychological

- Patients may be extremely anxious and/or agitated
- Give consistent, calming explanations, offer reassurance
- Sedation, anti-anxiety agents frequently indicated

## **Basic Ventilator Parameters**

# FiO<sub>2</sub> Fractional concentration of inspired oxygen delivered expressed as a % (21-100) Breath Rate (f)

R

R

The number of times over a one minute period inspiration is initiated (bpm)

#### ® Tidal volume (V<sub>T</sub>)

The amount of gas that is delivered during inspiration expressed in mls or Liters. Inspired or exhaled.

#### <sup>®</sup> Flow

The velocity of gas flow or volume of gas per minute

## PEEP

#### ® Definition

- Positive end expiratory pressure
- Application of a constant, positive pressure such that at end exhalation, airway pressure does not return to a 0 baseline
- Ised with other mechanical ventilation modes such as A/C, SIMV, or PCV
- Referred to as CPAP when applied to spontaneous breaths

## PEEP

- Increases functional residual capacity (FRC) and improves oxygenation
  - Recruits collapsed alveoli
  - Splints and distends patent alveoli
  - Redistributes lung fluid from alveoli to perivascular space



## **CPAP**

#### ® Definition

- Continuous positive airway pressure
- Application of constant positive pressure throughout the spontaneous ventilatory cycle
- No mechanical inspiratory assistance is provided
  - Requires active spontaneous respiratory drive
- ® Same physiologic effects as PEEP


- ® May decrease WOB
- ® Tidal volume and rate determined by patient



**CPAP: continuous positive airway** pressure

**PEEP:** positive end exspiratory presure

Most frequently used at present time:

PsIMV x CPAP/PS

## Indications

 RF
 + other causes: circulatory failure brain oedema multiple trauma to decrease energetic comsuption

### some notes:

RF

Impaired oxygenation Impaired ventilation

#### some notes :

- Ventilation ....?
- Respiration ....?
- Regulation of breathing ... brain stem...

pO<sub>2</sub> pCO<sub>2</sub> pH

- Compliance.....?
- Resistence

relationship.....Pressure v. Flow
( stenosis of upper airways )

## Mechanical ventilation

- Intubation : orotracheal nasotracheal tracheostomy
   Bypas of airways to warm
  - to make wet
  - to make wet
  - elimination of secrets

# medication

- Analgesics.....sufentanil
- Hypnotics.....midazolam
   propofol
- Muscle relaxants ?

## Start versus end of MV

- Start .....quick
- End.....weaning.....sometimes takes time

## ARF

RDS resp. distress sy
ARDS adult respiratory distress sy
ARDS acute respiratory distress sy
ALI acute lung injury

#### Definition of Acute Respiratory Dystress Syndrom

- Acute onset of respiratory dystress
- Hypoxemia
  - ALI:  $PaO_2/FiO_2 \leq 300$
  - ARDS: PaO<sub>2</sub>/FiO<sub>2</sub>  $\leq 200$
- Bilateral consolidation of chest radiograph
- Absence of clinical findings of cardiogenic pulmonary edema















• Clinical signs....RF...blood gases...quick onset

- Xray picture....wet lung(shock lung)
- Dif dg: cardiac failure pulmonary edema

# etiology

- Direct damage of the lung
  - aspiration
    difuse infection
    inhalation of toxic gases
    lung contusion
- Indirect damage of the lung
  - sepsis, necrosis, inflamation
    multiple trauma without lung injury, burns
    shock, hypoperfusion
    acute pancreatitis
    cardiopulmonary bypass
     ( mediators)

## **Treatment of ARDS**

- Nothing special
- Monitoring and hemodynamic managament
- Treatment of infection + nutrition
- Avoiding iatrogenec complicatios:
- Support of other organ system functions
- Mechanical ventilation

#### Oxygen v. Inspiratory pressure of MV

## Mechanical ventilation

#### • Lung protective strategy:

- Limit the size of VT 6ml/kg or end-insp.plateau airway pressure lower than 25cm H20
- Increase RR
- Level of PEEP
- Recruitment of the lung
- Permiseve hypercapnia
- Mode of ventilation pressure v. volum-control ventilation, mandatory v. spontaneuos, etc

## **MOD/MOF**

Culmination of general excesiv imune, neuroendocrinne and inflammatory reaction of organism on inzult, leading to failure of individual organs :

- circulatory failure shock
- lung ALI, ARDS
- CNS encefalopathy
- GIT gastritis, colitis, pancreatitis
- coagulation DIC
- metabolism
- imunity
- kidney ARF/AKF
- liver liver dysfunction/failure