Acute Respiratory Failure

István Lőrincz MD, PhD
Division of Emergency Medicine, Institute of Internal Medicine, University of Debrecen
Acute respiratory failure

- The goal of breathing is to fill the blood with the sufficient amount of oxygen necessary for the tissues and clear the blood of carbon dioxide.

- ARF = the insufficiency of the breathing to fulfill the above task— that is insufficient respiratory performance of the lungs.
Acute respiratory failure

- Not an independent entity - it is always a consequence of various pathologic processes

- The cause can be mechanical insufficiency of the breathing or alveolo-capillary dysfunction (hypercapnic and hypoxic types of respiratory insufficiency)
Acute respiratory failure

Classification

- Acute/ chronic respiratory failure
  + acute exacerbation of a chronic process
- Partial or total (global) ARI
  (hypoxia alone or + hypercapnia)
- Ventilation/ Diffusion/ Perfusion abnormalities
- Obstructive or restrictive RI
Alveolar phase of breathing

**Diagram:**
- Alveolus
- Surfactant
- Alveolar epithelium
- Basal membrane
- Capillarisendothel
- Plasma
- Erythrocyte
- Membrane
- Intracellular fluid
- Hgb molecule
- $O_2$
- $CO_2$
Causes of ventilation problems

- Central: CNS - spinal cord
  - Injuries
  - Drug action - e.g. opioids!

- Neurologic, neuromuscular, muscular failures
  - E.g. myasthenia gravis, Gillain Barré sy., muscle relaxants

- Mechanical causes
  - Thoracic cage - rib fractures, burns, scars...
  - Compression of the lungs - hydrothorax, hemothx, pneumothx

- Airway obstruction
  - Upper airway obstruction - foreign body, stenosis...
  - Lower airways - bronchospasm, asthma.....

- Problems in the lung-parenchyma itself
Acute respiratory failure

Acute Lung Injury, Acute Respiratory Distress Syndrome (ALI/ARDS)
Acute bronchospasm – severe asthma
Acute on chronic airflow limitation acute exacerbation of COPD
Severe pneumonia
Pulmonary embolism
Pulmonary edema
Aspiration, inhalation
Clinical signs of respiratory insufficiency

- dyspnoea
- use of ventilatory auxiliary muscles
- cyanosis
- progressive elevation of the resp. rate
- tachycardia
- agitation, confusion, somnolentia, coma
Diagnosis

- Inspection – dyspnoea, thoracic movements, etc.
- Respiratory rate
- Pulsoximetry
- Blood gases (arterial, venous) – repeated!
  - Reaction to oxygen inhalation?
- Asthma: peak flow
- Further investigations:
  - Thorax X ray? CT, MRI
  - Sputum - bacteriology, serology
  - Laboratory testing
  - ECG, US (TEE?)
Acute lung injury, Acute respiratory distress syndrome (ALI/ARDS)

Diffuse lung disease with severe hypoxia—characterized by loss of ventilated alveoli (loss of surfactant activity; edema of the lung tissue)

→ reduced ventilated lung-capacity
→ reduced compliance
→ severe hypoxemia (intrapulmonary shunts)
ALI/ARDS

Diagnosis:
- Thorax x-ray / CT
- Severe hypoxia – not reacting on oxygen inhalation
- $\text{PaO}_2/\text{FiO}_2 < 300$ (ALI) or 200 (ARDS)
- Lung compliance $\downarrow$

Diffuse bilateral infiltration caused not by LV insufficiency ($\text{Paop} < 18 \text{ Hgmm}$)

American/European Consensus Committee 1994
## Causes of ALI/ARDS

### Pulmonary:
- infektion/pneumonia
- aspiration/inhalation
- near drowning
- contusion

### Extrapulmonary:
- sepsis
- trauma
- TRALI
- CPB

**TRALI** = transfusion related acute lung injury  
**CPB** = Cardiopulmonary bypass
ALI/ARDS

A complex interaction between the cells and the inflammatory mediators - lesion of epithelial cells, alveolar macrophags and endothelial cells

- exsudation - edema, inflammation, coagulation disorders
- proliferative phase - regeneration
- fibrotic phase -
Acute bronchospasm, severe asthmatic attack

Components of the insufficiency
- Bronchospasmus
- Edema of the bronchiolar mucous membranes
- Secretion - sticky secretions
- Obscruption of small bronchioli
- Air trapping - Exhalation incomplete

The pressure never returns to zero! "dynamic hyperinflation" (TLC↑, RV↑, FRC↑)

Lung inflation - intrinsic or autoPEEP

Respiratory work elevated - Exhaustion!
Acute asthma: assessment

Pts with severe asthma and one or more adverse psychosocial factors (psychiatric illness, alcohol or drug abuse, unemployment) have t mortality. Measure the peak expiratory flow rate and compare it against that expected. The peak flow acts as an immediate triage tool: remember that pts with life-threatening asthma may be too dyspnoeic to do this. Make an initial assessment of the severity of acute asthma based upon a combination of clinical features, peak flow measurement and pulseoximetry as outlined below.
Moderate exacerbation of asthma

• Increasing symptoms.
• Peak flow 50--75% best or predicted.
• No features of acute severe asthma (below).

Acute severe asthma

Any 1 of:
• Inability to complete sentences in 1 breath.
• Respiratory rate 2:25/min.
• Heart rate 2:110/min.
• Peak flow 33-50% best or predicted.
Life-threatening asthma

A patient with severe asthma with any 1 of:

- Cyanosis.
- Exhaustion. Confusion, coma.
- Feeble respiratory effort.
- SpO2<92%.
- Silent chest.
- Bradycardia, arrhythmia, hypotension.
- pO2<8kPa.
- Normal pCO2 (4.6-6.0kPa).
- Peak flow <33% best or predicted.
Near fatal asthma

• pCO2 and/or requiring mechanical ventilation with inflation pressures.
Other investigations

Obtain ABG if SpO2<92% or if there are other features of life-threatening asthma.

Obtain a CXR (Without delaying treatment) if there is:
• Suspected pneumomediastinum or pneumothorax.
• Suspected consolidation.
• Life-threatening asthma.
• Failure to respond to treatment satisfactorily.
• Requirement for ventilation.
Acute asthma: management

Initial treatment

Follow BTS/SIGN guidelines summarized as follows:

• Provide high flow O2.
• Put the trolley back and side rails up so the pt is sitting up and holding on to the side rails (to use pectoral muscles as accessory m. of respiration).
• If the patient cannot talk, start treatment, but get senior ED and ICU help in case intubation and ventilation are required.
• Check trachea and chest signs for pneumothorax.
• Ask about previous admissions to ICU.
Acute asthma: management

Initial treatment 2

• Administer high dose nebulized β2 agonist (e.g. salbutamol 5mg or terbutaline 10mg), or 10 puffs of salbutamol into spacer device and face mask. For severe asthma or asthma that responds poorly to the initial nebulizer, consider continuous nebulization.

• Give a corticosteroid: either prednisolone 40-50mg PO or hydrocortysone (as sodium succinate) 100mg IV.

• Add nebulized ipratropium bromide (500mcg) to beta2 agonist treatment for patients with acute severe or life-threatening asthma or those with a poor initial response to beta2 agonist therapy.
Acute asthma: management

Initial treatment 4

- Consider a Single dose of IV Mg-sulphate (1.2-2g IVI over 20min) for pts with acute severe asthma without a good initial response to inhaled bronchodilator th. or for those with near-fatal asthma.
- Use IV aminophylline. Some individual pts with near-fatal or life-threatening asthma with a poor response to initial th. may gain additional benefit. The loading dose of IVI aminophylline is 5mg/kg over 20min unless on maintenance th, in which case check blood theophylline level and start IV of aminophylline at 0.5-0.7mg/kg/hr.
Acute asthma: management

Initial treatment

• IV salbutamol is an alternative in severe asthma, after consultation with senior staff. Draw up 5mg salbutamol into 500mL 5% dextrose and run at a rate of 30-60mUhr.

• A patient who cannot talk will be unable to drink fluids and may be dehydrated,

• Avoid 'routine' antibiotics.

• Repeat ABG within an hour.

• Hypokalaemia may be caused or exacerbated by beta2 agonist and/or steroid therapy.
Criteria for admission

Admit patients with any features of:

A life-threatening or near-fatal attack.

Severe attack persisting after initial treatment.
Refferral to intensive care unit

Refer any patient requiring ventilatory support or with acute severe or life-threatening asthma failing to respond to therapy, evidenced by:

- Drowsiness, confusion.
- Exhaustion, feeble respiration.
- Coma or respiratory arrest.
- Persisting or worsening hypoxia.
- Hypercapnoea.
- ABG showing ↓pH.
- Deteriorating peak flow.
Cardiac arrest in acute asthma

The underlying rhythm is usually PEA. This may reflect one or more of the following: prolonged severe hypoxia (secondary to severe bronchospasm and mucous plugging), hypoxia-related arrhythmias or tension pneumothorax (may be bilateral). Give advanced life support according to the guidelines in Cardiac arrest, and treat tension pneumothorax if present. Aim to achieve tracheal intubation early in view of the higher than normal required lung inflation pressures and the attendant risk of gastric inflation in the absence of a tracheal tube.
PEAK FLOW METER
## Arterial blood gases

<table>
<thead>
<tr>
<th>Severity</th>
<th>$\text{PaO}_2$</th>
<th>$\text{PaCO}_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Life danger!</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Pneumonia
– infective infiltration of the lungs

- Epidemiology:
  - Home acquired
  - Community acquired (CAP)
  - Hospital acquired (HAP)
  - Ventilator acquired (VAP)

- Infective agent
  - Bakterial - pneumocc., haemophylus, stacc., mycoplasma
  - Viral pneumonia (influenza, adenovirus, etc.)

- Clinical appearance
  - Typic pneumonia (sudden beginning, high fever, productive cough...)
  - Atypic pneumonia (less characteristic symptoms)
Pulmonary edema

Dynamic balance state:
intravascular – interstitial – alveolar compartments

Starling equation
(fluid movement through semipermeable membranes):

\[ Q_f = \frac{K}{(P_c - P_i)} - \frac{\sigma(P_c - P_i)}{K} \]

- \( Q_f \): filtration rate
- \( K \): filtration coefficient
- \( \sigma \): protein permeability
- \( P_c, P_i \): capillary + interstitial oncotic pressure

Factors:
- Alveolocapillary membrane permeability
- Hydrostatic pressure in the capillaries
- Onkotic pressure in the interstitium
- Capacity of the lymph-system
Common causes of pulmonary edema:

- **Cardial edema:** main cause is the elevated hydrostatic pressure in the pulmonary vessels
  (AMI, IHF, CMP, MS, MI, hypertensive crisis...)

- **Nono cardiac causes:**
  - Chemical irritation (gases, fumes, aspiration of acidic gastric content, etc.)
  - Fluid overload
  - Followinf upper airway obstruction, near drowning
  - Pneumothx (interstitial neg.pressure↓), re-expansion
  - High altitude
  - Infection, sepsis
  - Pharmacons, toxins
    (sedato-hypnotica, salicylate overdose, paraquat...)
  - .....
Therapy

- Elimination of the cause
- Half-sitting position
- Oxygen therapy
- Positive pressure ventilation (IPPV, PEEP)
  - in case of hypercapnia, severe hypoxia Gyógyszeres th:
    - Morphine 5-10 mg IV
    - Furosemide 20-40 mg IV
    - TNG sublingual 0,3-0,6 mg, or spray or IV infusion
Oxygen Supplementation
low flow systems  1-10 LPM

- 100% O2 mixes with room air to determine FIO2 - definition
- FIO2 varies with patient’s breathing pattern
  - Rapid inspiration entrains more room air
  - Deep breaths entrain more room air
  - Rapid respiratory rate entrains more room air
  - Patients in more distress get lower FIO2
- FIO2 is unknown since amount of entrainment is unknown
- Any humidity in gas comes from entrained air- wall O2 has 0% relative humidity
- Low flow devices
  Simple Nasal Cannulas
  Simple masks
High Flow O2 Devices > 20 - 60 lpm

- Device provides 100% of gas to patient - definition
- No entrainment of room air if mask fits
- FIO2 is known and exact
- Relative humidity depends on the device
- High flow devices:
  - High flow nasal cannula
  - Venturi mask
  - Aerosol mask – heated or cool
  - Nonrebreather mask – some characteristics of both high and low
O2 Devices

1. Oxygen Mask
2. Oxygen Mask
3. Oxygen Tank with Bag
4. Oxygen Tubing
Aerosol O2 devices
BiPAP or NPPV

Bilevel positive airway pressure (BiPAP) is a proprietary name of Respironics, Inc. For continuous positive airway pressure (CPAP) with pressure support breaths. It is used during noninvasive positive pressure ventilation. It delivers a preset inspiratory positive airway pressure (IPAP) during inspiration and expiratory positive airway pressure (EPAP). BiPAP can be described as a continuous positive airway pressure system with a time-cycled or flow-cycled change of the applied pressure level. CPAP, BPAP and other non-invasive ventilation modes have been shown to be effective management tools for COPD, acute and chronic respiratory failure. Another term for bilevel positive airway pressure, and the term becoming increasingly adopted by the medical community, is non-invasive positive pressure ventilation (NIPPV) or non-invasive ventilation (NIV).
The setup for BIPAP using a mechanical ventilator
BiPAP or NPPV

- **Contraindications**
  - Cardiac or respiratory arrest
  - Inability to cooperate, protect the airway, or clear secretions
  - Nonrespiratory organ failure, esp shock
  - Facial surgery, trauma, or deformity
  - Prolonged duration of mechanical ventilation anticipated
  - Recent esophageal anastomosis

- A need for emergent intubation is an absolute contraindication to NPPV

- Set inspiratory pressure (IP) and exp pressure (PEEP)

- Mean pressure determines oxygenation

- IP – PEEP determines ventilatory assist
Thank you !