REASONS OF HYPOXEMIA IN POSTOPERATIVE PERIOD

- FiO$_2$ decrease?
- V/Q disturbances – the most frequent
- Lung shunts – secretions, atelectases
- Hypoventilation – anesthesia effects
- Diffusion disturbances – interstitial lung oedema
- Hypoxia from diffusion $N_2O$ 40 x > $N_2$
PREDISPOSITION FACTORS FOR RETENTION OF SECRETIONS

- Surgery site (thoracic, epigastrium > hypogastrium)
- Preexist respiratory disease (infection, hypersecretion)
- Smoking
- Obesity ($\downarrow$FRC, $\uparrow$WOB)
BRONCHIAL SECRETIONS RETENTION IN POSTOPERATIVE PERIOD

- Expectoration insufficiency (painful wound, ↑sedation, muscle weakness ↓K⁺, ↓P)
- ↓ bronchial ciliary activity (dry inspir. gases, anesthetics)
- Antisialologic medicaments (premedication)
- Infection (washing mechanisms failure)
  = ↑atelectases, ↑WOB, hypoxemia, global respir. insuficiency
CONSEQUENCES OF SECRETION RETENTION

• **Atelectasis:**
during 24 h post surgery, fever 38-39°C, tachycardia, tachypnoe, cough, cyanosis?, x-ray - spot obscuration

• **Bronchopneumonia:**
Unusual lobar, elder pats., slower onset than in atelectatic cause, fever, tachypnoe, x-ray – more dense obscuration, especially basally
LABORATORY INVESTIGATIONS

- $p_aO_2$, $p_cO_2$, $p_vO_2$ (ABR + pO$_2$)
- possible mistakes and artefacts
- steady state, taking of blood samples, storage, laboratory
CHANGES OF FRC AND PaO$_2$ IN POSTOPERATIVE TIME
POOSTOPERATIVE PNEUMONIA TREATMENT

- **ATB** according to sputum cultivation
- **Oxygen** $\text{FiO}_2$ 0.3-0.4 according to ABB
- **Artif. ventilat.**, non-responders to $O_2$ and activation of auxiliary respiratory muscles, $\downarrow p_aO_2, \uparrow p_aCO_2$
Fig. 23.2 Gas exchange during hypoventilation. Note the relatively rapid increase in alveolar partial pressure of carbon dioxide ($P_{CO_2}$) compared with the slow decrease in arterial oxygen saturation. $P_{O_2}$ = Partial pressure of oxygen.
WEST’S LUNG ZONES

Zone 1: $P_A > P_a > P_v$

Zone 2: $P_a > P_A > P_v$

Zone 3: $P_a > P_v > P_A$

arterial

venous

alveolar

Distance

Blood flow
VENTILATORY MECHANICS

SPONTANEOUS BREATHING

zone > ventilation

ARTIFICIAL BREATHING

zone > perfusion

ATELECTASES
SITUATIONS FOR LONGER OXYGEN THERAPY DURING POSTOPERATIVE PERIOD

• Hypotension
• IHD
• ↓ C.O.
• Anaemia
• Obesity
• Shivering
• Hypothermia
• Hyperthermia
• Lung oedema
• Airway obstruction
• After large procedures
OXYGEN CASCADE
OXYGEN THERAPY

• Every pt. **10 min** after general anaesthesia should obtain 100% oxygen

• Cave:
  Recovery room
  Postoperative dpt., ICU...
HYPERBAROxIA

1. Air or Gas Embolism
2. Carbon Monoxide Poisoning
3. Clostridal Myositis and Myonecrosis (Gas Gangrene)
4. Crush Injury, Compartment Sy, & Acute Traumatic Ischemias
5. Decompression Sickness
6. Enhancement of Healing in Selected Problem Wounds
7. Exceptional Blood Loss (Anemia)
8. Intracranial Abscess
9. Necrotizing Soft Tissue Infections
10. Osteomyelitis (Refractory)
11. Delayed Radiation Injury (Soft Tissue and Bony Necrosis)
12. Skin Grafts & Flaps (Compromised)
13. Thermal Burns

EQUIPMENT FOR OXYGEN THERAPY

Spont inspiration - Peak Inspiratory Flow = 20 - 30 l/min

- Nasal sonds
- Oxygen „eye-glasses“
- Face mask
- Venturi mask 4l ≈ 28%, 8l ≈ 40%, 15l ≈ 60%
- CPAP ≈ 10 cmH₂O
- Artificial ventilation
VENTI MASK
SELF-EXPANDING BAG WITH O₂

Inflow O₂
10 - 13 l/min

<table>
<thead>
<tr>
<th></th>
<th>O₂ l/min</th>
<th>FiO₂ %</th>
<th>Vₜ x f</th>
</tr>
</thead>
<tbody>
<tr>
<td>adult</td>
<td>13</td>
<td>85-100</td>
<td>1000 x 15</td>
</tr>
<tr>
<td>-</td>
<td>4</td>
<td>&gt;40</td>
<td>dtto</td>
</tr>
<tr>
<td>child</td>
<td>5</td>
<td>85-100</td>
<td>300 x 20</td>
</tr>
<tr>
<td>-</td>
<td>2</td>
<td>&gt;40</td>
<td>dtto</td>
</tr>
</tbody>
</table>
NECESSITY OF FiO₂ CONTROL

• Normally inspiratory force is regulated by PaCO₂ level (5.3 kPa)
• Patients regulated inspiratory force by paO₂ (chronic bronchitis), 1-2 l/min O₂, 28% O₂

• ARDS: paO₂/FiO₂ = shunt amount
Fig. 23.5  Response of arterial partial pressure of oxygen ($P_{O_2}$) to increased inspired oxygen concentrations in the presence of various degrees of shunt. Note that arterial $P_{O_2}$ remains well below the normal value when 100% oxygen is breathed. Nevertheless, useful increases in arterial oxygenation occur with a shunt of up to 30%.
Fig. 1.10 The oxyhaemoglobin dissociation curves for normal adult haemoglobin at normal pH and with acidosis and alkalosis.
Regulation of inspiratory force by PaO₂ decrease

In higher FiO₂ threat respiratory depression

Fig. 23.9 Effect of controlled oxygen therapy on oxygen saturation in a hypoxaemic chronic bronchitic patient. A small increase in inspired oxygen concentration produces a modest increase in arterial oxygen tension (PaO₂) but a substantial increase in arterial oxygen saturation.
BYPASS OF NASAL CAVITY

Water vapour content r.h. 100% in $37^\circ$C = 44 mg/l
$V = 10$ l/min $\Rightarrow$ 0,4 ml H$_2$O/min, i.e. 24 ml H$_2$O/hod

$\approx 600$ ml/24 hod

+ hyperventilation !
+ fever !

Inspiration of dry air =

= losses 700-1000 ml/day

Aim: humidification to 100% r.h.
warming 37$^\circ$C
CONSEQUENCES OF INSUFFICIENT HUMIDIFICATION OF INSPIRATORY GASES

• Losses of clear water from airway
• Concentration of secretions in airway
  – failure of mucociliary transport
  – airway obstruction
  – development of atelektases
  – airway infection
AIMS OF VENTILATORY SUPPORT

• Bypassing of critical time during illness.
• Achievement of acceptable oxygenation and ventilation parameters.
• Elimination of side effects of artificial ventilation.
PHYSIOLOGICAL AIMS

1. Alveolar ventilation, \( \text{paCO}_2 \), pH,
2. Oxygention support, \( \text{paO}_2 \) above 8 kPa, \( \text{SaO}_2 \) above 90\%, \( \text{CaO}_2 \). Transport blood capacity for oxygen (\( \text{HB} \times \text{SaO}_2 \times \text{C.O.} \))
3. Increase of lung volume (FRC) - PEEP (against athelectases, improving of oxygenation, ARDS, postoperative cases)
4. Decreasing work of respiratory muscles.
CLINICAL AIMS

1. Treatment and reverse of hypoxaemia
2. Treatment of acute respiratory acidosis
3. Solution of respiratory distress
4. Prophylaxis and treatment of atelectases
5. Help for respiratory muscle weakness
6. Permission of pts. sedation / relaxation
7. Systemic and / or myocardial decreasing of oxygen consumption (cardiogenic shock, ARDS)
8. Decreasing of ICP through \(\text{paCO}_2\)
9. Thoracic wall stabilisation (flail chest)
INDICATIONS FOR ARTIFICIAL VENTILATION

• **Mechanics**: f above 35/min, VC below 15 ml/kg, Insp.neg pressure below 25 cmH₂O.

• **Oxygenation**: paO₂ below 70 mmHg at FiO₂ 0.4 by mask, AaDO₂ above 350 mmHg at FiO₂ 1.0 or Qs/Qt above 20% (paO₂/FiO₂ below 200)

• **Ventilation**: Apnoe, paCO₂ above 55 mmHg (except pat with chron. hyperkапnia), Vd/Vt above 0.60.

• **Crucial are clinical findings!**
PHYSIOLOGICAL AND PATHOPHYSIOLOGICAL PRINCIPLES OF ARTIFICIAL VENTILATION

- Airway securing during ventilation
- Apparatuses and equipment used during ventilation
- Tactics of ventilation, ventilatory modes
- Monitoring of ventilation
- Complications of ventilation
- Weaning from ventilator
## SOME PARAMETERS USED AS INDICATORS OF ARTIFICIAL VENTILATION

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Physiologic values</th>
<th>Indications for artificial support</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity (ml/kg)</td>
<td>60 – 70</td>
<td>10 – 15</td>
</tr>
<tr>
<td>$V_D/V_T$</td>
<td>0,3</td>
<td>0,6</td>
</tr>
<tr>
<td>Inspiratory force (cmH$_2$O)</td>
<td>– (80 – 100)</td>
<td>&lt; – 20</td>
</tr>
<tr>
<td>Lung shunt (%) ($Q_s/Q_t$)</td>
<td>5</td>
<td>&gt; 15</td>
</tr>
<tr>
<td>$paO_2$ (kPa) (at FiO$_2$ = 0,21)</td>
<td>13</td>
<td>&lt; 6,6</td>
</tr>
<tr>
<td>$p(A – a)DO_2$ (kPa) (at FiO$_2$ = 0,21)</td>
<td>1,3 – 4</td>
<td>&gt; 350</td>
</tr>
<tr>
<td>$paO_2/FiO_2$ (kPa)</td>
<td>&gt; 40</td>
<td>&lt; 40</td>
</tr>
<tr>
<td>$paO_2/FiO_2$ (mmHg)</td>
<td>&gt; 300</td>
<td>&lt; 300</td>
</tr>
<tr>
<td>$paCO_2$ (kPa)</td>
<td>5,3</td>
<td>7,3 – 8</td>
</tr>
<tr>
<td>pH</td>
<td>7,35 – 7,45</td>
<td>&lt; 7,25</td>
</tr>
<tr>
<td>Respiratory frequency</td>
<td>12 – 18</td>
<td>&gt; 35 – 40</td>
</tr>
</tbody>
</table>

|
**Phases of the ventilatory cycle**

Mechanical ventilation breaths can be distinguished between controlled, assist-control, assisted-spontaneous and (fully) spontaneous breaths, according to the kind of management of:

- Breath initiation
- Inspiration
- Cycling to exhalation

<table>
<thead>
<tr>
<th>Breath type</th>
<th>Breath initiation</th>
<th>Inspiration</th>
<th>Cycling to exhalation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled</td>
<td>Machine Frequency setting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assist-control</td>
<td>Patient Inspiratory trigger Machine Frequency setting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assisted-spontaneous</td>
<td>Patient Inspiratory trigger Pressure control Pressure support setting</td>
<td>Patient Expiratory trigger</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Patient Inspiratory trigger Pressure control PEEP/CPAP setting</td>
<td>Patient Expiratory trigger</td>
<td></td>
</tr>
</tbody>
</table>
AYRE T-PIECE

FGF < 2 x MV

Ø 22 mm (20 cm ≈ 80 ml)

Inlet $O_2$

Face mask

Tube - orotracheal
- nasotracheal
- tracheostomy

Atmosphere

Ø 22/15 mm
(S)CMV, Assist Control

- I:E $\approx$ Peak Flow $\approx$ %Ti
- Pause (%) $\approx$ Tip (s) from total respir. cycle
AIRWAY PRESSURE (cm H₂O)

SV
CPAP

CMV
Assist / control ventilation

IMV
IMV + CPAP

PSV
PSV + CPAP

IRV
APRV
BLOOD GASES INFLUENCE BY ARTIFICIAL VENTILATION

\[ p_{aCO_2} \]
- ↑ alveolar ventilation
  (VT, f, P_{insp} ...)

\[ p_{aO_2} \]
- ↑ FiO₂
- ↑ mean P_{aw}
  (PEEP, I:E ≈ Peak Flow ≈ %Ti,
  Pause (%) ≈ Tip (s))