Oxygen therapy and mechanical ventilation
Gas exchange at the alveolo-capillary level

- **Alveolo-capillary driving force:** $P_{\text{part.}}$ difference
- **Diffusion ability of the gases:**
  - For $O_2$: 20 ml/min/mmHg $\Delta P_{O_2}$
  - For $CO_2$: 250 ml/min/mmHg $\Delta P_{CO_2}$
- **Surface of the (open) alveoli** (70$m^2$)
- **Diffusion distance** (alv.-capillary)
- **Pulmonary capillary circulation**
Maintainance of proper CO$_2$

- Appropriate tidal volume
- Appropriate frequency
Appropriate tidal volume

- **Force of ventilation**
  - Central drive
  - Peripheral force

- **Static and elastic properties of the lung (underlying condition)**

- **Extrapulmonary causes (thoracic cavity, abdominal pressure)**
  - Obesity
  - Stiffness of the thoracic cavity (Parkinsonian disease)
  - Scoliosis
Appropriate frequency

- **Central regulation**: pH of the CSF in the lower brainstem → acidosis causes increase in
  - TV
  - Respiratory rate
- **Blockade of the lower respiratory center by**:
  - Drugs
  - Diseases
# Types of respiratory insufficiency

<table>
<thead>
<tr>
<th></th>
<th>PaO₂</th>
<th>PaCO₂</th>
<th>AaDO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>partial, type 1. (oxygenisation failure)</strong></td>
<td>↓</td>
<td>n (↓)</td>
<td>↑</td>
</tr>
<tr>
<td><strong>global, type 2 (ventilatory failure)</strong></td>
<td>↓</td>
<td>↑</td>
<td>n</td>
</tr>
<tr>
<td><strong>mixed, type 3</strong></td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
RI pathomechanisms

Ventilatory failure

Low FiO₂

vent-perf distribution failure

Diffusion failure

ventilation failure

hypovent

hypervent

Simple distrib. failure

shunt

Dead space

atelectasis

central

peripheral
**Ventilatory disturbances**

- Primary CO\(_2\) change
- *alveolar hypoventilation*

<table>
<thead>
<tr>
<th>Central</th>
<th>peripheral</th>
</tr>
</thead>
<tbody>
<tr>
<td>brainstem ischemia</td>
<td>Muscle diseases (myasthenia, CIMP)</td>
</tr>
<tr>
<td>herniation</td>
<td>Radicular and peripheral nerve diseases (MS, GB, CIP)</td>
</tr>
<tr>
<td>Drug effect</td>
<td>Spinal cord injury</td>
</tr>
<tr>
<td>severe metab. alkalosis</td>
<td>Thoracic wall injuries</td>
</tr>
<tr>
<td>or acidosis</td>
<td>Restrictive disorders (callus, HTX, abdominal distension)</td>
</tr>
<tr>
<td></td>
<td>Obstructive disorders (emph, asthma, bronchitis)</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
</tr>
<tr>
<td></td>
<td>Medications</td>
</tr>
<tr>
<td></td>
<td>Electrolyte disturbances</td>
</tr>
</tbody>
</table>
Decision making algorithm

• RI: yes or no?
• Type?
  – PaO$_2$
  – PaCO$_2$
• What is the severity?
  – Physical signs and symptoms
  – Progression
  – pH
  – Horowitz-index (PaO$_2$/FiO$_2$)
Causes of hypoxemia

- Normal
- Low FIO₂
- Ventilation-perfusion disturb. (atelectasis)
- Ventilation-perfusion disturb. (pulmonary embolism)
- Diffusion disturb.
- Shunt
If only $\text{paO}_2$ is low

- Can it be normalized by nasal or face mask oxygen?
- Progression? Type 1 RI turns into type 2 after a certain time, when manifest respiratory muscle hypoxemia is present (respiratory muscle weakness)
- Verify and treat the cause
- CPAP, BiPAP
- NIV?
Oxygen treatment

• Additional measures
  – Determinants of DO$_2$ has to be treated also (CO, Hgb, saturation)
  – Treat the cause of the increased O$_2$ demand (fever, WOB, sepsis)

• Aim
  – Increase O$_2$ concentration
  – Increase oxygen reserve (e.g. before intubation)
  – Improve PaO$_2$
Oxygen treatment

• indications
  – Resuscitation
  – Cardiac insufficiency or AMI
  – Any forms of shock
  – Increased metabolic demand (burns, polytrauma, sepsis)
  – postoperative
  – CO poisoning
  – RI (Type 1 and 2)
    • PaO$_2$ < 60 mmHg
    • satO$_2$ < 90%
Ideal device for oxygen treatment

- Sufficient FiO$_2$
- Controlled FiO$_2$
- Independent from patients breathing activity
- Able to provide 100% oxygen
- Avoidable CO$_2$ retention
- Minimal respiratory effort needed
- Tolerable
# Oxygen treatment devices

<table>
<thead>
<tr>
<th>Fix oxygen delivery</th>
<th>Unstable or uncertain oxygen delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Devices with unknown capacity</td>
</tr>
<tr>
<td></td>
<td>Nasal cannula &lt; 2 l/min</td>
</tr>
<tr>
<td></td>
<td>Low flow breathing circuits</td>
</tr>
<tr>
<td></td>
<td>Nasal cannula &gt; 2 l/min</td>
</tr>
<tr>
<td></td>
<td>Simple oxygen mask</td>
</tr>
<tr>
<td></td>
<td>Tracheostomy mask</td>
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<tr>
<td></td>
<td>T-piece</td>
</tr>
<tr>
<td></td>
<td>Face tent</td>
</tr>
<tr>
<td></td>
<td>Reservoir face mask</td>
</tr>
<tr>
<td></td>
<td>Oxygen tent</td>
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</tbody>
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# Oxygen treatment

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<tr>
<th>device</th>
<th>O2 flow (l)</th>
<th>O2 concentration (%)</th>
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<tbody>
<tr>
<td>Nasal cannula</td>
<td>2-6</td>
<td>25-40</td>
</tr>
<tr>
<td>Pharyngeal cannula</td>
<td>4-15</td>
<td>35-70</td>
</tr>
<tr>
<td>Simple face mask</td>
<td>6-10</td>
<td>35-50</td>
</tr>
<tr>
<td>Reservoir bag mask</td>
<td>5-15</td>
<td>70-95</td>
</tr>
<tr>
<td>T-piece</td>
<td>2-10</td>
<td>21-90</td>
</tr>
<tr>
<td>Anaesth circuits</td>
<td>1-8</td>
<td>21-100</td>
</tr>
<tr>
<td>oxygen tent</td>
<td>7-10</td>
<td>60-80</td>
</tr>
<tr>
<td>Venturi mask</td>
<td>6-12</td>
<td>24, 28, 25, 40, 50, 60</td>
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## Oxygen treatment devices

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Oxygen treatment, side effects

– hypercapnia (dead space increase)
– hypercapnia (COPD)
– absorption atelectasis
– oxygen toxicity (100%, > 60%)
Reabsorption atelectasis

- Develops with time
- Regional gas uptake > delivery
- No gas left in pulmonary units → the opening pressure is higher
Alveolo-capillary diffusion

- **Driving force:** partial pressure difference between alveoli and pulmonary capillaries
- Partial pressure and NOT concentration difference!
- Determinants of diffusion:
  - Driving force (partial pressure difference)
  - Other determinants of diffusion
Other determinants of diffusion

- Alveolar surface (alveoli getting in contact to capillaries ~ 70 m²)
- Diffusion distance
- Diffusion coefficient of the gas: $\text{CO}_2$ diffuses easier
Diffusion capacity

• $V^*O_2 = D O_2 \times (P_{AO2} - P_{pulcap-O2})$

• Diffusion capacity (D)
  – Proportional to the surface and to the diffusion coefficient
  – Inversely related to the diffusion distance

• Oxygen: 20 ml/min/mmHg $\Delta P_{O2}$

• CO$_2$: 250 ml/min/mmHg $\Delta P_{CO2}$
Consequences

- **Oxygen** the main determinant of alveolar gas exchange is the diffusion
- **CO₂** the main determinant of the alveolar gas exchange is the chemical reaction in the blood (because alveolo-capillary diffusion occurs easily)
Diffusion disturbance

- $O_2$-therapy increases alveolo-arterial pressure difference
- Mechanical ventilation:
  - Increases pressure difference and
  - Diffusion surface area
Indications of mechanical ventilation

- Reverse hypoxemia
- Reverse acute respiratory acidosis
- Relieve respiratory distress
- Prevent or reverse atelectasis
- Reverse respiratory muscle fatigue
- Permit sedation and/or neurom. blockade
- Decrease systemic or myocard. O2 consumption
- Reduce intracranial pressure
- Stabilize chest wall
Basic forms of mechanical ventilation

• Patient effort
  – None: controlled mode
  – Needs support: assisted mode
  – Uncertain or changing: assist-control mode

• According to invasivity
  – Invasive
  – Non-invasive

• Administered pressure
  – Positive
  – Negative (iron lung)
Breath delivery principles

- Start of inhalation: triggering mechanism
- Gas delivery principle: flow/volume or pressure
- Termination of inhalation: cycling mechanism
- Maintainance of expiratory pressure level
Respiratory cycle

2
Trigger

1
Inspiration:
- Control:
  - Flow
  - Pressure
- Limit:
  - Pressure
  - Time

Expiration:
- Pressure (PEEP)

Chatburn classification

3
Cycle

1
Control and Limit

- Inspiration:
  - Control:
    - Flow
    - Pressure
  - Limit:
    - Pressure
    - Time

- Expiratory trigger:
  - Time
  - Flow
  - Pressure
  - Volume

• Inspiratory trigger:
  - Time
  - Pressure changes
  - Flow changes
Components of breath delivery

Flow

Time

Triggering
Inspiration
Cycling
Expiration
Start of inhalation: time trigger
Start of inhalation: patient trigg.

Pressure Trigger

Flow Trigger
Why pressure limit (and not volume)?
Cycling

- Volume (Flow x Ti)
- Pressure
- Time (Ti)
- Flow (ETS)
Maintenance of exp. pressure

Paw

PEEP

time
Controlled mechanical ventilation

- All parameters are controlled by the physician:
  - **MV**: TV x frequency
  - **TV**: 10 ml/kg BW for healthy lung, 6-8 ml/kg BW for diseased
  - **Frequency**: ~ 15/min (higher if HV is necessary)
  - **FiO₂**: (21-100%; < 60% is desired)
  - The pressure in the expiratory phase (PEEP)
- Excellent in non-breathing patients because minute volume is guaranteed
- In neurocritical care it is rarely indicated:
  - No spontaneous breath
  - Spontaneous breath is blocked by deep sedation/relaxation
Assist-control ventilatory mode

Assist/Control Ventilation

- Controlled breath
- Pressure
- Assisted breath
- No patient effort
- Patient effort
- Volume
- Time, sec
- Pressure, cm H₂O
- Volume, mL
Most frequently used assisted modes

- CPAP
- BiPAP (Bi-Level, DuoPAP)
- APRV
- Spontaneous mode (ASB)
- SIMV
CPAP

- The zero-line of the airway pressure is elevated
- Inspiration and expiration occur at this higher pressure level
- Frequency is determined by the patient
- MV (TV) is not guaranteed
- Rationale: avoiding the closure of the airways
- May be used both in invasive and non-invasive modes
BiPAP mode

- Bi-level CPAP
- Total frequency is determined by the patient
- Preset frequency: blow-up (rate/min)
- MV is not guaranteed
- Rationale: re-open the parts of airways that almost collapse at lower CPAP levels
- May be used both in noninvasive and invasive modes
Spontaneous mode (ASB)

- Patient determines both the frequency and TV
- Every initiated breath initiates pressure support breath
- MV is not guaranteed
- May be used both for noninvasive and invasive modes
Intermittent mandatory ventilation (IMV)

• Mandatory positiv pressure breaths in preset time intervals (TV is guaranteed)

• Spontaneous breaths from a reservoir between mechanical breaths

• Problem: no synchrony between patient effort and mechanical breaths (danger of lung overdistension)
SIMV

- Minute volume is guaranteed
- Total frequency is determined by the patient
- Preset SIMV frequency is for mandatory breaths
- Excellent:
  - for patients with unstable spontaneous breathing
  - for suppressing central hyperpnea and
  - for weaning
Ventilator-Induced Diaphragmatic Dysfunction

Prolonged Controlled Mechanical Ventilation

- Diaphragmatic Atrophy and Contractile Dysfunction
- Diaphragmatic Oxidative Stress (Increased ROS production & decreased antioxidant capacity)

- Altered Diaphragmatic Gene Expression
- Diaphragm Fiber Ultrastructural Changes
Non-invasive ventilation

- Different modes may be used
- Cooperating patients
- Different devices
- Possibilities of FiO\textsubscript{2} are limited in home devices
- Excellent for intermittent use
- Improves weaning
Devices we are using for NIV

Intrahospital care

Home care
Most important parameters to preset

- TV
- frequency
- Sensitivity 8trigger)
- FiO2
- PEEP
- Inspiratory flow
Tidal volume

- No ALI: 10 ml/kg
- ARDS: outcome is better in case of low TV
  - 6 ml/kg
  - 12 ml/kg
- Why: large volumes may cause lung injury
  - Microvascular permeability increases
  - Pulmonary edem
  - Lung rupture
- Ideal plateau pressure (where no overdistension develops)?
  - 35 cmwater
  - 5 ml/kg TV is needed
- Permissive hypercapnia: low TV decreases ventilation and increases $\text{PaCO}_2$
Frequency

- ACV: 4/min (only for backup is needed)
- SIMV: 10/min. at the beginning, should be increased when needed
- PSV: no frequency preset is necessary
Sensitivity

- May be:
  - Flow
  - Pressure
  - Time
- Usually it is negative pressure: 1-3 H2Ocm
- Appropriate trigger sensitivity is important: low sensitivity → too frequent triggering → danger of respiratory alkalosis
FiO2

- High FiO2 potentially toxic
- Limit: 0.6
- Goal: the lowest FiO2, resulting in acceptable oxygenation
- What is acceptable?
  - 60 mmHg O2
  - O2 saturation above 90%
PEEP

• In healthy adults: end-tidal intrathoracic pressure is nearly equal to atmospheric pressure, intrapleural pressure is slightly negative (this is why there is FRC)
• FRC= reservoir, from where necessary pCO2 and pO2 can be maintained during exspiration and during respiration pause
• ARDS: FRC usually decreases
  – Decreased surfactant production
  – Alveolar instability (alveolar collaps)
• Goal of treatment: increase of the median airway pressure → PEEP
RI pathomechanisms

Ventilatory failure
- hypovent
  - central
- hypervent
  - peripheral
- Low $\text{FiO}_2$
- vent-perf distribution failure
  - Simple distrib. failure
- Diffusion failure
  - shunt
  - Dead space
  - atelectasis
Ventilatory failures

• Types:
  – Hyperventilation
  – Hypoventilation

• CO₂ change is primary: ↓or↑

• Hypoventilation: respiratory drive decreases due to central or peripheral cause → support is necessary by one of the assisted modes (SIMV, BiPAP, CPAP, APRV)

• Hyperventilation
  – Treat the cause (metabolic acidosis)
  – Decrease frequency (WOB↓)
    • SIMV frequency with or without sedation
    • Sedation/relaxation temporary → average SIMV frequency

• TV usually can be 10 ml/tskg (spontaneous + support)
How to decrease pCO$_2$ during mechanical ventilation?

- Higher TV
- Higher frequency
- Other measures (mucolysis, desuction)
Causes of hypoxemia

- Normal
- Low fiO2
- Ventilation-perfusion disturb.
  (atelectasis)
- Ventilation-perfusion disturb.
  (pulmonary embolism)
- Diffusion disturb.
- Shunt
What are the main problems in alveolo-capillary gas exchange?

- Transssudate, or exsudate within the alveoli
- Thickening of the alveolo-capillary membrane
- Increased alveolo-capillary diffusion distance, shunt
Diffusion disturbance

- $O_2$-therapy increases alveolo-arterial pressure difference (FiO2)
- Mechanical ventilation:
  - Increases pressure difference and
  - Diffusion surface area
How to improve $pO_2$ during mechanical ventilation?

- Increase $FiO_2$
- Increase mean alveolar pressure
- Increase PEEP
What is the first measure to improve alveolo-capillary gas exchange?

• Transsudate, or exsudate in the alveoli
  – FiO$_2$
  – pressure
• Thickening of the alveolo-capillary membrane: FiO$_2$
• Increased alveolo-capillary distance, shunt
  – FiO2
  – Pressure
ARDS

Acute Respiratory Distress Syndrome (ARDS)

Normal Anatomy

CO₂

Normal gas exchange across thin alveolar walls allowing the uptake of fresh oxygen and the release of carbon dioxide.

Cut-section through Alveoli at Terminus of Bronchi

Acute Respiratory Distress Syndrome (ARDS)

CO₂

CO₂

CO₂

CO₂

Fluid releasing from capillaries filling the alveolar space and preventing gas exchange.
TV and ARDS mortality

- 6 ml/kg
- 12 ml/kg
Uneven distribution of PEEP increase
Types of atelectasis in ARDS

- Compression atelectasis
- Reabsorption atelectasis
Compression atelectasis

- Due to increased lung weight
- Develops immediately
- Collaps of the small airways at end-expiration
- The opening pressure is relatively low (12-20 cmH₂O)
Reabsorption atelectasis

- Develops with time
- Regional gas uptake > delivery
- No gas left in pulmonary units → the opening pressure is higher
P/V curve

Alveolar closure

LIP

UIP

Distension of open alveoli

overdistension
Recruitment

- UIP
- Overdistension
- Safe zone
- Alveolar closure
- LIP
- PEEP
- VT
Other important measures

- Hemodynamic monitoring and support
- Bronchodilator- desuction
- Antibiotics
- Monitoring (no tempomat)
- Breathing excercise
- Weaning protocols