# Complications of UPV/HS (nurse)

- Complications arise due to securing the airways (complications of intubation, tracheostomy,...)
- From an improperly prepared inhaled mixture.
- From prolonged exposure of the respiratory tract to high concentrations of O2.
- Infectious complications (decrease/loss of DC reflexes, deterioration of mucociliary transport function,...).
- Pulmonary complications due to positive pressure ventilation.
- Extrapulmonary complications due to positive pressure ventilation.

## Complications related to intubation

- Damage to the oral cavity, larynx, trachea, vocal cords.
- Irritation of the vagus nerve, increased ICP, BP, arrhythmia.
- Introduction of a cannula into one bronchus.
- Introduction of the cannula into the esophagus .
- Laryngospasm, bronchospasm.
- Aspiration of gastric contents.

### Related to long-term OTI

- Tube obstruction - kinking, herniation of the balloon, obturation with secretions, blood.
- Pressure lesions caused by the cannula - lips, nasal and oral mucosa, trachea.
- Formation of tracheoesophageal fistulas.
- Inflammation of the facial sinuses.
- Damage to the vocal cords, formation of stenoses.
- Occurrence of atelectasis due to insufficient ventilation and DC toilet.
- Microaspiration with the development of pneumonia.

## Complications related to tracheostomy

- Bleeding from a wound.
- Incorrect insertion of the cannula (into the surrounding tissues).
- Pneumothorax.
- Infection.
- Cannula obstruction.
- Formation of tracheoesophageal fistulas.
- Tracheal stenosis, tracheomalacia.

## Complications caused by inappropriately adjusted breathing mixture

- Atelectasis occurs when the mixture is insufficiently heated and moistened . The density of sputum increases, the secretion stagnates in the lung areas and is not moved towards the upper respiratory tract.
- Excessive heating of the mixture can cause burns to the endothelium of the bronchi.
- Excessive moistening leads to the formation of a large amount of sputum. Fluid accumulates in the alveoli and gas permeability is very difficult. (The patient is "drowning".)

## Infectious complications

- Pneumonia or bronchopneumonia develops as a result of accumulation of secretions in the airways with insufficient displacement . The cough reflex is usually dampened by deep sitting, which makes it difficult to move secretions outwards.
- Lung damage due to inflammatory reactions is called Biotrauma (Tremblay and Slutsky).
- Release of inflammatory mediators during lung tissue damage by improperly conducted UPV.

## Extrapulmonary complications

### Cardiovascular system

- Influence of cardiac output, redistribution of blood flow to organs, ...
- During positive pressure ventilation:
  - →During inspiration – increase in lung volume above the end-expiratory level.
  - →Spontaneous unsupported breath – intrathoracic pressure drops during inspiration.
  - →During positive overpressure ventilation - intrathoracic pressure rises during inspiration.
- Effect of pulmonary inflation:
  - → Changes in vegetative tone.
  - → Changes in pulmonary vascular resistance.

    - → Mechanical interactions between the lungs and the heart.
- Hemodynamic consequences of changes in intrathoracic pressure:
    - → Transfer of pressure from airways to intrathoracic pressure.
    - → Reduction of venous return.
    - → Affecting the function of the right and left ventricles.

### Water and ion metabolism and renal function

- Aldosterone, adiuretin, natriuretic peptide.
- After initiation of UPV, urine output, renal blood flow, sodium excretion and glomerular filtration are usually reduced by up to 30%.
- The exact mechanism is unknown, several factors are assumed to be involved:
    - Decreased cardiac output.
    - Redistribution of renal blood flow.
    - Increased pressure in the veins.
    - Changes in the tone of the autonomic nervous system (a decrease in mean arterial pressure leads to sympathetic activation).

### Hormonal changes

- Increased secretion of adiuretin leads to redistribution of blood and fluid retention, increased secretion of aldosterone, decreased secretion of atrial natriuretic factor.
- Again, the exact mechanism is unknown, several factors are assumed to be involved:
    - Decreased cardiac output – decreased blood flow to the splanchnic and liver.
    - Increase in hepatic vascular resistance.
    - Increased pressure in the veins.
    - Increased intra-abdominal pressure.
    - Increased pressure in the biliary tract.

## Pulmonary complications

- Aggressive PPV (positive positive pressure ventilation) leads to lung damage - pulmonary edema, atelectasis, hemorrhage, etc.
- VILI = ventilator-induced lung injury = lung damage caused by UPV: in an experiment on animals with healthy lungs.
- VALI = ventilator-associated lung injury = lung damage caused during UPV: in people on UPV, where pre-existing lung pathology cannot be excluded (in practice).

### 3 mechanisms involved in the development of lung damage

- Structural disruption.
- Surfactant dysfunction.
- "Biotrauma" - damage caused by inflammatory response mechanisms.
    - Excessive expansion of the lungs - leads to ruptures of the epithelium, endothelium or all layers of the alveoli and an increase in the permeability of the alveolocapillary membrane.
        - In UPV with high end-inspiratory lung volume.
    - Shear forces – there are very high pressure gradients in the walls of the airways and alveoli at the boundary between aerated and non-aerated areas.
        - For UPV with low PEEP (= positive end-expiratory pressure).
    - Surfactant dysfunction – damage to the surfactant film on the surface of the alveoli by high inspiratory pressure; repeated alveolar collapses at UPV with low PEEP lead to "pumping" of surfactant from the alveoli into the bronchial tree, and the penetration of serum proteins into the alveolar spaces leads to changes in surfactant structure and function, and locally produced pro-inflammatory cytokines lead to a decrease in surfactant production .
    - Lung damage by inflammation = biotrauma.
        - Greater tissue stress - directly causes ruptures of lung structures - subsequently an inflammatory response to the damage will occur.
        - Less tissue stress – activation of the inflammatory response as a result of changes in cell structures.

### Lung damage demonstrably associated with UPV - VILI

- The presence of gas outside the alveolar space, pulmonary edema, destruction of alveoli,
    - → gradually to pseudocysts, pseudoemphysema, bronchodysplasia.
- Factors for the emergence:
    - high end-inspiratory lung volume (EILV),
    - insufficient end-expiratory lung volume (PEEP),
    - endotoxinemia (the presence of G-bacteria toxins in the blood),
    - temperature,
    - pulmonary capillary pressure (cardiac output),
    - respiratory rate,
    - genetic predisposition,
- Mechanism of formation:
    - Excessive tension of alveoli and capillary walls,

    - → high EILV = end-expiratory lung volume (=volotrauma) leads to over-expansion of the lungs → excessive tension of the lung structures,
    - → low PEEP = end-expiratory lung volume (=atelectrauma) – shear forces at the border of ventilated and non-ventilated alveoli.
    - Mechanical bronchial trauma → repeated collapses and opening of small airways (presence of atelectasis).
    - High transcapillary pressure:

→ high intravascular pressure, → normal intravascular pressure in combination with excessive tension of the alveoli and capillary walls will cause an increase in the permeability of the endothelium.

- !!!Manipulation of EILV and PEEP = basis of so-called protective ventilation strategies

# Oxygen toxicity

- The toxicity of oxygen depends on its proportion in the inhaled mixture and on the duration of its action.
    - Oxygen concentration above 60% lasting more than 72 hours is already toxic.
- The lungs react first.

### Lungs

- It causes lung fibrotization, the development of the picture of ARDS.
- A high concentration of oxygen in the alveoli leads to vasoconstriction → a decrease in blood flow in the alveolar capillaries → damage → an increase in cell permeability.
- Surfactant is lost from the damaged alveoli, which leads to the instability of the alveoli and their collapse → pulmonary atelectasis, which cannot be developed even by UPV.
- Symptoms: DC irritation, dry mouth, chest pain, cough, shortness of breath, nausea, vomiting, decrease in lung compliance, vital capacity, increase in capillary permeability with increase in extravascular lung water with subsequent hypoxemia and hypercapnia.

### Sight

- Retinal damage in premature infants due to vascular ingrowth into the vitreous.
- Cataract development.
- Narrowing of the visual field due to vasoconstriction of the retinal vessels.

# Complications from self-ventilation

- Increased intrathoracic pressure.
- Excessive expansion of the lungs.
- At the boundary between the aerated and non-aerated parts of the lungs, small airways are damaged by shearing forces.
- UPV can lead to surfactant inactivation.
- Transfer of surfactant to the bronchi.
- Occurrence of Barotrauma or Volumotrauma.

# Links

### Related Articles

- Atificial pulmonary ventilation/SŠ (nurse)

# Literature


- MUDR. PETR VOJTÍŠEK, *Komplikace při UPV* [přednáška k předmětu Modul UPV, obor Sestra pro intenzivní péči - postgraduální studium, Vyšší odborná škola zdravotnická škola Střední a vyšší zdravotnická škola Ústí nad Labem]. Ústí nad Labem. 20.12. 2012.
- DOSTÁL, Pavel, et al. *Základy umělé plicní ventilace.* 2. edition. Praha : Maxdorf, c2005. ISBN 80-7345-059-3.
- Jan Máca, *VILI - co vlastně poškozuje plíce při UPV?*, Akutně 2018 (https://www.akutne.cz/res/publikace/vili-co-poskozuje-plici-final.pdf)