

Complications of UPV/High School (nurse)

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

Complications related to intubation

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

Related to long-term OTI

- Obstruction of the tube – kink, herniation of the balloon, obturation by secretions, blood.
- Pressure lesions caused by the cannula – lips, nasal and oral mucosa, trachea.
- The formation of tracheoesophageal fistulas.
- Inflammation of the facial sinuses.
- Damage to the vocal cords, the emergence of stenosis.
- Formation of atelectasis in insufficient ventilation and toilet DC.
- Microaspiration with the development of pneumonia.

Complications related to tracheostomy

- Bleeding from the wound.
- Poor introduction of the cannula (into surrounding tissues).
- Pneumothorax.
- Infection.
- Cannula obstruction.
- The formation of tracheoesophageal fistulas.
- Stenosis of the trachea, Tracheomalacia.

Complications caused by inappropriately modified breathing mixture

- With insufficient heating and moistening of the mixture, Atelectases are formed. The density of sputum increases, the secretion stagnates in the lung areas and is not shifted towards the upper respiratory tract.
- Excessive heating of the mixture can cause a burn of the bronchial endothelium.
- Excessive moisturization leads to the formation of a large amount of sputum. The fluid accumulates in the alveoli and the permeability of gases is very difficult. (The patient is "drowning.")

Infectious complications

- As a result of the accumulation of secretions in the airways with insufficient shift, Pneumonia or bronchopneumonia develops. The coughing reflex is suppressed by deep sedation, which makes it difficult to move the secretion outwards.
- Lung damage due to inflammatory reactions is called Biotrauma (Tremblay and Slutsky).
- Release of mediators of inflammation in case of damage to lung tissue by inappropriately conducted UPV.

Extrapulmonary complications

Cardiovascular system

- Influencing the size of cardiac output, redistribution of blood flow to organs, ...
- When ventilated by positive overpressure:
 - →During inspiration – increase in lung volume above end expiratory levels.
 - →Spontaneous unsupported inhalation – intrathoracic pressure decreases in inspiration.
 - →When ventilated by positive overpressure – in inspiration, on the contrary, the intrathoracic pressure rises.
- 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 the airways to intrathoracic pressure.
 - → Reduction of venous return.
 - → Affecting the function of the right and left ventricles.

Water-ion metabolism and renal function

- Aldosterone, adiuretin, natriuretic peptide.
- After initiation of UPV, urine output, renal blood flow, sodium excretion and Glomerular filtration rate are usually reduced by up to 30%.
- The exact mechanism is unknown; several factors are assumed:
 - Decrease in cardiac output.
 - Redistribution of renal blood flow.
 - Increase in 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.
 - Increase in 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 formation of pulmonary damage

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

Lung damage proven to be related to IPV - VILI

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

- Mechanism of formation:
 - Excessive tension of the alveolus and capillary wall,
 - → high EILV = end inspiratory lung volume (=volumotrauma) leads to excessive lung expansion → excessive tension of lung structures,
 - → low PEEP = end expiratory lung volume (=atelektrauma) – shearing forces at the border of ventilated and unventilated alveoli.
 - Mechanical bronchial trauma → repeated collapses and opening of small airways (the presence of atelectasis).
 - High transcapillary pressure:

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

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

Oxygen toxicity

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

Lungs

- It causes fibrotization of the lungs, the development of an ARDS picture.
- 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 damaged alveoli, which leads to alveoli instability and 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, increased capillary permeability with increased extravascular lung fluid with subsequent Hypoxemia and Hypercapnia.

Eyesight

- Damage to the retina in premature babies due to the growth of blood vessels into the vitreous.
- Development of cataracts.
- Narrowing of the visual field due to Vasoconstriction of retinal vessels.

Complications caused by self-ventilation

- Increase in intrathoracic pressure.
- Excessive stretching of the lungs.
- On the border between the aerated and non-airy parts of the lungs, small airways are damaged by shearing forces.
- UPV may lead to inactivation of Surfactant.
- Moving Surfactant to the bronchi.
- The emergence of Barotrauma or Volumotrauma.

Links

Related Articles

- Artificial lung ventilation/secondary school (nurse)

Literature

- MUDR. PETR VOJTÍŠEK, *Complications at UPV* [lecture on the subject Module UPV, field of study Nurse for intensive care - postgraduate studies, Higher Vocational School of Nursing School Secondary and Higher Medical School Ústí nad Labem]. Ústí nad Labem. 20.12. 2012.
- DOSTÁL, Pavel. *Basics of artificial lung ventilation*. 2nd edition. c2005. ISBN 80-7345-059-3.
- Jan Máca, *what actually damages the lungs during UPV?*, Akutně 2018 (<https://www.akutne.cz/res/publikace/vil-i-co-poskozuje-plici-final.pdf>)