ARDS and specifics of APV/HS (nurses)

- Acute respiratory distress syndrome (ARDS) is defined as diffuse cellular dysfunction of the lung parenchyma caused by factors that are part of the body's overall inflammatory response to direct or indirect damage (insult).
- The syndrome itself is then characterized by an abnormal finding in blood gas values and a typical X-ray image of the lungs..
    - Based on the quantification of these abnormal findings, two basic clinical units are defined today – **acute lung injury (ALI)** and **acute respiratory distress syndrome (ARDS)**.
- Mortality 20–60%.

Cause

- Difficult identification of a single insult..
- Shock states.
- Trauma.
- Aspiration.
- Inhalation of toxic substances.
- Systemic intoxication.
- DIC, massive transfusion.
- Others – pancreatitis, malignancies, eclampsia.

Pathophysiological notes

- Increased capillary permeability → non-cardiac swelling of the alveoli.
- Alveolocyte damage → alveoli filled with proteins, tissue debris and cells of the inflammatory reaction

  → compression atelectasis.
- Disorder of ventilation-perfusion balance → hypoxia.
- Dysfunction of surfactant → collapse of alveoli, consolidation → synthesis of collagen, fibrotization → decrease in lung tissue compliance (compliance).
- Bronchoconstriction causes a decrease in functional residual capacity (FRC) and total lung capacity → normal tidal volume. increases ventilation of regional lung areas (baby lung phenomenon) → increased risk of volume trauma.
- The lung parenchyma responds to injury with stereotyped responses.
- During inhalation, some alveoli behave as airy, others permanently non-airy, but after an increase in inspiratory pressure, they become aerated.

Progress

1st phase = acute inflammatory responses.

- **Duration**: hours, days.
- Stimulation of macrophages to produce TNF (tumor necrosis factor) and interleukins.
- Aggregation of neutrophils in pulmonary capillaries, stimulation of protease production.

→ Edema, increase in lung permeability, development of pulmonary hypertension.

2nd phase = subacute phase

- **Duration**: days to two weeks.
- Stimulation of fibroblasts → remodeling of lung tissue → decrease in lung compliance → oxygenation disorder.
- Capillary obstruction due to epithelial damage and a disturbed balance between coagulation and fibrinolysis.
- Pulmonary parenchyma damage by mechanical ventilation.
- Reduction in surfactant production.

3rd phase = chronic

- **Duration**: two weeks to months.
- Proliferative changes → the possibility of pseudocysts, pneumocoeles, bronchodysplasias, etc.
- Fibrotic transformation of the lungs → reduced effectiveness of PEEP application.
- Deposit of hyaline membranes.
- Remodeling of the lung parenchyma.

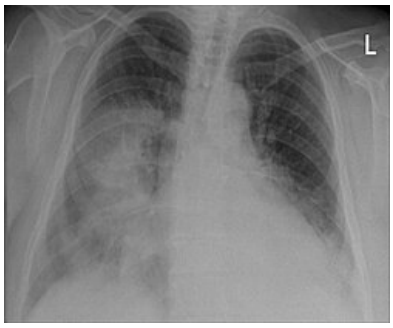
Clinical picture

- Affected by the precipitating cause.

- It usually develops within 48 h of the insult (up to 5 days).
- 1st phase: The initial symptom is tachypnea followed by hypoxemia, exertional dyspnea, slightly weakened respiratory phenomena, tachycardia, restlessness, fear, pallor, sweating.
- 2nd phase: Involvement of auxiliary respiratory muscles, auditory phenomena (rumbles), tachypnea above 30 D/min., tachycardia with possible arrhythmias.
- 3rd phase: Pulmonary hypertension, hypotension with tachycardia, acidosis, hypoxia, progression of infiltration on X-ray.
- X-ray image of bilateral lung involvement.

Investigation

- Subjective symptoms.
- Objective: pulmonary and extrapulmonary, clinical examination + laboratory.
- Blood gases – Astrup + lactate (PCWP).
- X-ray of the chest.



X-ray image of the chest

Complications

- Non-specific complications.
- Organ dysfunction syndrome.
    - kidneys 40–55 %,
    - liver 12–95 %,
    - GIT 7–30 %,
    - hematology 0–26 %,
    - circulation 10–23 %.
- complications associated with "treatment" procedures.

Treatment

- There is no universally recommended treatment with a proven effect.

1. Identification and control of insult → treatment of infection.
2. Pulmonary dysfunction therapy → ventilation support, optimization of ventilation and oxygenation.
3. Therapy of extrapulmonary dysfunction → optimization of organ perfusion, metabolic support.

- There is no specific treatment (sepsis – MOF – ARDS).
- Efforts not to further damage the lungs with our procedures (fluids, VILI).

UPV (artificial lung ventilation)

- Tidal volumes of 5-7 ml at a higher frequency.
- PEEP.
- Open lung concept (open and keep open) → ventilation with a higher inspiratory pressure to open the alveoli and to keep them open, the inspiratory pressure is reduced to the lowest possible value, at which no alveolar collapse occurs again.
- Prevention of VILI.
- Oxygen toxicity.
- Volotrauma.
- Biotrauma.
- Shear forces (atelectrauma).

Non-ventilatory procedures to improve oxygenation

- ECMO – the goal is oxygenation.
- $ECCO_2R$ – the aim is to eliminate CO_2 and reduce ventilation, IVOX,...
- Pronation position.
- Minimization of pulmonary edema (fluids, diuretics, beta mimetics, dopamine, dobutamine).

Pronation position

- Nursing demanding.
- It is less risky to turn the patient back and forth.
- Input fixation!
- A variant is a semi-pronation position of 135°.
- Better tolerated side pressures and complications, without the need for special equipment.
- Clinically similar effect.

Rescue procedures

- Ventilation and non-ventilation methods.

- Steroids.
- NO (dependency !!!).
- ECMO.
- High-frequency artificial lung ventilation
- CVVHD.

Links

- ARDS
- Shock
- High-frequency artificial lung ventilation
- Artificial pulmonary ventilation
- Pathophysiology of the respiratory system

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