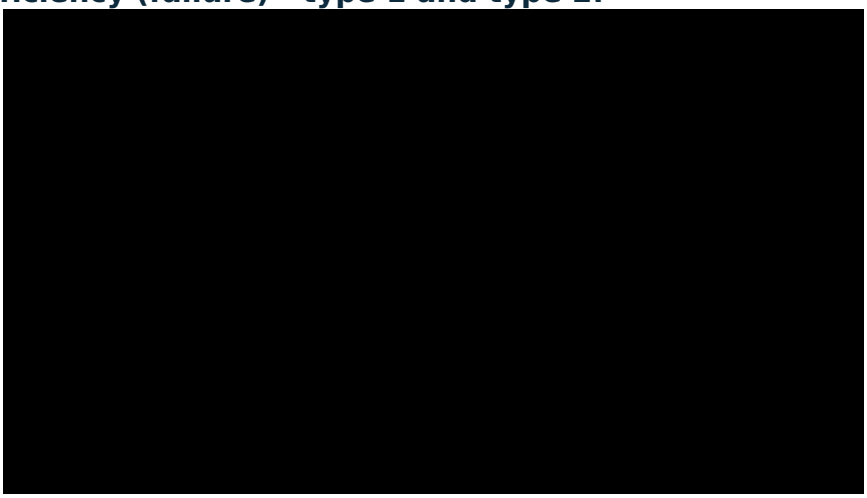
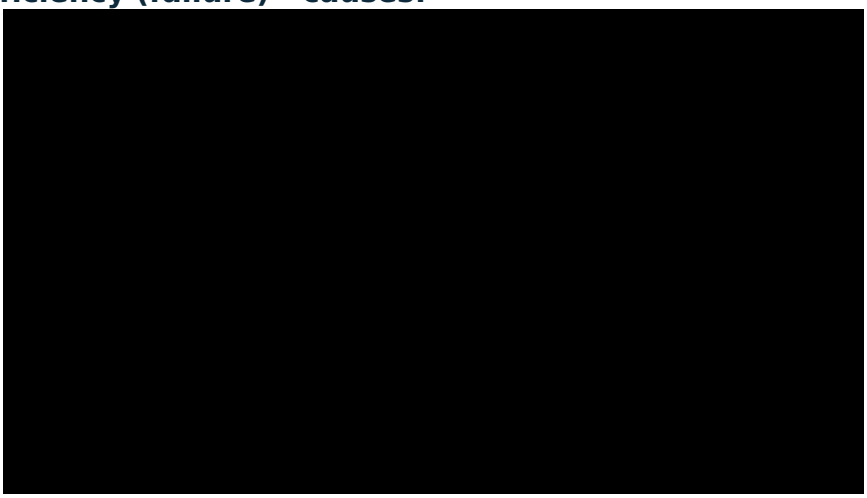
Respiratory insufficiency

Template:Zkontrolováno

Respiratory insufficiency (failure) - type 1 and type 2:

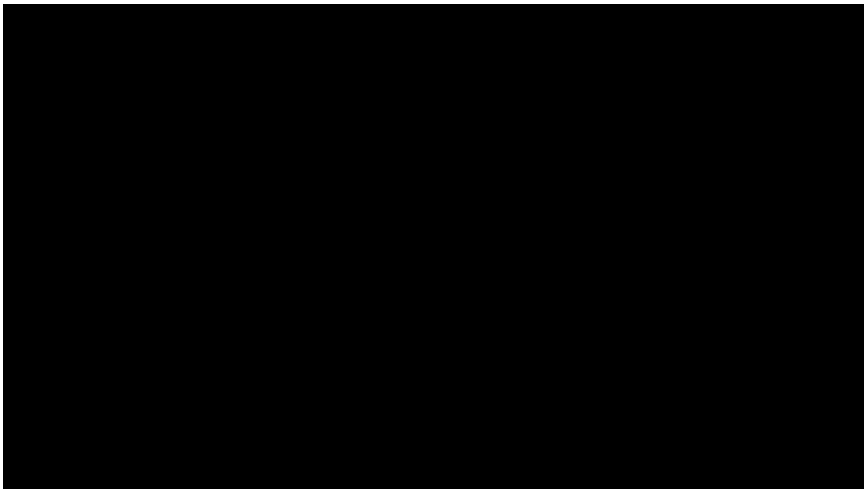


Respiratory insufficiency (failure) - causes:



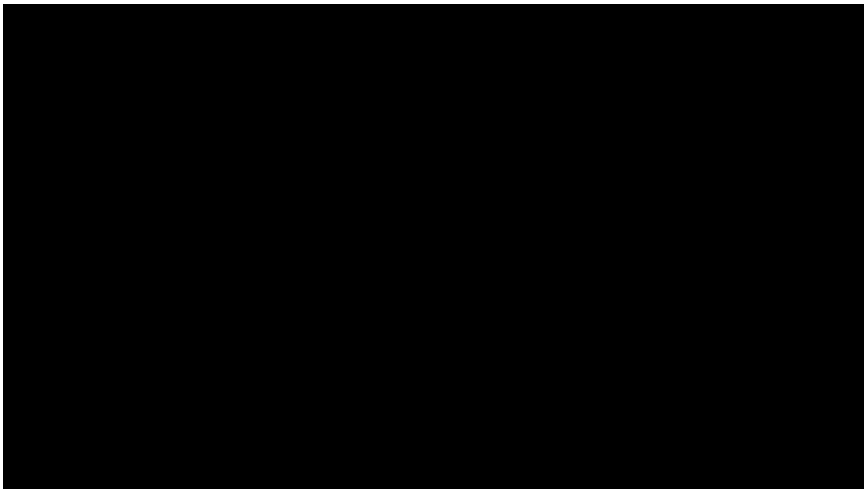
Hypoventilation:

Obstruction:

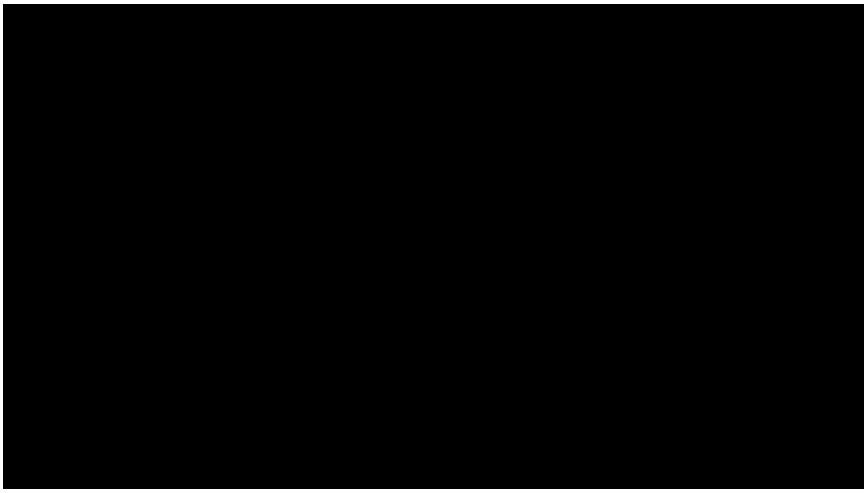


Diffusion:

V/Q mismatch:



Horovitz index (P/F ratio):



Respiratory insufficiency (RI) or **respiratory failure** is the inability of the respiratory system to ensure adequate gas exchange. The disorder can affect all functions of the respiratory system - ventilation, diffusion and perfusion of the lungs. The result is hypercapnia (insufficient secretion of carbon dioxide from the blood), hypoxemia (insufficient supply of oxygen to the arterial blood) or both at the same time.[1]

Epidemiology

- The incidence of **acute RI** is about 8 / 100,000 according to current criteria.
- Chronic lung diseases (COPD, bronchial asthma and others) are very common in the population and in about 5% of cases lead to **chronic RI**.[2]

Types

According to the development over time:

- **acute** - acute compensatory mechanisms are applied - hyperventilation, tachypnea, tachycardia;
  - etiology: ARDS;
- **chronic** - long-term compensatory mechanisms apply - renal compensation of acid-base balance and compensation of polycythemia hypoxia;
  - etiology: COPD, bronchial asthma, pulmonary fibrosis.[2]

According to the event:

- **latent** - decrease pO_2 conditioned by effort;
- **manifest** - decrease of pO_2 and possible increase of pCO_2 even at rest.[2]

According to blood gas changes:

- **hypoxemic** (type I respiratory insufficiency, partial)
  - decrease in pO_2 , pCO_2 may also decrease due to hyperventilation → respiratory alkalosis;
  - typical of acute RI (at least initially);
  - paO2 <7.3-7.9 kPa, paCO2 <5.3, alveoloarterial oxygen difference (AaDO2, paO2 difference in alveolus and arterial blood) increased;[1]
- **hypoxemic-hypercapnic** (type II respiratory insufficiency, global)
  - decrease in pO_2 and increase in pCO_2 due to hypoventilation → may lead to respiratory acidosis;
  - typical of chronic conditions (COPD, idiopathic pulmonary fibrosis); it can be compensated or decompensated;
  - paO2 <7.3-7.9 kPa, paCO2> 6.6 kPa, AaDO2 normal or elevated [1].[2]

Etiology

- lung failure: diseases of the respiratory tract, alveoli, alveolocapillary membrane or pulmonary artery;
- extrapulmonary failure: disorders of the respiratory center, elongated spinal cord, respiratory muscles or their innervation, chest wall disease.[1]

Pathophysiology

Hypercapnia

- decreased alveolar ventilation - CNS depression (unconsciousness, status epilepticus, narcotics), peripheral nervous system involvement (cervical spine injury, Guillan-Barré syndrome), respiratory muscle failure (muscular dystrophy)), respiratory muscle fatigue (shock states, increased breathing work), upper airway

obstruction (laryngitis, foreign body, tracheomalacia), decreased lung compliance (pulmonary edema, fibrosis), increased airway resistance (asthma, cystic fibrosis), increased compliance chest wall (chest trauma), disrupted pleural space (pneumothorax);
- increased dead air ventilation - decreased pulmonary circulation (pulmonary hypertension, decreased cardiac output), alveolar overdistension (asthma);
- increased CO2 production - increased metabolism (burns), impaired respiratory quotient (high glucose intake).[1]

Hypoxemia

- pulmonary shunt with increased pulmonary vascular resistance (PVR) - intraalveolar fluid diffusion, pulmonary vessel obliteration (ARDS);
- pulmonary shunt without increased PVR - intraalveolar fluid locally (lobar pneumonia);
- decreased ventilation / perfusion ratio with increased PVR - bronchospasm with pulmonary hypertension (meconium aspiration syndrome);
- decreased ventilation / perfusion ratio without increased PVR - intraalveolar fluid (cardiogenic pulmonary edema);
- intracardiac short circuit with increased PVR - right-left short circuit with pulmonary hypertension (endocardial defects);
- intracardiac short circuit without increased PVR - right-left short circuit (pulmonary stenosis with ventricular septal defect);
- hypoventilation - decreased alveolar ventilation (upper airway obstruction);
- diffusion disorder - enlargement of the interstitium (fibrosis);
- decreased saturation of fusion venous blood - increased oxygen extraction (cardiogenic shock).[1]

Clinical symptoms

- tachypnoea, dyspnoea, involvement of auxiliary respiratory muscles;
- tachycardia;
- listening finding with weakening or disappearance of respiratory murmurs;
- cyanosis;
- anxiety, agitation, quantitative disorder of consciousness.[1]

Hypoxemia irritates peripheral chemoreceptors, causing hyperventilation. Sympathetic activation results in *tachycardia, tachypnoea, anxiety and increased sweating*. As hypoxemia progresses, neurological symptoms such as *decreased mental performance, confusion,* and possibly *circulatory instability* (changes in blood pressure and heart rate) are added. Ventilation also fails with further progression, leading to hypercapnia with *CNS depression*.[2]

Hypercapnia is manifested by changes in consciousness (*drowsiness* can be replaced by *restlessness, tremors and headaches*). Severe hypercapnia may have *intracranial hypertension*. As pCO_2 rises again, a *coma* begins. Central *cyanosis* is common in chronic patients with polycythemia.[2]

Diagnostics

- **Examination of acid-base balance and blood gases:**
  - early: respiratory acidosis (decreased pH and increased pCO2);
  - later: respiratory acidosis with hypoxia (reduced paO2).[1]
- **History** - we are interested in the circumstances of dyspnea, intoxication, infection; we find out the state of consciousness (in chronic patients with dyspnea it can be significantly altered due to decompensated hypercapnia).
- **Physical examination** - various lung sound phenomena may occur depending on the underlying disease, cyanosis.
- **X-ray, CT** - often significant changes according to the underlying disease.
- Depending on the situation, spirometry, ECG, ECHO and other examinations may be performed.[2]

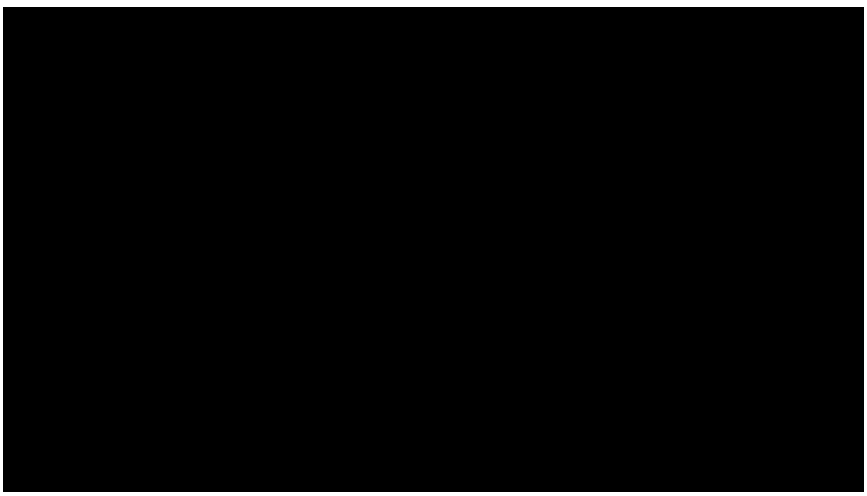
Therapy

- treatment of the underlying disease;
- treatment of hypoxia and hypercapnia - oxygen supply (heated by humidified oxygen mask, nasal cannulas or endotracheal cannula);
- adequate cardiac output, SpO2> 90%, hematocrit> 30%;
- acceleration of lung repair - nutrition, minimization of oxygen toxicity (lowest possible FiO2 to maintain normal paO2), elimination of nosocomial infection.[1]

The most common causes of respiratory failure in children

- craniocerebral trauma, intracranial hemorrhage, intoxication;
- spinal cord injury, Guillan-Barré syndrome, mysthenia gravis, paresis of the phrenic nerve;
- epiglottitis, laryngitis, foreign body aspiration, asthma, bronchiolitis;
- dystrophy (atrophy) of the respiratory muscles, muscle fatigue, cachexia;
- pneumonia, pulmonary edema, ARDS;

- pulmonary embolism, persistent pulmonary hypertension;
- pneumothorax, hemothorax, chylothorax.[1]



Links

Related articles

- Respiratory system monitoring

References

1.
2.