Ventilatory failure (pediatrics)

Pathophysiology

Respiratory failure is classified based on the pathophysiological mechanisms that lead to hypoxemia and/or hypercapnia. In a simplified view, these are disorders of ventilation, distribution, diffusion and perfusion. The following abnormalities lead to hypoxemia and/or hypercapnia:

- V/Q disproportion (ventilation-perfusion ratio)
- intrapulmonary PL shunts (intrapulmonary shunt)
- hypoventilation
- abnormal gas exchange at the alveolocapillary membrane
- reduced concentration of inspired O 2
- increased venous blood desaturation with cardiac dysfunction plus one or more of the above 5 factors.

🔎 *For more information see Ventilatory failure (pathophysiology).*

Definitions and terms

Arterial hypoxemia can be caused by a low O2 concentration in the inspired mixture, hypoventilation, oxygen diffusion disorder, ventilation-perfusion imbalance, shunt, desaturation of mixed venous blood. The causes of arterial hypoxemia are basically the same as those of cyanosis.

Distribution of respiratory insufficiency

	Partial respiratory insufficiency	**Global respiratory insufficiency**
pO2	< 8 kPa (60 torr)	< 8 kPa (60 torr)
pCO2	< 5,3 kPa (40 torr)	> 6,6 kPa (50 torr)
A-aDO2	extended gradient	normal or extended gradient

Indications for APV in childhood

Clinical indications	**Laboratory indications**
▪ apnea ▪ cyanosis with adequate oxygen therapy ▪ excessive work of breathing ▪ "quiet" chest ▪ alteration of circulation	▪ paO2 < 8 kPa = 60 torr when applying 60% O2* ▪ paCO2 > 8 kPa = 60 torr**

Criteria for respiratory failure

clinical criteria	**laboratory criteria**
tachypnea, bradypnea, apnea, irregular breathing	
pulsus paradoxus > 30 torr	
weakened or disappeared respiratory phenomena during auscultation	paO_2 < 8 kPa = 60 torr when applying 60 % O 2*
stridor, wheezing, grunting	$paCO_2$ > 8 kPa = 60 torr**
significant retraction of the chest and involvement of the auxiliary respiratory muscles	RAC with pH < 7,3
cyanosis at FiO2 > 0,4*	vital capacity < 15 ml/kg
impaired consciousness, reduced response to pain	maximum inspiratory pressure < −25 cm H_2O
weak cough or its absence, dtto laryngeal reflexes	
insufficient muscle strength	

Based on these criteria, respiratory failure is likely if 2 clinical or 1 laboratory criteria are met.

**does not apply to cyanotic heart defects **does not apply in the presence of chronic lung disease*

Terms and definitions

CONCEPT	DEFINITION
Acute respiratory failure	inability to maintain the exchange of blood gases, adequately to the metabolic needs of the organism.
Respiratory failure based on arterial blood gas values:	pO_2 < 8 kPa (60 torr) and pCO2 > 8 kPa (60 torr), or SaO_2 < 90 % with adequate oxygenation
Apnea	respiratory arrest for > 15–20 seconds, the cause can be central, reflex (via the vagus nerve, e.g. in GER) or obstruction in the DC
Dyspnea	subjective feeling of labored breathing, objective sign is chest retraction
Asphyxia	suffocation, it is an inability to exchange gases with severe hypoxemia and hypercapnia
Hypoxemia	lack of oxygen in arterial blood, PaO2 < 9 kPa
Hypercapnia	insufficient elimination of CO2 from arterial blood, PaCO2 > 6 kPa
Partial respiratory insufficiency (failure of oxygenation, hypoxemia)	a serious pulmonary oxygen transport disorder that does not allow sufficient oxygen supply to the tissues.
Global respiratory insufficiency compensated	hypoxemia + hypercapnia + normal pH
Global respiratory insufficiency decompensated	hypoxemia + hypercapnia + acidosis
Latent respiratory insufficiency	respiratory disorder, when normal blood gases are present only in the patient at rest, i.e. the ventilation reserve is limited
Manifest respiratory insufficiency	we find pathological values of blood gases even at rest
Acute respiratory failure	development within minutes and days, RAL or RAC, is immediately life-threatening
Chronic respiratory failure	development over months and years, pulmonary hypertension, compensatory MAL, potentially life-threatening
Indications for UPV: :	apnea, pCO2 > 8 – 9 kPa, cyanosis or paO2 < 60 torr (8 kPa) at FiO2 0,6, shunt > 20%.

Indications for UPV: the only absolute criterion for starting UPV is apnea. Other indications are strictly individual, when it is necessary to take into account circulatory stability, adaptation to hypercapnia in chronic patients or the variant of elective UPV, e.g. in the context of a septic state, when we follow other criteria (hyperventilation with excessive work of breathing, lactic acidosis, development of MODS...) than blood gas values.

Normal values of heart and respiratory rate

age	normal respiratory rate (per minute)	normal heart rate (per minute)
newborns	40–60	100–180
infants	30–50	80–150
toddlers	25–40	80–130
preschoolers	25–35	80–120
younger schoolchildren	20–30	70–100
older schoolchildren	12–20	60–100
an adult	12–16	60–90

Etiology

CNS lesions

- pharmacy → narcotics, barbiturates, sedatives, anesthetics, poisons
- neuroinfection→ meningitis encephalitis, abscess
- hypercapnia
- alkalosis
- tumors
- trauma
- intracranial hypertension
- central alveolar hypoventilation
- central sleep apnea

Neuromuscular lesions

- pharmacy → curariform drugs, aminoglycosides
- tumors
- tetanus
- trauma
- myasthenia gravis

- multiple sclerosis
- muscular dystrophy
- Guillain-Barré syndrome
- motor neuron disease
- bilateral lesions of the phrenic nerve

Upper respiratory tract lesions

- tonsillar adenoid hyperplasia
- VVV

infection → retropharyngeal abscess, papular tonsillitis, epiglottitis, laryngotracheitis, diphtheria

- trauma → burns, smoke inhalation, foreign body
- polyps
- tumors
- paralysis of the vocal cords
- acquired laryngomalacia
- acquired tracheomalacia
- swelling of the mucous membrane -> Quincke's angioneurotic edema, hereditary angioneurotic edema
- obstructive sleep apnea

Chest wall lesions

- severe kyphoscoliosis
- severe spondylitis
- trauma → burns with keloids
- expansion of the interpleural space → PNO, hemothorax, fluidothorax, chylothorax
- scleroderma

Lesions of the lower respiratory tract

- infection → bronchiolitis, bronchopneumonia, abscess, bronchiectasis
- VVV
- trauma → lung contusion
- distress syndromes → RDS from immaturity, ALI/ARDS
- bronchial asthma
- interstitial lung disease
- atelectasis
- cystic fibrosis
- pulmonary embolization
- Heart Failure

Other

- hyperglycemia
- ion imbalance → hyponatremia, hypocalcemia, hypomagnesemia, hypophosphatemia
- myxedema
- Pickwick syndrome

Clinic

Anamnesis

- Factors that may increase the risk of respiratory failure: young age, prematurity, immunodeficiency, chronic lung or heart disease (cystic fibrosis, bronchopulmonary dysplasia, uncorrected VCC), neuromuscular disease spinal muscular atrophy (pediatrics) = M. Werdnig–Hoffmann, myasthenia gravis, Guillain–Barré syndrome), severe sepsis sepsis (can lead to respiratory failure in the context of pulmonary edema, ARDS or with extremely increased O_2 consumption during tachypnea.
- The indication of the duration of the difficulties is important. The course of RSV infection may worsen after 3-5 days of a relatively mild course of respiratory infection. If the patient has pain, we must think of pleurisy or aspiration of a foreign body.
- With symptoms of neuromuscular weakness or paralysis, we must rule out the following diseases: myasthenia gravis (bulbar dysfunction), Guillain–Barré syndrome (distal paresis and hyporeflexia), apnea due to cervical spinal cord injury.
- A patient arriving with respiratory insufficiency could also aspirate due to convulsions.
- In patients with chronic hypercapnia, we find headaches at night or early in the morning after waking up.

Physical examination

- The position the patient takes spontaneously. (The smallest children cannot use the compensatory position, as they are often carried by their parents in their arms, i.e. in a position that does not respect their compensatory requirements).

    - Patients with pneumonia take the so-called. ***Splinting position*** of the chest, i.e. they try to keep the chest stretched and developed as much as possible.
    - With severe bronchial obstruction, we see ***orthopnea*** in children (an image of objective shortness of breath associated with the involvement of the axillary muscles and a relief position, usually sitting with the upper limbs retracted). Orthopnea is otherwise rare in childhood.
    - In case of severe obstruction of the upper respiratory tract, children take the so-called. **sniffin position** ("sniffing" position).
- Assessment of chest shape:
    - The increased anteroposterior dimension together with the bulging of the supraclavicular pits is referred to as **the volume of the pulmonum auctum** and is found in severe asthma or cystic fibrosis. Scoliosis and pectus excavatum represent a significant reduction in the compliance of the chest wall and restriction of the lung parenchyma.
    - Chest asymmetry can also be found in pneumothorax, paresis of the diaphragm, alar pneumonia, in atelectasis of the entire lung wing, in large unilateral fluidothorax.
- Painless expansion of the connective tissue of the distal joints of the phalanges = "mallet fingers " is a non-specific finding that indicates chronic hypoxia (VVV heart with PL shunt, cystic fibrosis).
- Under pathological circumstances, we can find **intensified breathing** (pneumonia, increased work of breathing, acute bronchitis) or **weakened breathing** (effusion, atelectasis, pneumothorax). The so-called **tube breathing** it is characterized by exhalation noisier than inhalation, reminiscent of the sound "ch". Physiologically, it is heard above the trachea, in pathological conditions it is found above the infiltrated lung tissue (with "complete" obturation of the alveoli). Secondary breath murmurs include moist phenomena - crackles and crepitus - they are audible during inspiration and originate in the alveoli. Crackles are also moist phenomena audible during inspiration and early expiration, originating in the bronchi. Wheezing and wheezing are audible during expiration, they also originate in the bronchi (the cause is inflammatory edema of the mucous membrane or spasm of the smooth muscle of the bronchi).

The frequency of respiratory failure in young children is higher than in older children and adults for a number of reasons.

Specifics of breathing in children

- The airways are narrower, and even a slight swelling in an inflammatory condition can lead to critical obstruction.
- Newborns and infants up to 6 months of age breathe mainly through the nose due to the close proximity of the epiglottis and nasopharynx. Nasal congestion at this age can lead to significant respiratory distress.
- Small children have a large tongue that fills their small oropharynx. Their larynx is located higher, i.e. opposite the C3−C4 vertebrae (in older children and adults, the larynx is opposite C6–C7).
- The epiglottis is long, slender and U-shaped
- The narrowest place in children < 8 years is the subglottic space (ring cartilage area), in children > 8 years then the glottis
- The trachea has a third diameter compared to adults.
- Infants and young children have significantly fewer alveoli than children > 8 years of age. The number of alveoli increases from 20 million to 300 million around the age of 8. Therefore, small children have a smaller area for gas exchange.
- There are less developed elastic fibers in the alveoli. The alveoli therefore have a greater tendency to collapse, resulting in a ventilation-perfusion imbalance.
- Small children are characterized by high compliance of the chest wall and, on the contrary, low compliance of the lungs. The intercostal muscles are immature, they show uncoordinated movements during REM sleep, the ribs are placed horizontally, the diaphragm works "more", but less efficiently.
- Children < 6 years of age have a value of the so-called closing capacity (the volume of air in the lungs that prevents the collapse of the small airways) higher than the FRC. This fact explains the predisposition of young children to the formation of atelectasis.
- Collateral ventilation is not fully developed. Therefore, atelectasis is far more common in children than in adults. During childhood, anatomical channels are formed that allow collateral ventilation of the alveoli. These collaterals exist between alveoli (pores of Kohn), bronchioles and alveoli (Lambert's ducts), additional bronchioles represent additional collaterals. Collateral ventilation enables gas exchange even with distal airway obstruction.
- The respiratory center in the smallest children is immature. This poses a risk for breathing irregularities and apnea. Reflexes inhibiting respiration predominate, especially the Hering-Breuer reflex, which is a response to lung expansion. Risk factors are sleep, cold, medication, various metabolic deviations.

Changes in the breathing pattern in relation to the patient's age

- Toddlers and older children achieve the necessary minute ventilation with **restrictive lung disease by increasing the respiratory rate at a reduced tidal volume.**
- **Conversely, with obstruction** the necessary gas exchange is achieved by deepening the respiratory volume at a reduced respiratory rate {{This pathophysiological formula is actually only applied to chronic patients - most often COPD, in children we already see tachypnea in the context of obstruction - the reason is the stimulation of the respiratory center during concomitant hypoxemia.}}
- Newborns and infants have an almost horizontal position of the ribs, i.e. the chest is already physiologically in the inspiratory position. Therefore, when the minute ventilation needs to be increased, the breathing frequency is practically always increased without an adequate increase in the respiratory volume. Thus, the universal response of children of this age group to respiratory distress is **tachypnea**. This pathophysiological note must be taken into account when analgosedation is indicated. Sedation of the child limits compensatory

tachypnea and may thus induce ventilatory failure in previously compensated respiratory distress.

Respiratory Distress Clinic

- In hypoxemia, we expect respiratory stimulation, i.e. tachypnea and/or dyspnea. The exception is premature babies, who on the contrary react with hypoventilation and apnea. Reduced respiratory effort in a child with hypoxemia most likely indicates CNS depression, a neuromuscular disorder, but the possibility of exhausting respiratory effort in a patient with prolonged respiratory distress must also be taken into account.
- Respiratory tachypnea is usually caused by the presence of fluid in the alveoli or interstitium.
- Primary tachypnea in a child with minimal chest retraction and no secondary auscultation phenomena most likely indicates a non-respiratory cause (CNS pathology including intoxications, sepsis, respiratory compensation MAC = Kussmaul breathing).
- In these cases, we often find reduced values of glycemia, calcium and hypocapnia. On the contrary, we can demonstrate hypercapnia in heart defects with significant pulmonary congestion.
- Respiratory distress in heart failure can only be associated with tachypnea without chest retraction. In advanced pulmonary edema, however, we find rales (intraalveolar edema), wheezing (bronchial wall edema), retraction of the soft parts of the chest.
- Involvement of auxiliary respiratory muscles = retraction, indicative of reduced lung compliance or airway obstruction. Likewise, retraction/falling in of the jugular (in eupnoea, the jugular fossa does not retract), retraction of the supra or infraclavicular fossae, intercostal, epigastrium, attachment of the diaphragm and alar flexure = expansion of the nostrils leads to a reduction in the resistance to the passage of air and thereby reduces the work of breathing. ⚠
- A special sign of respiratory distress is **grunting** Retraction with grunting means reduced lung compliance and loss of FRC. Grunting is caused by the glottis clenching mechanism. It is an attempt to increase autoPEEP and thereby increase functional residual capacity. A restrictive disorder is characterized by an accelerated rate and reduced tidal volume.
- Retraction with inspiratory stridoris accompanied by obstruction of the extrathoracic airways, mixed stridor means obstruction of the intra + extrathoracic airways (larynx, trachea), wheezing with the auscultatory finding of squeaks/gurgles is accompanied by obstruction of the lower, i.e. intrathoracic airways. * Mild obstruction of the airways will be manifested by a reduced respiratory rate and, conversely, an increased respiratory volume. Severe airway obstruction is characterized by increased respiratory rate, chest retraction, involvement of accessory muscles, anxiety, cyanosis. It should always be remembered that the resolution of severe upper airway obstruction may be followed by the development of pulmonary edema.
- On the other hand, it should be noted that compensatory expiratory effort in a dyspneic patient may be counterproductive in extreme cases. Patient anxiety increases oxygen consumption, strenuous respiratory effort can cause dynamic airway obstruction. If it lasts for a long time, respiratory muscle fatigue develops, hypoventilation occurs with a rise in pCO2. Forced inspiration will significantly increase intratracheal negative pressure, which can worsen upper airway obstruction, while positive intrapleural pressure during forced expiration can lead to intrathoracic airway collapse. When the closing capacity is exceeded, smaller or larger lung regions collapse.
- A critically serious form of breathing is **gasping** which is characterized by a low and irregular respiratory rate, variable respiratory volume, and respiratory pauses. It occurs in the terminal phase of hypoxia, sepsis or shock from any cause, before cardiopulmonary arrest. This breathing pattern is predictive for the immediate initiation of cardiopulmonary resuscitation.
- The respiratory compensation of severe MAC (diabetes mellitus, intoxication) is pronounced hyperpnea with extremely increased minute ventilation, the so-called Kussmaul breathing. Mistaking it for dyspnea in pneumonia or bronchitis is dangerous for the patient in this case! ⚠ **It should always be borne in mind that the development of pulmonary oedema may follow the resolution of severe upper airway obstruction.**

Within the CNS causes of respiratory distress, two specific breathing patterns should be noted

• Cheyne-Stokes breathing: it has an ascending (crescendo) phase, when the frequency and depth of breathing increases, and a descending phase (decrescendo), which ends with an apneic pause (apnea is not a condition, however). The cause is a delayed reaction of the respiratory centers to changes in blood gases. Cerebral, thalamic, or hypothalamic control of breathing is impaired, but brainstem control is still intact. This occurs when there is insufficient perfusion of the CNS or when respiration is controlled by a lack of oxygen. The Cheyne-Stokes respiratory pattern is found in CNS injury, intoxication (depression of the respiratory center) uremia, immaturity, heart failure, increased intracranial pressure.

• Biot's breathing: periodic breathing with respiratory effort followed by apnea. It is an expression of damage to the neurons controlling breathing. It is accompanied by CNS involvement in the region of the posterior fossa of the cranium or brainstem, it can be observed when the irritability of the respiratory center decreases during meningitis or encephalitis.

Other clinical signs of respiratory distress are changes in auscultatory phenomena on the lungs and cyanosis. We also often find disorders of consciousness - anxiety, agitation (arising as a result of hypoxia with subsequent sympathetic activation), somnolence (developing as a result of fatigue or hypoperfusion of the CNS). Cardiovascular symptomatology is also characteristic of respiratory distress (tachycardia, bradycardia, hypotension or mild hypertension, arrhythmia).

⚠ In general, sepsis should be excluded at any manifestation of respiratory distress, especially in children < 1 year.

Links

Resources

- HAVRÁNEK, Jiří: Ventilační selhání.

Related Articles

- Examination of the child's respiratory system
- Binding of oxygen to hemoglobin
- Pulmonary ventilation - perfusion ratio
- Ventilatory failure (pathophysiology)