# Cyanosis (neonatology)

Cyanosis is a blue to bluish-purple coloration of the skin and mucous membranes caused by insufficient oxygenation of the blood and a rise in reduced hemoglobin above 50 g/l.[1]

**Central cyanosis**

- clinical picture: overall bluish coloration of the skin and mucous membranes;
- etiopathogenesis: insufficient oxygenation of arterial blood in the lungs (heart defects, severe pneumopathy).

**Peripheral cyanosis**

- clinical picture: bluish coloration of the skin, but normal coloration of the mucous membranes;
- etiopathogenesis: low temperature or methemoglobinemia (Hb $Fe^{3+}$, chocolate brown blood).

**Acrocyanosis**

- clinical picture: bluish coloration only of the palms and soles;
- etiopathogenesis: impaired peripheral tissue perfusion during circulatory failure (hypovolemia).[1]

## Pathophysiology

**Central cyanosis** is a bluish discoloration of the skin, mucous membranes and tongue, which is evident when the level of deoxygenated hemoglobin (deoxy Hb) rises. Blood gas examination shows a low oxygen partial pressure ($PaO_2$) and pulse oximetry shows a low hemoglobin oxygen saturation ($SaO_2$).[2]

Oxygenated hemoglobin (oxy Hb) is bright red, while deoxy Hb is dark blue or purple. Cyanosis depends on the absolute concentration of deoxy Hb, not on the ratio of oxy Hb to deoxy Hb, therefore cyanosis in the polycythemic neonate is seen at a higher $SaO_2$ than in the anemic neonate. Newborns have a different ratio of fetal and adult hemoglobin - in a newborn with a greater proportion of adult hemoglobin, cyanosis will be evident at a higher $SaO_2$ than in a newborn with a greater proportion of fetal hemoglobin.[3][2]

**Peripheral cyanosis** is a bluish discoloration of the skin, but not of the mucous membranes and tongue. $PaO_2$ is usually normal. Due to the impaired blood flow through the capillaries, there is a higher extraction of oxygen from the hemoglobin, and therefore the level of deoxy Hb on the venous side of the capillary network increases. Vasomotor instability and vasoconstriction can be caused by cold, low cardiac output, and polycythemia. Acrocyanosis (peripheral cyanosis of the limbs) is a relatively common finding in newborns. It is usually caused by vasoconstriction during transient hypothermia. Sepsis can be a serious cause of peripheral cyanosis.[2]

## Diagnosis

### Anamnesis

- gestational diabetes mellitus → the cause of cyanosis is then transient tachypnea of the newborn (TTN), respiratory distress syndrome of the newborn (RDS), hypoglycemia, transposition of the great arteries (TGA);
- oligohydramnios → pulmonary hypoplasia;
- hypertension in pregnancy → IUGR, polycythemia, hypoglycemia;
- lithium use → Ebstein's anomaly;
- higher maternal age → trisomy 21 associated with various congenital heart defects;
- PROM, fever, GBS positivity → sepsis;
- sedatives/anesthesia → depression of the respiratory center, apnea;
- caesarean section → transient tachypnea of the newborn (TTN), persistent pulmonary hypertension (PPHN)
- premature newborn → RDS
- meconium in amniotic fluid → meconium aspiration syndrome (pneumonia).[2]

### Physical exam

- determine whether it is central or peripheral cyanosis;
- check vital signs: signs of respiratory distress (tachypnea, retractions, alar flexion, grunting);
- rule out clinical signs of sepsis: peripheral cyanosis, tachycardia, rise in respiratory rate, drop in blood pressure, rise/fall in body temperature (diff. dg. hypoplastic left heart syndrome, critical aortic stenosis, severe coarctation of the aorta);

- rule out choanal atresia (by inserting a nasogastric tube);
- determine if a systolic murmur is present (accompanying most cyanotic congenital heart defects); examine the abdomen (a sunken abdomen typical of a diaphragmatic hernia);
- neurological disorders associated with immaturity: apnea, periodic breathing;
- convulsions.[2]

# Differential diagnosis

## Ventilation/perfusion failure

- respiratory tract: transient tachypnea of the newborn (TTN), respiratory distress syndrome of the newborn (RDS), pneumonia, aspiration (meconium, blood, amniotic fluid), atelectasis, congenital diaphragmatic hernia, pulmonary hypoplasia, pulmonary apoplexy, congenital cystic adenomatoid malformation (CCAM);
- external oppression of the lungs: pneumothorax, pleural effusion, hemothorax.

## Right-left short circuit

- cardiac (5 T): Tetralogy of Fallot, tricuspid valve atresia, transposition of great arteries, total anomalous pulmonary venous return, truncus arteriosus; also pulmonary atresia, Ebstein's anomaly (abnormal tricuspid valve), hypoplastic left heart;
- large vessels: persistent pulmonary hypertension of the newborn;
- pulmonary: pulmonary arteriovenous malformation.

## Alveolar hypoventilation

- CNS depression: asphyxia (hypoxic-ischemic encephalopathy), maternal sedation, intraventricular hemorrhage, convulsions, meningitis, encephalitis; airway obstruction: choanal atresia, laryngomalacia, Pierre Robin syndrome;
- neuromuscular disease: phrenic nerve injury, neonatal myasthenia gravis.

## Oxygen diffusion disorder

- pulmonary edema: left-sided obstructive heart disease (aortic stenosis), cardiomyopathy;
- bronchopulmonary dysplasia, pulmonary fibrosis, pulmonary hypoplasia, congenital diaphragmatic hernia.

## Decreased affinity of hemoglobin for oxygen

- methemoglobinemia (congenital, drug induced).

## Impaired peripheral circulation

- sepsis, shock, polycythemia, hypothermia, hypoglycemia, low cardiac output (hypocalcemia, cardiomyopathy, etc.).[2][4]

# Examination

- blood count and differential: leukocytosis/leukopenia – sepsis, hematocrit above 65% – polycythemia;
- blood sugar;
- ABR: arterial $PaO_2$ to confirm central cyanosis; increased $PaO_2$ – lung or heart disease, heart failure; low pH – sepsis, circulatory shock, severe hypoxemia; methemoglobinemia (decreased $SaO_2$, normal $PaO_2$, chocolate brown blood);
- hyperoxic test: administration of 100% oxygen for at least 10 minutes;
  - $PaO_2$ > 100 mmHg: probably pulmonary in origin;
  - $PaO_2$ < 70 mmHg, rise < 30 mmHg or $SaO_2$ unchanged: likely cardiac cause (right-to-left shunt);
  - total anomalous pulmonary venous return may correspond;
  - pulmonary disease with massive intrapulmonary shunt may not match;
- pre- and postductal $PaO_2$ and $SaO_2$ measurements;
  - higher $PaO_2$ (by 10-15 mmHg) in the right radial artery than in the umbilical artery – right-left ductal shunt (persistent pulmonary hypertension);
  - also $SaO_2$ can be compared (significant difference is > 10=15%);
- Chest X-ray: pneumothorax, pulmonary hypoplasia, diaphragmatic hernia, pulmonary edema, pulmonary effusion; cardiomegaly and vascular congestion in heart failure;
  - typical heart shape in TOF, TGA, TAPVR;
- echocardiography.[2]

# Therapy

- vital signs monitoring;
- providing ventilation (oxygen therapy, artificial lung ventilation);
- provision of vascular access (insertion of umbilical catheters or peripheral artery and vein cannulation);
- if sepsis is suspected, culture sampling (blood culture, urine) and antibiotic therapy with broad-spectrum

antibiotics; if a cardiac cause is suspected, give prostaglandin E1, as it could be a ductus-dependent congenital heart defect.[2]

# Sources

## Related articles

- Central cyanosis • Peripheral cyanosis

## References


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