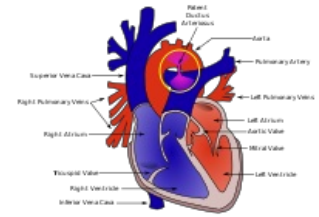


Patent ductus arteriosus

Patent Ductus Arteriosus (persistent ductus arteriosus, PDA) is an acyanotic congenital heart defect in which the ductus arteriosus (ductus Botalli) remains open. Ductus arteriosus is a vessel that connects the pulmonary artery and aorta. It is important in the fetal circulation, as it allows for blood to bypass the lungs by directing the deoxygenated blood from the pulmonary artery to aorta.



Patent ductus arteriosus

In healthy full-term neonates, a functional closure of the duct occurs shortly after the birth. Complete closure occurs within 24 hours in almost 50% of cases. Out of the remaining neonates, closure occurs within 48 hours in 90% of cases, and within 96 hours after birth, the duct should be closed in all full-term newborns. The failure to close this duct is called patent ductus arteriosus or persistent ductus arteriosus (PDA).^[1]

PDA is more common in preterm infants. Widths and lengths of the persistent duct can vary among individuals, therefore the severity of the symptoms of the PDA varies from case to case – it can manifest as a murmur in mild cases and as severe acute heart failure in severe cases.^[2]

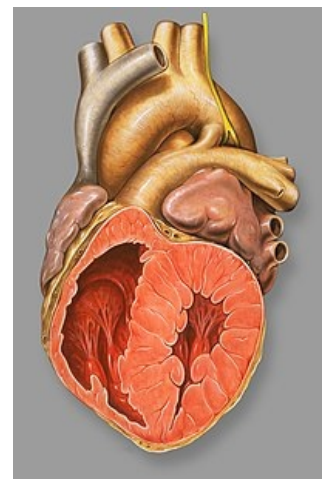
Risk Factors

Higher incidence of PDA is associated with the following factors:

- Preterm birth (80% of neonates with a birth weight below 1000 g)^[3]
- Respiratory Distress Syndrome (RDS) and its surfactant treatment
- High intravenous fluid administration in the first days of life,
- Asphyxia
- Congenital rubella, trisomy 13, trisomy 18, ...
- high altitude,
- Congenital heart defects (aortic coarctation, pulmonary atresia, transposition of the great vessels , ...).^[3]

Lower incidence of PDA is associated with the following factors:

- Use of antenatal corticosteroids (induction of pulmonary development in premature neonates),
- IUGR
- Premature amniotic fluid outflow.^[3]



Ductus arteriosus

Pathophysiology

The ductus arteriosus shunts the blood from the pulmonary artery to **descending aorta**, bypassing the pulmonary circulation which has a high vascular resistance. Various factors influence whether the duct closes or persists. Closure of the duct is stimulated by the presence of oxygen in the blood flow, and the duct is kept open by prostaglandin E2. Sensitivity to these opposing factors differs with **gestational age**. The younger the fetus is, the more sensitive it is to prostaglandin E2 and indomethacin, while the effect of oxygen is small. indomethacin is a medication used to stimulate the closure of the ductus arteriosus. ^[1]

Clinical Manifestation

It is often asymptomatic in mild cases. In severe cases, its consequences can be growth retardation, failure to thrive, collapsing pulse (Watson's water hammer pulse), hypotension (this can be an initial symptom in ELBW), ventilation problems, heart failure (with pulmonary edema and hepatomegaly) Peripheral pulses are bounding and forceful and hyperdynamic precordium is often seen. Resting tachycardia and tachypnea are also apparent.

Wide and short ductus arteriosus result in heart failure, pulmonary hypertension, and differential cyanosis (refers to cyanosis in both lower extremities but with acyanotic upper extremities). A moderate size of the duct may result in recurrent respiratory infections and failure to thrive. A narrow ductus arteriosus is asymptomatic. **All patients** are at risk of infectious endocarditis.

In premature infants with RDS (respiratory distress syndrome), PDA may lead to dependence on artificial ventilation. In infants, it causes circulatory failure, manifesting as dyspnea as a result of pulmonary edema.

⚠️ Auscultation finding: continuous "machine-like" heart **murmur** (or three-period rhythm) can be heard in **left subclavicular region**. The murmur can be heard best during the second heart sound^[4] (it can be described as a low frequency sound of "pouring coke").

Diagnosis

Diagnosis of PDA is based on:

- Findings during physical examination
- Echocardiogram, including doppler echocardiography
- X-ray of the heart shows cardiomegaly, blood flow to the lung^[2]

Differential diagnosis

Patients with an aortopulmonary septal defect (direct communication of the ascending aorta and pulmonary trunk, aortopulmonary window) have a similar clinical manifestation.

Treatment

- **Asymptomatic PDA** – continued monitoring. In most cases, the duct close spontaneously (especially in premature infants)^[2]
- **Symptomatic PDA:**
 - Limited fluid intake
 - maintenance of sufficient oxygen saturation in the blood
 - Treatment of heart failure: furosemide
 - Possible pharmacological closure of the duct: indomethacin, ibuprofen,
 - absolute contraindication: concurrent heart defects which require PDA for survival (e.g., aortic coarctation),
 - side effects: oliguria, fluid retention and hyponatremia due to decreased renal blood flow, decreased blood flow to the brain, gastrointestinal complications (bleeding, ulceration), bleeding (impaired platelet function), jaundice
 - Surgical treatment (ligation, excision).^[2]
 - Cardiac catheterization (Amplatzer duct occluders)

References

Related Pages

- Congenital heart defects
- Acquired heart defects

External Links

- Otevřená Botallova dučej- Šelest - Audio nahrávky (TECHmED) (<https://www.techmed.sk/kontinualny-selest/>)

Citations

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2. TASKER, Robert C. – MCCLURE, Robert J. – ACERINI, Carlo L.. *Oxford Handbook of Paediatrics*. 1. edition. New York : Oxford University Press, 2008. pp. 175. ISBN 978-0-19-856573-4.
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4. http://int-prop.lf2.cuni.cz/zof/vysetreni/srdceva_n.htm#od

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- TASKER, Robert C. – MCCLURE, Robert J. – ACERINI, Carlo L.. *Oxford Handbook of Paediatrics*. 1. edition. New York : Oxford University Press, 2008. pp. 175. ISBN 978-0-19-856573-4.

Classifications and References

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- MeSH ID D004374 (<https://www.medvik.cz/bmc/link.do?id=D004374>)
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