



Patent ductus arteriosus in the neonate

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KEYWORDS

Ductus arteriosus;
Infant premature;
Echocardiography;
Indomethacin;
Ibuprofen

Summary Early ductal shunting in preterm infants has traditionally been viewed as insignificant because of pulmonary hypertension. Doppler echocardiography has given us a window on preterm haemodynamics that questions the current relevance of this thinking. It has been shown that early postnatal ductal constriction is very variable and that the shunt direction is usually left to right. Where constriction fails, the adverse haemodynamic influence of the duct might be most significant in the first 12–24 h. Failed ductal constriction in the early hours after birth predicts haemodynamic significance at the time and also predicts later persisting patency requiring treatment. The only approach to the treatment of patent ductus arteriosus (PDA) that has been shown to make a difference to any outcome is prophylactic early indomethacin. However, the short-term benefits of reduced major IVH and PDA ligation have not been demonstrated to translate into better neurodevelopmental outcome. More research is needed into the role of targeting indomethacin on the basis of early ductal constriction as a refinement of the prophylactic approach.

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Practice points

- Risk of PDA increases with lower gestation, lack of antenatal steroids and need for ventilation
- Clinical signs are of limited diagnostic accuracy in the first 4 days after birth.

Early diagnosis requires high degree of suspicion and echocardiography

- The haemodynamic impact of ductal shunting may be at its most important in the first 24–48 h
- Indomethacin or ibuprofen are the first line treatment with surgery if medical treatment fails or is contraindicated. When medical treatment should commence is not clear from the evidence
- Prophylactic indomethacin reduces the incidence of major IVH and need for ductal ligation but does not affect long-term neurodevelopmental outcomes

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Research directions

- The potential to shorten courses of indomethacin or ibuprofen by using serial echocardiography to monitor ductal closure
- Is ibuprofen safe and effective if given orally
- Comparison of surgical ligation versus conservative management after failed medical treatment of PDA
- The role of targeting early treatment on the basis of echocardiographic assessment of ductal constriction as a refinement of the prophylactic approach

Introduction

This small fetal blood vessel creates some of the most difficult diagnostic and therapeutic dilemmas in neonatal medicine. These dilemmas are most apparent in the care of the very preterm baby where, in about a third of babies born before 30 weeks, the normal postnatal constriction of the ductus arteriosus (DA) will fail. The resultant haemodynamics can have important consequences in both the systemic and pulmonary circulations of these babies. Consequences that may relate to a range of adverse preterm outcomes have been associated with patent ductus arteriosus (PDA).

Normal physiology

The DA links the fetal main pulmonary artery with the descending aorta. High pulmonary vascular resistance is maintained by constricted pulmonary arterioles and preferentially directing the right ventricular output from right to left through the DA and hence back to the placenta. Fetal ductal patency is maintained by high circulating levels of prostaglandins, particularly PGE₂. During the last trimester, in preparation for closure at birth, the vessel wall of the DA becomes more muscular and that muscle becomes less sensitive to the dilating effects of PGE₂ and more sensitive to the vasoconstricting effect of oxygen.¹

After birth, the rising arterial pO_2 causes powerful constriction of the muscle in the wall of the DA. Complete closure occurs in two phases, the first is functional closure when the muscle constriction obstructs the flow of blood. The second is structural

closure, which results from ischaemia and necrosis of the intima.² During the phase of functional closure, it is possible for the ductus to re-open, either spontaneously as can occur in premature babies, or therapeutically in response to prostaglandins.

Doppler echocardiography of the duct

Much of our current understanding of normal and abnormal behaviour of the DA comes from studies with Doppler echocardiography, so it is important for the reader to be familiar with what these techniques can assess. Although Doppler echocardiography is now the gold standard of PDA diagnosis, its wider use is limited by access to ultrasound skills and equipment. With application, echocardiographic skills should be attainable by anyone practising or training in newborn intensive care and published learning resources for neonatologists are now available.^{3,4} The other limitation to echocardiography can be the technical difficulty of obtaining good ultrasound windows in ventilated babies with overinflated lungs.

The DA can be directly imaged with 2D ultrasound. Anatomically, the DA is a continuation of the main pulmonary artery, which is slightly offset to the left reflecting the arch it describes into the

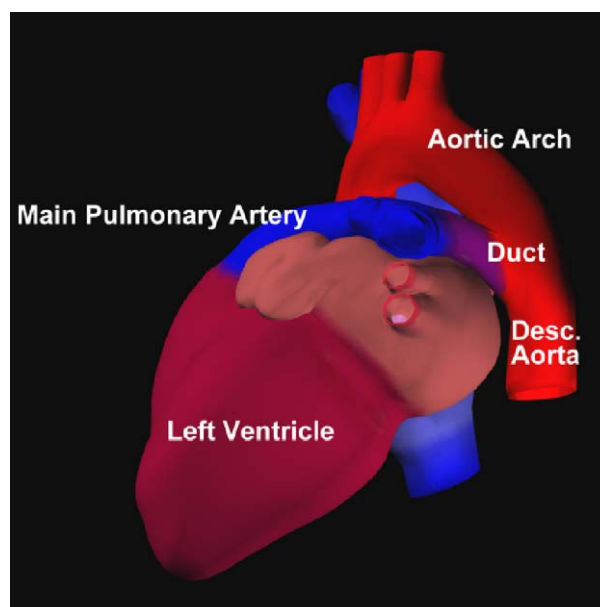


Figure 1 Image of a model of the heart viewed from the left-hand side to demonstrate the anatomical relationships of the ductus arteriosus. It can be seen how the ductus is a continuation of the main pulmonary artery, which is slightly offset to the left to allow it to describe an arch into the descending aorta.

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