

Patent Ductus Arteriosus

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Four shunts (the foramen ovale, ductus arteriosus, ductus venosus, and the umbilical vessels) permit much of the fetal blood to bypass the lungs and liver.

However, immediately after birth, when circulation of fetal blood through the placenta ceases, the lungs begin to function, and the sphincter in the ductus venosus constricts so that all the blood that reaches the liver must pass through the hepatic sinusoids (Figures 1 and 2).

Postnatal aeration of the lungs is associated with thinning of the walls of the pulmonary arteries, a dramatic drop in pulmonary vascular resistance, and a marked increase in pulmonary blood flow. These phenomena occur as the infant's first few breaths increase lung capacity, which stretches and thereby thins the walls of the pulmonary arteries. Pulmonary blood flow then increases, causing the pressure in the left atrium of the heart to rise above the pressure in the right atrium. Strong left-atrial pressure closes the foramen ovale by pressing the valve of the foramen ovale, formed by the septum primum, against the septum secundum (Figure 2).

In the fetal heart, the right ventricle works significantly harder than the left, causing the right ventricular wall to thicken. However, by the end of the first month after birth, the left ventricular wall becomes thicker because it now sends blood to the systemic circulation. The right anterior wall atrophies and hence, becomes thinner at this stage. The ductus arteriosus should constrict within the first 10 to 15 hours after birth, but in premature infants and infants with persistent hypoxia, it may remain open significantly longer.

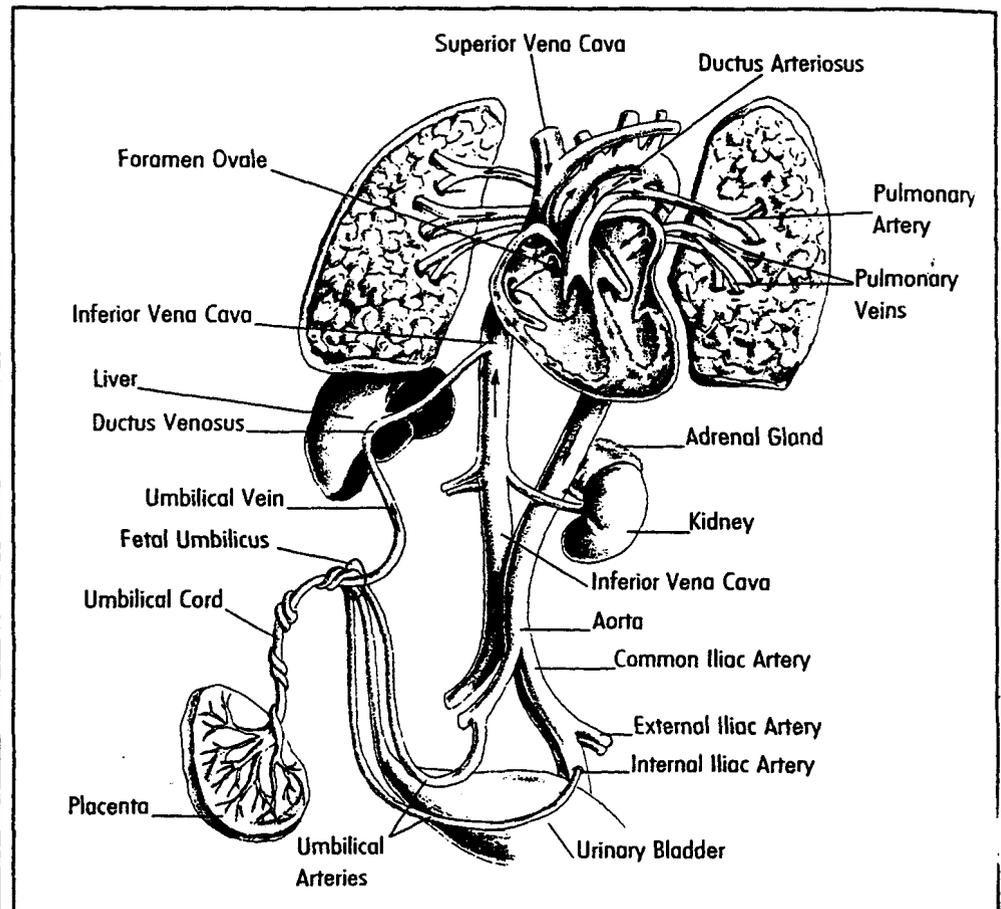


Figure 1. Fetal circulation.

Bradykinin—a substance released from the lungs during initial inflation—mediates closure of the ductus arteriosus. Patency of the ductus arteriosus before birth is controlled by locally produced prostaglandins that cause the muscle cells in the wall of the ductus arteriosus to relax. It therefore follows that prostaglandin inhibitors, such as indomethacin, can cause constriction of a patent ductus arteriosus in premature infants.

Patent ductus arteriosus (PDA) is the most common congenital malformation

associated with maternal rubella infection during early pregnancy. It appears two to three times more frequently in females than in males.

Hemodynamics

When aortic pressure increases, blood can flow through the ductus from the aorta to the pulmonary artery (Figure 1). The degree of shunting depends on the size of the ductus and the pressure gradient between the aorta and the pulmonary artery. In extreme cases, shunting can be

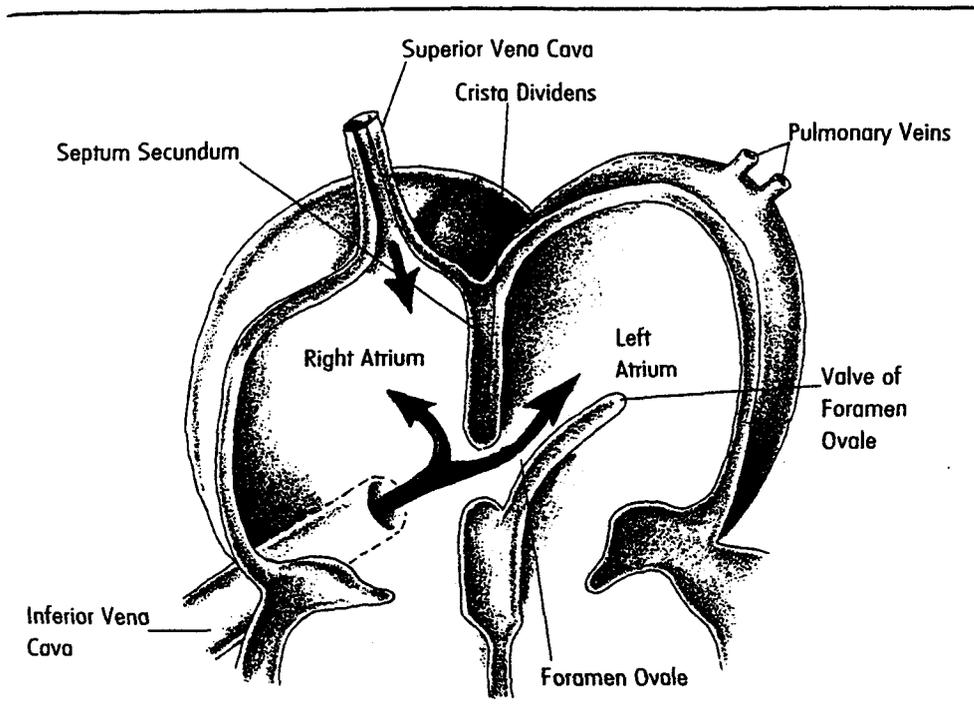


Figure 2. Postnatal heart: strong left-atrial pressure closes the foramen valve.

as much as 50% to 65% of left ventricular output through the ductus to the pulmonary circulation. As pressure in the pulmonary circulation increases, changes that lead to clinical symptoms can occur in the right ventricle, right atrium, and pulmonary artery.

Clinical Manifestations

Symptoms associated with PDA may develop at any age and may begin with slowly progressive exertional dyspnea (shortness of breath) followed by left ventricular or congestive cardiac failure. Retardation of physical growth is the most obvious external manifestation.

When the ductus is small, the heart is normal in size; however, it becomes moderately to grossly enlarged when the patency of the ductus creates a substantial communication between the aorta and the pulmonary artery. A classic heart murmur, audible through auscultation at the second intercostal space to the left of the sternum, sounds like machinery or rolling hummer. It begins soon after the first heart sound, reaches its maximum intensity at the end of systole, and wanes in late diastole.

The electrocardiogram reading is usually normal, but if the ductus is large, ventricular hypertrophy may be evident. Roentgenographic studies usually provide a normal result as well; but again, the result depends on cardiac size.

Patent Ductus Arteriosus in Infancy

An uncomplicated PDA may occasionally produce symptoms of left-sided heart failure or severe congestive heart failure during the first year of life. These symptoms are frequently precipitated by respiratory infections. As the child grows, the presence or absence of a murmur depends on the pressure relationship between the aorta and the pulmonary artery. Diagnosing symptomatic, uncomplicated PDA in infancy is critical to prolonging a child's life. Surgical treatment is indicated in all symptomatic patients regardless of age.

Prognosis and Complications

Many patients with minimal PDA live a normal life span with minor, if any, cardiac problems, but a number have developed clinical complications. The most frequent complication in late

childhood is infectious endarteritis. Rarer complications include aneurysmal dilation of the pulmonary artery or of the ductus itself; paradoxical emboli; and acquired rheumatic heart disease. Congestive cardiac failure, which may be preceded by episodes of left ventricular failure, can occur at any age, but is more common in the third decade of life.

Surgical Closure of Patent Ductus Arteriosus

The surgeon performs a thoracotomy, thus permitting the placement of a suture (3-0 silk) through the edges of the pleura. The assistant applies a hemostat to the ends of the suture and retracts the pleura. The surgeon carefully dissects between the aorta and pulmonary artery with Metzenbaum scissors to expose the ductus. A heavy silk suture mounted on a passer may be passed around the ductus. The surgeon continues the dissection until the ductus is totally isolated.

Straight or slightly angled vascular clamps are placed across the ductus—one close to the aorta and the other near the pulmonary artery. When performing the procedure on infants, the surgeon simply ties the ductus with size 0 silk suture because of the small size of the ductus and the critical condition of such patients.

The surgeon cuts halfway through the ductus using a No. 11 knife blade or Potts scissors. A 5-0 or 6-0 Prolene suture is used to begin closure of the ductus on the aortic side. The surgeon then completes the cutting of the ductus and continues the suture to close the entire ductus on the aortic side. Once the ductus is closed, the vascular clamp is removed slowly. Stay sutures are placed if any leaks are found. The same procedure is conducted on the pulmonary artery side. If bleeding occurs, a hemostatic agent can be used along the suture line. Finally, the surgeon closes the mediastinal pleura with continuous 3-0 or 4-0 silk or chromic gut sutures. Then, an appropriate-sized chest tube is placed, and a standard chest closure is completed.

Postoperative Care

With an uncomplicated ductus, the operative risk is surprisingly small. When patent ductus is associated with other abnormalities—a condition encountered in infants with cardiac failure—operative mortality is higher.

Convalescence following operation is usually uneventful, with most patients leaving the hospital in 7 to 10 days. The electrocardiogram reading usually returns to normal within a few months. Once the ductus has been surgically obliterated, cardiac function becomes normal over the ensuing decades of life.

Surgical Problems

The case fatality rate with surgical treatment is less than 1%. The risk without surgery is significantly greater than with the surgery. Patients in their third and fourth decade of life present

with pulmonary hypertension and sclerosis, or calcification of the ductus. These patients constitute a technically difficult and dangerous surgical problem because of friability of the ductus—especially at its junction with the pulmonary artery. Lacerations in this artery may quickly result in a fatal hemorrhage. Thus, the time for surgical correction is early in the child's life before complicating factors arise. Δ

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