UNDERSTANDING FETAL CIRCULATION AND THE TRANSITION TO POSTNATAL CIRCULATION: SHUNTS, PLACENTA AND CONGENITAL HEART DEFECTS

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Annotation. Do you know how blood circulates within the fetus? Is the mother's blood mixed with the fetus's blood? If the mother has AIDS, why doesn't the fetus get infected with AIDS? Fetal circulation is an intriguing process that differs from blood circulation in adults. For instance, adult blood circulation involves two distinct circulatory systems: systemic circulation and pulmonary circulation. In contrast, fetal circulation exhibits a mixed blood circulation.

Keywords: Fetal circulation, placenta Shunts, postnatal circulation, congenital heart defects, changes in circulation.

Introduction. People breath with lungs as well as children, from alveoli oxygen penetrates through diffusely to capillaries then with blood with abundant oxygen comes to all organs. Fetus breathing is different, fetus gets oxygen from his mother through **"placenta"**. Placenta is organ which provide oxygen and nutritions that acquired by diffusion from the mother. Placenta have chorionic villies which provide **"hemato-placentar barrier"** (contributes to the protection of the penetration of maternal blood), any disasters with hemato-placentar barrier contributes penetration infection diseases if mother has (for instance AIDS). Abundant oxygen within blood goes with two arteries through **"umbilical cord"** (ligament inside which there are two arteries going from "mother" to fetus and one vein which going from fetus to mother, but does not mix with mother's blood).

Methods: To explore the topic of fetal circulation and the transition to postnatal circulation, we conducted a comprehensive review of literature on the anatomy and physiology of the fetal and adult circulatory systems. This involved searching electronic databases including PubMed, Embase, and Scopus for relevant articles, as well as consulting textbooks and medical journals.

Blood with abundant oxygen comes from placenta within umbilical cord, the minor part of the blood going to liver by that developing liver lobes. The major part of the blood going to IVC via "**ductus venosus**" (1st shunt), high saturated (oxygenated) blood comes to IVC inside which deoxygenated blood, as a result of this high saturated blood become mixed and merges into right atrium. Mixed blood fills right atrium and the minor part of the blood passes through tricuspid valve and merges to the right ventricle. Major part of the blood passes through "**foramen ovale**" (2nd shunt) which is located between right and left atrium

and merges to the left atrium. Blood in the right ventricle passes through pulmonary trunk and comes to lungs, but lungs does not participate in respiration yet. There are 3rd shunt **"ductus arteriosus"** which is located between aorta and pulmonary trunk, the main function of this shunt is passing mixed blood from pulmonary trunk to aorta. The main reason for this passing is supplying blood each organs that is passed from aorta, because fetus "breathes" with oxygen bringing from umbilical cord, while lungs is not formed yet.

Waste products from the fetal blood are transferred back across the placenta to the mother's blood.

- Mixed blood enters the right atrium. This is the chamber on the upper right side of the heart. When the blood enters the right atrium, most of it flows through the foramen ovale into the left atrium.
- Blood then passes into the left ventricle. This is the lower chamber of the heart. Blood then passes to the aorta. This is the large artery coming from the heart.
- From the aorta, blood is sent to the heart muscle itself and to the brain and arms. After circulating there, the blood returns to the right atrium of the heart through the superior vena cava. Very little of this less oxygenated blood mixes with the oxygenated blood. Instead of going back through the foramen ovale, it goes into the right ventricle.
- This less oxygenated blood is pumped from the right ventricle into the pulmonary artery. A small amount of the blood continues on to the lungs. Most of this blood is shunted through the ductus arteriosus to the descending aorta. This blood then enters the umbilical arteries and flows into the placenta. In the placenta, carbon dioxide and waste products are released into the mother's circulatory system. Oxygen and nutrients from he mother's blood are released into the fetus' blood. At birth, the umbilical cord is clamped and the baby no longer gets oxygen and nutrients from the mother. With the first breaths of life, the lungs start to expand. As the lungs expand, the alveoli in the lungs are cleared of fluid. An increase in the baby's blood pressure and a major reduction in the pulmonary pressures reduce the need for the ductus arteriosus to shunt blood. These changes help the shunt close. These changes raise the pressure in the left atrium of the heart. They also lower the pressure in the right atrium. The shift in pressure stimulates the foramen ovale to close.

Pathophysiology. In fetal circulation, the right side of the heart has higher pressures than the left side of the heart. This pressure difference allows the shunts to remain open. In postnatal circulation, when the baby takes its first breath, pulmonary resistance decreases, and blood flow through the placenta ceases. Blood commences flowing through the lungs, and the pressure on the left side becomes higher than on the right. As a result, the shunts mentioned above close.

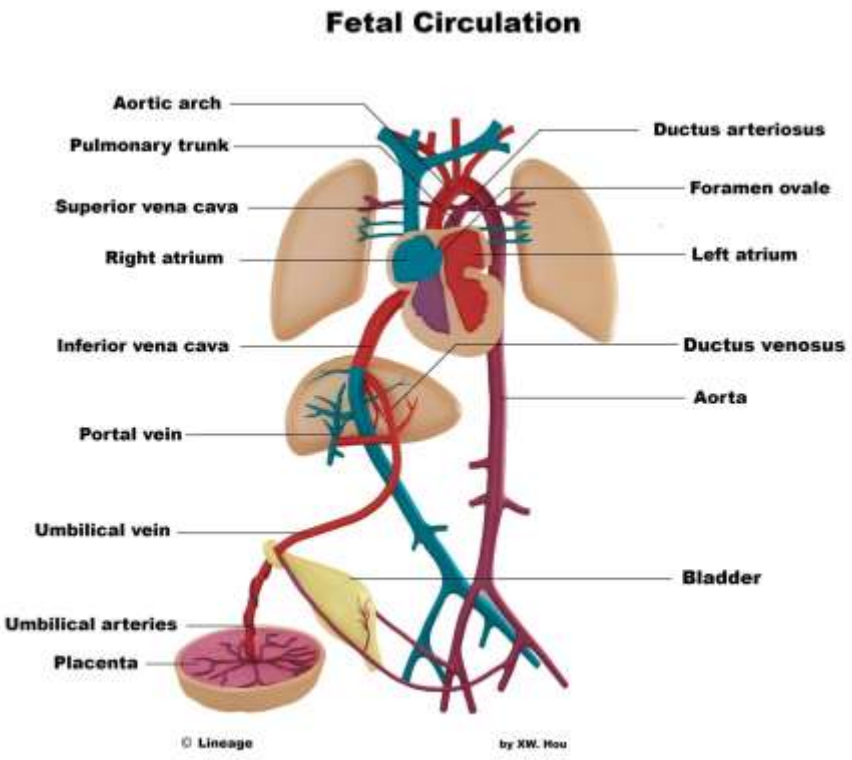

Figure 1. Fetal circulation

Congenital heart defects arise when shunts fail to close after birth. Abnormalities in the anatomy of the heart can also alter the proper flow of blood. These defects can be cyanotic or acyanotic. Cyanotic heart defects are typically from right-to-left shunts in blood after birth. The baby can appear blue at birth, with deoxygenated blood bypassing the lungs and entering the systemic circulation. Examples of cyanotic heart defects are tetralogy of Fallot (TOF), transposition of the great arteries (TGA), persistent truncus arteriosus, tricuspid atresia, and total anomalous pulmonary venous return (TAPVR). Acyanotic heart defects are typically left-to-right shunts in blood after birth. Because the left side contains oxygenated blood, no deoxygenated blood enters the systemic circulation. Instead, some oxygenated blood goes to the right side of the heart and travels through the lungs again. As a result, the baby does not initially appear blue at birth. Examples of acyanotic heart defects are atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), and patent foramen ovale (PFO).

However, the shunt can reverse later in life if the left-to-right shunt goes uncorrected. With the left-to-right shunt, there can be a severe overload of the right heart due to increased blood flow, causing an increase in pulmonary vascular resistance, which causes pulmonary hypertension. Eventually, the right ventricle hypertrophies and the pressure on the right side of the heart becomes more significant than on the left side. As a result, the shunt reverses and becomes right-to-left. Deoxygenated blood starts entering systemic circulation, and the baby can present with cyanosis. This switching of flow from left-to-right to right-to-left is known as Eisenmenger syndrome.

The table 1 provides an overview of patient demographics and the prevalence of congenital heart defects in the United States related to understanding fetal circulation and the

transition to postnatal circulation. The estimated total number of cases is 1,000,000, with patients ranging from 0 to 18 years of age. The gender distribution shows an equal split between males and females, with 500,000 cases each.

Among the congenital heart defects, there are a total of 100,000 cases. Cyanotic defects, characterized by right-to-left shunting of deoxygenated blood, account for 40,000 cases. The most common cyanotic defects include Tetralogy of Fallot (12,000 cases), Transposition of the Great Arteries (8,000 cases), Tricuspid Atresia (5,000 cases), and Total Anomalous Pulmonary Venous Return
(4,000 cases). Acyanotic defects, involving left-to-right shunting of oxygenated blood, make up 60,000 cases. Prominent acyanotic defects include Atrial Septal Defect (20,000 cases), Ventricular Septal Defect (18,000 cases), Patent Ductus Arteriosus (15,000 cases), and Patent Foramen Ovale (7,000 cases). There are also 5,000 cases of other congenital heart defects.

Table 1.

Patient Demographics	Number of Patients
Total Number of Cases	1,000,000 (est.)
Age Range	0-18 years
Gender Distribution	
- Male	500,000
- Female	500,000

Congenital Heart Defects	Number of Cases
Total Cases	100,000
Cyanotic Defects	40,000
- Tetralogy of Fallot	12,000
- Transposition of the Great Arteries	8,000
- Tricuspid Atresia	5,000
- Total Anomalous Pulmonary Venous Return	4,000
Acyanotic Defects	60,000
- Atrial Septal Defect	20,000
- Ventricular Septal Defect	18,000
- Patent Ductus Arteriosus	15,000
- Patent Foramen Ovale	7,000
Other Congenital Heart Defects	5,000

Developing endocardial cushions is essential in understanding why certain cardiac defects develop. The endocardial cushions contribute to the emergence of the atrial and ventricular septa, the mitral and tricuspid valves, the conotruncal septum, and the atrioventricular septa. When there is an endocardial cushion defect, it can cause cardiac

malformations like ASD and VSD. These defects are also common in patients with trisomy 21 and fetal alcohol syndrome. ASDs arise when there is a hole in the atrial septum after birth. An ASD leads to communication between the right and left atria. The primum type of ASD is due to inadequate development of endocardial cushions and is seen less often than the secundum type. VSD arises when there is a hole in the ventricular septum after birth. A VSD leads to communication between the right and left ventricles.

Conotruncal septal defects are accountable for persistent truncus arteriosus, TGA, and TOF. In persistent truncus arteriosus, a single arterial trunk originates from both the right and left ventricles. It is not able to divide into the aorta and pulmonary artery distally. Because of a failure of neural crest cell migration, the conotruncal ridges are not able to form, resulting in this defect, which results in the deoxygenated blood from the right ventricle mixing with the oxygenated blood from the left ventricle, causing cyanosis. In TGA, the aorta and pulmonary artery switch locations. The aorta, in this case, originates from the right ventricle, and the pulmonary artery arises from the left ventricle. As a result, two independent blood circuits do not mix due to the conotruncal septum failing to spiral during development. Deoxygenated blood returns to the right side of the heart, travels through the aorta and goes out to the body.

On the other hand, oxygenated blood returns to the left side of the heart from the lungs and then travels through the pulmonary artery to go back to the lungs. A shunt is needed for survival in this case due to the lack of oxygenated blood being delivered to the body. In TOF, there is an anterior displacement of the conotruncal septum. It is characterized by pulmonary stenosis, a VSD, an overriding aorta, and hypertrophy of the right ventricle. The pulmonary stenosis forces the deoxygenated blood to travel through the VSD from the right side to the left side, leading to right ventricular hypertrophy. Because of the deoxygenated blood crossing over into systemic circulation, the baby presents with early cyanosis.

Vascular malformations may also result in congenital defects. Coarctation of the aorta develops when there is constriction of the aortic arch distal to where the subclavian artery branches off. Pre-ductal indicates that the constriction is before the ductus arteriosus and post-ductal indicates that the constriction is after the ductus arteriosus. In pre-ductal coarctation of the aorta, deoxygenated blood travels from the right atrium to the right ventricle and then through the pulmonary artery. Because a PDA is present, the deoxygenated blood crosses over to the aorta after the point of constriction. In post-ductal coarctation of the aorta, deoxygenated blood travels from the right atrium to the right ventricle and then through the pulmonary artery. Because there is no PDA present, the deoxygenated blood does not cross over to the left side.

Blood circulation after birth.

The closure of the ductus arteriosus, ductus venosus, and foramen ovale completes the change of fetal circulation to newborn circulation. Furthermore, we discussed the significance of endocardial cushions in cardiac development and how defects in these structures can contribute to conditions such as atrial and ventricular septal defects. We also touched upon conotruncal septal defects, which affect the proper division of the aorta and pulmonary artery.

The discussion highlighted the potential complications associated with uncorrected shunts, leading to conditions like Eisenmenger syndrome, where the direction of blood flow reverses, causing cyanosis.

Finally, we briefly mentioned the closure of the ductus arteriosus, ductus venosus, and foramen ovale, which signifies the completion of the transition from fetal to newborn circulation.

Understanding fetal circulation and the transition to postnatal circulation is crucial for identifying and managing congenital heart defects. This knowledge aids in diagnosing and treating these conditions early, improving outcomes for affected individuals. Further research in this field can lead to advancements in prenatal screening, intervention, and long-term management of congenital heart defects.

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