What is fetal circulation pdf

During pregnancy, fetal circulation works differently than after birth:The fetus is connected by the umbilical cord to the placenta, the organ that develops and implants in the mother's uterus during pregnancy. Through the blood vessels in the umbilical cord, the fetus receives all the necessary nutrition, oxygen and life support from the mother via the placenta. Waste and carbon dioxide from the fetus are sent back through the umbilical cord and placenta to the mother's circulatory system to be eliminated. The fetal circulatory system uses two right to left shunts, which are small passages that direct blood that needs to be oxygenated. The purpose of these shunts is to bypass certain body parts? in particular, the lungs and liver? that are not fully developed while the fetus is still in the womb. The shunts that bypass the lungs are called the foramen oval, which moves blood from the right atrium from the heart to the left atrium, and the ductus arteriosus, which moves blood from the pulmonary artery to the aorta. Oxygen and nutrients from the mother's blood are transferred through the placenta to the fetus. The enriched blood flows through the umbilical cord to the liver and splits into three branches. The blood then reaches the inferior vena cava, an important vein connected to the heart. Most of this blood is sent through the ductus venosus, also a shunt that passes very oxygenated blood through the liver to the inferior vena cava and then to the right atrium of the heart. A small amount of this blood goes directly to the liver to give it the oxygen and nutrients it needs. Waste from the fetal blood is transferred through the placenta to the mother's blood. In the fetal heart: Blood enters the right atrium, the room in the upper right of the heart. When the blood enters the right atrium, most of it flows through the foramen oval into the left atrium. Blood then passes into the left ventricle (lower chamber of the heart) and then to the aorta, (the major artery from the heart). From the aorta, blood is sent to the heart muscle itself next to the brain. After circulation there, the blood returns to the right atrium of the heart by the superior vena cava. About two thirds of the blood will pass through the foramen oval as described above, but the remaining one-third will enter the right ventricle, toward the lungs. In the fetus, the placenta does the work of breathing instead of the lungs. As a result, only a small amount of blood remains on the lungs. Most of this blood is bypassed or driven away from the lungs via the ductus arteriosus to the aorta. Most of the circulation to the lower body is provided by blood passing through the ductus arteriosus. This blood then enters the navel arteries and flows placenta. In the placenta, carbon dioxide and waste are released into the mother's bloodstream, and oxygen and nutrients from the mother's blood are in the blood of the fetus. At birth, the umbilical cord is clamped and the baby no longer receives oxygen and nutrients from the mother. With the first breaths of life, the lungs begin to expand. As the lungs expand, the alve vesicers in the lungs are cleared of fluid. An increase in the baby's blood pressure and a significant reduction in lung pressure reduces the need for the ductus arteriosus to shun blood. These changes promote the closure of the shunt. These changes increase the pressure in the left atrium of the heart, which decreases the pressure in the right atrium. The shift in pressure stimulates the foramen oval to close. The closure of the ductus arteriosus and foramen ovale completes the transition from fetal circulation to newborn circulation. The fetal circulation system is clearly different from the adult circulation. This complicated system allows the fetus to receive oxygenated blood and nutrients from the placenta. It consists of the blood vessels in the placenta and the umbilical cord, which contains two umbilical arteries and an umbilical cord. Fetal circulation bypasses the lungs via a shunt known as the ductus arteriosus; the liver is also bypassed via the ductus venosus and blood can travel through the oval oval from the right atrium to the left atrium. Normal fetal heart rate is between 110 and 160 peat per minute. Compared to adults, fetuses have reduced ventricular filling and decreased contractility. [1] Fetal circulation undergoes a rapid transition after birth to accommodate the extra-uterine life. Human understanding of fetal circulation originated from fetal sheep, but ultrasound and magnetic resonance imaging (MRI) during the fetal period now provide detailed information. [2] There are clear differences in fetal circulation which, if not properly formed, can lead to childhood or adult diseases. With advancing medical technology babies are viable on earlier weeks of pregnancy than ever before – immature hearts' transition to newborn circulation has longevity-long effects. Adults, born prematurely, were studied and were found to have increased heart muscle mass, decreased room length, and impaired function. These limitations are deeper in those born deeply premature; they were found to have 50% more ventricular heart muscle mass than those born over time. These patients are at increased risk of ischemic heart disease and heart failure. [1] Another area of concern is when certain shunts do not close after birth, the baby may be born with congenital heart defects that are present with varying signs and symptoms. This will be discussed below. The yolk sac initiates erythropoiesis until the liver can take over at 5 weeks gestation and then the bone marrow eventually contributes to 6 months gestation. The relative activates hypoxia inducible factor-1 compared to the mother to stimulate the production of erythropoietin in the kidneys, which improves the capacity of fetal blood for oxygen. Fetal hemoglobin hemoglobin has a higher affinity for oxygen compared to maternal hemoglobin. However, fetal tissue has adapted ways to release oxygen from the higher hemoglobin affinity when in the fetal tissue by creating an acidic environment. By the age of 4 to 6 months, the baby will have an adult level of hemoglobin and will have no fetal hemoglobin. After birth, erythropoiesis will also slow down. [2] [3] [4] The fetal heart initiates after 22 days; this indicates the opening of fetal circulation. Gas exchange takes place initially in the yolk sac until the placenta completely takes over. This transition occurs around 10 weeks gestation. Maternal oxygen blood mixes with placental blood that contains little oxygen before they go to the fetus. As a result of this mingling, the fetus is relatively hypoxic compared to maternal, arterial blood. [2] When the baby is born, the cardiovascular system undergoes a rapid, drastic change. With his first breath, the baby's pulmonary vascular resistance decreases significantly, which is a reaction to the oxygen that is now present in the lungs and the physical act of breathing. With the clamping of the umbilical cord after birth, the systemic vascular resistance increases and the blood flow to the lungs helps. The ductus arteriosus has a flow from left to right within 10 minutes. The smooth muscle in the ductus arteriosus responds to oxygen by increasing calcium channel activity causing narrowing and eventually shunt closure. The increased systemic resistance also increases the pressure in the left atrium to be higher than the right atrium, and this causes the foramen oval to close. [2] The mother's uterus promotes the environment for fetal growth and placental vitality. Every organ system is involved in the process of fetal circulation, because as the fetus grows and develops, it needs oxygen and nutrients that the blood provides. The fetal blood will reach every aspect of the growing fetus, except for the liver and lungs that are bypassed. However, the fetal arterial system receives waste from those organs. The fetal circulatory system provides the fetus with nutrients and oxygen, while also removing waste and carbon dioxide from fetal circulation. The placenta connects the fetus to the wall of the uterus. It supplies oxygen and nutrients from the mother to the growing fetus and also removes metabolic waste and carbon dioxide from the fetus through the blood vessels in the umbilical cord. The umbilical cord develops from the placenta and is attached to the fetus. Oxygenated blood from the mother in the placenta flows through the umbilical cord and into the inferior vena cava (IVC), bypassing the liver via the ductus venosus. From the IVC, oxygenated blood travels to the right atrium of the heart. There is a pressure in the right atrium compared to the left atrium in fetal circulation; therefore most of the blood from the right atrium to the left atrium is called by an opening called foramen foramen Once in the left atrium, blood travels through the left ventricle into the aorta and systemic circulation. The oxygen-deprived blood goes back to the placenta through the navel arteries to be oxygenated by the mother. In addition, some oxygenated blood in the right atrium can also enter the right ventricle and then the pulmonary artery. Because there is a high resistance to the blood flow in the lungs, the blood of the pulmonary artery in the aorta is sununted through the ductus arteriosus, bypassing the lungs. Blood then enters systemic circulation, and the oxygenated blood is recycled back to the mother through the navel arteries. [2] [5] [2] Tests to detect congenital heart defects can be completed during pregnancy. A fetal echocardiogram can visualize the fetal heart as early as 16 weeks of pregnancy. Some defects do not manifest until after birth. In these cases, evaluation consists of a thorough history and physical examination, an echocardiogram and possibly cardiac catheterization. Near-infrared spectroscopy (NIRS) is a tool to measure non-invasive oxygen formation of fetal tissues that doctors can then calculate by blood flow and oxygen supply. [2] Other tests may be performed, depending on the presenting signs and symptoms. In tetralogy of Fallot, a boat-shaped heart due to right ventricular hypertrophy may be present on the chest X-ray. Rib notching may be present on the chest x-ray in patients with coarctation of the aorta, due to the intercostal arteries enlarging as the patient ages. An echocardiogram shows any structural or valve abnormalities in the fetal or newborn heart. In fetal circulation, the right side of the heart has a higher pressure than the left side of the heart. This pressure difference ensures that the shunts remain open. In postnatal circulation, when the baby blows out his first breath, lung resistance decreases and blood flow through the placenta stops. Blood begins flowing through the lungs, and the pressure in the left side becomes higher than the right. As a result, the shunts mentioned above close. Congenital heart defects occur when shunts fail to close after birth. Abnormalities in the anatomy of the heart can also alter the right blood flow. These defects can be cyanotic or acyanotic. Cyanotic heart defects are usually right to left shunts in blood after birth. With oxygenated blood bypassing the lungs and entering systemic circulation, the baby may appear blue at birth. Examples of cyanotic heart defects include tetralogy of Fallot (TOF), conversion of the major arteries (TGA), persistent truncus arteriosus, tricuspid atresia and total abnormal pulmonary recurrence (TAPVR). Acyanotic heart defects are usually left to right shunts in after birth. Because the left side contains oxygenated blood, there is no oxygenated blood in systemic circulation. Instead, some oxygenated blood goes to the right side of the heart and travels through the lungs again. As a result, it may be possible to baby does not initially appear blue at birth. Examples of acyanotic heart defects include atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA) and patent foramen oval (PFO). However, the shunt may reverse life later in life if the left-to-right shunt goes uncorrected. With the shunt from left to right, there may be a severe overload of the right heart due to an increase in blood flow, causing an increase in pulmonary vascular resistance, which causes lung hypertension. Eventually, the right ventricle hypertrophies and the pressure on the right side of the heart becomes more important than the left side. As a result, the shunt reverses and is moved from right to left. Oxygenated blood begins in systemic circulation, and the baby can present it with cyanosis. This switch from left to right is known as Eisenmenger syndrome. The development of endocardial pillows is essential to understand why certain heart defects develop. The endocardial cushions contribute to the emergence of the atrium and ventricular septa, the mitral and tricuspid valves, the conotruncal septum, and the atrioventricular septa. When there is an endocardial pillow defect, it can cause cardiac deformities such as ASD and VSD. These abnormalities are also common in patients with trisomy 21 and fetal alcohol syndrome. ASDs occur when there is a hole in the atrium septum after birth. An ASD leads to communication between right and left atria. The primum type ASD is due to insufficient development of endocardial pillows and is seen less often than the secondary type. VSD occurs 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 responsible for persistent truncus arteriosus, TGA and TOF. In persistent truncus arteriosus, a single arterial torso comes from both the right ventricle and left ventricle. It is unable to distribute distally in the aorta and pulmonary artery. Due to a failure of neural crest cell migration, the conotruncal ridges cannot form, resulting in this defect, resulting in the oxygenated blood from the right ventricle that mixes with the oxygenated blood from the left ventricle, causing cyanosis. In TGA, the aorta and pulmonary artery change location. The aorta, in this case, comes from the right ventricle, and the pulmonary artery emerges from the left ventricle. As a result, there are two independent circuits of blood that do not mix due to the conotruncal septum not to spiral during development. Oxygenated blood returns to the right side of the heart and then travels through the aorta and goes out to the body. On the other hand, oxygenated blood returns to the left side of the heart of lungs and then travels through the pulmonary artery to get back to the lungs. A shunt is needed to survive in this case due to the lack of oxygenated blood being delivered to Body. In TOF there is an anterior displacement of the conotruncal septum. It is characterized by lung stenosis, a VSD, a compelling aorta, and hypertrophy of the right ventricle. The lung stenosis forces the oxygenated blood to travel from the right side to the left side through the VSD and leads to the right ventricular hypertrophy. Because of the oxygen-oxygenated blood crossing in systemic circulation, the baby presents itself with early cyanosis. Vascular deformities can also lead to birth defects. Coarctation of the aorta develops when there is narrowing of the aortic imbalance to where the subclavian artery branches. Pre-ductal indicates that the narrowing before the ductus arteriosus and post-ductal indicates that the narrowing after the ductus is arteriosus. In pre-ductal coarctation of the aorta, oxygenated blood travels from the right atrium to the right ventricle and then through the pulmonary artery. Because a PDA is present, the oxygenated blood passes to the aorta after the point of narrowing. In post-ductal coarctation of the aorta, oxygenless blood travels from the right atrium to the right ventricle and then through the pulmonary artery. Because there is no PDA present, the oxygenated blood does not transfer to the left side. The clinical presentation of babies with ASD is related to the size of the hole. Patients are often asymptomatic and are only detected when hearing a murmur. However, a patient with a large ASD may present with heart failure, failure to thrive, or recurrent lung infections. The noise associated with ASD is a fixed split of S2, due to A2 occurring before P2, which is due to the increase in the right atrium and right ventricular volumes, which eventually increase flow through the pulmonic valve, delaying delay. [6] [7] [8] The clinical presentation of babies with VSD also depends on the size of the defect. Small VSDs are often asymptomatic. Larger VSDs with significant left-to-right shunting can lead to failure and congestive heart failure because the heart is unable to pump enough blood to meet body demand. A holosystolic murmur that is emoped to the left lower sternal boundary. On physical examination of babies born with PDA, patients have a continuous machine-like murmur, loudest in S2. It is best irrigated in the left infraclavicular area and associated with innate rubella or prematurity. In TOF, a child often presents with tet spells. These spells mean that cyanosis can develop when a child becomes agitated. After physical examination, the patient will have a blue tint to their lips and around their mouth, their skin will be moist or warm, and breathing will be fast. [9] A child with pre-ductal coarctation of the aorta can present himself with differential cyanosis. There will only be the lower extremity cyanosis because oxygenated blood is not of the aorta coming to the upper extremities. In post-ductal coarctation of the a child will not be present with cyanosis because a PDA is not present. A child with coarctation of the aorta may also be present with high blood pressure in the upper extremities due to the high pressure for narrowing and low blood pressure in the lower extremities after the point of narrowing. TreatmentProstaglandins of the placenta keep the shunts in the fetus open. During birth, the shunts usually close due to the loss of prostaglandins from placental separation and increase in oxygen due to breathing. However, in the management of PDA, NSAIDs such as indomethacine can be given to close the shunt because it blocks the production of prostaglandins. TGA is incompatible with life, except when there is another defect in the heart, which can mix blood. Prostaglandins can be given to allow the oxygenated blood and oxygenated blood to mix by keeping the ductus arteriosus open. In TOF, cracking can improve symptoms. Cracking kinks the femoral arteries, increasing systemic vascular resistance. As a result, the pressure of the left heart is greater than the pressure of the right heart, which reverses the shunt. However, early surgical correction is the recommendation. Secondary Education/Review QuestionsFetal circulation. Contributed by T. Silappathikaram 1.Finnemore A, Groves A. Physiology of fetal and transitional circulation. Semin Foetal Neonatal Med. 2015 Aug.; 20(4):210-6. [PubMed: 25921445] 2.Morton SU, Brodsky D. Fetal physiology and the transition to extrauterine life. Clin Perinatol. 2016 Sep;43(3):395-407. [PMC free article: PMC4987541] [PubMed: 27524443] 3.Feng SYS, Hollis JH, Samarasinghe T, Phillips DJ, Rao S, Yu VYH, Walker AM. Endotoxin-induced cerebral pathophysiology: differences between fetus and newborn. Physiol Rep. 2019 Feb.7(4):e13973. [PMC free article: PMC6381816] [PubMed: 30785235] 4.Vonck S, Staelens AS, Lanssens D, Tomsin K, Oben J, Dreesen P, Bruckers L, Gyselaers W. 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