

COARCTATION OF THE AORTA

In the normal heart, blood flows to the body through the aorta, which connects to the left ventricle and arches over the top of the heart. In coarctation of the aorta, the aorta is pinched (in medical terms, coarcted) at a point somewhere along its length. This pinching restricts blood flow from the heart to the rest of the body. Often, the baby also has other heart defects, such as a narrowing of the aortic arch (in medical terms, transverse aortic arch hypoplasia).

Usually no symptoms are apparent at birth, but can develop within a week of birth with the closure of the ductus arteriosus. The possible consequences of this condition are poor feeding, an enlarged heart, an enlarged liver and congestive heart failure. Blood pressure also increases above the constriction.

Timing of surgery depends on the degree of coarctation. Surgery may be delayed with mild coarctation (which sometimes is not detected until adolescence). However, if a baby develops congestive heart failure or high blood pressure, early surgery is usually required. A baby with severe coarctation should have early surgery to prevent long-term high blood pressure.

The coarctation can be repaired in a number of ways without opening the heart. The surgeon can remove the narrowed part of the aorta and sew the ends together, thereby creating an anastomosis. (An anastomosis is the surgical formation of a connection between two normally distinct structures.) Alternatively, the surgeon can cut the narrowed section open and sew a patch over the opening.

INTERRUPTED AORTIC ARCH

In this condition, the aorta does not develop completely in the area of the arch. As a result, the aorta is divided into two parts that are not connected to each other. This prevents the flow of blood through the aorta. Because blood flow through the aorta is blocked, the blood supply to the lower body is compromised. At birth, the open ductus arteriosus allows blood to flow from the right ventricle to the lower body. There are often other coexisting heart defects, such as a VSD.

Interrupted aortic arch is usually diagnosed when the ductus arteriosus begins to close. With decreasing circulation to the lower body, serious damage can occur to the liver, kidneys and intestines. Prostaglandin is given to keep the ductus arteriosus open until surgery is performed, usually as soon after birth as possible.

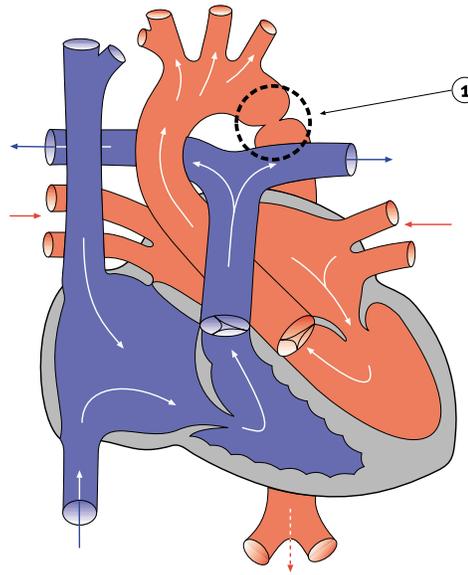


Diagram 2.9 Coarctation of the aorta

1 – pinched or coarcted aorta

Flow patterns are normal but are reduced below the coarctation. Blood pressure is increased in vessels leaving the aorta above the coarctation. The broken white arrow indicates diminished blood flow through the aorta.

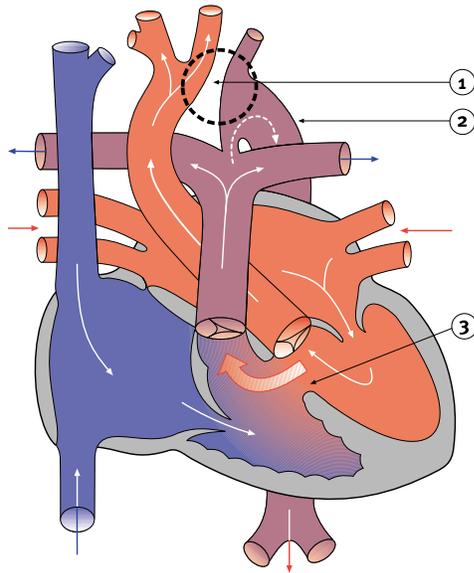


Diagram 2.10 Interrupted aortic arch

- 1 – interruption of aortic arch
- 2 – descending aorta connected to pulmonary artery by large patent ductus arteriosus
- 3 – ventricular septal defect

Flow patterns are normal to the upper body. However, there is no flow of oxygenated blood to the lower body unless there exist, as in this drawing, shunts such as a ventricular septal defect that allows oxygenated blood into the pulmonary artery, and a patent ductus arteriosus that allows the partially oxygenated blood to travel from the pulmonary artery to the descending aorta (as indicated by the broken white arrow).

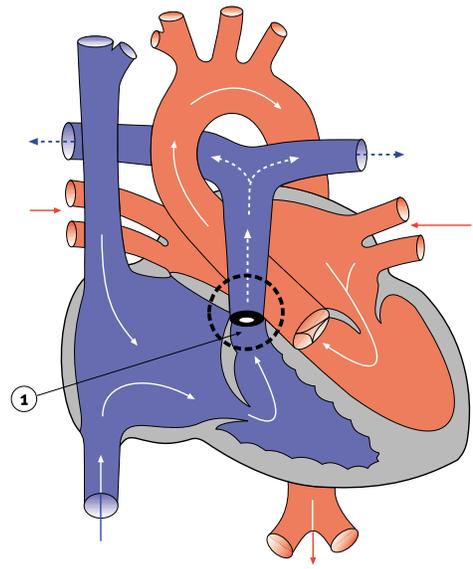


Diagram 2.11 Pulmonary stenosis (valvular)

- 1 – narrowed pulmonary valve

Blood flow patterns are normal but blood flow through the pulmonary artery is reduced as indicated by the broken white arrows.

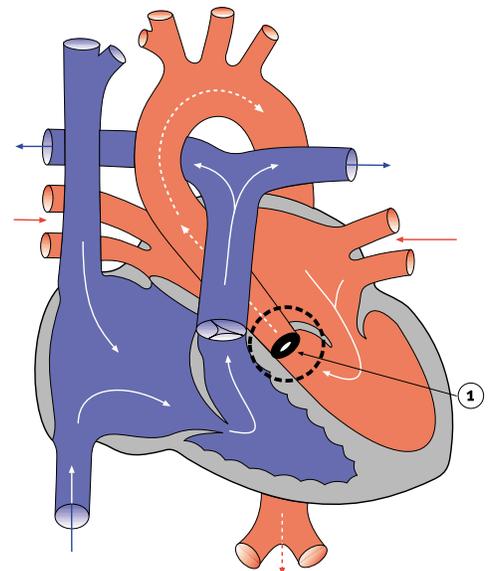


Diagram 2.12 Aortic stenosis (valvular)

- 1 – narrowed aortic valve

Flow patterns are normal but blood flow to the aorta is reduced as indicated by the broken white arrows.

Most often a one-stage repair is undertaken, patching the arch or connecting the two ends of the aortic arch together, closing any VSD, and tying off and dividing the ductus arteriosus into two parts.

PULMONARY STENOSIS AND AORTIC STENOSIS

Any one of the heart's valves may suffer from narrowing (or stenosis). This narrowing, which can occur at, above or below the valve, can block blood flow.

In pulmonary stenosis, the area around the pulmonary valve can become blocked or the blood vessel beyond the valve can become narrowed, causing the right ventricle to have to pump harder to get blood past the blockage to the pulmonary artery. If the stenosis is severe, there will be a lack of blood flow to the lungs and the baby can become cyanotic. The condition requires treatment when the pressure in the right ventricle is high.

In aortic stenosis, the aortic valve also can become narrowed, requiring the left ventricle to pump harder to get blood past the blockage to the aorta and out to the body. The normal aortic valve has three cusps or leaflets and is described as tricuspid. In contrast, a stenotic aortic valve may have only two cusps (bicuspid) or even one cusp (unicuspid). The cusps in a stenotic valve are usually thick and stiff.

One treatment for stenosis is to insert a special catheter containing a balloon through the valve. The balloon is then inflated, stretching the valve open. This procedure is known as a balloon valvuloplasty and is performed during a cardiac catheterization (a procedure that is discussed in the following chapter). In most children, a valvuloplasty may be sufficient to alleviate the stenosis. In other children, surgery may be needed.

TRICUSPID ATRESIA

Atresia means the absence or closure of a normal body opening or tubular structure. In tricuspid atresia, the tricuspid valve is missing, preventing blood from flowing from the right atrium into the right ventricle. Because the right ventricle has no blood to pump, it remains small and underdeveloped.

In this condition, the child's survival depends on the presence of two septal defects: an atrial septal defect (ASD) and a ventricular septal defect (VSD). The ASD allows the venous blood to flow from the right atrium into the left atrium. There, venous blood mixes with oxygen-rich blood from the lungs, flows to the left ventricle, into the aorta and out to the body. The rest of the mixture is pumped from the left ventricle through the VSD into the right ventricle, and on through the pulmonary artery back to the lungs.

Sometimes the baby will also have other cardiac defects. These include transposition of the great arteries (to be discussed later), coarctation of the aorta or pulmonary atresia.

The majority of babies with tricuspid atresia are cyanotic at birth. These children will require surgery to place a tube (known in medical terms as a shunt), which will provide an increase in blood flow to the lungs.

Other children have too much blood flowing to the lungs and need a procedure called pulmonary artery banding. In this procedure, the surgeon places a band around the pulmonary artery to narrow it, and to reduce the blood flow and resultant high pressure in the lungs.

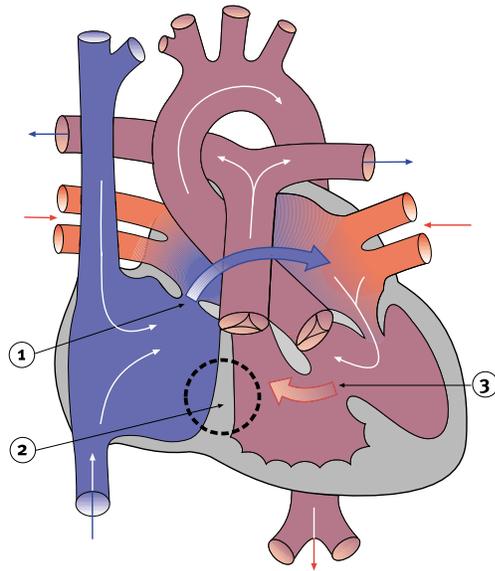


Diagram 2.13 Tricuspid atresia (showing no opening between the right atrium and ventricle)

- 1 – atrial septal defect
- 2 – absent tricuspid valve
- 3 – ventricular septal defect

Blood is shunted through an atrial septal defect to the left atrium and through the ventricular septal defect to the pulmonary artery. The shaded arrows indicate mixing of the blood.

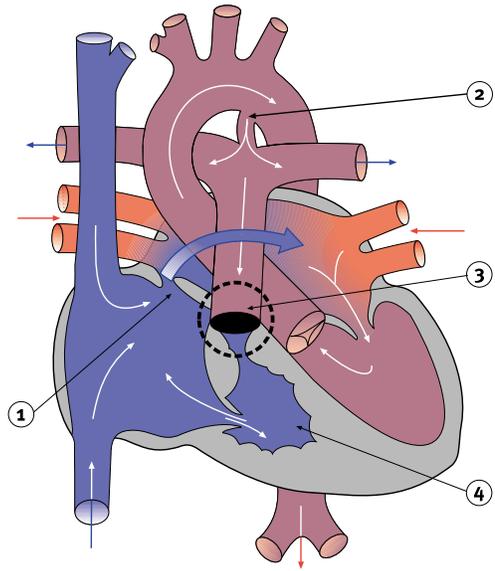


Diagram 2.14 Pulmonary atresia

- 1 – atrial septal defect
- 2 – patent ductus arteriosus
- 3 – absent pulmonary valve
- 4 – hypoplastic right ventricle

Abnormal blood flow (as indicated by the shaded blue arrow) is from the right atrium and right ventricle through an atrial septal defect to the left side of the heart. Blood can reach the pulmonary arteries only through a patent ductus arteriosus.

In other children the surgeon connects the right atrium and the pulmonary artery and closes the ASD. This procedure is known as a Fontan, named after the doctor who first perfected the procedure. The Fontan repair allows blood to flow passively from the right atrium to the pulmonary circulation without being pumped by the right ventricle. The functioning of this repair relies totally on the preservation of a low resistance in the pulmonary arterial vessels.

PULMONARY ATRESIA

In the condition known as pulmonary atresia, no pulmonary valve exists. Blood is therefore unable to flow from the right ventricle into the pulmonary artery and onward to the lungs. As a further result, the right ventricle usually remains hypoplastic. In addition, the tricuspid valve or right atrioventricular valve is often poorly developed.

With pulmonary atresia, the only means of getting blood flow to the lungs is through the ductus arteriosus, which connects the pulmonary artery and the aorta. The infant is usually cyanotic.

Blood returning to the right atrium and right ventricle therefore needs to shunt across to the left side of the circulation, through either an ASD or a patent foramen ovale. The foramen ovale is a natural septal open-

ing in the fetal heart that allows blood to flow between the atria. In most newborns this opening closes at birth. Failure of the foramen ovale to close results in a type of atrial septal defect, known as a patent foramen ovale (PFO). Where there is an ASD or a PFO, blood leaves the right atrium and mixes with the oxygen-rich blood in the left atrium. This blood is then pumped by the left ventricle into the aorta and out to the body.

A further problem arises when the ductus arteriosus closes, causing severe cyanosis. The infant is then usually given prostaglandin to keep the ductus arteriosus open. As a palliative measure, a surgeon may create a shunt between the pulmonary artery and the aorta to increase blood flow to the lungs. If the right ventricle is too small to be effective as a pump, a Fontan procedure is performed later, connecting the right atrium directly to the pulmonary artery. The ASD or PFO is also closed to relieve the cyanosis.

TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION

The pulmonary veins usually connect to the left atrium. However, in total anomalous pulmonary venous connection (TAPVC), there is no direct connection between any pulmonary vein and the left atrium. The pulmonary veins that normally bring oxygen-rich blood back from the lung are not connected to the left atrium. Instead, they drain through abnormal connections to the right atrium.

In the right atrium, the oxygen-rich blood from the pulmonary veins mixes with the oxygen-poor venous blood from the body. Some of this mixture then passes through an atrial septal defect or a PFO into the left atrium. The result is that the blood flowing to the body is poorly oxygenated. The rest of the poorly oxygenated mixture flows through the right ventricle into the pulmonary artery and on to the lungs.

Both congestive heart failure and pulmonary artery hypertension may develop. Signs of these may appear soon after birth and vary in severity. They include a lethargic appearance, pallor or cyanosis, poor feeding and weight loss. TAPVC is usually repaired in early infancy or when the abnormality is diagnosed. Surgical repair involves reconnecting the pulmonary veins to the left atrium and closing the atrial septal defect or PFO.

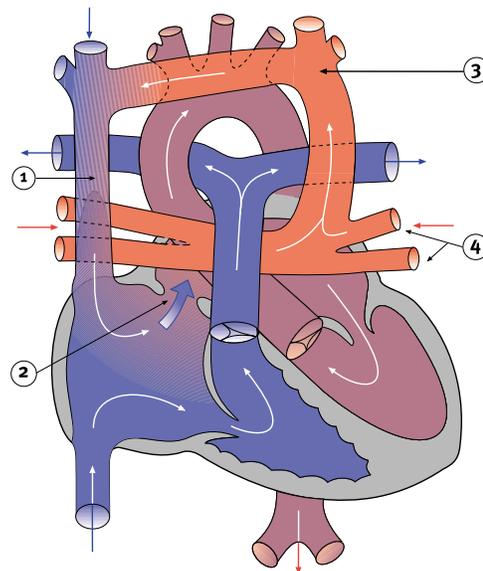


Diagram 2.15 Total anomalous pulmonary venous connection (showing pulmonary veins connected to the left innominate vein)

- 1 – superior vena cava
- 2 – atrial septal defect
- 3 – left innominate vein
- 4 – pulmonary veins

Oxygenated blood returning from the lungs is routed back into the superior vena cava, rather than the left atrium. The presence of an atrial septal defect is necessary to allow partially oxygenated blood to reach the left side of the heart.

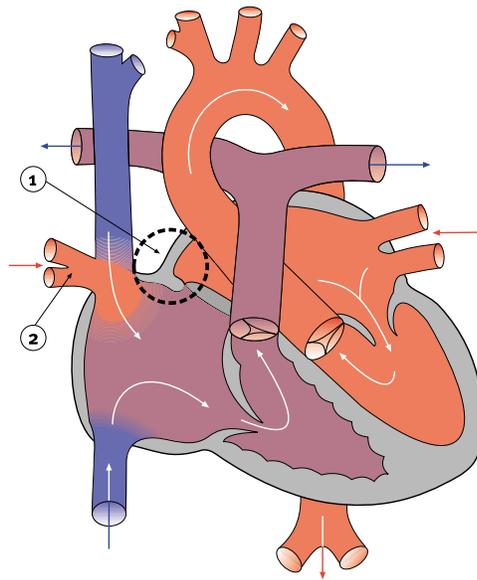


Diagram 2.16 Partial anomalous pulmonary venous drainage

- 1 – location where right pulmonary veins normally enter left atrium
- 2 – right pulmonary veins entering right atrium

Oxygenated blood flows from the right pulmonary veins into the right atrium instead of into the left atrium.

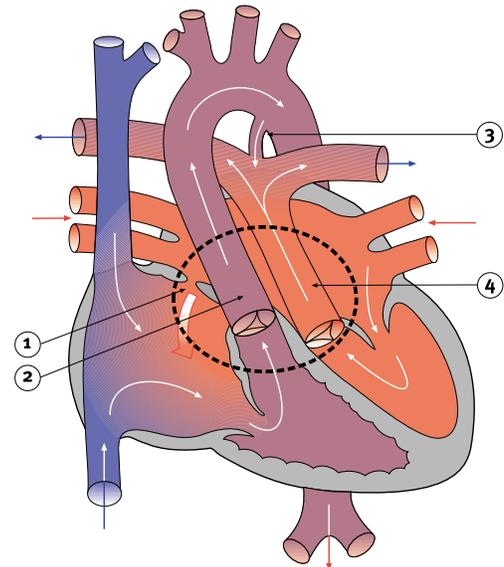


Diagram 2.17 Transposition of the great arteries

- 1 – atrial septal defect or patent foramen ovale
- 2 – aorta connected to right ventricle
- 3 – patent ductus arteriosus
- 4 – pulmonary artery connected to left ventricle

With the presence of an atrial septal defect, two parallel blood flows exist, with one recirculating oxygenated blood and one recirculating de-oxygenated blood. As shown in this diagram, mixing occurs via the atrial septal defect.

PARTIAL ANOMALOUS PULMONARY VENOUS DRAINAGE

Partial anomalous pulmonary venous drainage (PAPVD) exists when one or more of the pulmonary veins drain to the left atrium while the other vein is connected to the right atrium. The result is that some oxygen-rich blood is returned to the right atrium and mixes with venous blood, and is then returned to the lungs instead of flowing out of the heart to the body. The result of this anomaly is that the right side of the heart must work harder. PAPVD is often associated with an ASD or with more complex cardiac defects.

Surgery is needed when the child has increasing pulmonary pressures or develops congestive heart failure. Depending on the complexity of the PAPVD, surgery usually involves connecting the pulmonary veins to the left atrium.

TRANSPOSITION OF THE GREAT ARTERIES

In transposition of the great arteries (also sometimes called transposition of the great vessels), the aorta and the pulmonary artery (the great arteries) are each connected to the wrong ventricles. The aorta is connected to the right ventricle, so that venous blood is carried to the body instead of to the lungs. The pul-

monary artery is attached to the left ventricle, so that oxygen-rich blood is carried back to the lungs instead of to the body.

Newborns with transposition survive only if they have one or more connections that let oxygen-rich blood reach the body. These connections may be in the form of a hole between the two ventricles, a patent foramen ovale or a patent ductus arteriosus. Most babies born with transposition are extremely blue soon after birth because their bodies are not receiving enough oxygenated blood.

In order to immediately improve the body's oxygen supply, prostaglandin is given to keep the ductus arteriosus open. It is also possible to enlarge the patent foramen ovale by inflating a small balloon during a heart catheter procedure. This is known as a balloon atrial septostomy and allows more mixing between the right and left sides of the circulation.

A common surgical procedure for treating transposition is known as an arterial switch. In this operation, the great arteries are disconnected from the ventricles to which they are attached at birth and are reconnected to the appropriate ventricles. The pulmonary artery is connected to the right ventricle, while the aorta is connected to the left ventricle. The coronary arteries are also re-implanted. Depending on the condition of the baby, this procedure may be done in the first few weeks after birth. The repair is more complicated if there is also a large VSD or other defects. An arterial switch operation is considered a high-risk procedure.

DOUBLE OUTLET RIGHT VENTRICLE

Double outlet right ventricle (DORV) is a condition in which both the pulmonary artery and the aorta connect to the right ventricle. DORVs are usually accompanied by one of a number of ventricular septal defects. The three types of VSDs most likely to occur with DORV are subaortic VSD (the defect is located beneath the aortic valve), subpulmonary VSD (the defect is located beneath the pulmonary valve) and doubly committed VSD (the defect is located within the septal band and immediately beneath the leaflets of the aortic and pulmonary valves). Sometimes the baby will have other associated cardiac defects.

Symptoms tend to occur early in life, often within days of birth. The two major symptoms are congestive heart failure and cyanosis. In some cases, cyanosis can become severe because of reduced pulmonary blood flow.

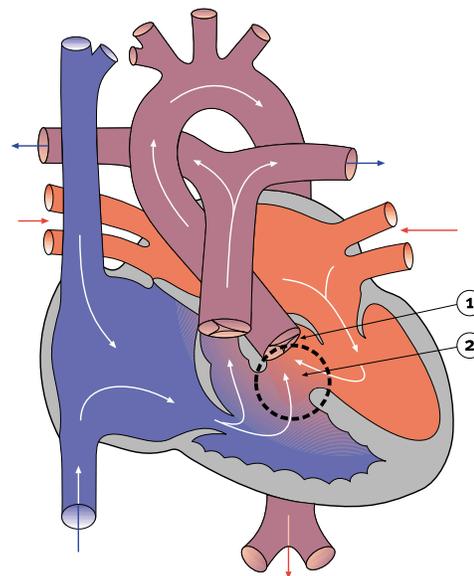


Diagram 2.18 Double outlet right ventricle

1 – overriding aorta

2 – ventricular septal defect

De-oxygenated blood enters the aorta from the right ventricle and is returned to the body.

The goal of the surgical treatment of DORV is a complete anatomic repair. The left ventricle is connected to the aorta, the right ventricle is connected to the pulmonary artery and the VSD is closed. Usually a complete repair is undertaken at as early an age as possible.

HYPOPLASTIC LEFT HEART SYNDROME

Hypoplastic left heart syndrome (HLHS) refers to the grouping of certain congenital cardiac defects. The main characteristics are the marked hypoplasia (underdevelopment) or even absence of the left ventricle and severe hypoplasia of the aorta. Often a localized pinching (coarctation) of the aorta is also present. The main pulmonary artery is enlarged, and gives rise to a large ductus arteriosus. This allows blood to flow from

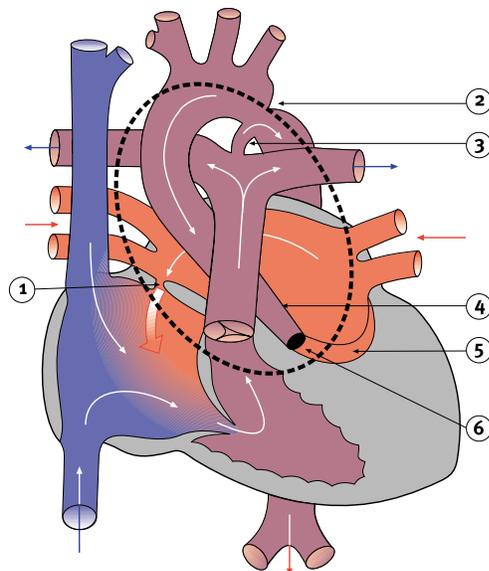


Diagram 2.19 Hypoplastic left heart syndrome (HLHS)

- 1 – patent foramen ovale
- 2 – pinched aorta
- 3 – patent ductus arteriosus
- 4 – narrowed aorta
- 5 – hypoplastic left ventricle
- 6 – aortic atresia

The lack of a developed left ventricle, plus the aortic coarctation leads to reversed blood flow through the aorta. Partially oxygenated blood reaches the aorta after travelling through the patent foramen ovale, up the pulmonary trunk and through the patent ductus arteriosus. The major blood flow to the systemic circulation is through the PDA.

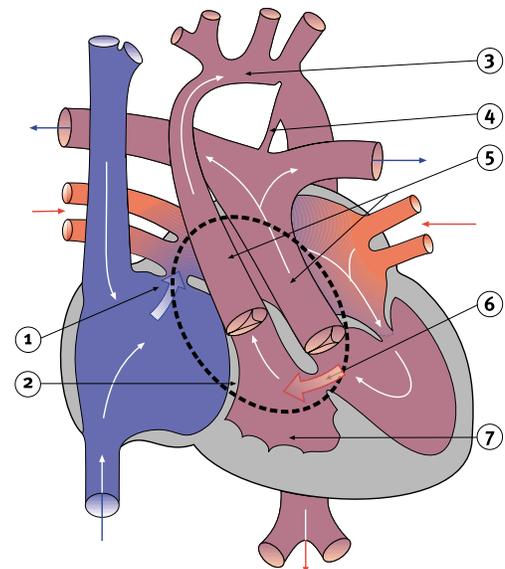


Diagram 2.20 Functional hypoplastic left heart syndrome

- 1 – patent foramen ovale or atrial septal defect secundum
- 2 – tricuspid valve atresia
- 3 – hypoplastic aortic arch
- 4 – patent ductus arteriosus
- 5 – transposition of the great arteries
- 6 – restrictive muscular ventricular septal defect
- 7 – hypoplastic right ventricle

De-oxygenated blood flows from the right atrium through an atrial septal defect into the left atrium. There it mixes with oxygenated blood returning from the lungs. A portion of this blood flows from the left ventricle up the pulmonary trunk to the lungs and through the patent ductus arteriosus to the body. Another portion of this blood flows through a ventricular septal defect, and is pumped by the hypoplastic right ventricle up the aorta.

the right ventricle into the aorta and out to the body. Other characteristics of HLHS often include a combination of aortic and mitral stenosis or aortic and mitral atresia.

HLHS is also used to describe cases in which the right ventricle is hypoplastic and the child also suffers from transposition of the great arteries. In such cases the hypoplastic right heart pumps into the same artery that the left ventricle normally pumps blood through, and therefore the circulation is the same as in HLHS. This is sometimes referred to as functional HLHS.

HLHS is a severe form of congenital heart disease. Without surgical intervention, HLHS is fatal. Infants are often diagnosed within 24 to 48 hours of birth. Symptoms appear when the ductus arteriosus begins to close.

There are three options for treating these children: supportive care until death occurs, staged reconstruction of the heart, or a heart transplant.

Supportive care

Before surgical treatment was developed, at least 90 per cent of infants with this condition died by the age of one month. Even today, some infants are not candidates for surgical therapy. For these infants, supportive care is the only option. Exactly what this care entails will depend on the condition of the infant, but would likely include assistance with breathing, the provision of fluids and management of pain.

Reconstruction of the heart

Staged reconstruction is the treatment of choice for hypoplastic left heart syndrome. Reconstruction takes place in three stages.

The first stage, known as a Norwood operation, is undertaken as soon as possible after birth. This stage calls for a homograft¹ to be used to enlarge the rudimentary aortic arch and then join it to the pulmonary trunk. This is known as an anastomosis. The pulmonary trunk will have been disconnected from the left and right pulmonary arteries. The newly created blood vessel functions as the

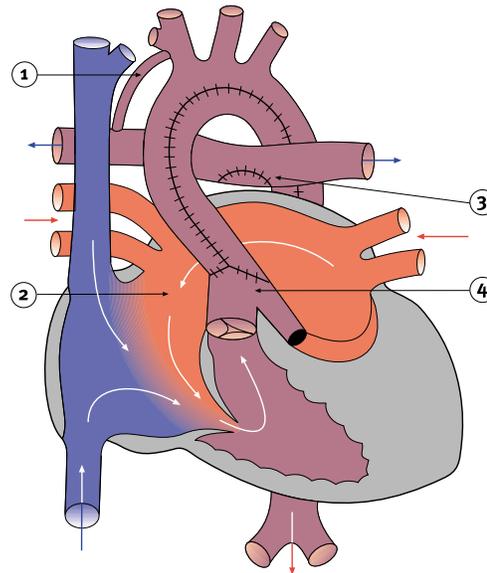


Diagram 2.21 Stage 1 of hypoplastic left heart syndrome reconstruction (Norwood)

- 1 – Blalock-Taussig shunt (temporary)
- 2 – atrial septum removed
- 3 – patch where pulmonary trunk is disconnected from left and right pulmonary artery
- 4 – aorta and pulmonary trunk anastomosed together and the aorta made larger

Blood flows through the anastomosed aorta and pulmonary trunk to the aortic arch. A shunt connects the aorta to the pulmonary arteries, providing the lungs with blood to oxygenate.

¹ A homograft is a graft of tissue between animals of the same species. In the case of humans, a homograft comes from a tissue donor. A homograft is in contrast to an autograft, which is a graft of tissue transferred from another part of the patient's body. With pulmonary homografts, the pulmonary artery is removed from a tissue donor shortly after death. The pulmonary homograft is prepared and stored indefinitely at -70 degrees Celsius in liquid nitrogen until needed, when it is thawed and prepared under sterile conditions.)

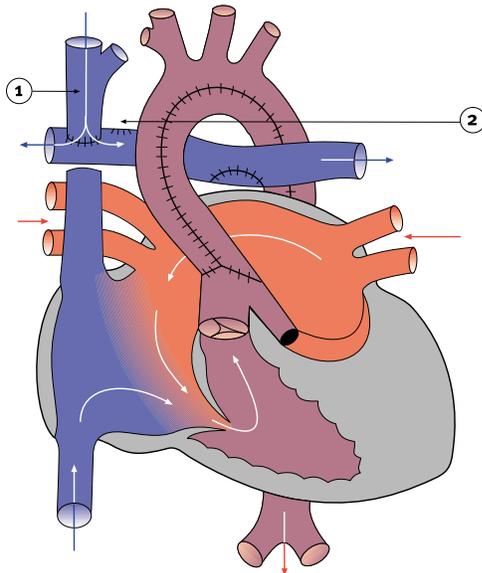


Diagram 2.22 Stage 2 of hypoplastic left heart syndrome reconstruction

1 – superior vena cava connected to pulmonary artery

2 – takedown of Blalock-Taussig shunt

De-oxygenated blood from the superior vena cava is routed directly to the lungs. A mix of oxygenated and de-oxygenated blood is sent to the body through the anastomosed aorta and pulmonary trunk.

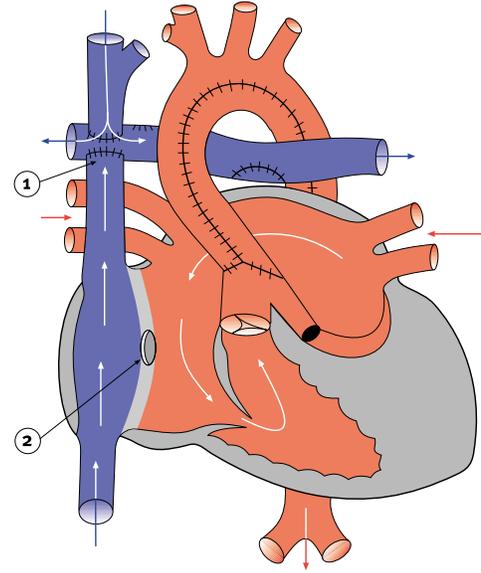


Diagram 2.23 Stage 3 of hypoplastic left heart syndrome reconstruction (Fenestrated Fontan)

1 – conduit joining inferior vena cava and superior vena cava to right pulmonary artery

2 – artificial wall containing small hole (to separate blue blood from red blood in atria and to channel blue blood from lower body to pulmonary artery).

De-oxygenated blood from the superior vena cava and inferior vena cava is routed directly to the lungs. Oxygenated blood is sent to the body through the anastomosed aorta and pulmonary trunk.

patient's aorta. The right ventricle then becomes a common ventricle, pumping blood through the aorta to the rest of the body.

Blood flow to the lungs is provided by means of a modified Blalock-Taussig shunt joining the innominate artery to the pulmonary artery. This increases the flow of oxygen-enriched blood that will be sent to the body. The Norwood operation also involves making a hole in the wall between the left and right atria. In medical terms this is known as an atrial septectomy.

After the Norwood operation, children require medical management with various drugs. Some drugs, such as digoxin, are needed to improve the strength of the heart's contractions, and other drugs, such as diuretics, are required to remove excess fluid from the body.

The second stage of reconstruction is undertaken between three and ten months of age. Stage two involves performing a bidirectional Glenn shunt or a hemi-Fontan. (This is also known as a partial Fontan or a Bidirectional Cavo-Pulmonary Shunt.)

A bidirectional Glenn shunt is performed by connecting the superior vena cava (SVC) to the right branch of the pulmonary artery. As a result, venous blood from the head and upper limbs will pass directly to the

lungs, bypassing the right ventricle. However, the venous blood from the lower half of the body will continue to return to the heart.

While avoiding the risk of failure of a complete Fontan operation, the partial Fontan helps to relieve symptoms. The procedure decreases the volume of blood delivered to the single ventricle, thus reducing the amount of work that the ventricle must perform. Post-operatively, oxygen saturation is improved.

In addition, because the bi-directional Glenn is a low-pressure shunt, it does not carry the risk of causing thickening and hardening of the blood vessels of the lungs. (This is a normal response of the lung's blood vessels to high pressures.) This operation creates a more favorable setting in which to complete a Fontan reconstruction at one and a half to two years of age.

At 18 to 24 months of age, if the patient does well after the Glenn shunt and pulmonary resistance remains low, the third stage of reconstruction is possible. This involves the completion of the Fontan procedure (as described in the earlier discussion of tricuspid atresia).

With the Fontan operation, the venous blood from the lower part of the body is also diverted to the lungs, thus creating the 'complete' Fontan circulation. However, if the child is not well or the pulmonary resistance is considered too high, then no further surgical treatment is possible.

Transplantation

Heart transplantation is carried out using the same basic techniques as for other transplants. In the case of pediatric heart transplants, the greatest problem is that infant donor hearts are in short supply. Thus transplantation is a treatment for only a limited number of babies with hypoplastic left heart syndrome. In addition, recipients can survive only with the assistance of heavy doses of expensive medication to prevent rejection of the transplanted tissue. These drugs also have significant side-effects, such as the risk of infection and lymphatic cancer, and must be taken by recipients for the rest of their lives. For those reasons, heart transplants are not considered the standard treatment for children suffering from HLHS.

SUMMARY

The defects mentioned in this chapter are not an exhaustive list of each and every defect or lesion that pediatric cardiac surgical programs are called upon to deal with. There are other defects not listed here that would be seen by professionals involved in the care of children with heart problems.

Nor is this chapter to be taken as being an authoritative statement of what the defects involve and how best they are treated. For such information, readers should refer to authoritative medical literature and texts. In addition, it is also important to note that frequently there are individual variations in each of these lesions. No two cases are alike.

This chapter is intended only to acquaint the reader with those defects that will receive the most attention in this Inquiry. As one reads through the following chapters, reference back to the information contained in this chapter (as well as in the Glossary and in Chapter 3) may be helpful.

RISK AND PEDIATRIC CARDIAC SURGERY

There is always risk involved in pediatric cardiac surgery. Various attempts have been made to estimate the level of risk associated with surgically treating each of the known pediatric congenital heart lesions.

During the course of 1994, parents in Winnipeg were given estimates by the doctors involved as to how many patients were likely to survive a particular procedure. At the time, there was no single book or list of percentages that contained a definitive estimation of the risk for each procedure, although information was available from pediatric cardiac surgeons and cardiologists working in other centres. Risk depends on many factors, including the specifics of the patient's condition, the skills and experience of the team, available technology and the overall growth of medical knowledge. One variable that recent research has focused attention on is the relationship between the numbers of cases a hospital deals with and results as measured by mortality.

Two academic articles presented to this Inquest examined this question. The articles were "In-Hospital Mortality for Surgical Repair of Congenital Heart Defects: Preliminary Observations of Variation by Hospital Caseload," by Kathy J. Jenkins MD MPH, Jane W. Newburger MD MPH, James E. Lock MD, Roger B. Davis ScD, Gerald A. Coffman MSc and Lisa I. Iezzoni MD MSc, published in the journal *Pediatrics* (Volume 95, Number 3 (March 1995), pages 323–330) and "Pediatric Cardiac Surgery: The Effect of Hospital and Surgeon Volume on In-Hospital Mortality," by Edward L. Hannan PhD, Michael Racz MS, Rae-Ellen Kavey MD, Jan M. Quaegebeur MD PhD and Roberta Williams MD, published in the journal *Pediatrics* (Volume 101, Number 6 (June 1998), pages 963–969).

Both articles came to similar conclusions. The Jenkins study made the following comment:

In our study population, the risk of dying in-hospital was much less for children who underwent surgical correction of a congenital heart defect at institutions with the highest volume of such patients, in comparison with lower volume centres. Outcomes at the highest volume institutions appeared clearly different than those for other institutions; below 300 cases annually, no consistent trends in mortality by annual case volume were observed. (Jenkins et al, pages 327–328)

The Hannan study commented:

Findings of the study were that annual hospital volume and annual surgeon volume were both significantly related to inpatient mortality rates, even after controlling for patient age and several clinical risk factors in addition to procedure complexity. The maximal differentiation in mortality rates between high and low-volume providers was 100 procedures annually for hospital and 75 procedures annually for surgeons. (Hannan et al, page 968)

This issue of the relationship between the number of procedures carried out in a pediatric cardiac surgery program and the mortality rate is one that will be returned to later in this report.