NCCU CLINICAL GUIDELINES
SECTION: 14

NEONATAL CARDIAC CONDITIONS:

MEDICAL AND SURGICAL MANAGEMENT

Section 14 Neonatal cardiac conditions
Neonatal circulation changes/unbalanced circulation
Date created: Oct 2014
Date revised:
Review date: Oct 2017

Neonatology Clinical Guidelines
King Edward Memorial/Princess Margaret Hospitals
Perth Western Australia
Authorisation and review by
Neonatal Coordinating Group

NEONATAL CIRCULATION CHANGES / UNBALANCED CIRCULATION

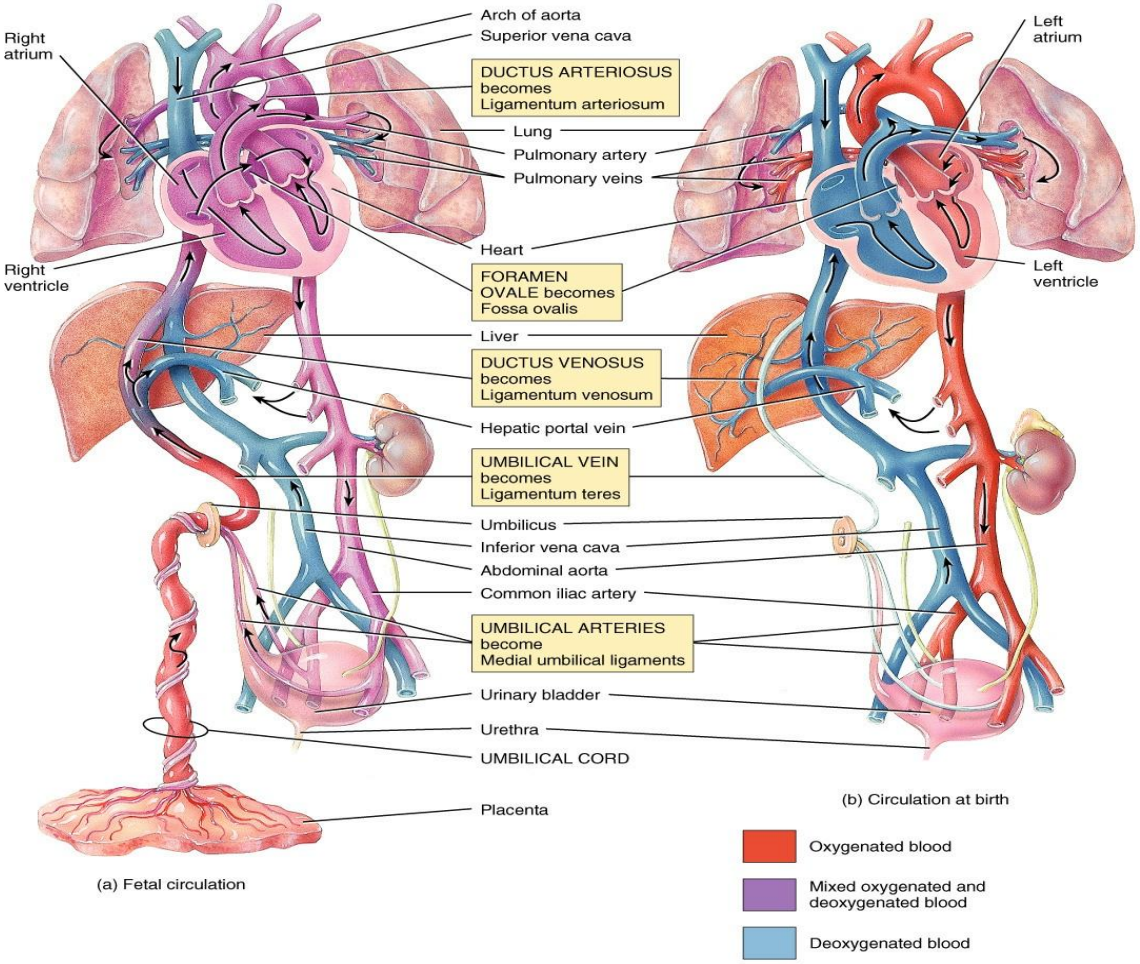
FETAL CIRCULATION

The basic differences between the fetal circulation compared with the postnatal circulation are:

- The presence of a low-resistance high-flow placental circulation.
- The pulmonary vascular resistance (PVR) is very high and the systemic vascular resistance (SVR) low. No more than 20% of the total cardiac output enters the pulmonary circulation at term (less earlier in gestation).
- Three vascular pathways, which are an integral part of the different blood flow pattern, and usually close shortly after birth – the **ductus venosus**, the **foramen ovale** and the **ductus arteriosus (DA)**. The first two allow oxygenated blood from the placenta to be channelled to the left atrium and thence to the aorta, coronary and cerebral circulations. The DA has pulmonary artery to aorta (right to left) flow due to the high pulmonary vascular resistance. The DA is kept open by circulating prostaglandins and as gestation progresses becomes increasingly sensitive to the constricting influence of oxygen.

THE FLOW OF BLOOD IN THE FETUS (SEE DIAGRAM BELOW)

- Blood from the placenta is carried to the fetus by the **umbilical vein**. About half of this enters the fetal **ductus venosus** and is carried to the **inferior vena cava** (IVC), while the other half enters the liver. The blood then moves to the **right atrium** of the heart.
- From the **right atrium**, most of the blood flows through the **foramen ovale** directly into the **left atrium**, thus bypassing the pulmonary circulation.
- The blood continues into the **left ventricle**, and from there it is pumped through the **aorta** to perfuse the **coronary arteries**, **head**, **brain** and **upper extremities** with only a small proportion entering the **descending aorta** to perfuse the rest of the body.
- Some of the blood moves from the **aorta** through the **internal iliac arteries** to the **umbilical arteries**, and re-enters the placenta, where carbon dioxide and other waste products from the fetus are taken up and enter the maternal circulation.
- Blood from the **head and neck** returns to the **right atrium** via the **superior vena cava** (SVC). It then passes through the **tricuspid valve** to the **right ventricle** and into the **pulmonary artery**. Only 15% of this blood goes to the **lungs** (due to the high PVR of the unexpanded lungs) and the remaining 85% flows through the **DA** into the **descending aorta** to perfuse the **abdominal viscera** and **lower extremities**.



CHANGES IN THE CIRCULATION AT BIRTH

As the umbilical cord is clamped, the SVR increases due to increased blood volume in the placenta. As the infant takes its first breath, the lung expand and the PVR drops, increasing the blood flow to the lungs which increases the oxygenation of the blood. The PVR continues to fall over the following weeks and ultimately takes about a month to fall to a mean level of 1/3 of mean systolic pressure.

DUCTUS ARTERIOSUS

The DA begins to constrict due to the increase in pO2, lowering levels of PGE2 and the decreasing blood flow through it. This process is functionally complete within 60 hours in 93% of term infants. Normally the duct then fibroses and closes within 2-3 weeks of birth and is then known as the *ligamentum arteriosum*.

FORAMEN OVALE

Increased pulmonary blood flow increases pulmonary venous return, resulting in an increase in the left atrial pressure which pushes the foramen ovale shut, becoming the *fossa ovalis*.

DUCTUS VENOSUS

The ductus venosus closes to become the *ligamentum venosum* which passes through the liver from the portal vein to the IVC. It can still provide vascular access to the right heart for a few days.

UMBILICAL VEIN

Once the cord is clamped, the umbilical vein constricts and obliterates becoming the *ligamentum teres*. Closure of all these structures may be delayed in pathological circumstances and may precipitate clinical deterioration in various structural cardiac conditions. Continued patency of the DA and foramen ovale may contribute to clinical problems in some conditions (see table below).

	CLINICAL PROBLEMS ASSOCIATED WITH CLOSURE AFTER BIRTH	**CLINICAL PROBLEMS ASSOCIATED WITH FAILED CLOSURE AFTER BIRTH**
FORAMEN OVALE	Poorer mixing in TGA Systemic/ pulmonary venous obstruction in right/ left heart obstructions	Allows L→R shunting in PPHN
DUCTUS VENOSUS	Worsening pulmonary venous obstruction in infradiaphragmatic TAPVD	No problem identified
DUCTUS ARTERIOSUS	Marked deterioration in duct-dependent pulmonary or systemic circulations	Associated with major respiratory and other problems in premature neonates Rarely clinically important L→R shunting in neonatal period in term infants

THE UNBALANCED CIRCULATION

THE NORMAL CIRCULATION	**THE UNBALANCED CIRCULATION**
Blood flow to systemic and peripheral circulations is equal: ***Qp (pulmonary flow) = Qs (systemic flow)*** i.e. ***Qp : Qs = 1:1***	When the ratio of ***Qp:Qs* is not 1:1** the circulation is 'unbalanced'. There can be too much pulmonary flow with too little systemic flow or too little pulmonary flow with high systemic flow. It is most common to see the unbalanced circulation with PPHN when there is too little blood flow to the lungs. Other causes are due to cardiac anomalies (see table) or post Blalock-Taussig (BT) shunt.

Too **little** blood flow to the lungs results in hypoxia.

- PPHN
- Severe tetralogy of fallot
- Pulmonary stenosis/ atresia
- Single ventricle anatomy/ DORV (some)
- BT shunt too small/ blocked

Too **much** blood flow to the lungs results in shock, lactic acidosis, pulmonary oedema, multiorgan failure.

- HLHS
- Truncus arteriosus
- Unbalanced AVSD
- Single ventricle anatomy/ DORV (some)
- BT shunt too large

WAYS TO INFLUENCE THE CIRCULATION

There are a number of different ways in which we can try to manipulate the circulation when it is unbalanced.

RESPIRATORY	CARDIOVASCULAR
• Mechanical Ventilation. • Oxygen – supplemental/ air/ hypoxic mixture. • pH/ pCO2 – manipulation of ventilation/ bicarbonate infusion. • Consistent MV. • PEEP • Nitric Oxide	• Filling • Inotropic Support. • Inodilators e.g. milrinone. • Vasodilators e.g. SNP, MgSO4. • Vasoconstrictors e.g. phenylephrine. • Ductal Patency. • Specific Procedures e.g. atrial septostomy.

OTHER

- Ensure no electrolyte abnormalities.
- Maintain haematocrit 0.4-0.5 for maximum O2 carrying capacity.
- Minimal handling.
- Sedation +/- paralysis.

MANAGEMENT OF QP<QS

- Give supplemental O2.
- Low normal pCO2/ high normal pH.
- NO if PHT.
- Prostin
- MgSO4
- Ensure adequately filled.
- Inotropic support – dopamine/ milrinone.
- If PS, normal cardiac function – phenylephrine (intense peripheral vasoconstrictor, forces blood to lungs).
- Sedation/ paralysis + minimal handling.

MANAGEMENT OF QP>QS

- Be watchful in any lesion prone to developing this problem – SaO2 'too good', regular gases and beware of metabolic acidosis/ rising lactate.
- Early ventilation in air, if needs hand-bagging use air or low FiO2 eg. 30%. Keep O2 sats 75-85%..
- High PEEP, consistent MV.
- High normal pCO2/ low normal pH.
- Hypoxic mix – eg. FiO2 19% (not usually used these days).
- Ensure adequately filled.
- Inotropic support of R ventricle – low dose dopamine or dobutamine at 5microg/kg/min.
- Correct acidosis with HCO3.
- Look for signs multiorgan failure and treat accordingly.