Guidelines

Circulatory support in the newborn

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Functional Sub-Group: Clinical Governance
Corporate Governance

Summary: Describes pathogenesis and management of circulatory failure in the newborn.

National Standard
Standard 1: Governance for Safety and Quality in Health Service Organisations
Standard 9: Recognising and responding to the deteriorating patient.

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Approved by: RPA Newborn Care Guideline Development Committee

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Replaces Existing Policies: RPA Newborn Care Guidelines: Hypotension and Circulatory Support

Previous Review Dates: March 2007

Note: Sydney Local Health District (LHD) and South Western Sydney LHD were established on 1 July 2011, with the dissolution of the former Sydney South West Area Health Service (SSWAHS) in January 2011. The former SSWAHS was established on 1 January 2005 with the amalgamation of the former Central Sydney Area Health Service (CSAHS) and the former South Western Sydney Area Health Service (SWSAHS).

In the interim period between 1 January 2011 and the release of specific LHN policies (dated after 1 January 2011) and SLHD (dated after July 2011), the former SSWAHS, CSAHS and SWSAHS policies are applicable to the LHDs as follows:

Where there is a relevant SSWAHS policy, that policy will apply

Where there is no relevant SSWAHS policy, relevant CSAHS policies will apply to Sydney LHD; and relevant SWSAHS policies will apply to South Western Sydney LHD.
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1. Introduction

Shock is the failure of the cardiovascular system to provide oxygen transport necessary to meet oxygen demand. This clinical state of acute circulatory failure can result from four basic mechanisms: reduced intravascular volume, failure of the cardiac pump, obstruction in the circulation, and loss of vascular tone or distributive disorders of the peripheral circulation. In the first three processes, the main hemodynamic feature is low systemic blood flow which triggers tissue hypoperfusion and microcirculatory abnormalities. In contrast, vasodilatory or distributive shock usually exhibits a normal or high systemic blood flow, and microcirculatory alterations play a primary role in the development of tissue hypoperfusion. Microcirculatory shock is the condition in which the microcirculation fails to support tissue oxygenation in face of normal systemic haemodynamics.¹

The risks addressed by this policy:

- Risk of not recognising circulatory failure and not supporting the circulation appropriately.

The aims / expected outcome of this policy

That neonates needing circulatory support will be managed appropriately.

2. Policy Statement

The goal of this guideline is to familiarise medical staff with the evidence, indications and practical management of a neonate with suspected circulatory failure.

3. Principles / Guidelines

3.1 Pathogenesis of circulatory failure in the newborn:

The general principles of circulatory failure as defined in the introduction, apply in the newborn. Assuming normal haemoglobin and arterial oxygen content, the adequacy of the systemic blood flow is defined by normal blood volume, good pump (myocardial) function and normal vascular resistance. In the newborn, each of these factors plays into the pathogenesis of shock to a different degree in different clinical situations. This, and the limited circulatory monitoring modalities for the newborn, creates unique challenges for diagnosis and management of circulatory failure.

Recognition of circulatory failure relies on the usual vital signs (blood pressure, heart rate, capillary refill) but these have limited accuracy so also, and probably more importantly, there is a need to recognise high risk clinical situations where circulatory competence needs to be proactively assessed with cardiac ultrasound.

3.2 Diagnosis of circulatory failure: Vital signs are important in the assessment of the competence of the circulation however all are limited in the newborn by being more specific than they are sensitive.

- Increased heart rate: An increase in heart rate is a usual compensatory response to circulatory failure. Heart rate is an unreliable marker in preterm transitional compromise² but an increased heart rate is more commonly seen with hypovolaemia or sepsis. Circulatory compromise should always be considered in babies with an increase in heart rate.
- **Prolonged capillary refill.** This is a widely used but poorly validated sign. Studies in preterm babies have shown that a capillary refill over 5 seconds is specific for low SBF but refill times less than 5 seconds have limited predictive accuracy. Capillary refill times over 3 seconds should be a trigger to assess for other markers of circulatory compromise.

- **Acid base or lactate.** Also widely used but with limited validation data. In preterm babies, we found no relationship to SBF. Ultimately circulatory failure will express with a lactic acidosis. This seems to be a late sign but one that should be taken seriously when found.

- **Low urine output and hyperkalaemia:** Circulatory failure will also eventually express with reduced urine output and, in the very preterm baby, hyperkalaemia but again by the time either of these are recognised, the haemodynamic pathology will be well advanced.

- **Low blood pressure** is an important vital sign but also a limited measure if viewed in isolation. Normal blood pressure range increases with gestational age and with postnatal age. There are complex tables which document this however, for clinical purposes, the pragmatic rule is that a normal mean BP will be above the baby’s gestation in weeks. Consistent data has questioned the long held neonatal tenet of a direct correlation between blood pressure and systemic blood flow in the preterm infant. Blood pressure is the product of systemic blood flow (SBF) and systemic vascular resistance (SVR) and it is the variation in the latter which makes BP an unreliable marker of SBF. BP can be low because SBF is low or because SVR is low or because both are low. Interpretation of a low mean BP needs a measure of SBF.

- **Low systemic blood flow:** This needs cardiac ultrasound. Because of the complexity of the transitional circulation, particularly the confounding effect of ductal shunting on LV output, the best measures of SBF are right ventricular (RV) output and superior vena cava (SVC) flow. Normal RV output will be above 150mls/kg/min and normal SVC flow will be above 50mls/kg/min. In this guideline, low SBF will be defined by either of these measures being below these cut offs.

- **Low pulmonary blood flow (PBF);** This also needs cardiac ultrasound. If the ductus is closed, then RV output will be PBF. If the ductus is patent, RV output no longer represents pulmonary blood flow. Because of this, velocity in the left pulmonary artery (LPA) can be used as an estimate of PBF. A mean LPA Doppler velocity of less than 20cms/sec has been proposed as a marker of low PBF in term baby. There is no published normal range for LPA velocity in preterm babies.

### 3.3 Diagnosis of circulatory failure: High risk clinical situations:

#### 3.3.1 The immediate postnatal period:

Babies with signs of circulatory failure in the immediate post-resuscitation are usually asphyxiated, hypovolaemic or, occasionally, both. Often, but not exclusively seen, in term babies. These two main pathologies can be difficult to differentiate on clinical grounds.

**Asphyxia** is usually diagnosed on the basis of the usual markers; fetal distress, low Apgars, need for resuscitation and lactic acidosis on the cord arterial blood. Such babies will usually appear pale and shut down in the immediate post-resuscitation period and in many, this will resolve with time and, if indicated by the acid base status, a slow bicarbonate half correction. However some of these babies remain at risk of circulatory failure due to compromised myocardial function.

- Because haemodynamic compromise will often improve with correction of acidosis, the immediate focus should be on checking acid base and correcting if needed. If the acidosis is mainly metabolic, it is reasonable to manage a pH>7.05 expectantly. If pH less than 7.05, consider respiratory and/or metabolic correction as indicated.

- Attend to respiratory, metabolic and neurological homeostasis. Then perform a cardiac ultrasound after stabilisation and assess myocardial function and SBF with SVC flow and RV output.
Hypovolaemia: True hypovolaemia is quite rare in the newborn but early after birth is the one time in which you can see real and life threatening hypovolaemia. Causes include:

- **Vasa Praevia** with fetal vessel damage at rupture of membranes. Low and velamentous cord insertion may have been identified ultrasound in these pregnancies but they present with brisk antepartum blood loss at rupture of membranes.
- **Feto-placental haemorrhage** this is seen in babies born with a tight nuchal cord or, occasionally, after vaginal breech deliveries. In both situations, slow compression of the cord obstructs the vein before the arteries, so blood pools in the placenta. If the cord is then cut to facilitate delivery, this blood is trapped in the placenta and the baby is born hypovolaemic. Not an easy diagnosis because the blood loss is occult.
- **Acute feto-fetal haemorrhage** can occur in monochorionic twins during labour, usually characterised by sudden change in the fetal trace of one twin and one very pale twin and one very pink twin at delivery. Chronic twin anaemia polycythaemia sequence (TAPS) is more common than acute and also results in one pale and one pink twin.
- **Acute feto-maternal haemorrhage** is more commonly chronic than acute but should be considered.
- **Subgaleal Haemorrhage** can be significant enough to cause hypovolaemia shortly after birth but more usually evolves over the early postnatal hours (see guideline).
- **Placental Haemorrhage** can occur spontaneously but more commonly when a caesarean section incision has to go through the placenta.

It is important to differentiate normovolaemic chronic anaemia from hypovolaemic acute anaemia. Both result in a clinically pale baby and both will have a low haematocrit. The hypovolaemic baby will usually have a tachycardia but cardiac ultrasound provides the most accurate differentiation. The chronically anaemic baby will have a dilated heart while the hypovolaemic baby will have a small chambered under-filled heart.

### 3.3.2 The first 12 hours of life in the very preterm baby

The early transitional period is one of exquisite circulatory vulnerability for the very preterm infant and 10-20% of babies born before 30 weeks will go into a low systemic blood flow state. The causes of this are complex and relate to many factors including immaturity, positive pressure ventilation and shunts out of the systemic circulation through the fetal channels mainly the ductus. It is predictive of mortality and a range of morbidities.²,⁸

Low SBF develops during the first 12 hrs and then improves spontaneously in almost all babies by 24 hours. It is poorly predicted by clinical parameters including blood pressure, so detection depends on prospective cardiac ultrasound measures of SBF during the high risk period of the first 12 hours. Low systemic blood flow is uncommon in preterm after 24 hours unless critically unwell and most hypotension after this time is vasodilatory e.g low blood pressure, normal or high flow.

The haemodynamic compromise of severe pulmonary hypertension (described below) is uncommon in preterm babies but can occur in certain subgroups, particularly babies born after prolonged rupture of membranes and oligohydramnios.⁹

In addition to monitoring of vital signs including blood pressure, consider the following groups of babies for prospective assessment with cardiac ultrasound between 3 and 9 hours of age to measure SBF and (as indicated in the PDA guideline) ductal diameter.

- Babies born before 28 weeks, particularly those with ongoing ventilation requirements.
- Babies born after 27 weeks with higher risk e.g. limited exposure to antenatal steroids and/or RDS requiring significant ventilator support.
• Any baby with severely or persistently low BP or other clinical signs of circulatory compromise.

3.3.3 The septicaemic baby: The haemodynamic compromise of late onset sepsis is almost always vasodilatory (low BP/normal or high SBF) in the newborn. In advanced septic shock, evidence of tissue ischaemia can persist despite normalisation of systemic haemodynamics. Although little studied in the newborn, this may be due to the microvascular dysfunction described in older subjects with sepsis.

The haemodynamic of early onset sepsis is more variable. It can be vasodilatory but pulmonary hypertension can be a significant factor in early onset Group B Streptococcal pneumonia.

All babies with suspected sepsis should be monitored for signs of circulatory compromise including invasive or non-invasive (as indicated clinically) blood pressure and monitoring acid base and lactate. Confirm the haemodynamic with cardiac ultrasound and manage as in the table below:

3.3.4 Term or late preterm baby with high ventilator/oxygen requirements and/or persistent pulmonary hypertension (PPHN): These babies are at high risk for low SBF particularly during the first 24 hours. The haemodynamics are as varied as the underlying pathologies and needs to be assessed individually with cardiac ultrasound. In some, the low SBF probably results from the negative circulatory effect of high positive intrathoracic pressure during the period of circulatory transition.

In babies with low pulmonary blood flow PPHN, the systemic compromise may be a consequence of the restricted pulmonary blood flow and will improve if the pulmonary vascular resistance can be reduced with inhaled nitric oxide or other vasodilators. Low pulmonary blood flow, as assessed with LPA velocity, predicts oxygenation response to iNO.

3.3.5 Structural and other cardiac problems: Babies with primary cardiac problems can also present as circulatory failure. These are not common but must be considered in any baby presenting with shock. The problems should become apparent on cardiac ultrasound and heart rate monitoring. Possibilities include:

- Ductal dependent obstructive cardiac abnormalities; such as critical aortic stenosis or coarctation. These may be detected on early saturation screening but otherwise present as acute circulatory failure after duct closure during the first week of life.
- Primary Cardiomyopathy; very rare but can present as circulatory failure.
- Supraventricular Tachycardia; the heart rate will be fixed and usually over 200 bpm.

3.4 Circulatory Support in the Newborn

3.4.1 The effects of support interventions: clinical trials provide little clear outcome based guidance in the management of circulatory compromise. In most studies, enrolment is based on low blood pressure and few of the studies have long term follow up data

- Placental transfusion from delayed cord clamping: will result in increased blood volume compared to immediate clamping. Delayed cord clamping increases blood volume, results in better BP, less use of inotropes, less need for transfusions and lower IVH rates. Study of the haemodynamic effects have produce contradictory results, several small RCTs and observational studies have shown better SBF while the larger echo sub-study of the large Australian Placental Transfusion Study (APTS) showed no benefit to SBF. RPA Newborn Care is enrolling in the APTS and use of this intervention should be within the trial.
- **Volume expansion**: will be life-saving in a baby with definite hypovolaemia as described above but, in other babies, the benefits are less clear. Routine early volume expansion in preterm babies does not change outcomes in the neonatal period. We know that volume expansion has little effect on blood pressure but does produce improvement in flow in preterm babies with low SBF. It is not known how long that improvement is sustained.

- **Dopamine**: has some central inotropic effect but also has vasoconstrictor (pressor) effects which are more marked at higher doses (above 10μg/kg/min). Dopamine is better than dobutamine at improving low blood pressure. Studies suggest that the effect on BP is more to do with the pressor effects than the inotrope effects. Dopamine would be a logical choice where the haemodynamic was vasodilatory.

- **Dobutamine**: has a short half-life of about 2 minutes. It has central inotropic and chronotropic (heart rate) effects and its peripheral effects are more vasodilatory. Hence it improves SBF while having limited effect on BP. Dobutamine is better than dopamine at improving low systemic blood flow. Dobutamine is the logical choice where there is low systemic blood flow, particularly with evidence of myocardial dysfunction. There is no evidence that either dopamine or dobutamine offer any advantage in terms of long term outcomes.

- **Adrenaline**: Has similar effects to dopamine with pressor effects predominating at doses above 0.2 micrograms/kg/min. Adrenaline and dopamine have similar effects on cerebral tissue oxygenation index and there is no difference in long term outcomes.

- **Noradrenaline**: is less well studied in the newborn but has predominantly peripheral pressor effects. There is observational data showing improved blood pressure in term babies with refractory septic shock. An observational study in term babies with pulmonary hypertension pointed to a beneficial effect on the haemodynamic causing systemic constriction and pulmonary dilatation. Noradrenaline can be considered in babies with PPHN and low blood pressure and in septic shock.

- **Vasopressin**: has been shown to have equivalent effect on blood pressure to dopamine in small RCT of preterm infants (n=20). It has been reported to improve blood pressure in a cohort of mainly septic ELBW babies with hypotension refractory to dopamine adrenaline and corticosteroids.

- **Milrinone**: has central inotropic effects with peripheral vasodilatory effects, sometimes called an inodilator. It does not prevent low SBF in preterm babies. There is some observational data of improved haemodynamics and oxygenation in babies with PPHN unresponsive to iNO. It should be used with caution in babies with low BP because of its vasodilatory effects and its long half-life of about 4 hours in term babies and 10 hours in preterm babies.

- **Corticosteroids**: will increase blood pressure but in an observational study, there was no effect on SBF. Hydrocortisone is as effective as dopamine at increasing blood pressure and may be effective in hypotension refractory to other inotropes but there is a lack of long term outcome data. Steroids probably work as a peripheral pressor by up-regulating sympathetic receptors.
3.5 Management:

In the absence of clear evidence, the recommendations in this guideline are largely based on identifying the haemodynamic in an individual baby of concern using cardiac ultrasound together with other vital signs. Then applying a logical support based on what we know about the physiological effects of the interventions. Defining the haemodynamic involves integrating information from clinical history, vital signs and the cardiac ultrasound. Diagnosis and management of each haemodynamic is defined in the table below.

3.5.1 Integrating flow and pressure to define the haemodynamic: Pressure in either the pulmonary or the systemic circulation is important only in as much as it reflects in flow. Pressure is the product of flow and resistance. So when pressure is low (usually in the systemic circulation), it can be because flow is low; or because resistance is low; or because both are low. When pressure is high (usually in the pulmonary circulation), it can be because flow is high or because resistance is high or because both are high. Because resistance cannot be measured, it must be estimated in either circulation using measures of pressure and flow.

3.5.2 Refractory low systemic blood flow

This is usually seen in the very preterm infant with low SBF in the transitional period, where there can be limited response to the strategies suggested above. Experientially, this situation is difficult to manage, there is often limited response whatever intervention is used and predicts a high risk of morbidity, particularly IVH. See table for suggested intervention.

3.5.3 Refractory hypotension

This is uncommon but situations in which this can occur include:

- The very preterm infant
- Septic shock
- After severe asphyxia and/or hypovolaemia

The haemodynamic is usually vasodilatory but maybe combined with low flow particularly in the severely asphyxiated baby. In the very preterm infant, resistant hypotension may be a marker of large patent ductus arteriosus or relative corticoadrenal insufficiency. In septic and post-asphyxial shock, it is probably a marker of microvascular dysfunction causing loss of vascular tone. See table for suggested interventions.
Table: Diagnosis and management of common neonatal haemodynamics. Note these haemodynamics are not mutually exclusive and babies may have features of more than one haemodynamic. Criteria in italics indicate the key feature of each haemodynamic. For more details of drug dosage regimens, see NEOMED guidelines (currently being developed).

<table>
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<tr>
<th>Haemodynamic</th>
<th>Clinical situations</th>
<th>Vital signs</th>
<th>Cardiac US Flow measures</th>
<th>Other cardiac US findings</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolaemic haemodynamic</td>
<td>• Perinatal fetal blood loss</td>
<td>• Pallor</td>
<td>• Low or low normal SBF depending on volume loss.</td>
<td>• Biventricular poor filling.</td>
<td>• Give 20 ml/kg N-saline IV over 5–10 minutes depending on severity.</td>
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<td></td>
<td>• Subgaleal haemorrhage</td>
<td>• Tachycardia</td>
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<td>• If acute blood loss (see above), follow with 20 ml/kg of un-crossmatched O-ve blood (cross match only if time) over 10-30 minutes depending on severity.</td>
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<td>• Abdominal emergencies</td>
<td>• Low BP</td>
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<td>• Monitor impact with FBC, coagulation vital signs and cardiac ultrasound.</td>
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<td>• Further volume expansion/blood as indicated, consider FFP if evidence of coagulopathy.</td>
</tr>
<tr>
<td>Low systemic flow haemodynamic</td>
<td>• Very preterm &lt;12h</td>
<td>• Variable,</td>
<td>• Low SBF</td>
<td></td>
<td>• Give 10mls/kg normal saline over 30 minutes.</td>
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<td></td>
<td>• Any baby high ventilation/oxygen need.</td>
<td>• BP low or normal</td>
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<td>• At the same time, commence dobutamine infusion at 10µg/kg/min</td>
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<td></td>
<td>• Asphyxia.</td>
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<td>• Monitor impact with cardiac US and/or heart rate.</td>
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<td>• If flow not improving, increase dobutamine to 20µg/kg/min</td>
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<td>• If flow not improving, consider adding adrenaline at 0.01µg/kg/min increasing to 0.5µg/kg/min depending on response. Note, low SBF in preterm babies may be very resistant to inotropic support.</td>
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<td>• Review cardiac US at 24 hrs, particularly in very preterm babies, and wean dobutamine over 4-6 hours if flow measures have normalised (which they usually will).</td>
</tr>
<tr>
<td>Vasodilatory haemodynamic</td>
<td>• Sepsis</td>
<td>• BP low</td>
<td>• Normal or high SBF</td>
<td></td>
<td>• If early postnatal in a preterm baby, it may be reasonable to observe, depending on BP and other vital signs. Spontaneous improvement is common. Otherwise,</td>
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<td></td>
<td>• Preterm baby after 24 hrs, sometimes earlier.</td>
<td>• Other vital signs variable</td>
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<td></td>
<td>• Give 10mls/kg normal saline over 30 minutes. Consider more in probable septic shock.</td>
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<td></td>
<td>• Recovery from shock or asphyxia</td>
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<td>• At the same time, commence dopamine at 5µg/kg/min. Monitor impact with BP.</td>
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<td>• If not improving, increase dopamine in 2µg/kg/min increments to a maximum of 15µg/kg/min in order to achieve a minimally acceptable MBP (&gt;GA).</td>
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<td>• Reduce dopamine if MBP goes significantly above the minimally.</td>
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</tbody>
</table>

Compliance with this Guideline is recommended
### Vasodilatory Haemodynamic (cont)

<table>
<thead>
<tr>
<th>Combined low flow/vasodilatory haemodynamic</th>
<th>Low BP</th>
<th>Low SBF</th>
<th>Variable</th>
<th>Acceptable MBP (&gt;GA).</th>
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<tbody>
<tr>
<td>• Very preterm &lt;12h</td>
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<td>• If BP does not improve, consider.</td>
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<tr>
<td>• Asphyxia</td>
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<td>• <strong>In septic shock:</strong> Adrenaline or Noradrenaline at starting at 0.05 µg/kg/min to a maximum of 1.0µg/kg/min. If not responding to this, consider vasopressin at 0.02 units/kg/hr increasing to a maximum of 0.04 units/kg/hr depending on BP response.</td>
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### Low pulmonary blood flow PPHN

<table>
<thead>
<tr>
<th>Low pulmonary blood flow PPHN</th>
<th>High FiO2 with relatively normal lungs (clinically and on CXR).</th>
<th>Poor oxygenation</th>
<th>Normal or low SBF</th>
<th>Low mean velocity in LPA.</th>
<th>Exclude CHD</th>
<th>Dilated poorly contracting RV, Poorly filled LV</th>
<th>High PAP</th>
<th>Dominant Rt to Lt bidirectional shunt thro ductus and/or FO.</th>
<th>Commence inhaled nitric oxide (iNO) 5-10ppm (as in PPHN guideline).</th>
<th>Monitor effect with oxygenation and cardiac ultrasound, both will usually improve.</th>
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<tbody>
<tr>
<td></td>
<td>• BP normal or low</td>
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<td>• Other vital signs variable</td>
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<td>• Often low SBF on day 1.</td>
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<td>• Normal mean velocity in LPA.</td>
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<td>• Exclude CHD</td>
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<td>• Usually normal contractility</td>
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<td>• Moderate to high PAP.</td>
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<td>• Bidirectional shunt thro ductus and/or FO.</td>
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<td>• Optimise respiratory management, ventilation, surfactant etc.</td>
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<td>• Consider iNO depending on oxygenation and pulmonary artery pressure.</td>
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<td></td>
<td>• Monitor effect with oxygenation and cardiac ultrasound. The effect of iNO is more variable.</td>
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<td>• If low systemic blood flow, consider saline and dobutamine as above.</td>
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<td>• If low blood pressure, particularly if oxygenation varying with MBP, consider noradrenaline starting at 0.5 µg/kg/min increasing to 1.0 µg/kg/min or adrenaline starting at 0.05 µg/kg/min increasing to 1.0 µg/kg/min depending on BP response.</td>
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### Normal pulmonary blood flow PPHN

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<thead>
<tr>
<th>Normal pulmonary blood flow PPHN</th>
<th>High FiO2 with abnormal lungs (clinically and on CXR).</th>
<th>BP normal or low</th>
<th>Other vital signs variable</th>
<th>Often low SBF on day 1.</th>
<th>Normal mean velocity in LPA.</th>
<th>Exclude CHD</th>
<th>Usually normal contractility</th>
<th>Moderate to high PAP.</th>
<th>Bidirectional shunt thro ductus and/or FO.</th>
<th>Optimise respiratory management, ventilation, surfactant etc.</th>
<th>Consider iNO depending on oxygenation and pulmonary artery pressure.</th>
<th>Monitor effect with oxygenation and cardiac ultrasound. The effect of iNO is more variable.</th>
<th>If low systemic blood flow, consider saline and dobutamine as above.</th>
<th>If low blood pressure, particularly if oxygenation varying with MBP, consider noradrenaline starting at 0.5 µg/kg/min increasing to 1.0 µg/kg/min or adrenaline starting at 0.05 µg/kg/min increasing to 1.0 µg/kg/min depending on BP response.</th>
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<td></td>
<td>• Normal or low SBF</td>
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<td>• Low mean velocity in LPA.</td>
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### Definitions:

- **Low systemic blood flow (SBF):** RV output <150mls/kg/min and/or SVC Flow <50ml/kg/min
- **Low pulmonary blood flow (PBF):** Left pulmonary artery (LPA) mean velocity <20cms/sec
Key Points

<table>
<thead>
<tr>
<th>Key Points</th>
<th>Level of Evidence</th>
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<tbody>
<tr>
<td>There is no outcome based evidence on which to base recommendations for neonatal circulatory support.</td>
<td>Ia</td>
</tr>
<tr>
<td>Diagnosis and management requires integration of the clinical background, vital signs and cardiac ultrasound.</td>
<td>IIb</td>
</tr>
<tr>
<td>Hypovolaemia is rare but needs prompt support with volume replacement.</td>
<td>V</td>
</tr>
<tr>
<td>Low systemic blood flow is most likely to respond to dobutamine.</td>
<td>Ib</td>
</tr>
<tr>
<td>Vasodilatory low blood pressure is most likely to respond to dopamine.</td>
<td>IIb</td>
</tr>
<tr>
<td>Inhaled nitric oxide may improve systemic blood flow in babies with PPHN, particularly those with low pulmonary blood flow.</td>
<td>IIb</td>
</tr>
</tbody>
</table>

References

12. Evans NJ, Kluckow M, Currie A. The range of echocardiographic findings in term and near term babies with high oxygen requirements. Archives of Disease in Childhood, 1998;78:105-111.
15. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. Cochrane


Last Reviewed: March 2016 Nick Evans
Minimum list (nothing to do with management of neonatal circulatory support)

a. Legislative Compliance: Organisation, Management and Staff Obligations – Governing Body and Management manual, Policy Number 2.7.1

b. Code of Conduct – Governing Body and Management Manual, Policy Number 1.1