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**Aim**

This guideline focuses on the specifics of the post-op management and complications of a neonate following the insertion of a Blalock-Taussig (BT) shunt. For routine post-op cardiac care, general complications following cardiac surgery and for general information on tricuspid atresia/pulmonary atresia and Tetralogy of Fallot, and their pre-op management please refer to the following guidelines

- **Cardiac: Routine Post-Operative Care**
- **Cardiac: Complications Management Following Surgery**

A BT shunt is a systemic to pulmonary shunt which is a palliative procedure usually performed in neonates to establish a reliable pulmonary blood flow in patients with obstructed pulmonary blood flow e.g. tricuspid atresia, pulmonary atresia and severe Tetralogy of Fallot. The pulmonary blood flow will have been maintained pre-operatively by maintaining ductal patency with Prostaglandin E1 (PGE1). The definitive operation/s is/are performed later when the child is larger.
Cardiac: Blalock-Taussig Shunt (BT Shunt) Management Following Insertion

Surgical Procedure
The Classical BT Shunt (CBTS) was first performed in the 1940s and is created by division of the subclavian artery (left or right) and anastomosis to the ipsilateral pulmonary artery (PA). The main advantage is that the shunt grows with the patient and the main disadvantage is loss of pulses in the ipsilateral upper limb and resulting decreased growth and strength. The classical BT shunt is seldom used these days.

The Modified BT Shunt (MBTS) is now performed and consists of a Gore-Tex (PTFE) graft (3.5-5.0 mm diameter) interposed between the innominate or subclavian artery and the ipsilateral pulmonary artery (PA). This can be performed on the left or right side, but is routinely on the right side at PMH. It is usually a non-bypass procedure and is performed through a lateral thoracotomy.

Postoperatively, pulmonary and systemic vessels are fed from the same common source. The balance of systemic and pulmonary flows is of utmost importance, and is the principal determinant of systemic oxygen saturation (SaO₂). Ideally the post-op SaO₂ is 75-80%, and this gives a Qp:Qs ratio 1:1-1.8. Refer to Cardiac: Neonatal Circulation Changes/Unbalanced Circulation.

Routine Post-Op Management Following Neonatal BT Shunt Insertion
For more general post-op cardiac management, refer to Cardiac: Routine Post-Operative Care guideline.

Currently, neonates undergoing BT shunts procedures are cared for on PCC postoperatively, usually until extubated and stable. The following care remains relevant once the neonate returns to NICU.

Respiratory
- Will require ventilation at least overnight, however usually weaned to extubation on day 2 if otherwise stable (PCC).
- Keep SaO₂ 70-85% (ideal 75-80%).
- Gradually wean oxygen therapy down to air if SaO₂ > 80% (to avoid over-shunting).
- 6 hourly blood gases if stable

CVS
- Check for shunt murmur when arrives back from PCC and again anytime when there are concerns over the patient, particularly if they have low SaO₂.
- Aim for BP in normal range (Refer to Monitoring and Observation Frequency guideline) unless otherwise ordered.
- If inotropic support required, 1st line dopamine, 2nd line noradrenaline if low diastolic pressure/over-shunting or adrenaline to support cardiac performance.
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- An echocardiogram should be performed and documented in the notes by the cardiologist if there is a concern over shunt blockage or pulmonary over-circulation.

**Causes of Hypotension/Low Cardiac Output**
- Hypovolaemia/bleeding.
- ‘Over-shunting’ causing unbalanced circulation (particularly low diastolic pressure).
- Shunt blockage.
- Sepsis

**Fluids/Nutrition**
- Fluids should be restricted to 60-80 mL/kg/day on day 1 (PCC). Then can then be increased by 10-20 mL/kg/day depending upon the clinical status of the patient.
- Beware of over-restriction causing intravascular depletion as this predisposes to shunt thrombosis.
- Feeds can be considered the day after surgery if everything has been stable. Grade up feeds as per normal protocol.
- If there are concerns of ‘over-shunting’ and a poor systemic output, feeds should be withheld as there is a risk of NEC.

**Anticoagulation**
- Anticoagulation with heparin to prevent BT shunt thrombus is controversial. It is not routinely used in every case at PCH. The decision to use heparin is on a case-by-case basis and is discussed with the cardiac surgeon on returning from theatre. The heparin infusion is usually commenced at a rate of 10 units/kg/hr (can use up to 20 units/kg/hr). Changes in APTT are not usually targeted.
- Aspirin 3-5 mg/kg once a day is commenced once on feeds.

**Haemoglobin**
- Should be kept around 120 g/L. Avoid too high levels as this may predispose to shunt thrombosis. Avoid a low Hb as patients with cyanotic heart disease require slightly higher Hb levels than normal to optimise oxygen carrying capacity.

**Post-Op BT Shunt Complications** — Refer to Cardiac: Complications Management Following Surgery
- **Haemorrhage** from anastomosis may lead to haemothorax.
- **Poor shunt flow/blockage** with thrombus or shunt too small (unusual – refer to Low O₂ Saturations < 70% section below).
- ‘Over-shunting’ - additional blood supply from PDA (which is yet to close) or MAPCAs, or shunt too large.
- **Shunt infection** (rare) results in features of sepsis with elevated CRP and WCC.
- Discuss with cardiac surgeon before commencing antibiotics if this is felt to be a possibility.
- **Seroma** - Gore-Tex can ‘sweat’. Seen on CXR or echo.
- **Chylothorax** due to thoracic duct damage.
- **Vocal cord palsy** due to recurrent laryngeal nerve.
- **Diaphragmatic palsy** due to phrenic nerve damage.
- **Shunt narrowing** may occur at the site of anastomosis and may sometimes need to be treated with balloon angioplasty.
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**Low O_{2} Saturations (< 70%)**

Usually due to inadequate pulmonary blood flow.

**Causes**

- Shunt blockage with thrombus.
- Inadequate shunt flow due to systemic hypotension.
- Small feeding artery/ pulmonary arteries (PAs)/branch PAs.
- Shunt too small (unusual as the shunt is usually larger than the feeding artery to allow for growth of the baby and so the feeding artery is usually the limiting factor).
- Inadequate ventilation e.g. Pneumothorax, atelectasis, pneumonia, haemothorax, chylothorax, dislodged ETT.

**Signs**

A shunt blockage may present acutely or sub-acute and can be complete or partial. If there is an acute complete blockage it is an emergency and will present with:

- Sudden severe desaturation.
- Normal (or low) blood pressure.
- Normal ventilator parameters, normal air entry and normal CXR.
- No shunt murmur heard on auscultation.

**Management**

- If SaO_{2} < 65% and shunt malfunction suspected (i.e. no respiratory cause) call NICU consultant. Inform cardiac surgeon and cardiologist immediately.
- Review of ventilation status, blood gas and CXR should have been performed.
- Ensure haemodynamic status is adequate. May require volume expansion with normal saline. Give 5 mL/kg aliquots and titrate with response. It is essential to give smaller aliquots of fluid so as not to give too much fluid which could tip the delicate balance of a cardiac patient. If requiring > 20-30 mL/kg of fluid, reconsider your diagnosis.
- May require vasopressors to augment systemic pressure. Noradrenaline works well by intense peripheral vasoconstriction to increase the systemic driving pressure. However dopamine and/or adrenaline may be required to support the cardiac function.
- Ensure coagulation profiles are normal and blood available if trip back to theatre seems likely.

**High O_{2} Saturations (> 85% in Air)**

Usually due to high shunt flow, causing excessive blood flow to the lungs and an unbalanced circulation.

**Causes**

- Unnecessarily high FiO_{2}.
- PDA still open and so additional shunting of blood to the lungs*.
- MAPCAs as a source of additional blood flow to the lungs.
- Shunt too large.

*The ductus arteriosus (DA) is not routinely ligated at PMH as most procedures are right sided and access to the DA is not possible. Therefore the DA is patent post-op and causes additional blood flow to the lungs. PGE1 will have been ceased in theatre, and the PDA
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usually closes over hours/days. If the high \(O_2\) saturations are due to this reason, then they should improve as the ductus closes.

Other Signs
- **Rising lactate and metabolic acidosis in a ‘pink’ patient** (reduced systemic flow)
- Pulmonary plethora/oedema (may be unilateral).
- Cardiac failure.
- Systemic diastolic pressure low due to ‘run off’ to the lungs.
- May cause pulmonary haemorrhage.

Treatment
- Ensure ventilating in \(FiO_2\) 0.21.
- Inform NICU consultant.
- If \(SaO_2\) newly consistently > 90% inform cardiologist (will require review and echo)
- If not mechanically ventilated, consider ventilation and use higher PEEP/MAP.
- In more refractory cases manipulation of pH (permissive hypercapnia) may assist in increasing the pulmonary vascular resistance (Refer to Cardiac: Neonatal Circulation Changes/Unbalanced Circulation).
- Often fluid restriction and diuretics are beneficial.
- Sometimes volume loaded ventricles needs supporting with inotropes e.g. Dopamine.
- If poor systemic output, withhold feeds as at high risk of NEC.
- Occasionally urgent surgical revision of a large shunt is required (smaller diameter shunt inserted).

Prior to Discharge
Whenever a child with a BT shunt develops an illness where they may become dehydrated e.g. Gastroenteritis/vomiting or has a decreased oral intake or is sweating excessively, they are at risk of shunt thrombosis. They will need increased oral fluids e.g. Oral rehydration solution (ORS) if tolerated or early IV fluids. Parents should be warned of this prior to discharge and told to seek medical help early.

Also, if their child ever becomes blue then they need to take them to hospital immediately as it may indicate shunt blockage.

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