NTS Clinical Guideline

Title:	Management of Bradyarrhythmias		
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Foreword

This document provides guidance for the recognition and management of neonatal brady arrhythmias.

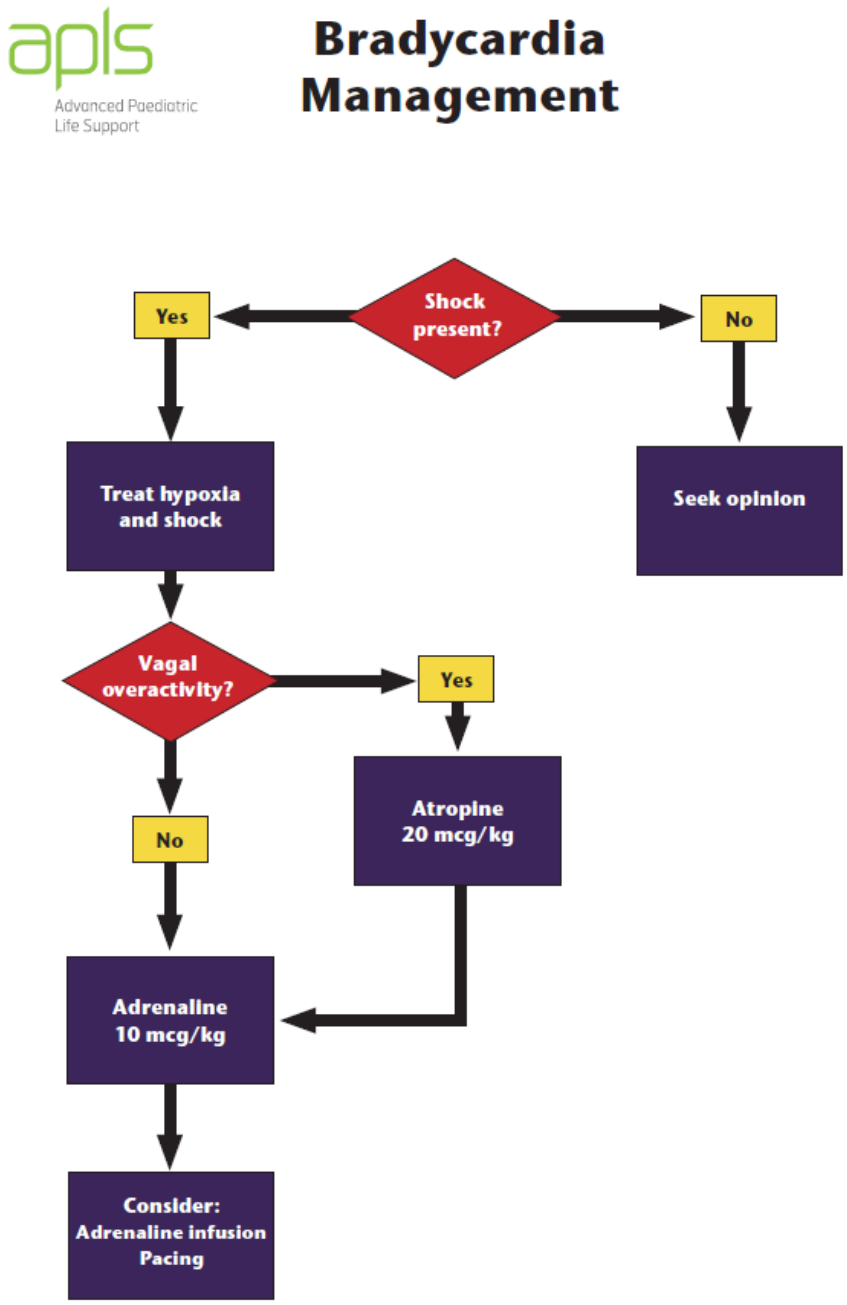
Infants with known cardiac lesions / arrhythmias who weigh ≥ 1.5kg, should normally be transferred by the Children's Acute Transport Service (CATS). However, it is possible that NTS may become involved in the management of these infants if CATS are unable to undertake the transfer, or if a cardiac lesion comes to light during the referral or assessment process.

Brady-arrhythmias

- Usually a pre-terminal finding in respiratory or circulatory insufficiency
- Sustained bradycardia may be due to congenital heart block (p waves not always followed by QRS complex), or secondary to hypoxaemia, acidosis, myocarditis, digoxin toxicity, hypothyroidism, raised ICP, or congenital heart defects

Management

- Assess ABC (including rhythm analysis / ECG) and whether in shock. If not in shock, discuss with Paediatric Cardiologist
- Follow APLS algorithm (below)



Background

- Arrhythmias may be detected during either the foetal or neonatal period
- They occur in around 1-2% of all pregnancies and around 1-5% of all newborns in the first 10 days of life.
- They are commonly classified as tachycardic, bradycardic or irregular arrhythmias
- In pregnancy the mother is often asymptomatic and many foetal arrhythmias do not cause problems, however, sustained tachycardia or bradycardia can cause cardiac failure and hydrops.
- Neonatal presentation is variable, with symptom development depending on rate and duration of the arrhythmia.
- The clinical picture ranges from the asymptomatic infant, to one with signs of congestive cardiac failure and cardiogenic shock. Brady-arrhythmias usually have a heart rate of <100 bpm.

Brady-arrhythmia

- Bradycardia is defined as a heart rate < 100 bpm.
- Transient bradycardia is not uncommon during sleep or vagal stimulating manoeuvres and is benign.
- The most common neonatal cause of sustained bradycardia is *congenital heart block.* This can occur in the presence of structural heart defects, or in infants born to mothers with connective tissue disorders such as SLE or Sjogren's, as a result of antibodies(anti-Ro and La) crossing the placenta and affecting the conduction pathway. On the ECG, p waves will not be followed by a QRS complex (AV dissociation). An escape junctional rhythm will generate a slow heart rate of around 50-80 bpm.
- Other causes include hypoxaemia, acidosis, myocarditis, digoxin toxicity, raised intracranial pressure, hypothyroidism, congenital heart disease and cardiac surgery.

Management (see algorithm above):

- *Stable patients:* Discuss with a Paeditric Cardiologist and assess for underlying causes
- *Shocked patients:* ABC approach. Consider atropine 20mcg/kg if vagal excitation, or adrenaline 10mcg/kg. An adrenaline infusion or cardiac pacing may be needed if ineffective.