

PAEDIATRIC ARRHYTHMIAS

BRADYARRHYTHMIA

Sinus node dysfunction

- criteria for sinus bradycardia

Table 1. ECG criteria

Age Group	Heart Rate
infants to < 3 years	<100 bpm
children 3 – 9 years	< 60 bpm
children 9 – 16 years	< 50 bpm
adolescents > 16 years	< 40 bpm

Table 2. 24 hours Ambulatory ECG criteria

Age Group	Heart Rate
infants to 1 year of age	< 60 bpm sleeping, < 80 bpm awake
children 1 – 6 years	< 60 bpm
children 7 – 11 years	< 45 bpm
adolescents, young adults	< 40 bpm
highly trained athletes	< 30 bpm

Systemic causes of sinus bradycardia:

- hypoxia
- intracranial lesions
- hypothyroidism
- electrolytes abnormalities i.e. hypokalaemia, hypocalcaemia
- sepsis
- acidosis
- anorexia nervosa

Causes of sinus node dysfunction

- right atrial dilatation due to volume loading
- cardiomyopathies
- inflammatory conditions: myocarditis, pericarditis, rheumatic fever
- post atrial surgery: Mustard, Senning, Fontan, ASD closure, cannulation for cardiopulmonary bypass

Atrioventricular block

Classification

- 1st degree - prolonged PR interval
- 2nd degree
 - Mobitz type 1 (Wenckebach): progressive PR prolongation before dropped AV conduction
 - Mobitz type 2: abrupt failure of AV conduction without prior PR prolongation
 - high grade – 3:1 or more AV conduction
- 3rd degree (complete heart block): AV dissociation with no atrial impulses conducted to ventricles

Note: 2nd degree (Type 2 and above) and 3rd degree heart block are *always* pathological

Aetiology

- congenital – in association with positive maternal antibody (anti-Ro and anti-La); mother frequently asymptomatic
- congenital heart diseases: atrioventricular septal defect (AVSD), congenital corrected transposition of great arteries (L-TGA), left atrial isomerism
- congenital long QT syndrome
- surgical trauma: especially in VSD closure, TOF repair, AVSD repair, Konno procedure, LV myomectomy, radiofrequency catheter ablation
- myopathy: muscular dystrophies, myotonic dystrophy, Kearns-Sayre syndrome
- infection: diphtheria, rheumatic fever, endocarditis, viral myocarditis

Acute Management of symptomatic bradycardia with haemodynamic instability

- treat the underlying systemic causes of bradycardia
- drugs:
 - IV atropine
 - IV isoprenaline infusion
 - IV adrenaline infusion
- transcutaneous pacing if available

Patients who are not responding to initial acute management should be referred to cardiologist for further management. Emergency transvenous pacing or permanent pacing may be required.

TACHYARRHYTHMIA

Classification

- atrial tachycardia – AF, EAT, MAT
- conduction system tachycardia or supraventricular tachycardia – AVRT, AVNRT, PJRT
- ventricular tachycardia – VT, VF

Description

- Atrial flutter (AF)
 - saw tooth flutter waves
 - variable AV conduction
- Ectopic atrial tachycardia (EAT)
 - abnormal P wave axis
 - P wave precedes QRS
 - variable rate
 - “warm up” and “cool down” phenomenon
- Multifocal atrial tachycardia (MAT)
 - irregularly irregular
 - multiple different P wave morphologies, bizarre and chaotic
 - no two RR intervals the same
- Atrioventricular re-entry tachycardia (AVRT)
 - P wave follows QRS

Figure 2. Atrial flutter



Figure 3. Ectopic atrial tachycardia



Figure 4. Multifocal atrial tachycardia



Figure 5. Atrioventricular re-entry tachycardia



Figure 6. Atrioventricular nodal re-entry tachycardia



- Atrioventricular nodal re-entry tachycardia (AVNRT)
 - P wave not visible, superimposed on QRS
- Permanent junctional reciprocating tachycardia (PJRT)
 - inverted P waves in II, III, aVF appear to precede QRS complex
 - long RP interval
- Ventricular tachycardia (VT)
 - wide QRS complex
 - P wave may be dissociated from the QRS complex
- Ventricular fibrillation (VF)
 - chaotic, irregular rhythm

Figure 7. Permanent junctional reciprocating tachycardia



Figure 8. Ventricular tachycardia

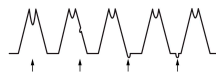
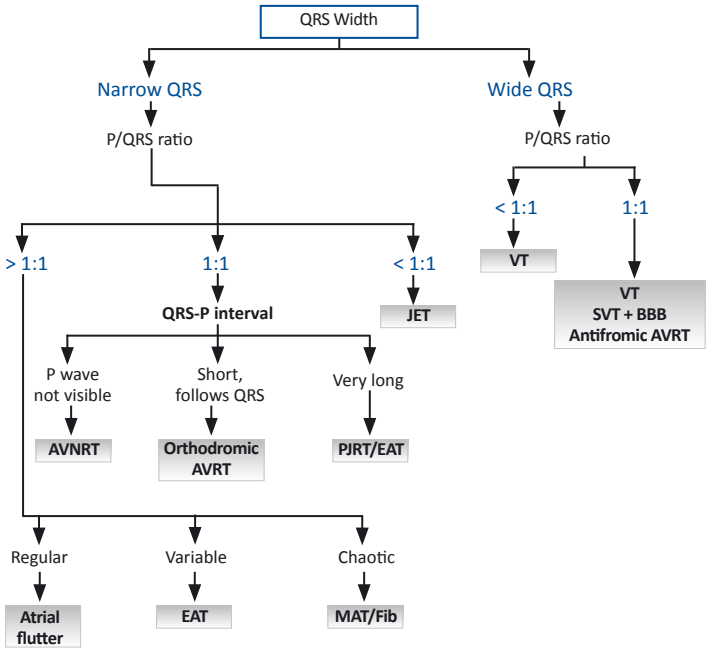


Figure 8. Ventricular fibrillation

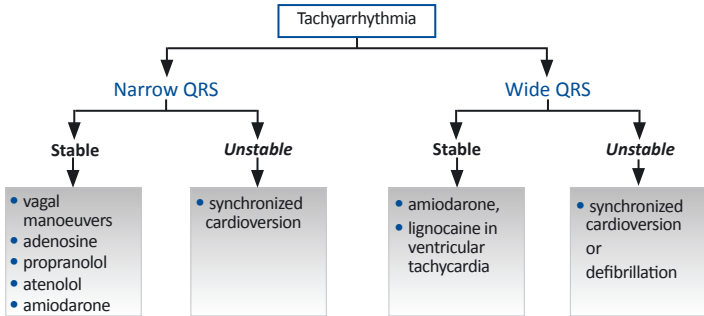


Figure 2. Algorithm for identifying tachycardia



Abbreviations. VT, ventricular tachycardia; JET< junctional ectopic tachycardia; SVT, supraventricular tachycardia; BBB, bundle branch block; AVRT, atrioventricular re-entry tachycardia; AVNRT, atrioventricular nodal re-entry tachycardia; PJRT, permanent junctional reciprocating tachycardia; EAT, ectopic atrial tachycardia; MAT, multifocal atrial tachycardia; Fib, fibrillation.

Figure 3. Algorithm for management of acute tachyarrhythmia



Narrow QRS complex tachycardia

Haemodynamically stable

- vagal manoeuvres:
 - icepack/iced water for infants – apply to face for a maximum of 30 seconds
 - Valsalva manoeuvres if child is old enough (blow into a pinched straw)
- IV Adenosine: 0.1mg/kg (max 6mg) rapid push. Increase by 0.1mg/kg every 2 mins until tachycardia terminated or up to a maximum of 0.5mg/kg (maximum: 18 mg).
- IV propranolol 0.02mg/kg test dose, then 0.1mg/kg over 10 minutes
- IV amiodarone: 25mcg/kg/min for 4 hours then 5 -15mcg/kg/min until conversion

Haemodynamically unstable

- synchronized DC conversion at 0.5 to 1 joule/kg

Wide QRS complex tachycardia

Haemodynamically stable

- IV amiodarone (same as above)
- IV procainamide
- IV lignocaine

Haemodynamically unstable

- synchronized cardioversion at 0.5 to 1.0 joule/kg
- in pulseless patients, defibrillate at 2 to 4 joules/kg

Pitfalls in management

- to consult a cardiologist if these acute measures fail to revert the tachycardia.
- in Wolff-Parkinson-White syndrome, digoxin is contraindicated because paroxysm of atrial flutter or fibrillation can be conducted directly into the ventricle.
- adenosine unmasks the atrial flutter by causing AV block and revealing more atrial beats per QRS complex
- in wide QRS complex tachycardia with 1:1 ventriculoatrial conduction, it is reasonable to see if adenosine will cause cardioversion, thereby making a diagnosis of a conduction system dependent SVT
- a follow up plan should be made in consultation with cardiologist