Department of Anesthesia Techniques

Arrhythmias

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Interpreting the ECG

1- Rate and rhythm

• regularly irregular (is there a pattern?) irregularly irregular (no pattern)

2: P wave Does the P wave appear before the QRS?

Is there one P wave for every QRS

Is the shape normal?

Are P waves missing?

2: PR interval Is the PR interval 3-5 small squares?

3: QRS complex Is the QRS width within 3 small squares? Is the axis normal (“positive QRS”)?

Is the S wave deep (“negative QRS”)?

Does it look normal?

4: ST segment Does the isoelectric line return between the S and the T? If not, is it:

elevated (> 1mm above isoelectric line) depressed (< 0.5 mm below isoelectric line)

5: T wave Does the T wave look normal?

6. QT interval Is QTc within 0.48 seconds?

- **Tachycardia (> 100 bpm) narrow complex (usually with P waves) = atrial (supraventricular) broad complex (without P wave) = ventricular**

- Normal conduction begins in the sinoatrial node, passing through atrial muscle to the atrioventricular node, then passing from the atrioventricular node through the ventricular conduction pathway and into ventricular myocytes. There are therefore three key stages in normal cardiac conduction and the ECG:

- **atrial (P wave)**

- **atrioventricular node (PR interval)**

- **ventricular (QRS complex, ST segment, T wave).**

Sinus tachycardia

- HR > 100bpm
- Causes:

Intra-cardiac causes

  - Ishcaemic heart disease
  - Valvular heart disease
  - Heart failure
  - Cardiomyopathy
  - Congenital heart disease

- Treatment
  - Treat the cause.

Extra-cardiac causes

- Drugs
- Alcohol
- Stimulants e.g. caffeine
- Stress
- Hyperthyroidism
- Infection/Sepsis

Sinus Bradycardia

- HR < 60bpm
- Causes

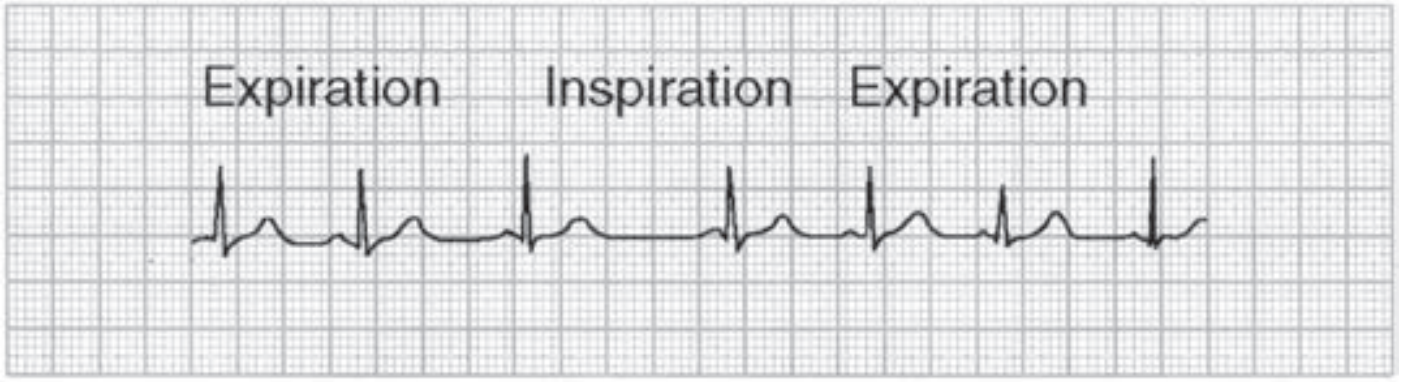
  Physiological (normal in athletic people)
  Iatrogenic (Beta blockers, Ca channel blockers, digoxin, anticholinergics)
  Hypothyroidism
  Metabolic e.g. hyperkalemia
  Hypoxia
  Hypothermia
  Acute MI/ischemia

- Treatment

  - Remove cause (ie drugs)
  - Treat cause (ie hypothyroidism)

Atrial dysrhythmias

- **Sinus arrhythmia** Inspiration significantly increases venous return, and so heart rate.
- **Sinus bradycardia**. sinus rate < 60 bpm
- **Sinus tachycardia.** sinus rate > 100 bpm. normal in young children, tachycardia is abnormal with adults



Supraventricular tachycardia

- any tachycardia originating above the ventricles rates exceeding 130 bpm, where decompensation causes hypotension.
- Narrow-complex tachycardia must be SVT,
- “atrial tachycardia” (i.e. originating in the atrium but not sinus), are sometimes used.
- Treatment. In the ICU, SVT is usually treated with amiodarone. Other drugs, such as beta-blockers, may sometimes be tried.

Atrial fibrillation (AF)

May occur acute or may be chronic

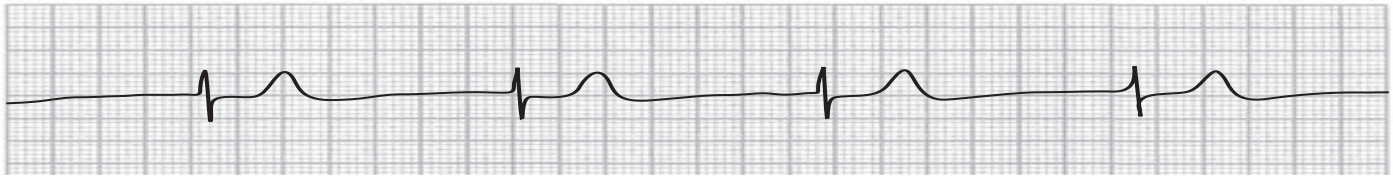
- causes chaotic atrial activity, seen by either a chaotically wavy or almost flat line between ventricular electrical activity.
- QRS complexes, ST segments and T waves are normal but timing (space) between ventricular contractions (QRS complexes) is erratic – irregularly irregular.
- Loss of atrioventricular synchrony
- reduces stroke volume by 5–15%
- which usually provokes compensatory tachycardia.
- Treatment. New-onset AF should be cardioverted either electrically or chemically by amiodarone
- rate control: aim < 110 bpm and anticoagulation: aim INR 2.5
- Digoxin, beta-blockers or calcium channel blockers for rate control.

Atrial flutter

- An ectopic pacemaker in the atrium causes rapid atrial waves with **sawtooth shapes ("F" or flutter waves)**
- **300 bpm. This rate exceeds possible AV nodes conduction, so a regular** block occurs, usually of an even ratio (e.g. 2:1, 4:1, 6:1 or 8:1). A 4:1 AV block with atrial rates of 300 creates ventricular responses of 75 bpm. But blocks can change suddenly, creating gross tachycardia (2:1 block = ventricular rate 150/minute) or bradycardia.
- Treatment. electrical cardioversion is usually preferred, but chemical cardioversion (e.g. amiodarone) may restore sinus rhythm.

Junctional (or “nodal”) rhythm

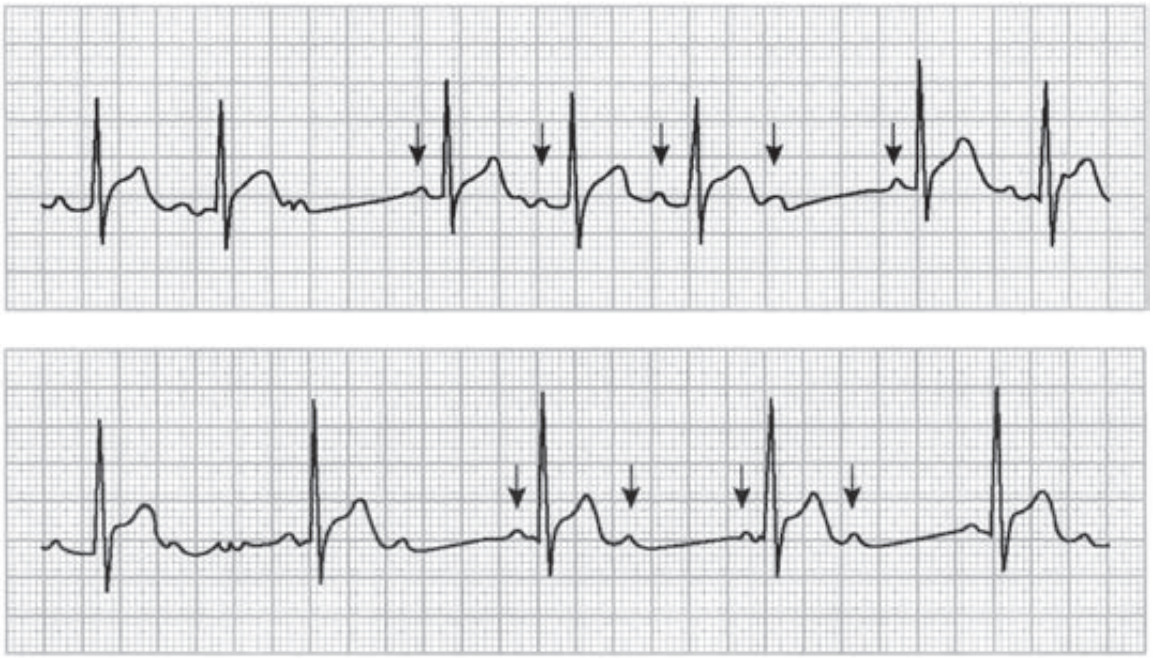
- impulses originating in the AV node or atrioventricular junction. Rate is slower than sinus/atrial rhythms.
- **P waves are not usually seen**, but if present are inverted, and may appear after QRSs. Irritation (oedema, mechanical – e.g. central lines in the right atrium) may cause junctional ectopics.
- Oedema from cardiac surgery often causes transient junctional rhythms
- Treatment. Junctional rates are often sufficient to support life but should be closely monitored. If bradycardia becomes symptomatic, treat as sinus bradycardia



Atrioventricular blocks

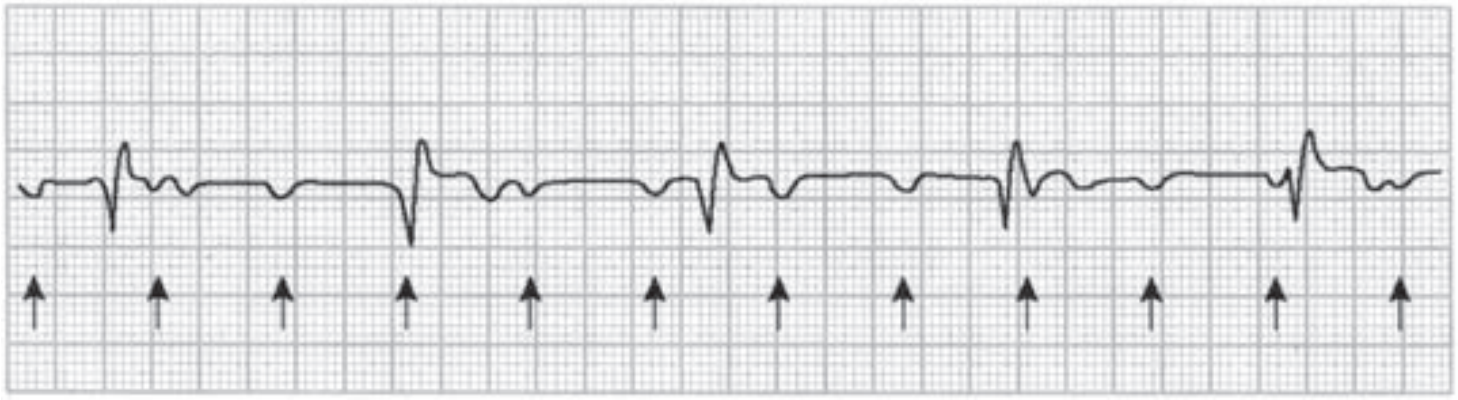
- Any conduction pathway may be blocked by: infarction, oedema or ischaemia.
- Blocks may occur at the atrioventricular node (first-, second- or third-degree) or in one of the bundle branches.
- **First-degree (AV node) block** delayed atrioventricular node conduction, prolongs PR intervals beyond 0.2 seconds (five small squares). Despite delay, every impulse is conducted, so a QRS complex follows each P wave.

- **Second-degree block**, or incomplete heart block, occurs when at regular intervals there is an unconducted P wave. There are two types of second-degree block:



Third-degree block

- **Complete heart block**, causes complete atrioventricular dissociation. Any atrial activity (e.g. P waves) is unrelated to QRS complexes; some P waves may be "lost" in QRS or T waves. Cardiac output and blood pressure are usually compromised.
- **Treatment. pacing**

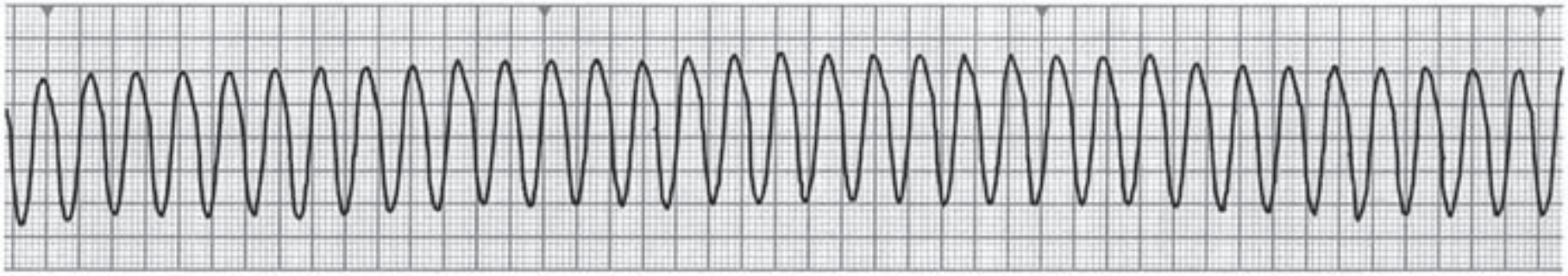


Bundle branch blocks

- conduction through one of branches from the Bundle of His is blocked. This creates two QRS complexes – a normal one from the intact branch, and a broadened ventricular-shaped complex from impulses spreading across the septum. This RSR, or biphasic QRS, wave creates the characteristic M or W shapes on ECGs.
- Left bundle branch block causes a W in early, and an M in late chest leads;
- right bundle branch block reverses this picture.

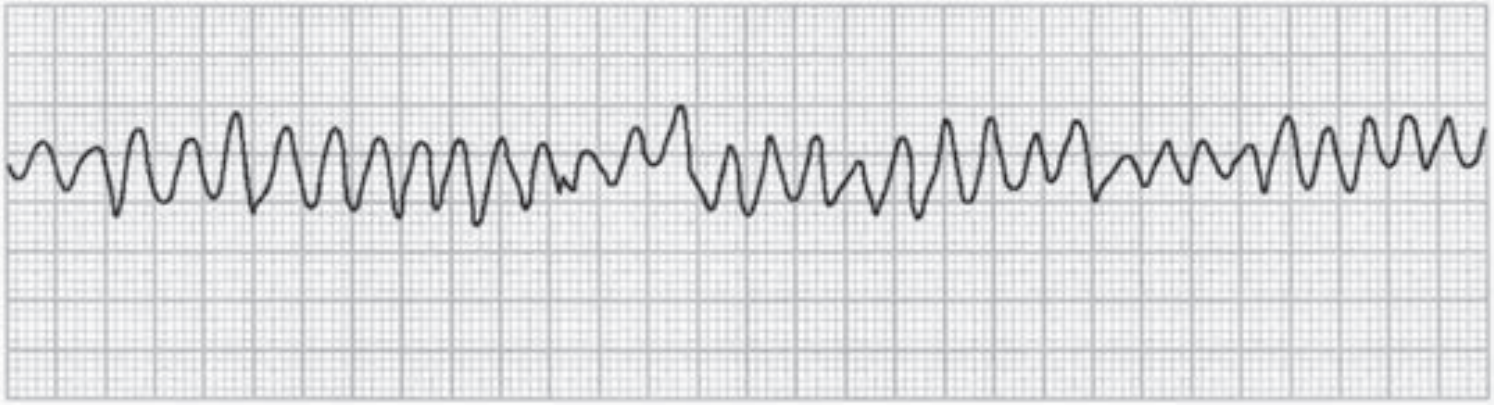
Ventricular dysrhythmia

- Ventricular impulses originate in ventricular muscle, so usually travel from muscle fibre to muscle fibre rather than through conduction pathways, making progress relatively slow, giving them typically broad QRS complexes.
- progress relatively slow, giving them typically broad QRS complexes. Impulses originating in or near conduction pathways may have narrow complexes.
- **Ventricular tachycardia** is a regular rhythm, with rapid unifocal impulses (typically 200–250 bpm)..
- Treatment. Ventricular tachycardia with a pulse may respond to drugs (e.g. amiodarone), but pulseless VT is a shockable rhythm, necessitating immediate resuscitation.



Ventricular fibrillation (VF)

- is almost fatal in two to three minutes. VF may be coarse or fine; fine ventricular fibrillation may appear like asystole, so increasing gain on ECGs shows whether “f” waves are present.
- Treatment. Ventricular fibrillation is a shockable rhythm, necessitating immediate resuscitation.



Asystole (= ventricular standstill),

- absence of systole, appears as an uninterrupted isoelectric line, Chest wall movement from breathing (including mechanical ventilation) typically causes slow undulations to the isoelectric line.
- Treatment. Asystole is not a shockable rhythm.
- Cardiac compressions and drugs are essential to maintain effective circulation.
- Pulseless electrical activity (PEA) results in whatever electrical activity is seen not being translated into pulses and arterial blood pressure traces

Cardiac Asystole