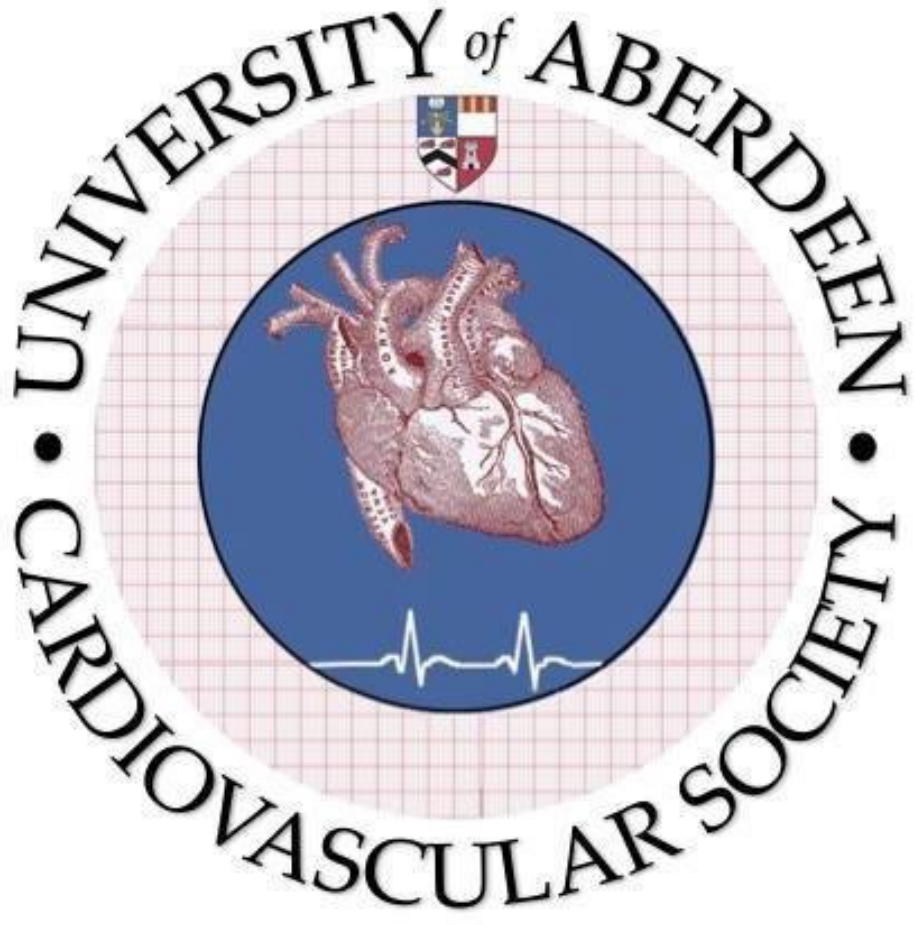
1 4 9 5

UNIVERSITY OF ABERDEEN



ECG Bootcamp

Prepared by: UoACVS

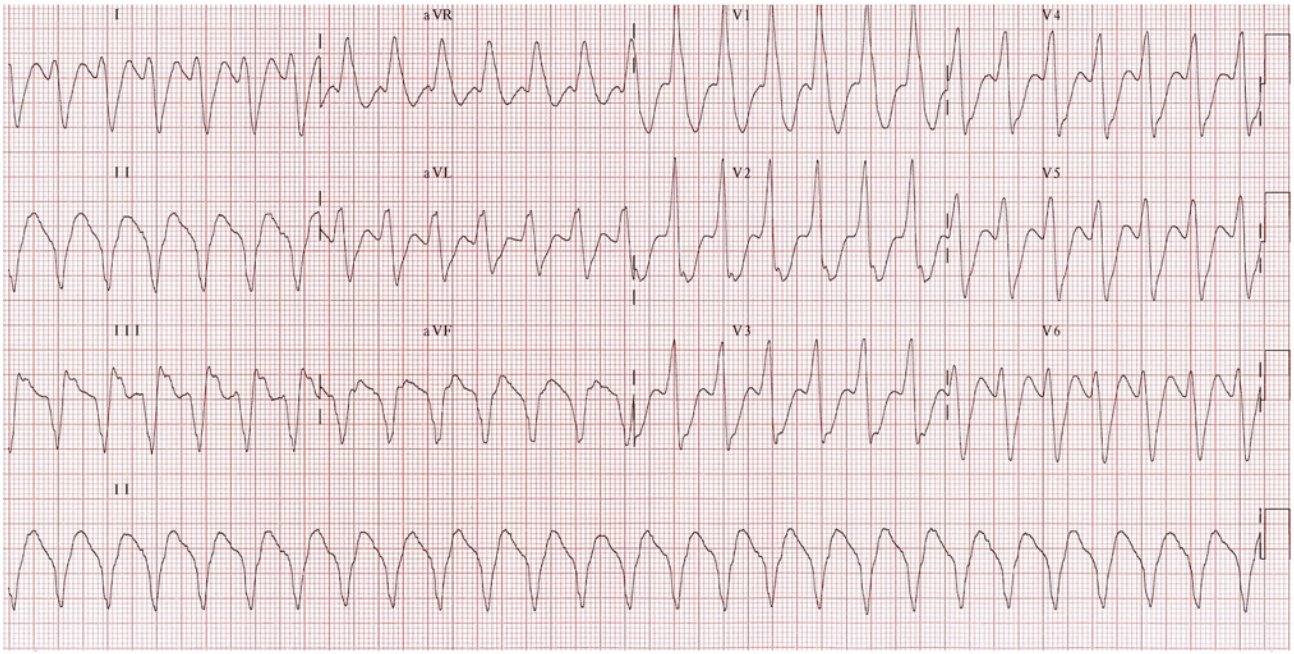
7 September 2016

Proposal number: 123-4567

ARREST RHYTHMS STATION

BACKGROUND

VT can either be monomorphic or polymorphic. In monomorphic VT the axis stays the same so each of the QRS complexes looks the same. In polymorphic VT the axis changes hence each of the wide QRS complexes look different. Torsades de Pointes is a type of polymorphic VT. In Torsades the axis keeps changing direction and so some of the QRS complexes are positive and then they change to negative and then back to positive etc.



1. Label the QRS complex, P waves and T waves

2. What features do you see on this ECG that would make you think that this is ventricular tachycardia? Wide QRS, extreme axis deviation that doesn't change, tachycardia, AV dissociation

3. Is it polymorphic or monomorphic?

monomorphic

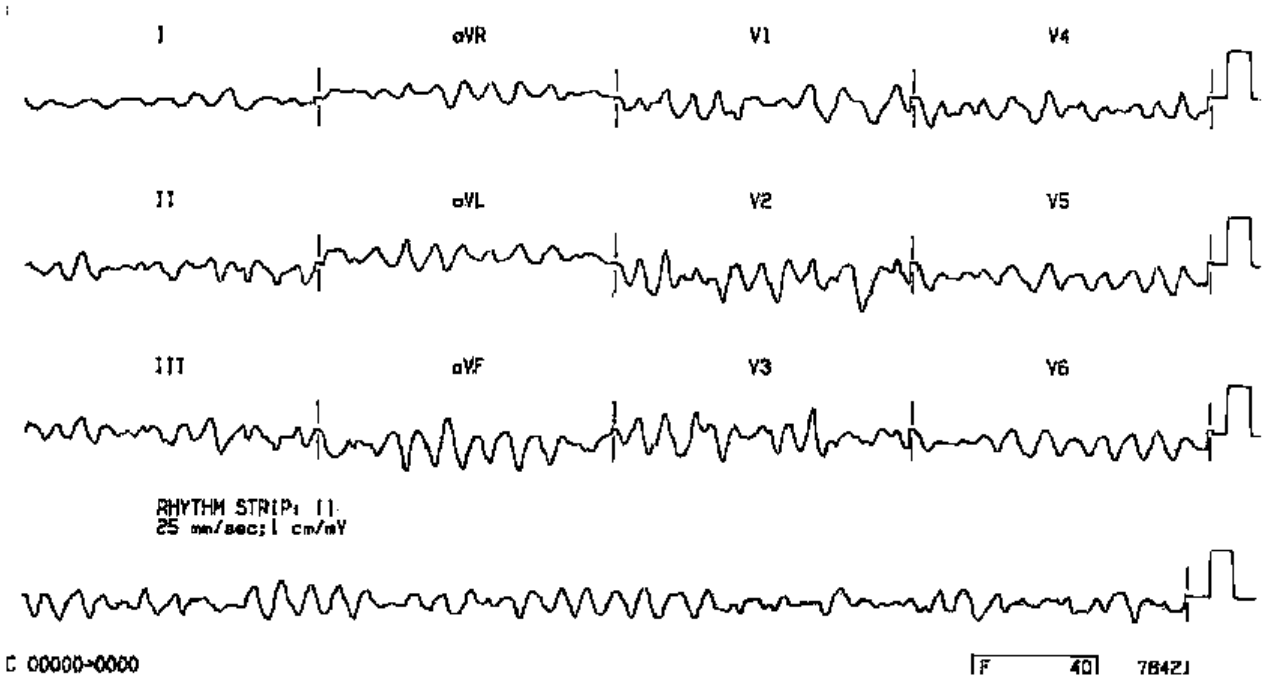
4. What is the axis (normal, left deviation, right deviation or extreme deviation) Hint: look at leads I and aVF ?

extreme deviation (aVF and I both negative)

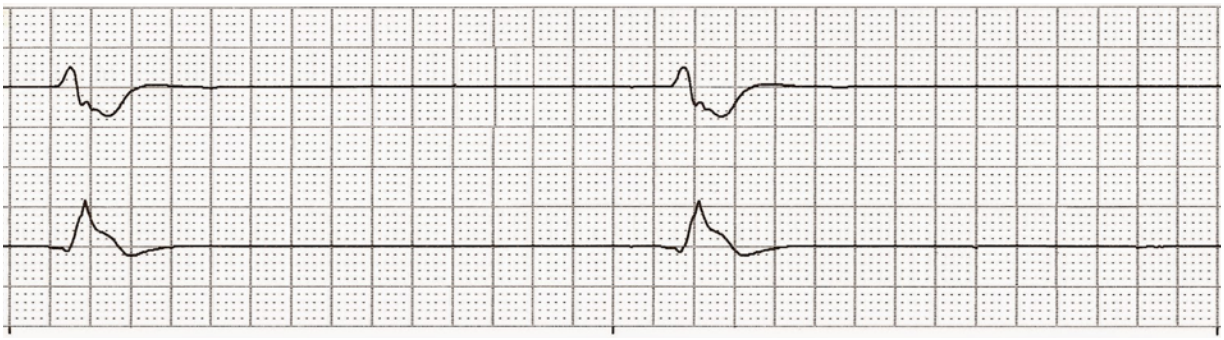
5. What is the management of this patient if they are unstable?

Defibrillation (if pulseless VT) CPR, ABC assessment

6. The VT has been going on for a long time and now the ECG tracing has changed. What has happened to the heart rhythm in the strip below? Changed to VFib



7. Eventually the situation worsens and this rhythm appears on the ECG strip. What is the name for this rhythm? Is it shockable? Why? Agonal rhythm (wide QRS, very very slow HR, peri death situation), No it is treated in the same way as asystole, when you shock you basically create asystole and then hope that the sinus node will take over, this will not work in this case



8. Name eight causes of a cardiac arrest

1. Hyper/hypokalaemia
2. Hypoxia
3. Hypothermia
4. Hypovolaemia
5. Thrombosis
6. Tamponade
7. Tension pneumothorax
8. Toxins
9. Hydrogen ions (acidosis),

HEART ATTACKS STATION

Background

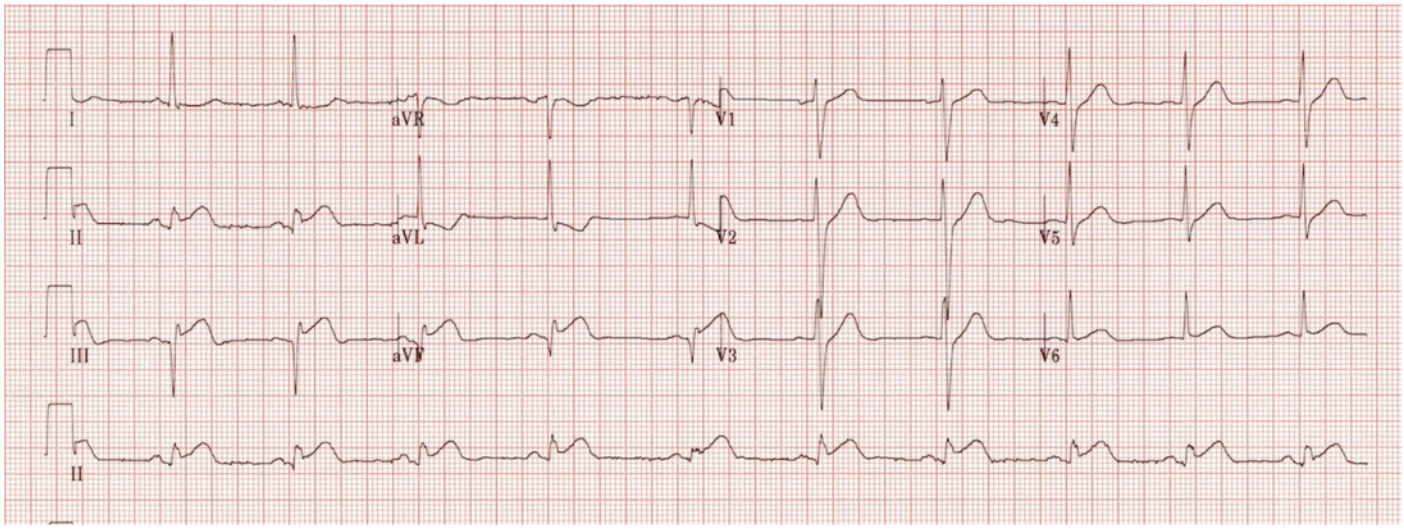
An MI is different from a cardiac arrest. In a cardiac arrest the patient is in a haemodynamically unstable rhythm. A patient having an MI can still be in sinus rhythm despite having death of an area of the heart. However heart attacks can cause cardiac arrests.

Fill in the table stating which leads look at which areas of the heart

Description	Leads
Anterior	V3 and V4
Septal	V1 and V2
Inferior	Lead II, III and aVf
Lateral	V5 and V6
High Lateral	Lead I and aVL

You cannot see the posterior aspect of the heart on a standard 12 lead ECG. How might you be able to tell if there was a posterior STEMI? Reciprocal ST depression in the leads at the from of the chest V1 and V2

This patient is having an MI. Which Region of the heart has been affected and which coronary artery has most likely become blocked? Inferior MI, most likely right coronary artery



Is there any evidence of an old MI within this ECG?

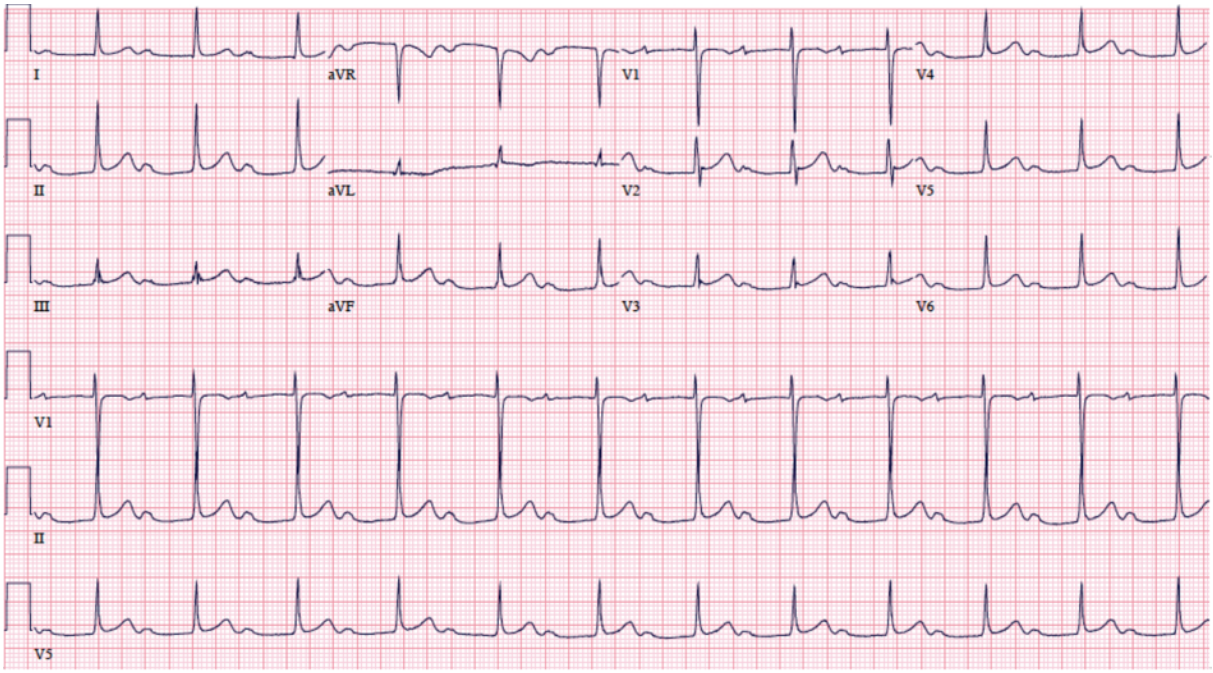
There are pathological Q waves in lead III and aVf suggesting an old inferior MI

HEART BLOCK (AV BLOCK) STATION

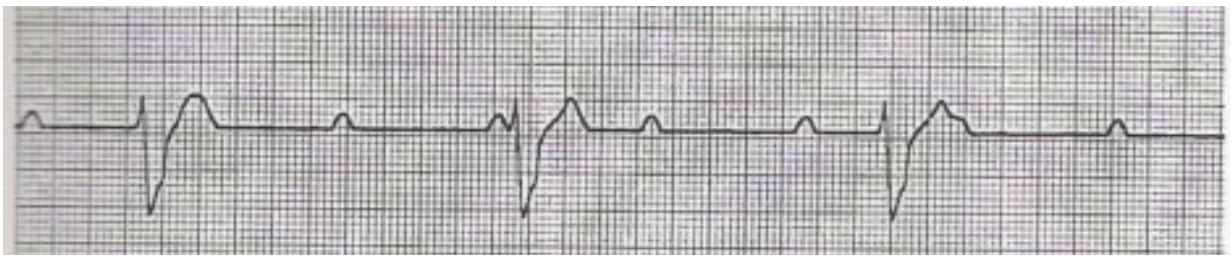
BACKGROUND

AV block can happen for many reasons e.g. specific drugs, disease of the AV node etc. In broad terms there are 3 degrees. In 1st degree all the p waves conduct to the ventricles. In 2nd degree some of the p waves conduct to the ventricles. In 3rd degree none of the p waves conduct to the ventricles.

Use ARI BAR to work out which type of heart block this patient has. Are they haemodynamically stable? One p wave to one QRS and so 1st degree heart block because PR interval is long



Another patient has a rhythm strip that looks like this. Which type of heart block do they have?
3rd degree



Why are the QRS complexes wide?

They aren't following the normal conduction pathway

Explain what escape rhythm (aka idioventricular rhythm) is

Ectopic beats generated when the SAN fails to initiate a beat. If the AV node is the generator of these beats it's called junctional escape but remember any heart cell can be a pacemaker

What is the definitive treatment for someone with this arrhythmia? Is it an emergency situation?

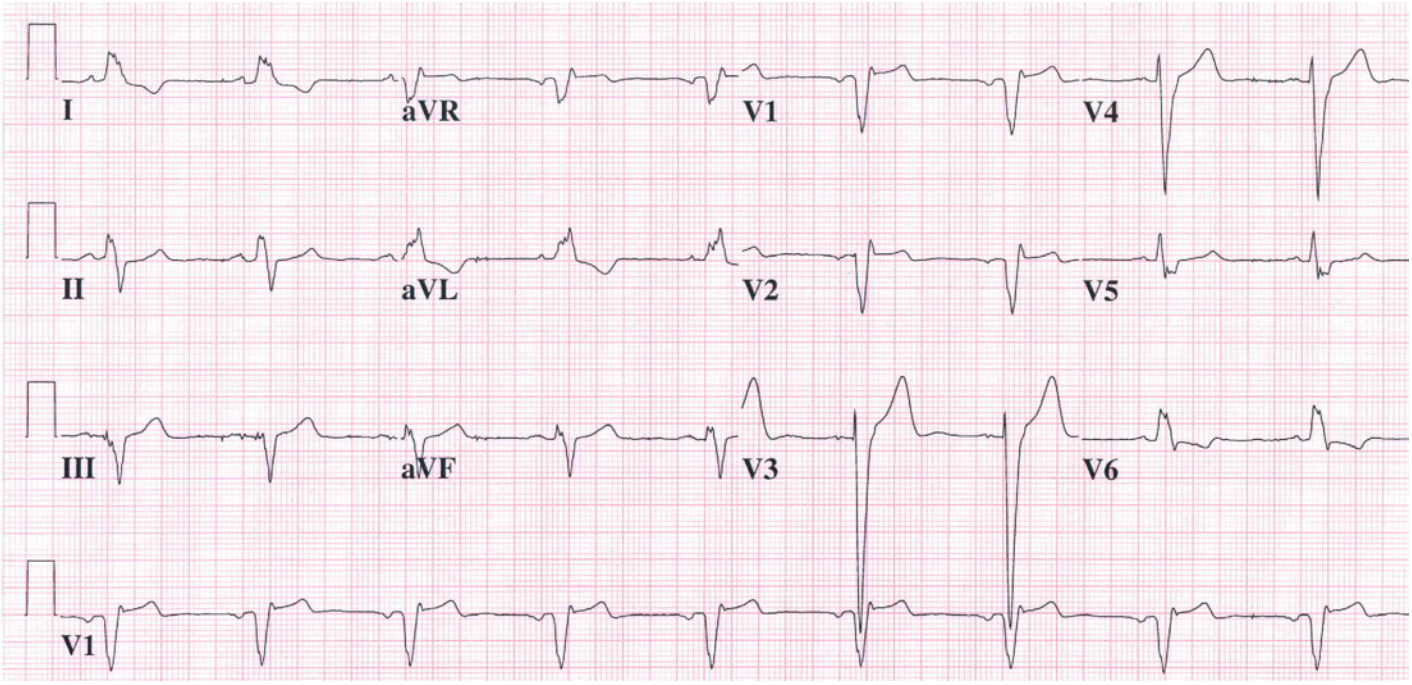
transcutaneous pacing

BUNDLE BRANCH BLOCKS STATION

BACKGROUND

A bundle branch block is a delay in the signal getting through the left or right bundle branch so instead the signal goes down the unblocked branch and spreads slowly across the septum rather than following the rapid His-Purkinje system.

Interpret this ECG fully using the layout below. If you are asked to describe an ECG on the ward this is the layout to use



A- electrical activity in all leads

R- roughly 60

I- regular

B- broad QRS

A- p waves present

R - one p wave to one QRS

PR length 0.12**s** **QRS duration** more than 0.12**s** **QT interval** 0.48**s**

Axis - left axis deviation

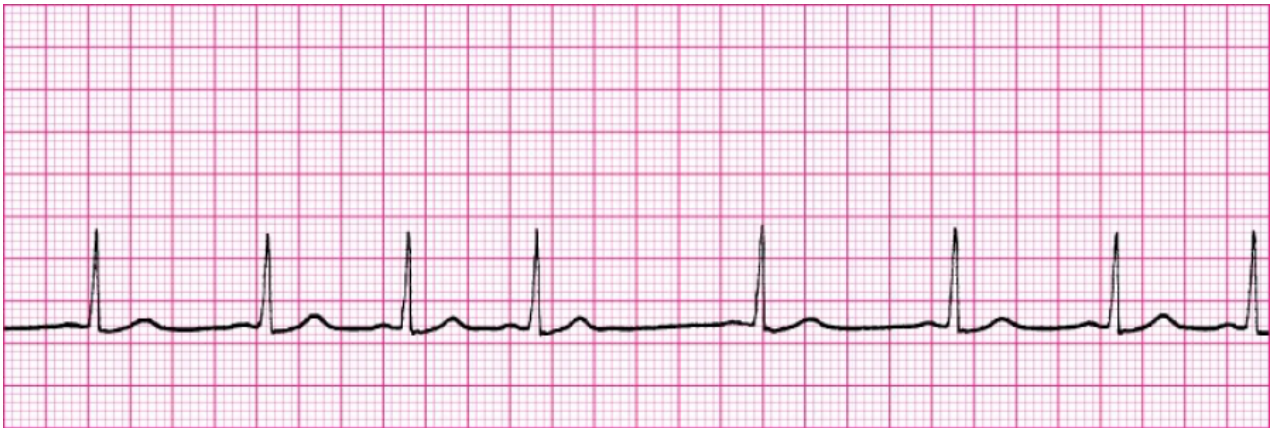
Any other Diagnostic Features - notch in QRS in leads V4-6

Diagnosis - left bundle branch block

ATRIAL RHYTHMS STATION

BACKGROUND

There are several different types of atrial rhythms but today we will focus on atrial fibrillation, Atrial flutter and SVT (which is a broad heading for tachycardias of atria origin). In atrial rhythms it is the AV node that determines what the ventricular response it. For example Afib can have a slow, moderate or fast ventricular response depending on how fast the AV is conducting.

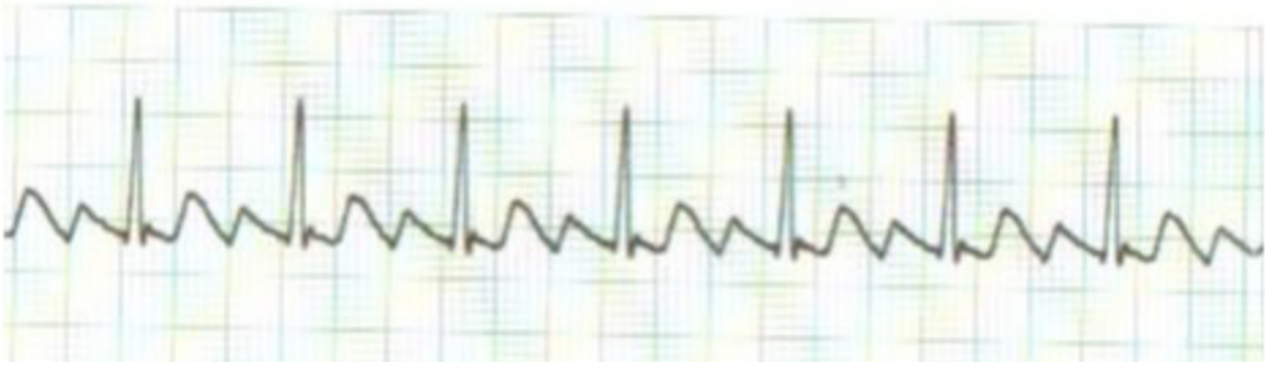


1) **Is this rhythm regular, regularly irregular or irregularly irregular?** Irregularly irregular

2) **What is the diagnosis here?** Sinus Arrhythmia NOT atrial fibrillation because there are p-waves visible. Sinus arrhythmia is caused by fluctuations in the signal from the vagus nerve but is a normal finding

3) **They have been admitted with breathlessness what is the management (brief description) of this patient?** ABC looking for other causes of breathlessness

4) **Can you cardiovert this rhythm?** NO but you can cardiovert unstable aFib



5) **What is the atrial rate in this rhythm and what is the ventricular rate?** atrial 300BPM and 100 BPM ventricular

6) **Why are they not the same?** the AV node will not allow conduction of every beat at such a high atrial rate

7) **What is the name of this arrhythmia?** atrial flutter

METABOLIC PROBLEMS STATION

BACKGROUND

Hyperkalaemia and hypokalaemia are both very dangerous and cause serious arrhythmias and arrests. These are the only two metabolic problems we will look at today but for your own interest it would be worth looking at how Ca+, magnesium and Na affect the ECG.

1. **What is a normal level of potassium in the body?** 3.5-5.5 mmol/l
2. **What symptoms would someone with hyperkalaemia have?** Frank muscle paralysis, Dyspnea, Palpitations, Chest pain, Nausea or vomiting, Paresthesias, asymptomatic
3. **What is the treatment for hyperkalaemia?** calcium gluconate, IV insulin/dextrose solution, Salbutamol, fluids, may need to actually give more potassium because the insulin tends to give hypokalaemia
   Dialysis

The ECG changes tend to correlate to how severe the hyperkalaemia is. There are early and late changes which show different patterns.

Early changes - K+ of 5.5-6.5 mmol/L

- Tall, peaked T waves with a narrow base, best seen in precordial leads (indicates a large voltage during repolarisation due to K+ repolarising the cells)
- Shortened QT interval (because repolarisation has happened more quickly)
- ST-segment depression

Mid Changes - K+ of 6.5-8.0 mmol/L

- Peaked T waves
- Prolonged PR interval
- Decreased or disappearing P wave
- Widening of the QRS
- Amplified R wave

Late Changes - K+ >8.0 mmol/L

- Absence of P wave
- Progressive QRS widening with a sine wave appearance
- Intraventricular/fascicular/bundle branch blocks
- Sine wave appearance

4. **Can you identify some/all of these on the ECG and estimate what this patient's potassium level is?**

- Peaked T wave
- Prolonged PR
- Wide QRS
- ST segment depression

K+ roughly 6.5