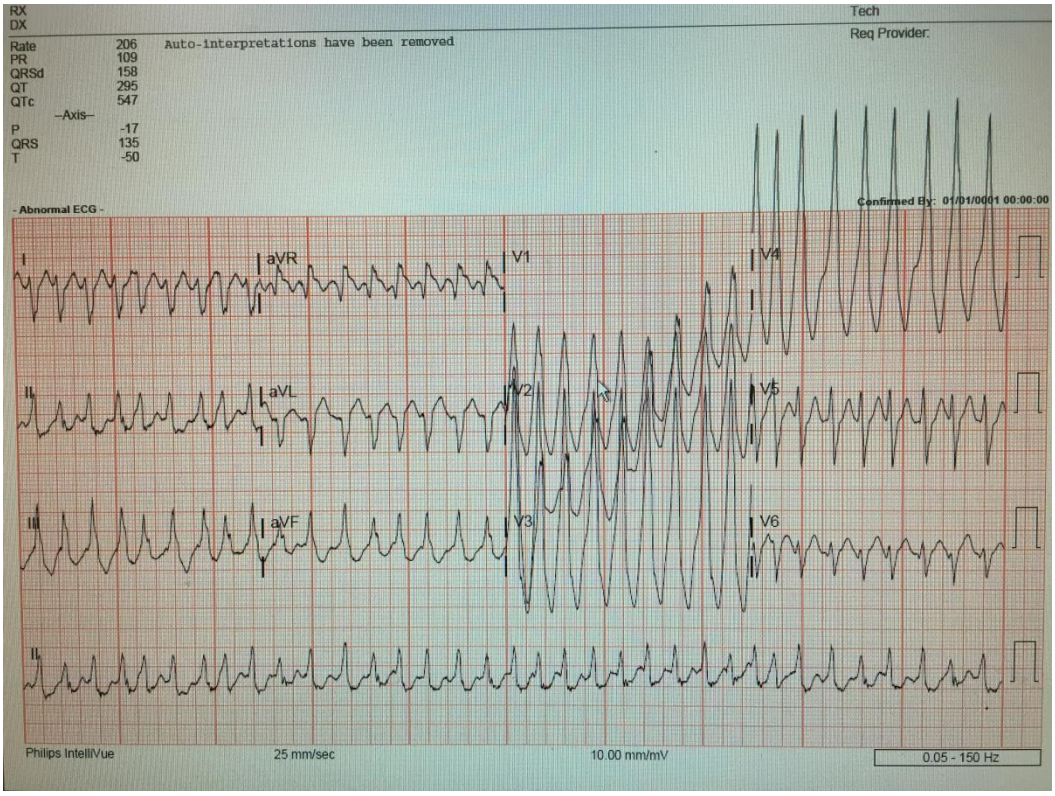
CASE BASED DISCUSSIONS 27/10/20 MEDICINE

CASE ONE

27yr old male, 120kg, presents with 24 hours of palpitations, dizziness, pre syncope. Has some vague chest pain. His vitals are:

HR 210-230bpm, BP 100/60, GCS 15, sats 98%

1. **Below is his ECG, interpret the ECG and discuss your management options and priorities?**



Irregularly irregular broad complex tachycardia – AF with aberrant conduction or underlying bundle. ? WPW . Differential could include polymorphic VT.

Wolff-Parkinson-White Syndrome with Atrial Fibrillation

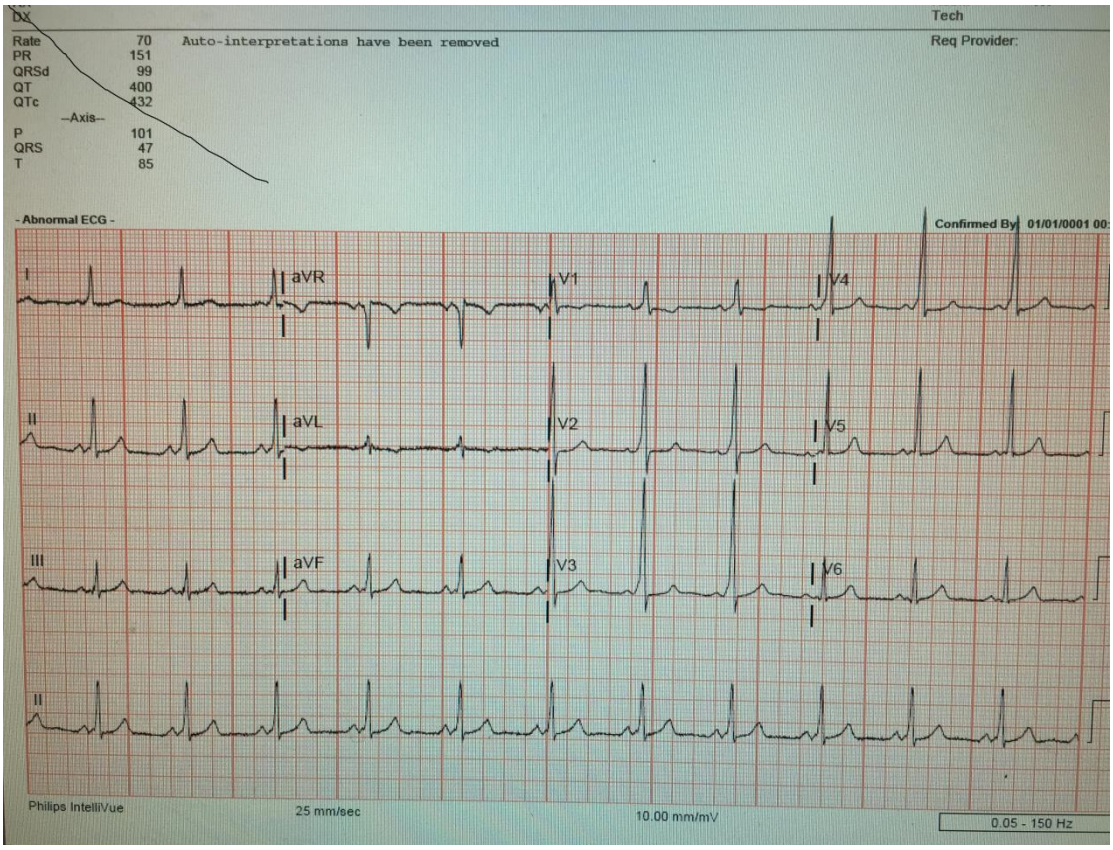
- Very rapid irregularly irregular tachycardia (rates may approach 300 beats/min) with wide QRS complexes that <u>vary in morphology</u>
- Often misdiagnosed as SVT, VT or atrial fibrillation with BBB
- Misdiagnosis and treatment with AVN blockers can be deadly!
- Treat with **preferably electrical cardioversion OR** procainamide
- **Key Point: Avoid all AV Nodal blockers**

- Management would be synchronized DC cardioversion

2. How do you carry out DC cardioversion?

- Consent and discussion
- Planning and equipment
- Staff
- Drugs
- Plan options for failure or success

3. Below is his post cardioversion ECG, interpret?



Delta waves, short PR

4. Discuss WPW patho-electrophysiology?

- In WPW the accessory pathway is often referred to as the **Bundle of Kent**, or atrioventricular bypass tract.
- The majority of pathways allow conduction in both directions, with retrograde only conduction occurring in 15% of cases, and anterograde only conduction rarely seen.

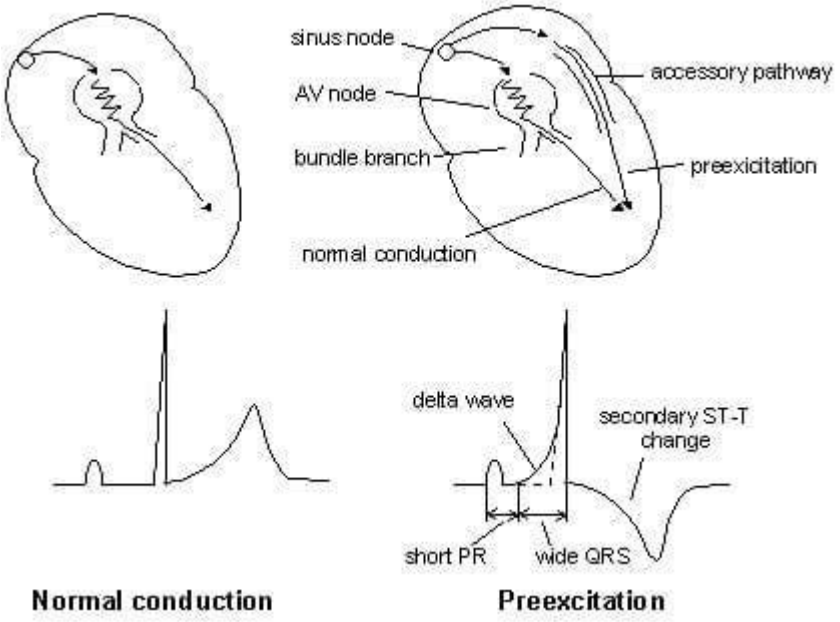
- The direction of conduction affects the appearance of the ECG in sinus rhythm and during tachyarrhythmias.
- Tachyarrythmia can be facilitated by the formation of a reentry circuit involving the accessory pathway, termed **atrioventricular reentry tachycardias (AVRT)**.
- Tachyarrythmia may also be facilitated by direct conduction from the atria to the ventricles via the accessory pathway, bypassing the AV node, seen with atrial fibrillation or atrial flutter in conjunction with WPW

ECG in Sinus Rhythm

The presence of a pre-excitation pathway results in a number of changes to the ECG in sinus rhythm.

ECG features of WPW in sinus rhythm are:

- PR interval <120ms
- Delta wave – slurring slow rise of initial portion of the QRS
- QRS prolongation >110ms
- ST Segment and T wave discordant changes – i.e. in the opposite direction to the major component of the QRS complex
- Pseudo-infarction pattern can be seen in up to 70% of patients – due to negatively deflected delta waves in the inferior / anterior leads ("pseudo-Q waves"), or as a prominent R wave in V1-3 (mimicking posterior infarction).



The features of pre-excitation may be subtle, or present only intermittently. Pre-excitation may be more pronounced with increased vagal tone e.g. during Valsalva manoeuvres, or with AV blockade e.g. drug therapy.

WPW may be described as type A or B.

- Type A: ***positive delta wave*** in all precordial leads with R/S > 1 in V_1
- Type B: ***negative delta wave*** in leads V_1 and V_2

In patients with retrograde-only accessory conduction all antegrade conduction occurs via the AV node, thus no features of WPW are seen on the

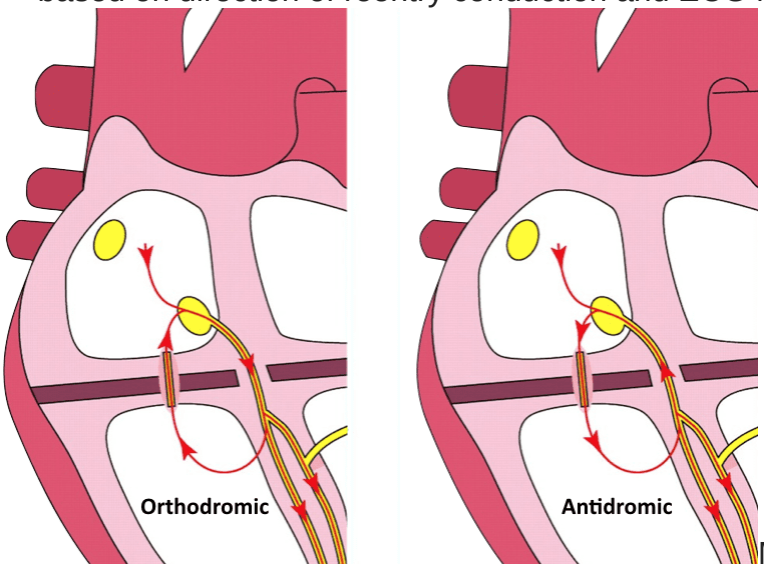
ECG in sinus rhythm (as no pre-excitation occurs). This is termed a “concealed pathway”.

Patients with a concealed pathway can experience tachyarrythmias as the pathway can still form part of a re-entry circuit

Atrioventricular Reentry Tachycardias (AVRT)

AVRT is a form of paroxysmal supraventricular tachycardia. A reentry circuit is formed by the normal conduction system and the accessory pathway resulting in circus movement.

- During tachyarrythmias the features of pre-excitation are lost as the accessory pathway forms part of the reentry circuit.
- AVRT often triggered by premature atrial or premature ventricular beats.
- AVRT are further divided in to **orthodromic** or **antidromic** conduction based on direction of reentry conduction and ECG morphology.



Mechanisms for orthodromic (left) and antidromic (right) atrioventricular re-entrant tachycardia

AVRT with Orthodromic Conduction

In orthodromic AVRT anterograde conduction occurs via the AV node with retrograde conduction occurring via the accessory pathway. This can occur in patients with a concealed pathway.

ECG features of AVRT with orthodromic conduction are:

- Rate usually 200 – 300 bpm
- P waves may be buried in QRS complex or retrograde
- QRS Complex usually <120 ms unless pre-existing bundle branch block, or rate-related aberrant conduction
- QRS Alternans – phasic variation in QRS amplitude associated with AVNRT and AVRT, distinguished from electrical alternans by a normal QRS amplitude
- T wave inversion common
- ST segment depression

Treatment of orthodromic AVRT

- Treatment of AVRT is based on the presence of haemodynamic instability e.g. hypotension, altered mental state, or pulmonary oedema.
- In patients who are haemodynamically stable vagal manoeuvres may be successful, followed by adenosine or calcium-channel blockers, and DC cardioversion may be considered if non-repsonsive to medical therapy.
- In a haemodynamically unstable patient urgent synchronised DC cardioversion is required.

AVRT with Antidromic Conduction

In antidromic AVRT anterograde conduction occurs via the accessory pathway with retrograde conduction via the AV node. Much less common than orthodromic AVRT occurring in ~5% of patients with WPW.

ECG features of AVRT with antidromic conduction are:

- Rate usually 200 – 300 bpm.
- Wide QRS complexes due to abnormal ventricular depolarisation via accessory pathway.

Treatment of antidromic AVRT

- AVRT with antidromic conduction results in a wide complex tachycardia which may be mistaken for Ventricular Tachycardia.
- For discussion on differentiating wide complex tachycardias see here, here, and here.
- Stable patients may respond to drug therapy including amiodarone, procainamide or ibutilide, but may require DC cardioversion
- In a haemodynamically unstable patient urgent synchronised DC cardioversion is required.
- If in doubt treat as VT

Atrial Fibrillation & Atrial Flutter in WPW

- Atrial fibrillation can occur in up to 20% of patients with WPW.
- Atrial flutter can occur in up to 7% of patients with WPW.
- The accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node.
- Rapid ventricular rates may result in degeneration to VT or VF.

ECG features of Atrial Fibrillation in WPW are:

- Rate > 200 bpm
- Irregular rhythm
- Wide QRS complexes due to abnormal ventricular depolarisation via accessory pathway
- QRS Complexes change in shape and morphology
- Axis remains stable unlike Polymorphic VT

Atrial Flutter results in the same features as AF in WPW except the rhythm is regular and may be mistaken for VT.

Treatment of AF with WPW

- Treatment with AV nodal blocking drugs e.g. adenosine, calcium-channel blockers, beta-blockers may increase conduction via the accessory

pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF

- In a haemodynamically unstable patient urgent synchronised DC cardioversion is required.
- Medical treatment options in a stable patient include procainamide or ibutilide, although DC cardioversion may be preferred.

CASE TWO

A 23yo mane has been found collapsed on his bedroom floor by friends. He was last seen well over 48hours ago while at a party. He has been transported into the ED with the Ambulance service. He has received no pre-hospital treatment so far.

Vitals: HR 40, BP 100, 34.5 C, BGL 4.5, GCS 12 (M5), 92% o/a, RR 20

1. **Discuss important further history and information you would like?**

- Background medical history if able
  - Concerto/records
  - Friends/family
  - Scene information from ambulance crew
  - Any mental health hx
  - Tox history
  - Past medical history
  - Medications

- Information surrounding party
  - Drug and alcohol use
  - Mental state
  - Trauma

He is placed in the Resuscitation bay. There are no external signs of trauma, and he is calmly agitated remaining settled if not stimulated.

2. **What are your differential diagnoses?**

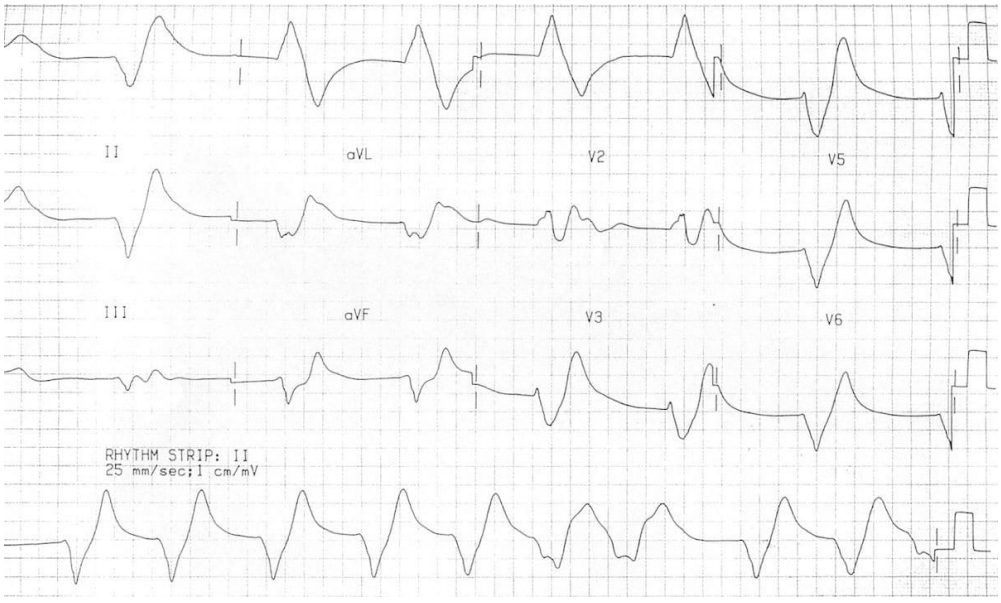
- Broad range
  - Medical event CNS, infection
  - Trauma
  - Toxicology
  - Environmental

3. **What investigations do you want at this stage?**

- Bedside
  - ECG (bradycardia)
  - BGL
  - MSU (MC+S, colour ? myoglobinuria)
- Labs
  - VBG – electrolytes, metabolic state, perfusion
  - Septic screen
  - TFTs, cortisol
- Radiology
  - CXR
  - CT head

4. **Below is his VBG result and ECG. Describe and discuss findings and concerns?**

pH 7.06, PCO2 4, HCO3 16, Na 129, K 7.2, AG 22, Lactate 4, Gluc 3.5



Severe mixed metabolic acidaemia, severe life threatening hyperkalaemia, raised anion gap, hypoglycaemia

Broad complex bradycardia, no p waves likely due to hyperkalaemia

5. **Outline your management for this man?**

- Supportive cares
  - Maintain airway, oxygenation as required
  - Thermal (external warming measures)
  - Correct hypoglycaemia
  - U/O with IDC
- Treat hyperkalaemia
  - Stabilise cardiac membrane Ca Gluconate 20ml (2g) or Ca chloride (1g)
  - Drive K into cells with insulin and dextrose, B agonists (?)
  - Eliminate K with IVF if hypovolaemia, Fruesmide if hypervolaemia, dialysis if severe/refractory/renal failure or oliguria
  - Sodium bicarbonate indicated if unstable, renal failure, peri arrest/arrest in the setting of acidaemia.
- Determine cause of hyperkalaemia
- Consider cover for sepsis
- Consider antidotes as appropriate

Below are his initial blood results

Cr 420, Na 128, K 6.9, CK > 50000, AST 320, ALT 290, Bili 35
Hb 175, WBC 22
INR 1.8
TFT normal, cortisol 300

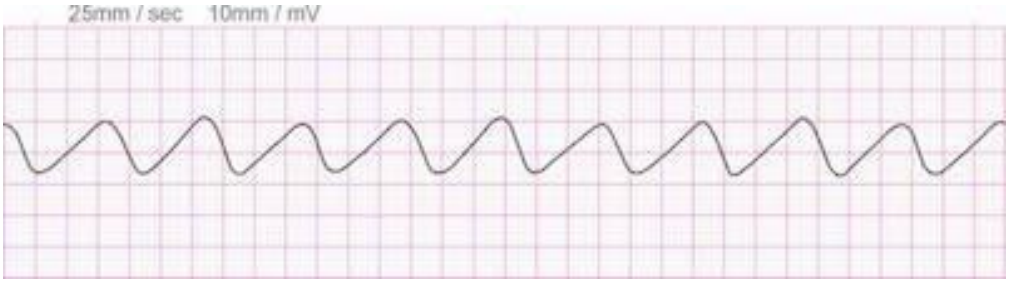
6. **Discuss the potential causes for his hyperkalaemia?**

- **Wide potential cause but substance use or toxicologic cause likely, with probable renal failure stemming from prolonged period on floor, dehydration and resulting rhabdomyolysis.**

- **Other causes**

1. **Medications:** ACEi, Potassium sparing diuretics, B-Blockers, NSAIDs, Trimethoprim (Septra) and Non-prescription salt substitutes
2. **Renal Failure**
3. **Cell death:** Secondary to rhabdomyolisis, massive transfusion, crush or burn injuries.
4. **Acidosis:** Consider Addisons crisis, primary adrenal insufficiency and DKA.
5. **Pseudohyperkalaemia (haemolysis)**

7. **He deteriorates despite receiving treatment 1L saline, 20ml calcium gluconate, 10IU actrapid + 100ml DW50. His current ECG and vitals are below, outline your management?**



- BP unrecordable, no palpable pulse, agonal breaths

- **Hyperkalemia in Cardiac Arrest**
- Based on the principles of treatment and indications discussed above, our experts recommend the following approach to suspected hyperkalemia (based on patient history and rhythm strip) or confirmed hyperkalemia (based on a point of care blood gas) in cardiac arrest in addition to usual ACLS measures:

- **Push 1 amp calcium chloride in well running peripheral IV or central line and repeat until the QRS is <100ms**

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- **Epinephrine 5-20 mcg q2-5 minutes (shifts K intracellularly)**

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- **Sodium Bicarbonate 1 amp IV (if suspect severe acidosis)**

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- **Bolus IV NS**

↓

- **Shift potassium with Insulin and Glucose followed by B-agonist**

↓

- **Dialysis**

- **Rebound Hyperkalemia**
- In cases of cardiac arrest due to hyperkalemia, perform CPR until the hyperkalemia is corrected. This may be a much longer time than usual. When ROSC is achieved, it will be primarily due to the effects of calcium rather than decreased potassium levels. The effect of calcium can last 20-30min. Since the stabilizing effects of calcium will wear off, you must promptly work on shifting the potassium and enhancing its elimination as described above. Consider repeating the calcium bolus if there are any worsening ECG changes. Repeat serial potassium measurements to monitor for rebound hyperkalemia, which occurs more often than we'd like.

- ***PEARL:*** *the patient in cardiac arrest with hyperkalemia should not be pronounced dead until their potassium level is normalized*

CASE THREE

A 17yo woman has been found collapsed in the streets. She was found with a plastic bag with strong smelling chemical odour next to her. She has been transported to the ED. Below are her vitals. She is floppy and has reduced tone and has shallow breathing.

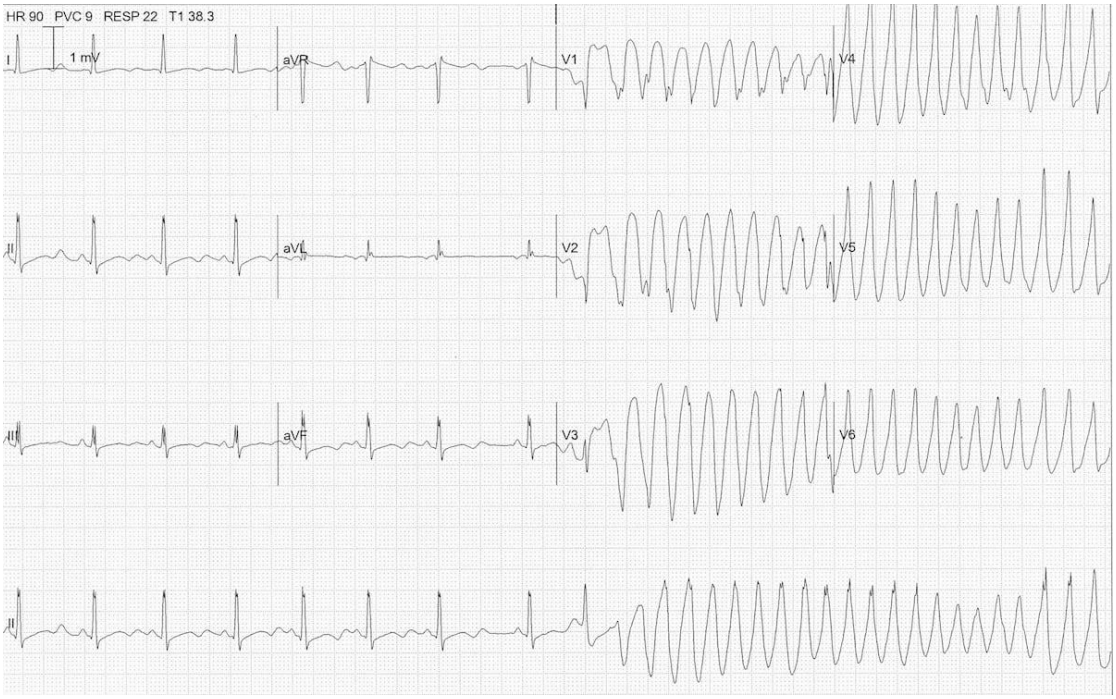
HR 100, BP 80, 35 C, RR 24, Sats 92%, BGL 5, GCS 10 (M5)

1. **What is your concern for this woman?**

- Probably solvent induced presentation
  - Concern for metabolic disturbance in particular hypokalaemia and arrhythmia and hypokalaemic paralysis risk impaired ventilation.
  - Consider seizures
  - Potential traumatic injury
  - Other substance use
  - May have other ingestions

2. **Below is her VBG result and initial ECG. Discuss your findings?**

pH 7.1, PCO2 3.5 Na 128, K 1.2, HCO3 12, AG 14, Lactate 2.2, Gluc 5.2



- **Severe normal anion gap metabolic acidaemia, with severe hypokalaemia**
- **Prolonged QT sinus rhythm with run of polymorphic VT (Torsades)**

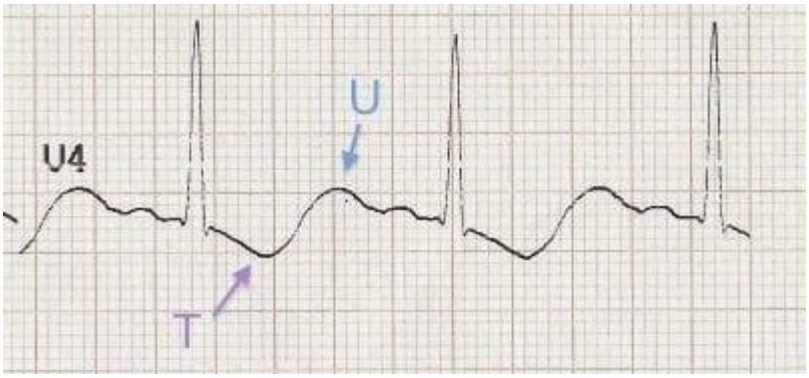
3. **Discuss the changes in the ECG you expect with hypokalaemia?**

ECG changes when K+ < 2.7 mmol/l

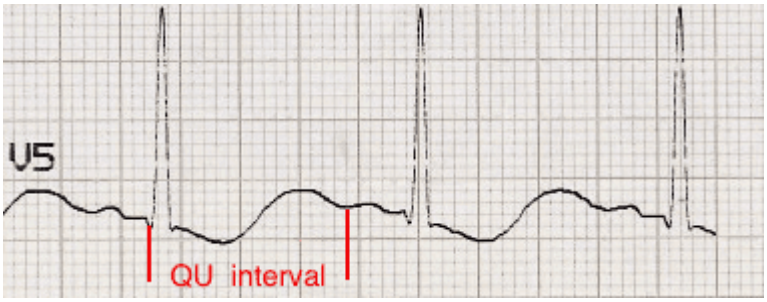
- Increased amplitude and width of the P wave
- Prolongation of the PR interval
- T wave flattening and inversion
- ST depression
- Prominent U waves (best seen in the precordial leads)
- *Apparent* long QT interval due to fusion of the T and U waves (= long QU interval)

With worsening hypokalaemia…

- Frequent supraventricular and ventricular ectopics
- Supraventricular tachyarrhythmias: AF, atrial flutter, atrial tachycardia
- Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes



T wave inversion and prominent U waves in hypokalaemia



Long QU interval in hypokalaemia

4. **What is the likely cause for her metabolic disturbance, and how are you going to manage her?**

- Likely Toluene toxicity
  - RTA1 distal tubular acidosis typically with hyperchloraemic, hypokalaemic, normal AG metabolic acidosis.
  - Risk of hypokalaemic paralysis, Rhabdomyolysis from hypokalaemia and hypophosphataemia, other organ injury
  - Altered mental status common

- Arrhythmia due to low K

- Urgent replacement of Magnesium and K required
  - 10-20mmol Mg SO4 (2 amps)
  - Stable 20mmol K over 60 minutes
  - 10-20mmol diluted over 15-20min if recurrent arrhythmia then review (Preferable via central access, large bore)
  - In refractory hypokalaemia arrest VT/VF/asytole bolus 20mmol over 2-3minutes and consider repeating if persistent refractory arrest/ventricular arrhythmia.

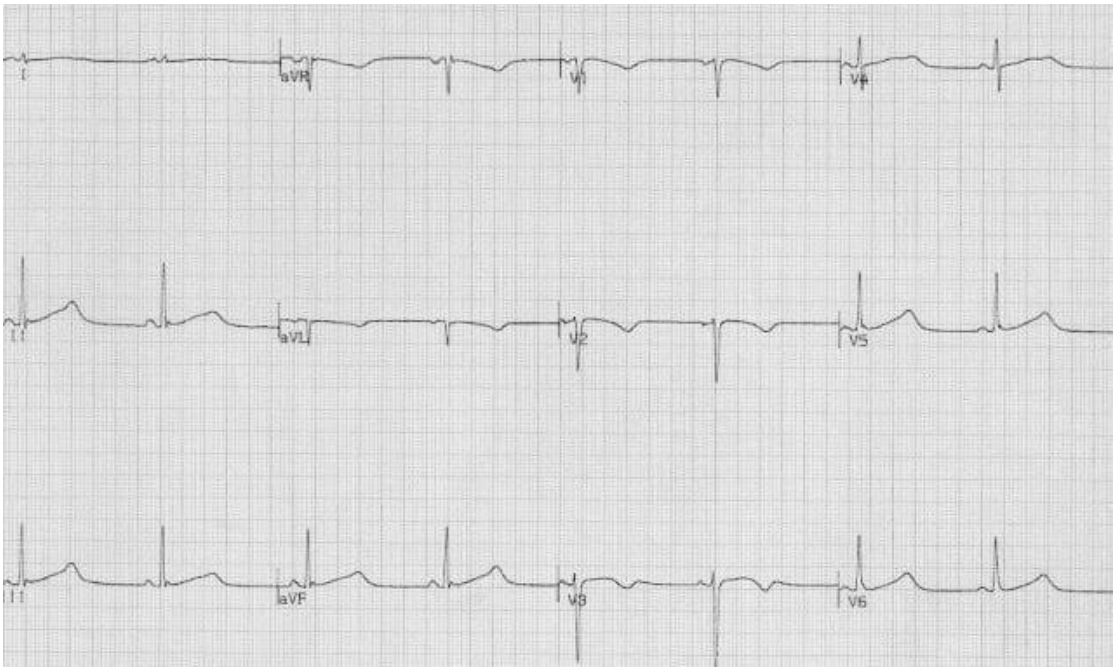
CASE FOUR

22yo male presents with two episodes of witnessed syncope during the morning, with brief LOC each time and collapse to the floor. He has no history of chest pain, SOB, and no family history of note. He denies and seizure history. He is on Erythromycin for a URTI started 5 days ago; he is not on any other medication. He has had some diarrhoea since starting the antibiotic.

1. **What are your differential causes for his syncope?**

- Concerning syncope history
  - Need to consider Cardiac cause
  - On macrolide may prolong QT interval
  - Possible congenital cardiac cause
  - Potential K/Mg depletion from GIT loss

2. **Below is his baseline ECG, interpret findings?**



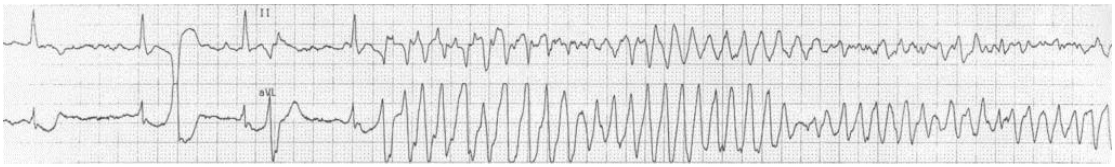
Sinus rhythm, prolonged QT interval, TWI anterior

3. **Outline potential causes of long QT intervals?**

Causes of a prolonged QTc (>440ms)

- Hypokalaemia
- Hypomagnesaemia
- Hypocalcaemia
- Hypothermia
- Myocardial ischemia
- ROSC Post-cardiac arrest
- Raised intracranial pressure
- Congenital long QT syndrome
- Medications/Drugs

4. **The patient suddenly loses consciousness while on his bed in the ED monitored area. He regains consciousness but his ECG shows an abnormal rhythm, interpret and outline your treatment?**



Torsades de Pointes

- Mg 1-2g IV slow bolus
- Attach defib – persistent or unstable immediate cardioversion (may need unsynchronised)
- Look for cause
  - Correct low K/Mg/Ca
  - Remove ppt drugs
  - Avoid QT prolonging drugs
  - Review history for any triggers (medications/overdose)

5. **His electrolytes are normal along with his VBG. Despite initial bolus of Mg 2g he has a further episode of above rhythm with LOC. Discuss your approach?**

- ACLS approach
- Further Mg bolus and DC cardioversion
- Prevention – ensure Mg level 1.5-2mmol/L (rpt bolus/infusion)
- Refractory – lidocaine 1.5mg/kg bolus (+/- infusion)
- Prevention with increasing HR for acquired prolonged QT
  - Increase rate (chemical or electrical)
  - Relatively contraindicated in congenital prolonged QT syndromes

Occasional patients will have recurrent episodes of torsades ("Torsades storm"). Each individual episode may be treated with magnesium or defibrillation, if needed

(Treatment step #1 above). However, additional therapies are required to stop recurrence and end the storm.

re-load magnesium PRN

- Recurrent torsades may reflects inadequate magnesium dosing (e.g. patient is bolused with 2-4 grams, without an infusion). The first step when managing recurrent torsades is therefore to ensure that the patient has truly received an adequate dose of magnesium.
- If the patient was bolused with magnesium a few hours ago without an infusion, re-load with 2-4 grams IV immediately (8-16 mM).
- If the patient is a candidate for magnesium infusion (GFR >30 ml/hr), this should be ordered.
- If the patient has renal failure and has already received 4-6 grams of magnesium (16-24 mM), then check magnesium levels and ensure that a high level is achieved. Note that a therapeutic level for torsades is roughly 3.5-5 mg/dL (1.5-2 mM) – not a "normal" level.
- More on magnesium above (Treatment step #2).

optimize the potassium

- The target potassium level here is probably >4.5-4.7 mEq/L.
- Giving potassium alone is unlikely to work, but it might help a bit.

speed up the heart

- Speeding up the heart rate will decrease the QT interval and reduce the risk of acquired torsades (but it may be contraindicated in some forms of congenital torsades).
- The usefulness of chronotropy depends on the patient's **baseline heart rate**.
    - Chronotropy is most beneficial for patients starting out with bradycardia.
    - If the patient is already significantly tachycardic, chronotropy is unlikely to provide benefit. The usual target heart rate is 100-110 b/m, but occasionally heart rates up to 140 b/m may be needed.[2] There's no high-quality data on this.
- **Medical chronotropy** is generally the easiest & fastest way to stabilize the patient. The ideal chronotrope depends on the patient's hemodynamics and baseline blood pressure:
    - Baseline severe hypotension: epinephrine infusion.
    - Baseline normotension or mild hypotension: dobutamine or isoproterenol infusion.
- **Electrical chronotropy** may be used if medical chronotropy fails:
    - Transcutaneous pacing may work, but this is painful for conscious patients.
    - Transvenous pacing is more comfortable, but this is more invasive and takes a bit longer to achieve.
    - Patients with a pacemaker may have the device rate increased.

lidocaine

- Lidocaine is the preferred anti-arrhythmic drug for torsades, although there isn't a ton of evidence supporting its use.
    - **Do not use amiodarone, procainamide, beta-blockers, or most other antiarrhythmics.** Most of these will stretch out the QT interval even further! Beta-blockers will slow down the heart rate, increasing the risk of torsades.
- Start with a loading dose of 1-1.5 mg/kg followed by a 1 mg/min infusion. For recurrent arrhythmias, re-load with another 1 mg/kg bolus and increase the maintenance infusion.

Consider an alternative diagnosis

- Torsades is generally fairly easy to control with a combination of high-dose magnesium, heart rate augmentation, and occasionally some lidocaine. Failure to respond to these interventions suggests an alternative diagnosis (e.g. polymorphic VT due to ischemia or catecholaminergic ventricular tachycardia).

CASE FIVE

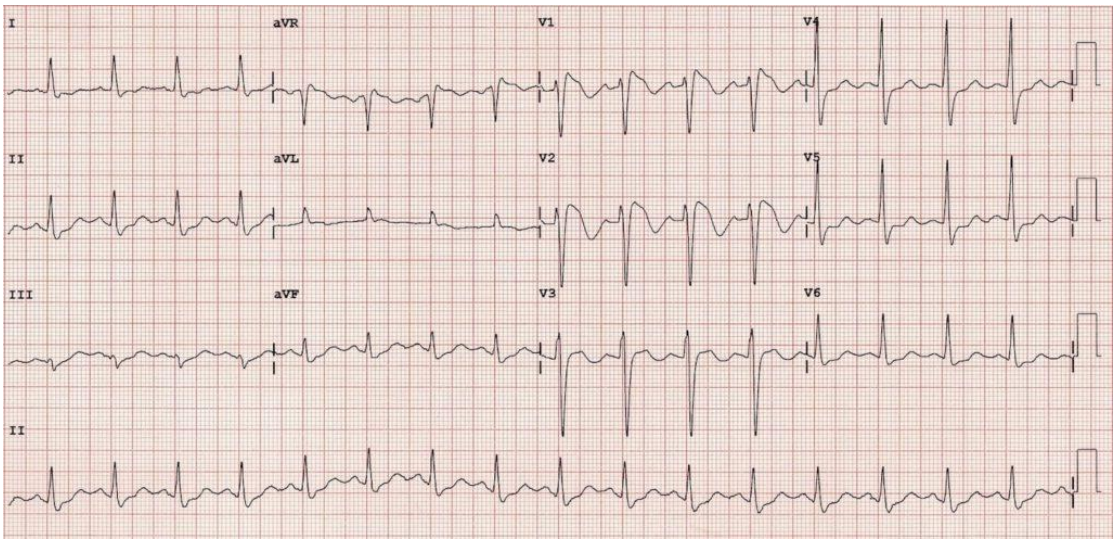
30 yo man presents to ED with URTI symptoms and a fever. Prior to presentation he had an apparent collapse or syncope with rapid return to baseline. He is otherwise well.

Obs 38.8, HR 120, BP 110, Sats 98%, GCS 15

1. What further history would you ask, what are your differentials, and what investigations do you want?

- History
  - Nature of collapse, triggers, pre syncopal symptoms,
  - Prior similar history
  - Medications or drugs
  - Fhx of syncope/collapse
- Investigations
  - ECG
  - Septic bloods +/- CXR/MSU
- Differentials
  - Broad causes

2. Below is his ECG please discuss findings and relevance to clinical picture?



Brugada type 1 coved ST elevation > 2mm with negative inverted T waves
Potential risk for lethal ventricular dysrhythmias

Mandates admission and further investigation, on monitor

3. What is Brugada, discuss the different morphologies and criteria for diagnosis?

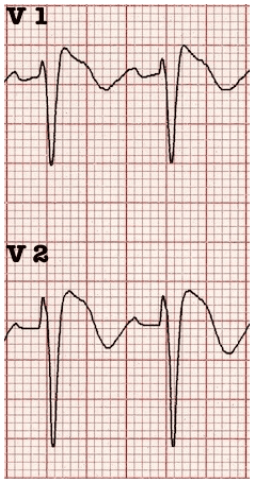
- Na channelopathy, 50% spontaneous, autosomal dominant inheritance
- ECG changes can be transient with Brugada syndrome and can also be unmasked or augmented by multiple factors:

- Fever
- Ischaemia
- Multiple Drugs
  - Sodium channel blockers eg: Flecainide, Propafenone
  - Calcium channel blockers
  - Alpha agonists
  - Beta Blockers
  - Nitrates
  - Cholinergic stimulation
  - Cocaine
  - Alcohol
- Hypokalaemia
- Hypothermia
- Post DC cardioversion

➢ ***Diagnostic Criteria***

Type 1

- Coved ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave.
- This is the only ECG abnormality that is ***potentially*** diagnostic.
- It is often referred to as **Brugada sign**.



This ECG abnormality ***must*** be associated with one of the following ***clinical criteria*** to make the diagnosis:

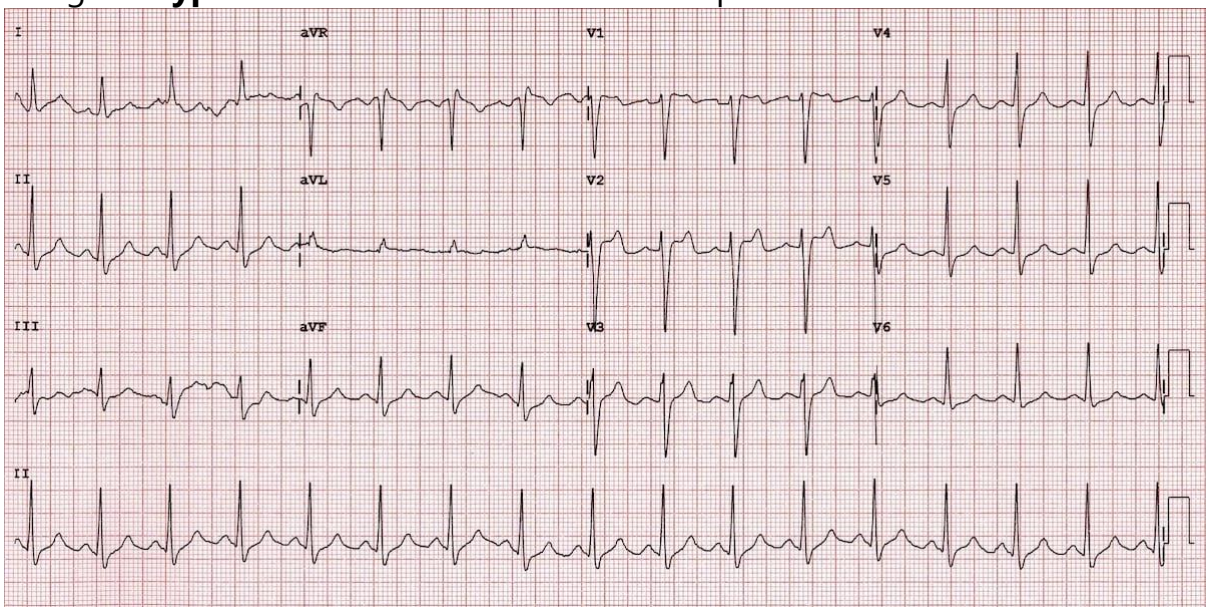
- Documented ventricular fibrillation (VF) or polymorphic ventricular tachycardia (VT).

- Family history of sudden cardiac death at <45 years old .
- Coved-type ECGs in family members.
- Inducibility of VT with programmed electrical stimulation .
- Syncope.
- Nocturnal agonal respiration.

The other two types of Brugada are non-diagnostic but possibly warrant further investigation (see discussion below).

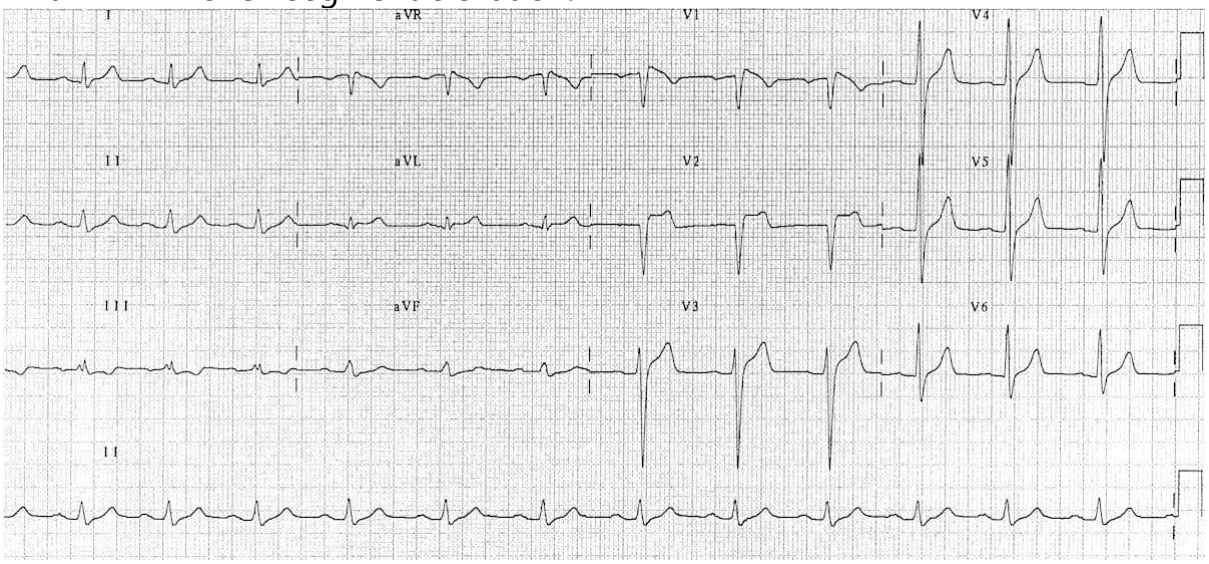
Type 2

- Brugada **Type 2** has >2mm of saddleback shaped ST elevation.



Type 3

- Brugada **type 3:** can be the morphology of either type 1 or type 2, but with <2mm of ST segment elevation.



Management

- The only proven therapy is an implantable cardioverter – defibrillator (ICD).

- Undiagnosed, Brugada syndrome has been estimated to have a mortality of 10% per year. Does this mean that a diagnosis in ED mandates admission? Probably yes for all type 1 patients if they present with suggestive clinical criteria.

- It may be appropriate for risk stratification on an outpatient basis with an electrophysiology study (EPS) to see if the patient has inducible ventricular tachycardia (VT) or fibrillation (VF)in the following settings:

- Asymptomatic patients with a type 1 ECG pattern.
- All type 2 + 3 ECG patterns.

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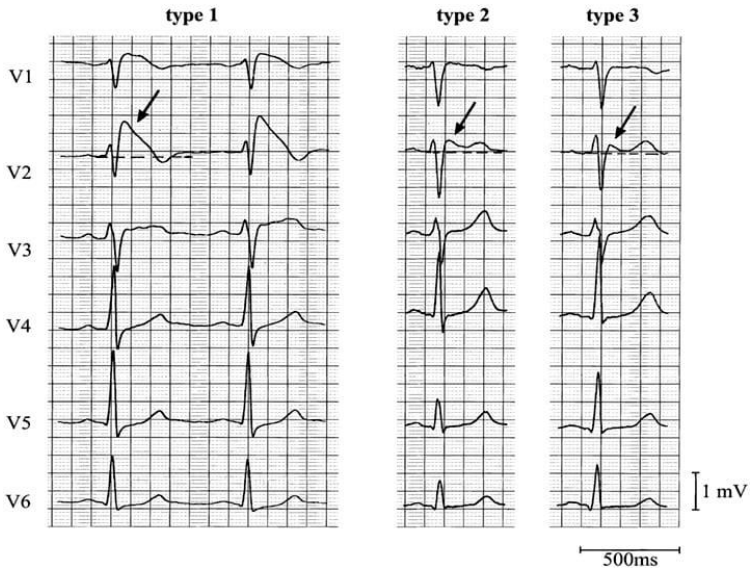
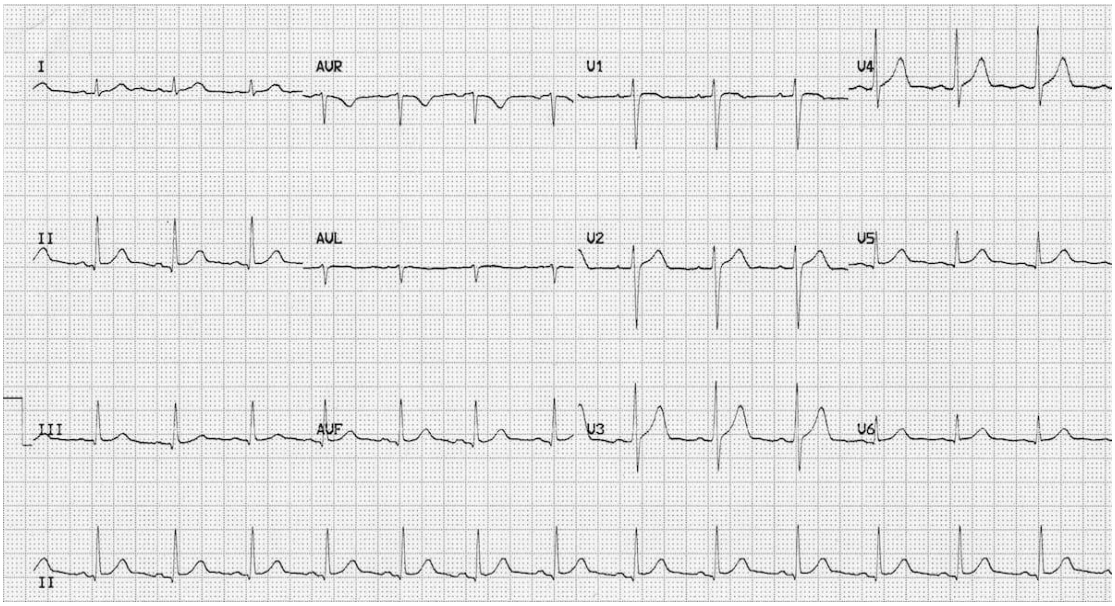


CASE SIX

A 35 year old Fijian Indian man presents at 2am with chest pain which last 10minutes, resolving prior to arrival. He has no personal medical history, and takes no medication. His father had an MI at the age of 46yrs old.

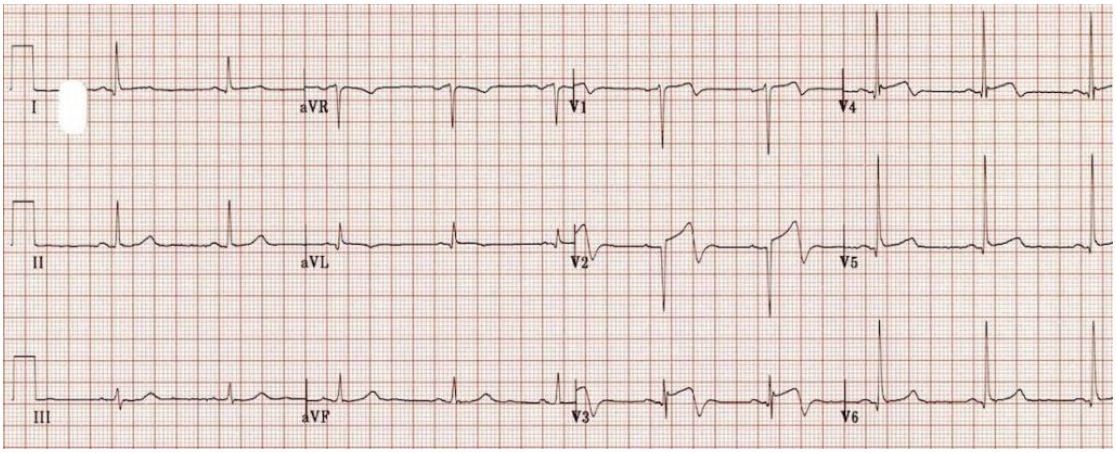
1. Discuss his ECG below and your investigation?



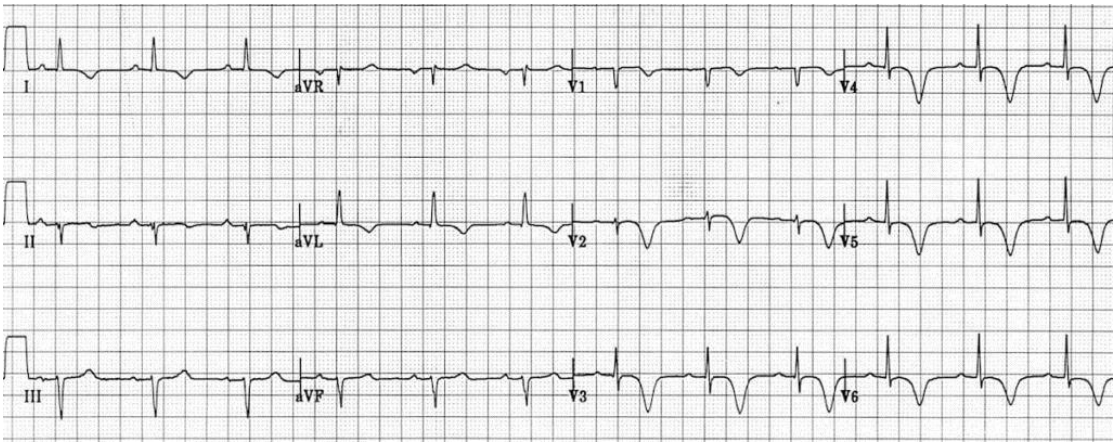
- NSR
- Consider PTX, Cardiac, PE, Infective, MSK

2. He develops further brief chest pain in ED resolving within 2-3 minutes, his repeat ECG's are below. Discuss your potential diagnosis?

- 1



- 2



- Biphasic T wave with STE in precordial leads followed by deep TWI in second ECG concerning for likely proximal LAD lesion and high risk of developing anterior/Lat STEMI

3. **His symptoms resolved and his ECG is now normalised, what would your next management be for this man?**

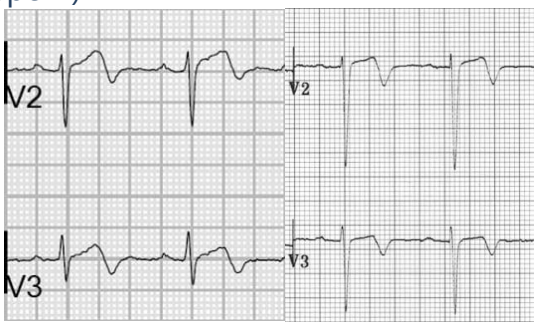
- Urgent discussion with cardiology for PCI

4. Discuss the patterns of Wellens Syndrome and diagnostic criteria?

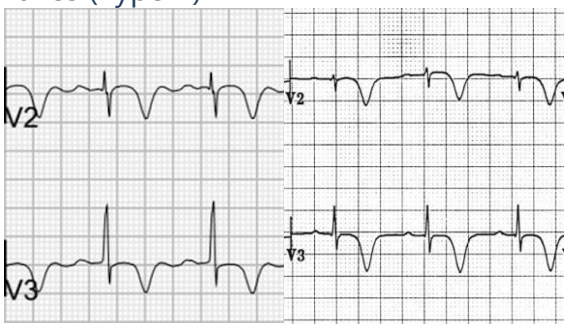
There are *two patterns* of T-wave abnormality in Wellens syndrome:

- **Type A** – Biphasic, with initial positivity and terminal negativity (25% of cases)
- **Type B** – Deeply and symmetrically inverted (75% of cases)

Biphasic T Waves (Type A)



Deeply Inverted T Waves (Type B)



Rhinehart et al (2002) describe the following diagnostic criteria for Wellens syndrome:

- Deeply-inverted or biphasic T waves in V2-3 (may extend to V1-6)
- Isoelectric or minimally-elevated ST segment (< 1mm)
- No precordial Q waves
- Preserved precordial R wave progression
- Recent history of angina
- ECG pattern present in pain-free state
- Normal or slightly elevated serum cardiac markers