CASE ONE

32 yo man sudden collapse at work. He is BIBA and will be in the department in 5 minutes with GCS of 10, RR 40, HR 160, BP 80.

History from the ambulance paramedic of sudden collapse at work. Immediate bystander CPR for 10 minutes before ambulance arrival with AED recommending no shock. On review by paramedics spontaneous breathing, atrial fibrillation 160, hypotensive.

Initial Observations:

HR 160, RR 32, BP 80, GCS 10 (M 5), 38 temp, BGL 8, Sats 88% on 15L mask

1. Discuss you immediate management and likely differentials?

- Broad differential : ACS, PE, Dissection, CNS, Tox
- Breathing and Circulation :
    - Help now
    - ? BMV ? NIV and Prep for need to intubate
    - IVF bolus +/- pressor
- Determine cause for shock
    - ECG/VBG/CXR/POCUS
    - History if available
    - PMhx

Further history reveals that he recently underwent an ORIF of a right ankle fracture and came out of cast 1 week ago. You decide to do a bedside USS to help determine the cause of shock.

2. Interpret the USS findings?

- Distended IVC hepatic veins
- Large RV + D sign and deviated IVS suggesting high R sided pressures

➢ Massive PE - hypotension with Rv strain

3. How are you going to manage this man and discuss the thrombolysis in PE?

- Expert advice consensus decision ED/ICU/Resp SMO to thrombolyse
    - Follow local guideline - remember contraindications
    - This man needs thrombolysis ASAP

Indications:

➢ Massive PE

- Haemodynamic instability
- BP <90mmHg for >15min
- BP fall by >40mmHg
- Cardiogenic shock

➢ Cardiac arrest secondary to PE

➢ Submassive PE

Normotensive patients with extensive clot burden on CT (saddle embolus or involving main pulmonary artery), consider if one or more of the following; RV strain plus troponin rise
RV strain on CTPA alone is probably insufficient.

- Acute RV strain on CTPA (marked enlargement of RV with flattening of the interventricular septum) without evidence of chronic pulmonary hypertension and organised clot
- Severe acute RV dysfunction on echocardiogram (echocardiogram not essential if other indications for thrombolysis already present)
- Elevated troponin
- Elevated BNP
- Significant hypoxaemia and large A-a gradient
- Free floating RA or RV thrombus
- Patients with a PFO

- Tenectaplase max 50mg (weight based) as bolus followed by heparin infusion (delayed if already has had enoxaparin)

Before he receives thrombolysis he becomes severely hypoxic and has a PEA arrest.

4. Discuss your management?

- ACLS - adrenaline
- Secure airway with minimum interruption of CPR (muscle relaxant)
- Bolus tenectaplase weight based max 50mg ASAP and continue CPR
    - Continue effective CPR prolonged after 20-30min consider second bolus tenectaplase
    - Prolonged resuscitation

- Ensure normothermia
- Correct hypoglycaemia and electrolytes
- Consider Sodum bicarbonate in setting of academia

You get ROSC after 30minutes. He remains shocked BP 70, HR 120, and saturations of 90% on 100% O2. He is intubated and ventilated.

5. Discuss options to manage his shock on going?

- Ensure adequate preload
- Pressor/inotrope
- Repeat echo ? Degree of dysfunction
- Resistant shock options ? ECMO/Bypass
- ? Embolectomy ? or further thrombolysis/targeted

CASE TWO

22 yo male presents to the ED with Fevers, chest pain and SOB for 24 hours. He has no medical history of note and takes no regular medications.

His initial observations are:

RR22, HR 110, BP 100, Temp 38, Sats 92% o/a, GCS 15

1. Discuss what further history would be important and your initial investigations?

- Details of clinical presentation
- Full medical history
  - Drug and alcohol, IVDU
  - Medications
  - Review of systems
- Travel history

➢ Initial investigations:

- ECG
- Cultures and septic screen
- MSU
- VBG
- CXR

On review you note track marks in his arms and feet. He admits to IV drug abuse. He has no localizing findings otherwise. His chest is clear and he has a soft abdomen but he does have a soft systolic murmur.

2. What are your concerns given this history?

➢ Staph aureus sepsis, coag negative staph, strep

- Possible Endocarditis
- Discitis/epidural abscess
- Lung abscess/pneumonia
- Other foci that may be occult

He develops worsening SOB and he is agitated. He is placed into resuscitation area.

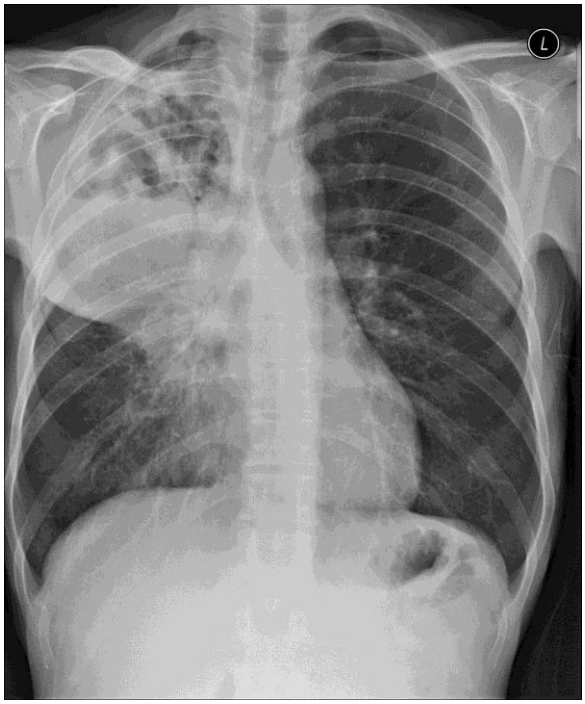
His observations are:

BP 70, HR 180, GCS 13 (M5), Sats 88% o/a, BGL 6

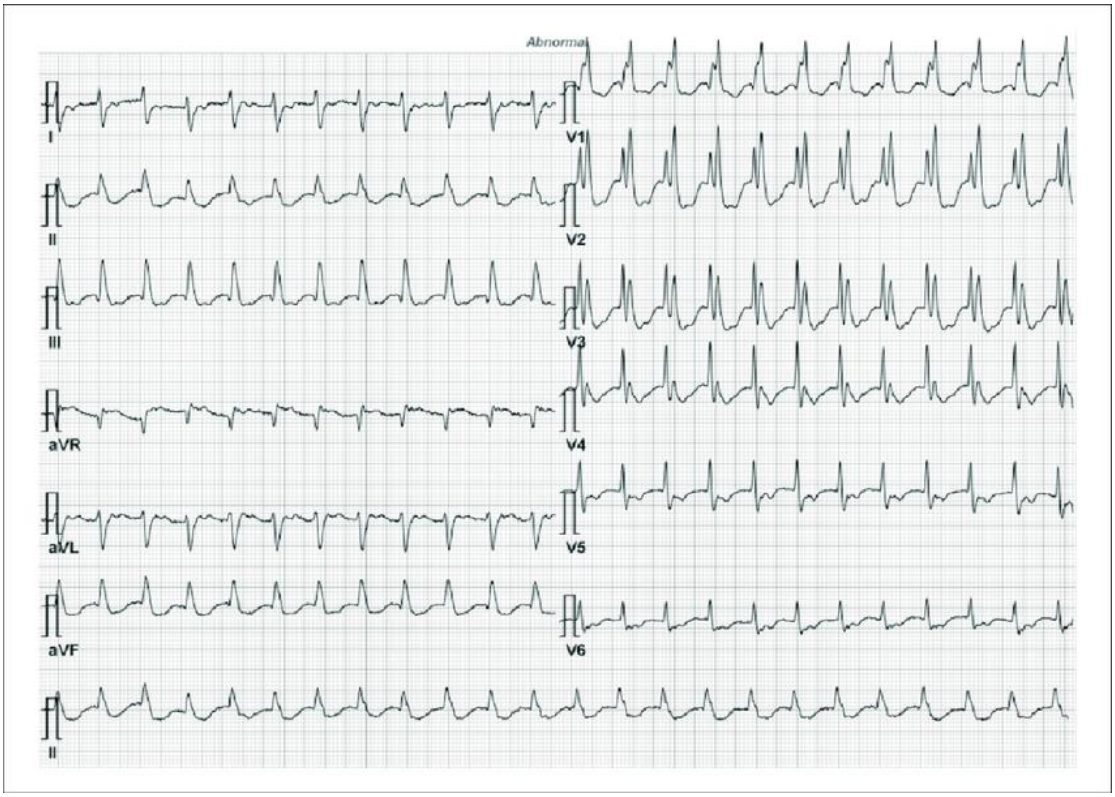
3. What are your concerns and how are you going to manage him?

- Shocked patient? septic shock most likely
- Get help , involve ICU early
- Severely tachycardic 180bpm – possible tachyarrhythmia needs ECG rpt
- High flow O2
- IVF bolus resuscitation
- Empiric broad spectrum Abs (consult ID) – MRSA cover
- CXR, repeat VBG

4. Discuss the ECG and CXR findings?



RUZ consolidation and fluid level: abscess BAD!



Tachycardia RBBB morphology, no P waves, likely junctional origin

5. His GCS is 10 (M5), BP is now 70 and HR still 180bpm despite 3L IVF. Discuss further management?

- Start pressors ideally noradrenaline
  - Can start with metaraminol as a bridge
  - ART line
  - CVL
- You have help !
- Prepare for possible need to intubate
  - Remember HOP KILLERS
  - Resuscitate before you intubate

- Consider echo/formal
- Cardioversion unlikely to be successful given underlying pathology

6. Despite 40mcg/min Noradrenaline, and total 4L IVF he remains hypotensive BP 70, HR 170, GCS still 10, with increasing O2 requirement. His beside echo image is shown. Discuss the echo and your further management?

- Severely impaired LV with mod dilation
  - ? acute secondary to sepsis
  - ? underlying cardiomyopathy due to IVDU
- Unlikely to tolerate further IVF
- Discuss with ICU and cardiology
  - Milrinone/Dobutamine ?
  - Avoid adrenaline probably given degree of tachycardia

He becomes increasingly drowsy and confused, with saturations of 80% on 15L O2. He is on high dose Noradrenaline with BP of 60 systolic.

7. How will you approach an RSI in this situation, discuss preparation, equipment, personnel, and RSI drugs?

- Experienced team
  - Delegated roles clearly and within comfort levels
- Airway plan clearly voiced and agreed on
- HOP KILLERS
  - Optimise resuscitation
  - NP O2, bridging with NIV, or BMV with PEEP if tolerated
  - ? Bicarb
  - Upright patient position optimise ventilation
- Have arrest drugs and defib available
- If agitated and difficulty pre oxygenating consider modified intubation
  - Small dose ketamine
  - NP O2
  - BMV
  - Delayed high dose paralysis
- Drugs for intubation
  - Low dose sedation
  - High dose paralysis

CASE THREE

24yo woman is brought into your resuscitation room by an ambulance. She has been found confused on the floor at home. According to family she has been unwell for a few weeks with some weight loss, diarrhoea, and more recently having fevers, and seemingly a bit confused in the last day. Her obs in the ambulance HR 140 sinus, BP 150/90, GCS 12 (M5), BGL 10, Sats 95% o/a, temp 38.

1. **What are your differential diagnoses and further history you want?**

- Sepsis- of any potential source
- Malignancy- hematologic possibly secondary infection
- Metabolic cause or endocrine : Diabetes, thyroid, adrenal
- Toxicological

- Full history

    - Recent hx of events
    - Travel and contacts
    - Past medical history
    - Social/drugs and alcohol
    - Trauma
    - Medications and allergies
    - Recent medical visits etc

2. **Shortly after arrival in the ED resus she deteriorates. Discuss your initial investigations and management?**

BP 180/100, HR 180, RR 30, Sats 90% o/a, GCS 10 (M5), BGL 10, 38.9

- High flow 02
- Emergency call get SMO help
- Empiric antibiotics – CNS cover ceftriaxone/vancomycin (MRSA)
- Consider steroids if concern for CNS pathology

- Investigations:

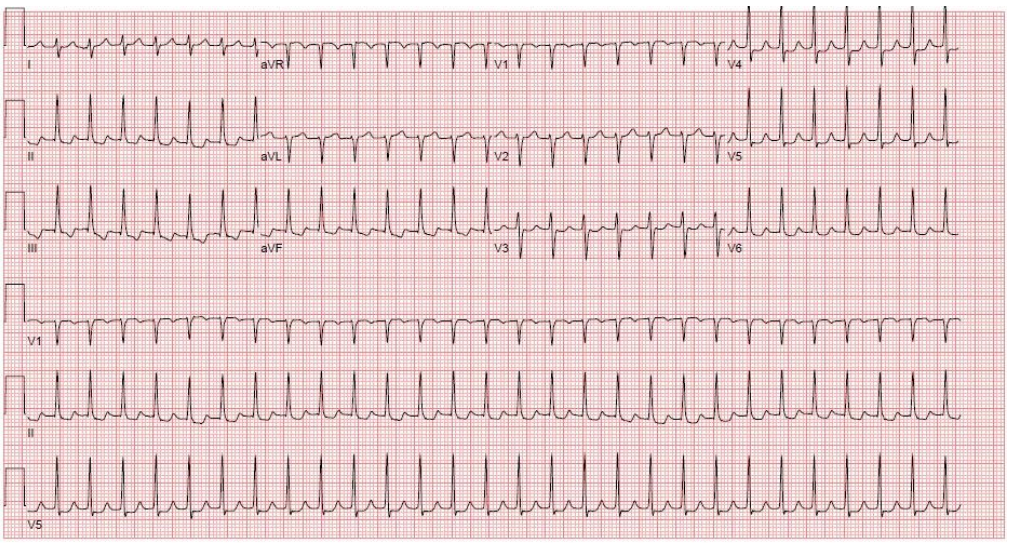
    - ECG ? tachyarrhythmia
    - VBG – pH, HCO3, lactate, Na, K
    - Bloods, cultures, TFTs, cortisol
    - IDC and urine
    - CXR
    - Bedside USS: volume status, pump status, lungs ? wet ? consolidation

A bedside USS is performed showing some LVH, normal LV size, with moderate LV impairment. The IVC is full, and there are symmetrical multiple B lines in both lung fields.

3. **Below is her VBG, ECG, Please interpret in context of the clinical presentation?**

VBG

pH 7.0
CO2 6
HCO3 14
Na 140
K 5
Gluc 9
Lactate 6.4
AG 22



- Metabolic AG lactic acidaemia (note CO2 – concerning decompensation)
- Narrow complex tacycardia with inferior ST depression ? ischaemic
- USS – wet lungs, euvolaemic or hyper, LVH suggest hypertension

Putting it together suggestive of high output state with SVT and LV failure/cardiogenic shock and pulmonary edema.

Her blood tests are available below:

WBC 20 , CRP 80, Cr 100,
TFT : TSH <0.2, T4 15 (8-22), T3 180
Cortisol 300 (random)

4. **What is your likely diagnosis and how are you going to manage this woman?**

- Hyperthyroid storm with high output cardiac failure
  - Medical emergency
  - Potential rapid deterioration and CV collapse

- Supportive cares
  - Manage airway and oxygenation
  - Urgent intervention to manage catecholamine effects
  - Temperature control
  - Antimicrobials
  - Fluid/volume management
- Aims to manage hyperthyroidism:
  - Block thyroid hormone release (PTU)
  - Block receptor effects of catecholamine surge (beta blockade)
  - Block peripheral conversion T4 to T3 (PTU, Hydrocortisone, propranolol)
  - Block mobilisation of stored hormone in thryroid (lugols iodine)
- Airway and breathing:
  - High flow O2 consider escalation NIV or RSI
  - Risk cardiac decompensation
  - Ideally start tx for hyperthyroidism and control hypertension/svt
- Hydrocortisone 200mg IV stat then regular dosing
  - Often secondary cortisol deficiency
  - Depression of the hypothalamic-pituitary axis commonly occurs in the setting of thyroid storm
  - Helps reduce acitivation of T4 to T3
- Beta blockade
  - Esmolol best option in this situation short ½ life, titrateable, B1 selective
  - Risk exacerbation failure so best avoid propranolol/metoprolol/labetalol
  - 50-200mcg/kg/min (can load with 50mcg/kg bolus) titrate to reasonable HR and BP
- PTU NG/PO/PR
  - Inhibit thyroid peroxidase, an enzyme involved in the production of T3 and T4 through the iodination of tyrosine residues on thyroglobulin
  - PTU preferred to (carbimazole/methimazole) as reduces T4-T3 conversion
  - Loading dose: 600 to 1000 mg
  - Maintenance dose: 200 to 250 mg q4

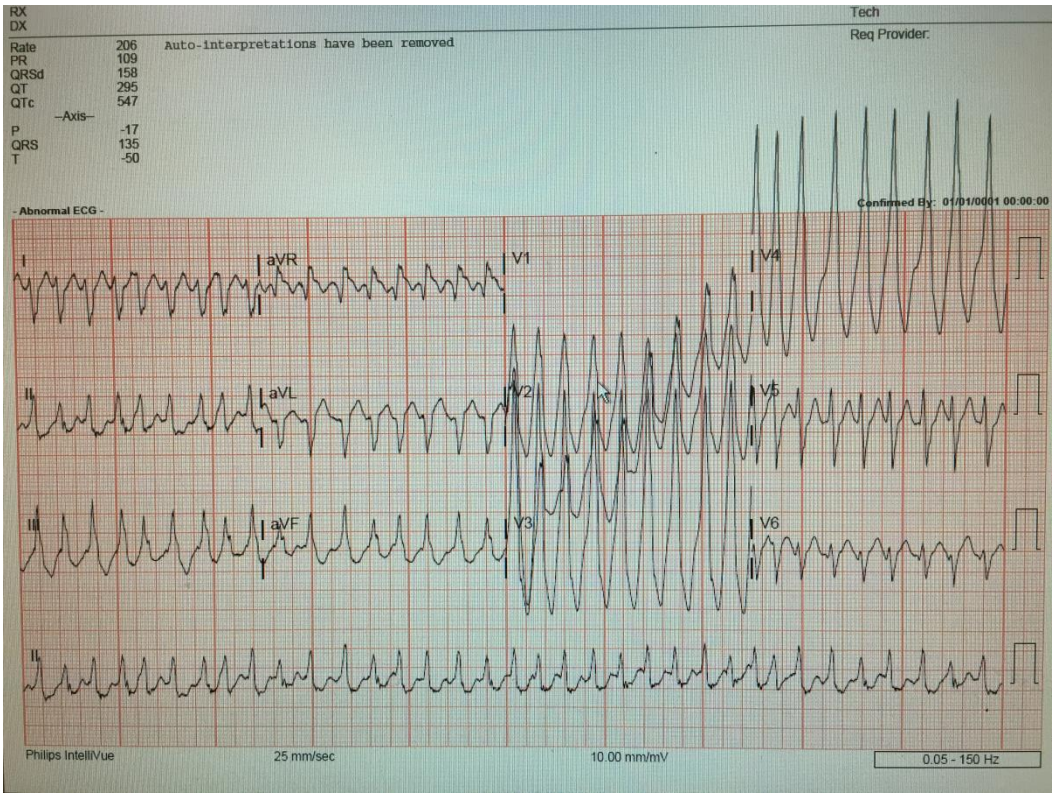
- Lugols iodine
  - prevent the release of pre-formed thyroid hormone from the thyroid gland
  - 8 drops PO, NG, or PR q6h
  - Should be delayed 1-2hrs post PTU to avoid exacerbation of thyroid hormone synthesis (iodine can be used a substrate by thyroid)

CASE FOUR

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

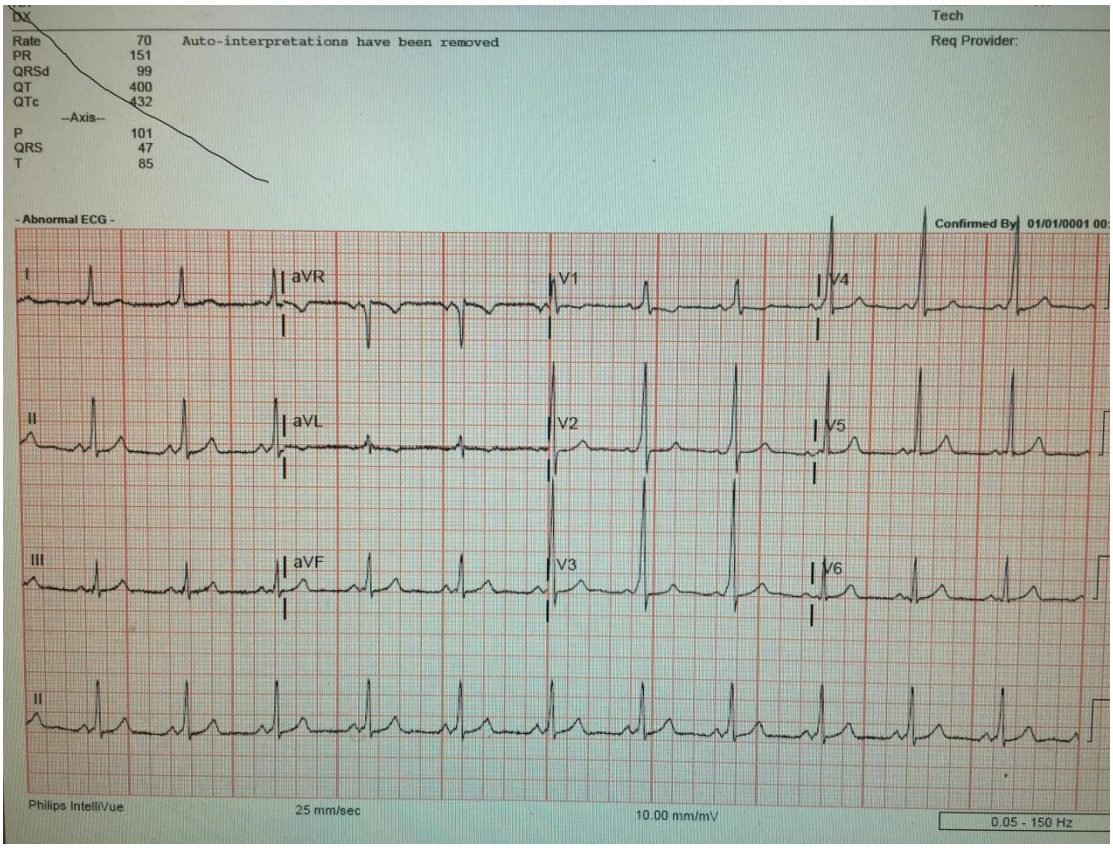
- Very rapid irregularly irregular tachycardia (rates may approach 300 beats/min) with wide QRS complexes that <u>vary in morphology</u>
- Often misdiagnosed as SVT, VT or atrial fibrillation with BBB
- Misdiagnosis and treatment with AVN blockers can be deadly!
- Treat with **preferably electrical cardioversion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

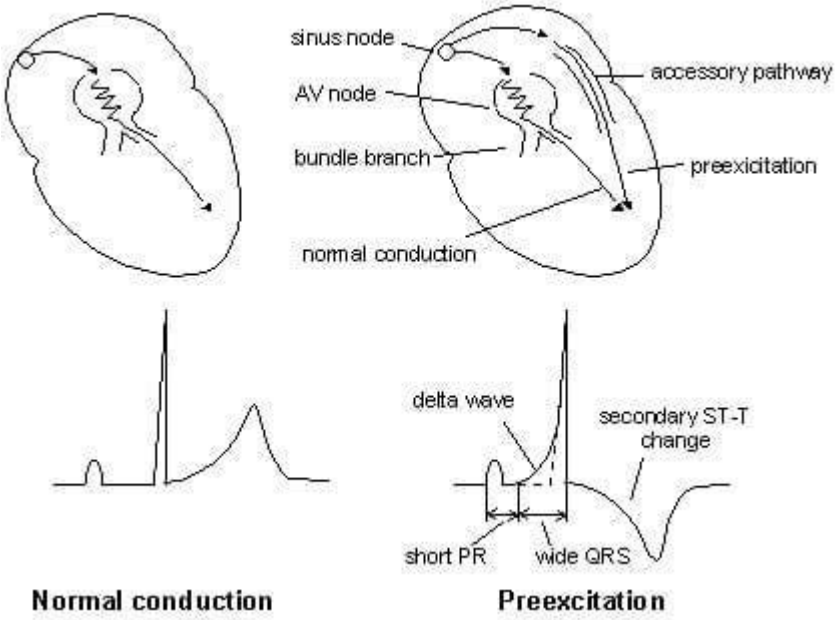
- Tachyarrythmia can be facilitated by the formation of a reentry circuit involving the accessory pathway, termed **atrioventricular reentry tachycardias (AVRT)**.
- Tachyarrythmia may also be facilitated by direct conduction from the atria to the ventricles via the accessory pathway, bypassing the AV 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

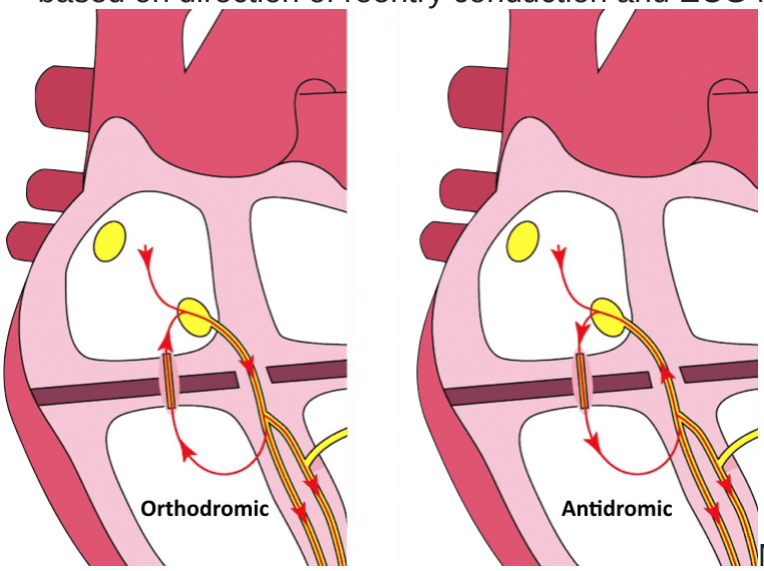
In patients with retrograde-only accessory conduction all antegrade conduction occurs via the AV node, thus no features of WPW are seen on the ECG in sinus rhythm (as no pre-excitation occurs). This is termed a "concealed pathway".

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.