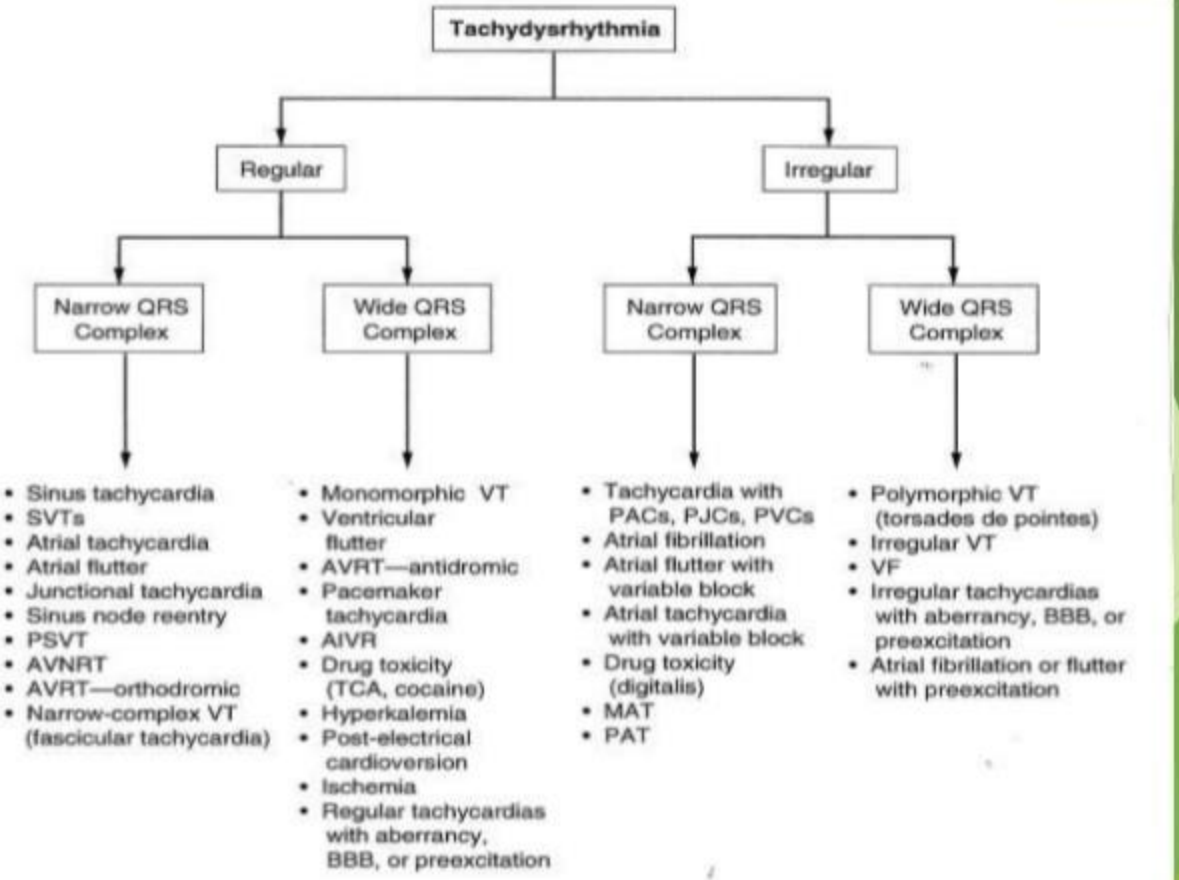
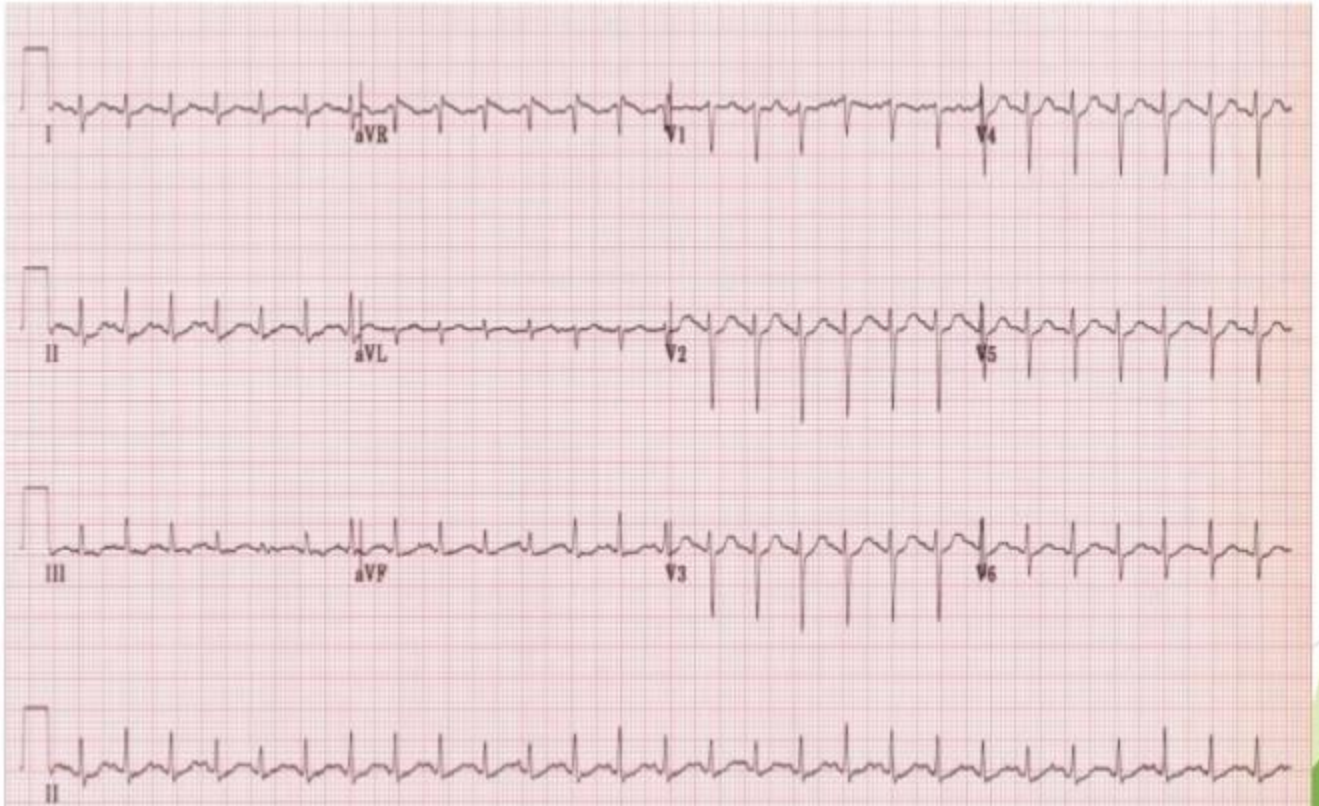
# APPROACH TO TACHYARRYTHMIAS

# Now the hard stuff:



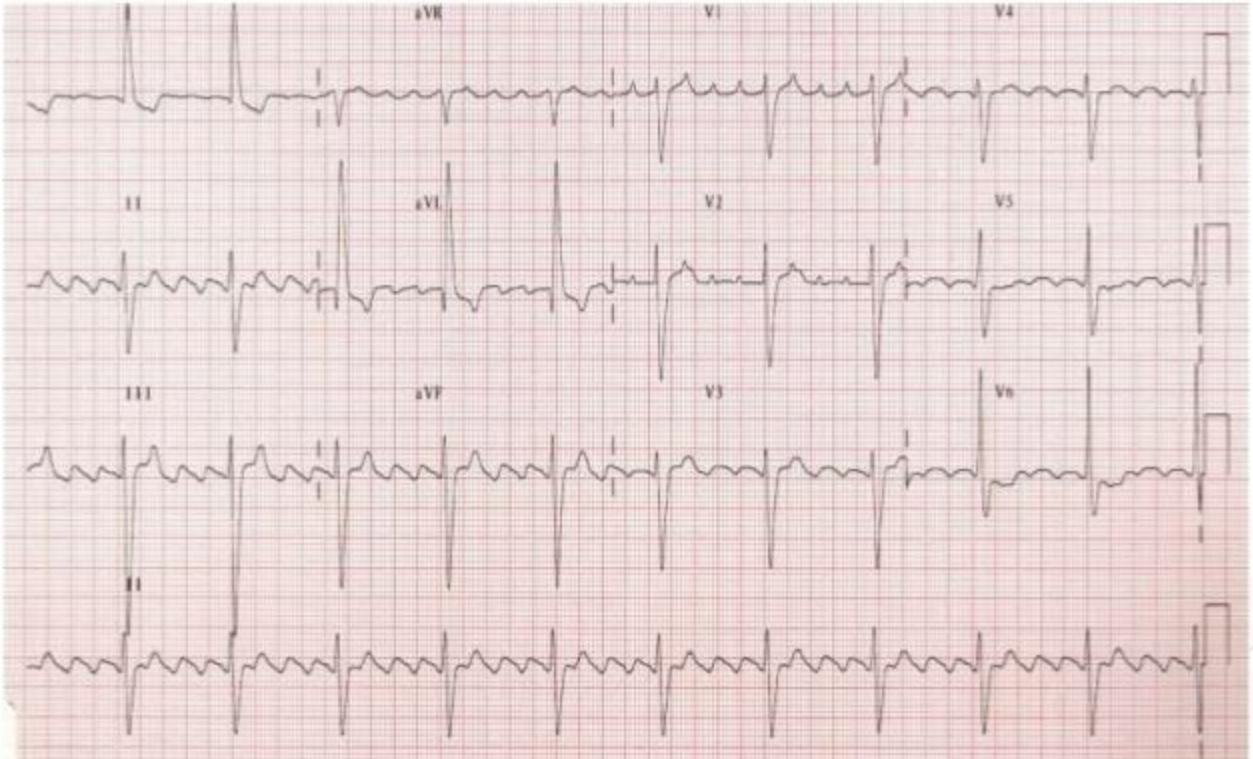
# Regular & Narrow Complex

# Sinus tachycardia



# Atrial Flutter

- Atrial rates 250-350. Slower ventricular rate e.g. 150 = 2:1 block

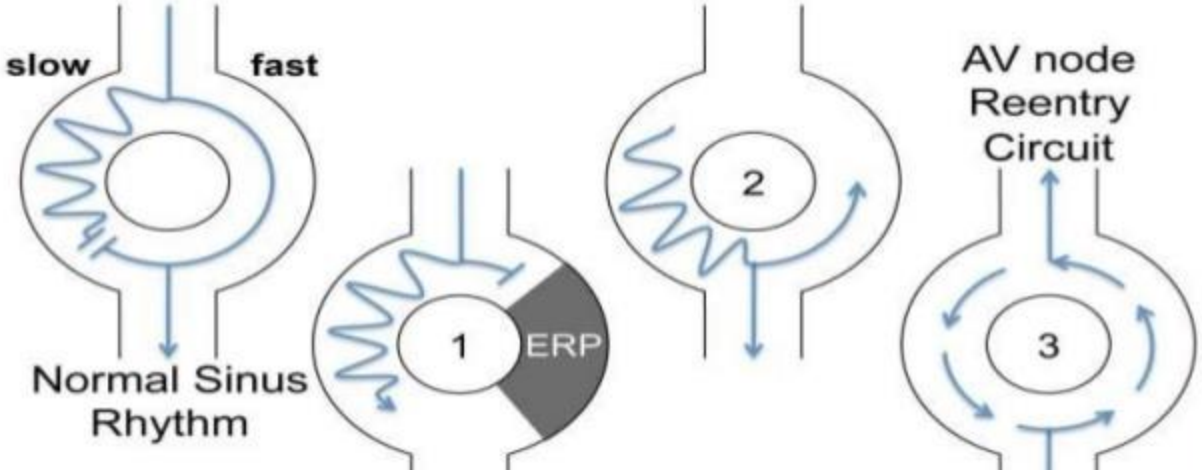


# Supraventicular Tachycardias

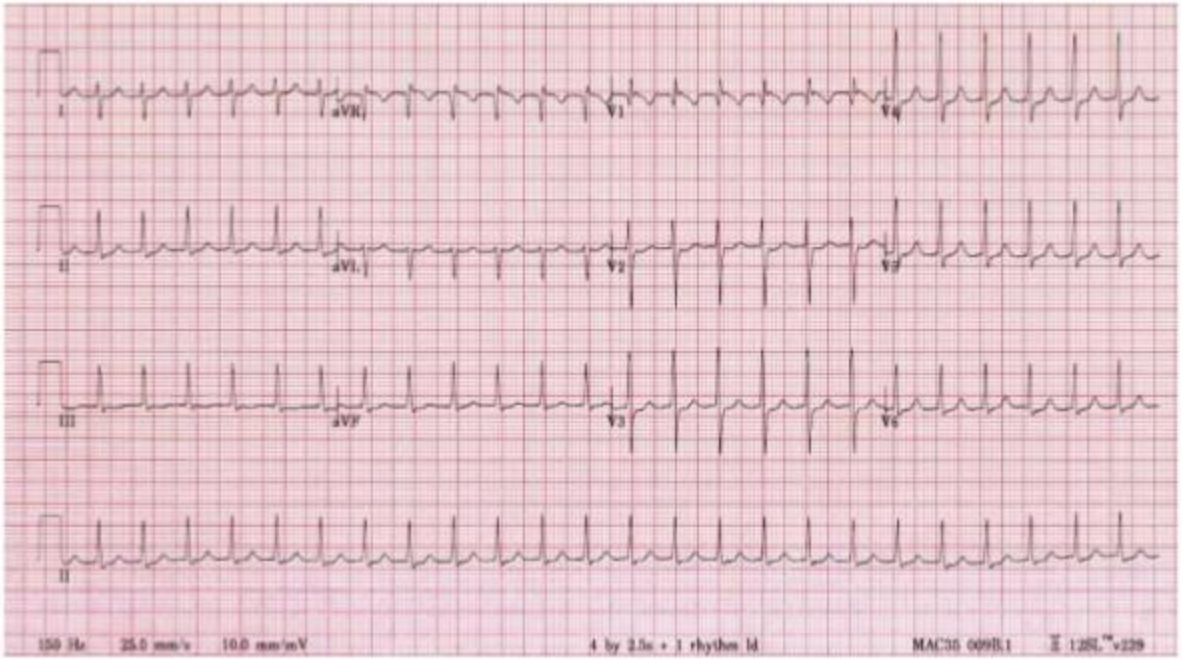
- Generally used to mean any tachyarrythmia with abrupt onset and offset involving a re-entrant pathway:
  - Atrioventricular nodal re-entrant tachycardia (AVNRT)
  - Atrioventricular re-entry tachycardia (AVRT) – orthodromic, antidromic
- AVNRT
  - Anatomy of the AV node: fast & slow fibres. Results in a *functional* re-entrant circuit within the AV node.
  - Narrow complex, rates of 140-280. Heart is structurally normal. More common in women. Sudden onset / offset. May respond to vagal manoeuvres.

# AVNRT

- Typically initiated by a PAC.
- Different subtypes (google LITFL…)
- ECG findings – narrow complex tachy, QRS alternans, retrograde conduction of P waves
- Management: vagal manoeuvres, adenosine, other anti-arrythmics (CCB, BB, amiodarone). Rarely requires DCCV*



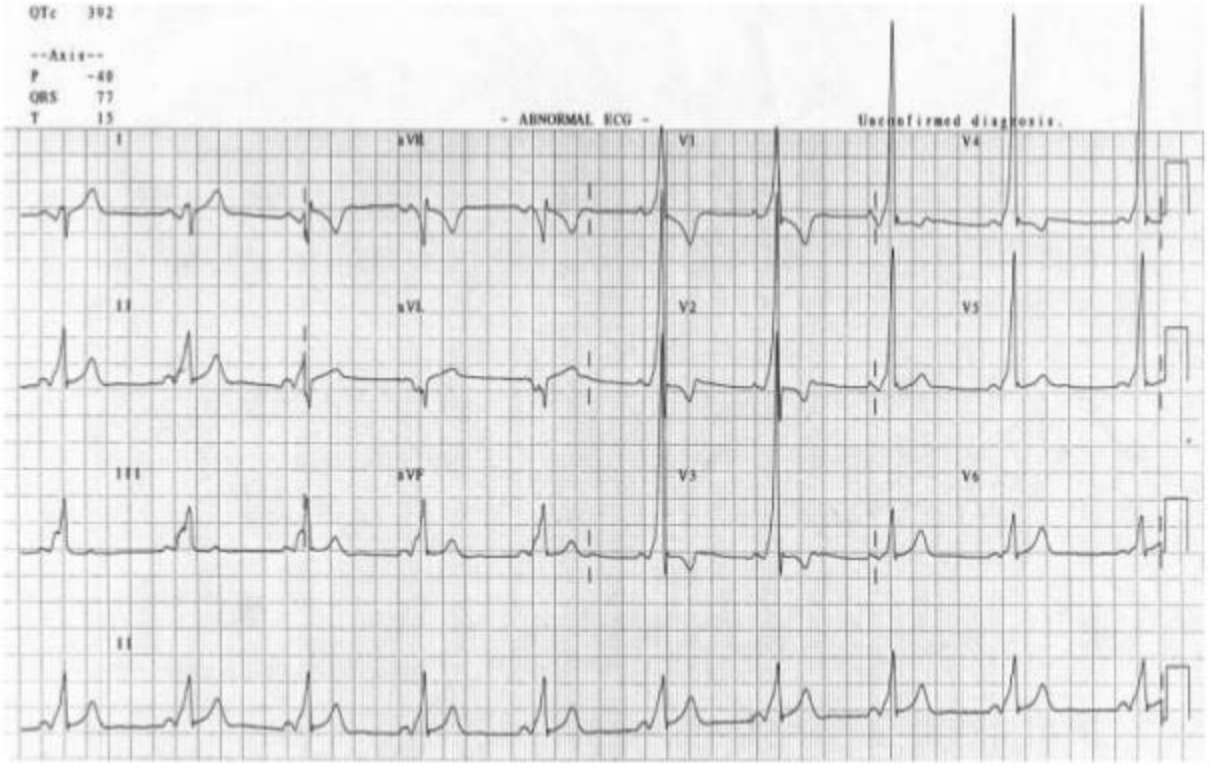
# ECG in AVNRT



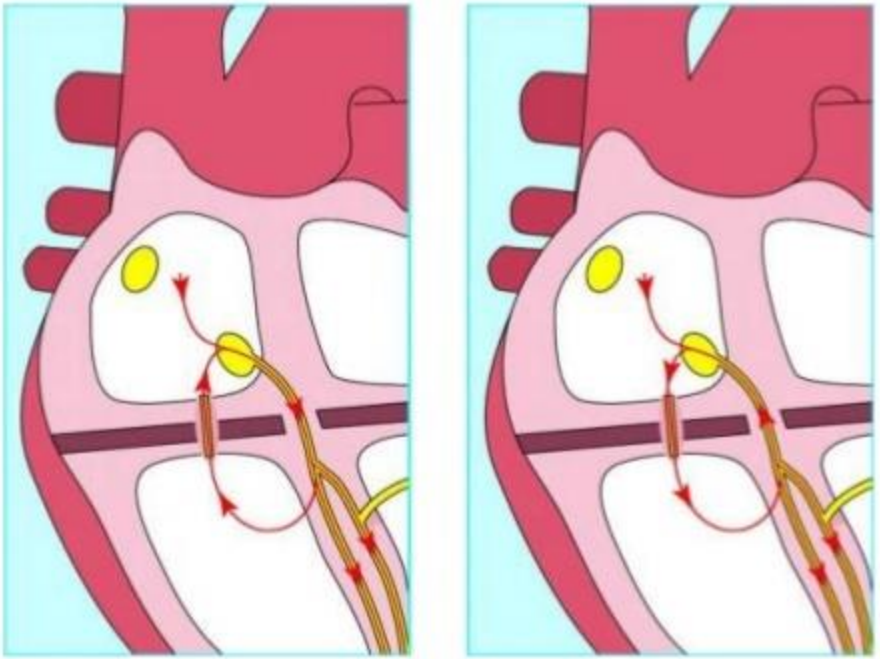
# AVRT

- Pre-excitation syndromes seen on ECG when patient in sinus rhythm (WPW changes). These are lost when AVRT is established
  - Short PR, delta wave, widened QRS
- Anatomical re-entrant pathway (Bundle of Kent). Circus movement between the AV node and accessory pathway.
- May be triggered by PAC or PVC
- Circus movement may by *orthodromic* or *antidromic*

# Pre-excitation

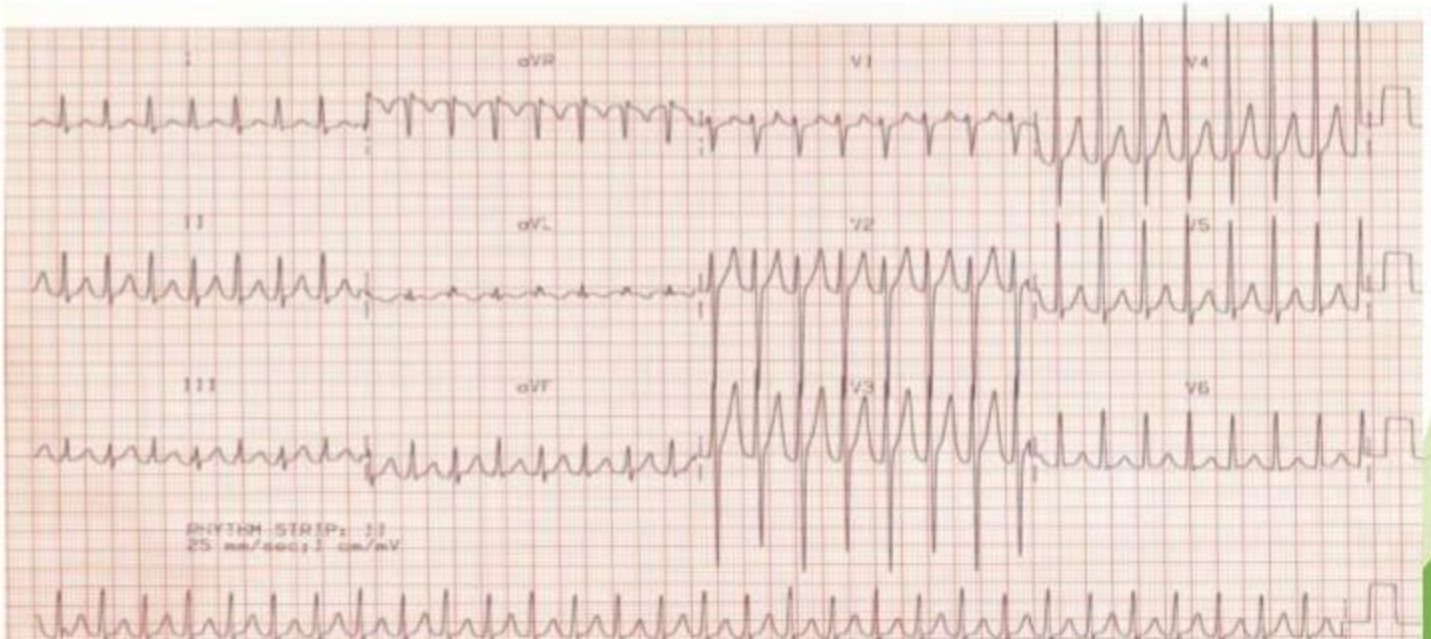


# Orthodromic vs Antidromic Conduction



# Orthodromic AVRT

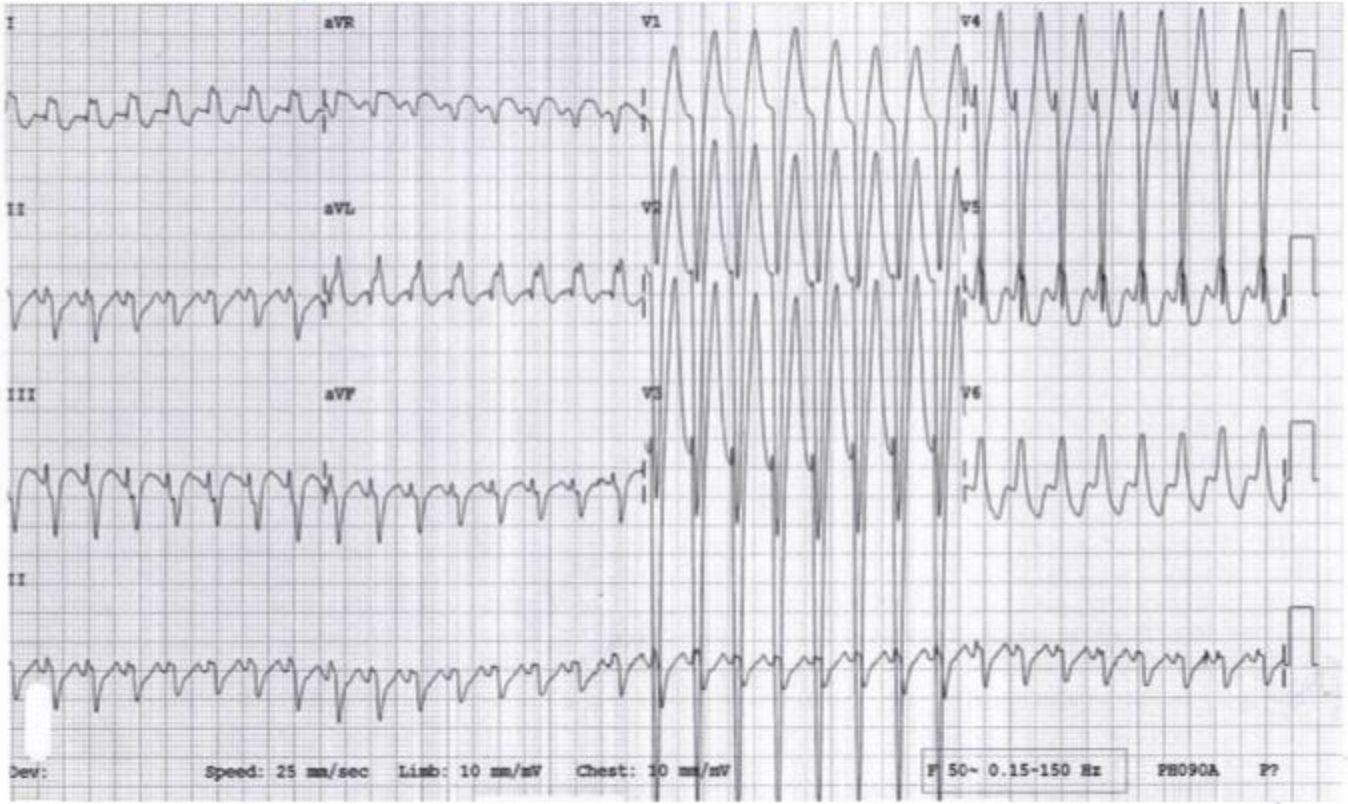
- ECG features: rate 200-300 bpm; buried or retrograde P waves, narrow complex*, QRS alternans, TWI, ST depression.
- Treatment – is pt stable? Can try vagal, adenosine, CCB. If pt compromised → DCCV!



# Antidromic AVRT

- Antegrade conduction via the accessory pathway, retrograde conduction via AV node.
- ECG features: rate 200-300, wide QRS (abnormal depolarisation of ventricles)
- Likely to be mistaken for VT. Treatment: DCCV

# Technically broad complex…



# Summing up the SVTs

- AVNRT – most common
  - Narrow complex*, rate up to 240, regular, responds to adenosine
  - Structurally normal heart – functional re-entrant pathway

- AVRT – seen in pre-excitation syndromes (WPW)
  - Anatomical re-entrant pathway due to presence of accessory bundle
  - Orthodromic → via AV node and back up (retrograde) via the bundle. Results in narrow complex tachy up to 300bpm, regular
  - Antidromic → via accessory pathway and retrograde via the AV node. Results in broad complex tachy up to 300bpm, regular.

# Regular & Broad Complex

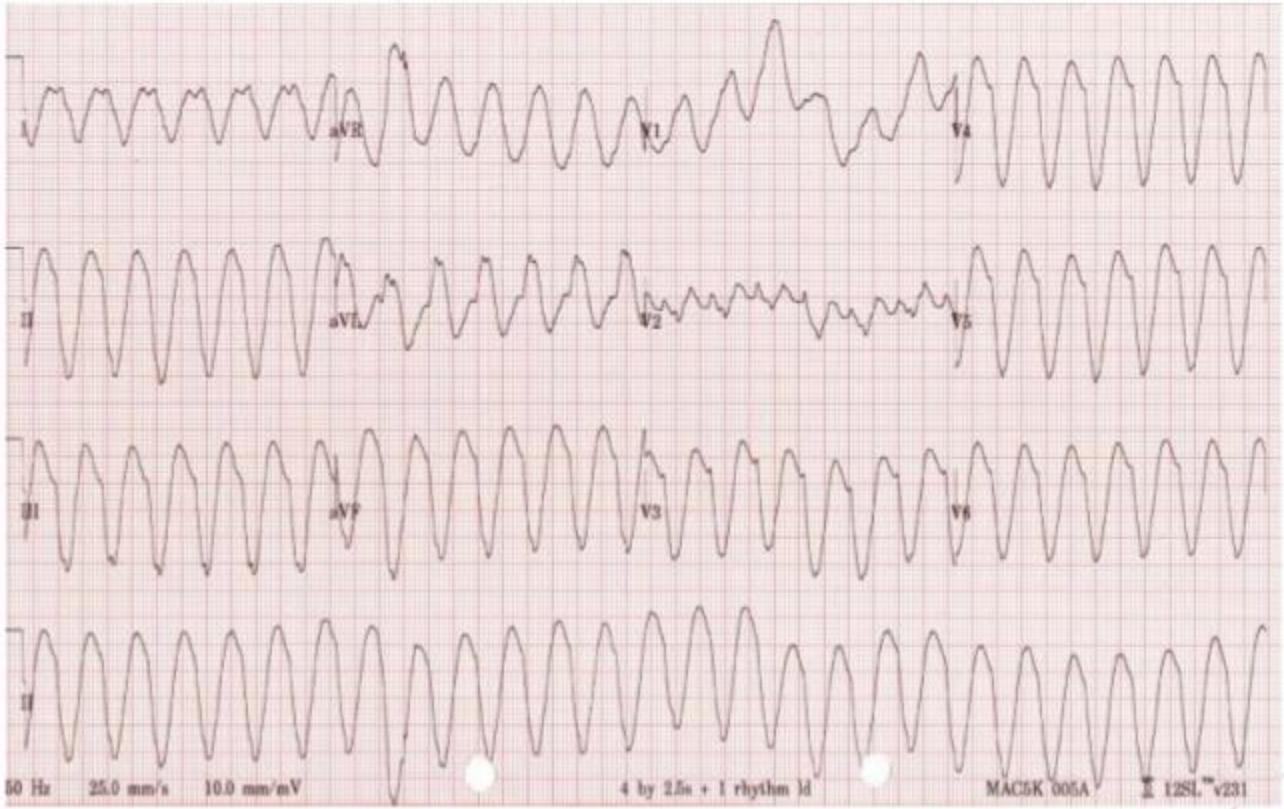
# Ventricular Tachycardia

- Ventricular tachycardia, rates up to 300bpm
  - Sustained / non-sustained; monomorphic, polymorphic
  - VT should be regular (sustained irregularity think SVT w BBB e.g. AF)
  - SVT with aberrancy vs VT
- Pt can be stable or unstable
- Features more suggestive of VT:
  - Age > 35
  - Pre-existing ischaemic heart disease
  - Previous VT
  - Absence normal BBB morphology
  - Hx structural disease, CCF, cardiomyopathy
  - FHx sudden cardiac death

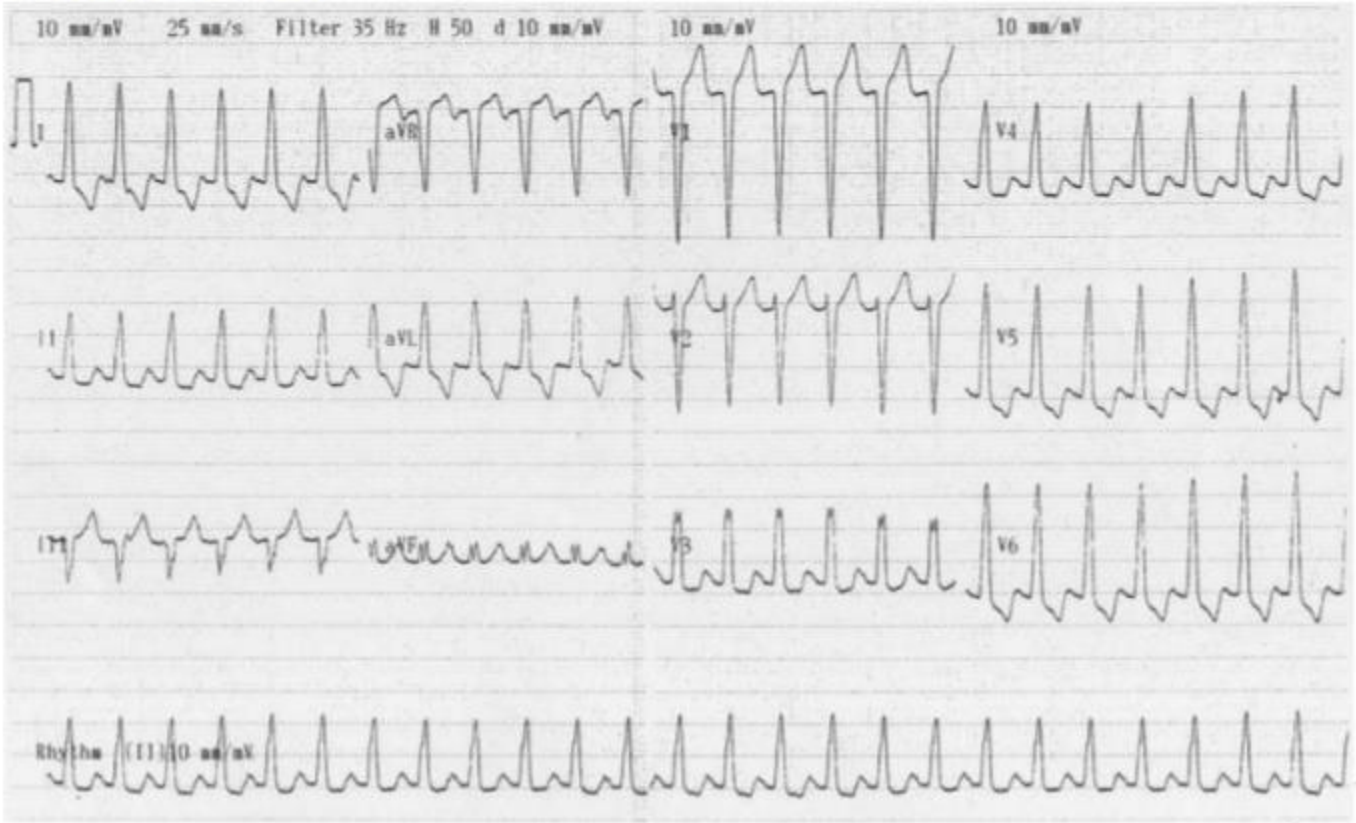
# SVT with aberrancy

- Looks like VT!
- Broad complex tachycardia originating above Bundle of His. Becomes broad due to pre-existing BBB. This will be seen on the baseline ECG!
- Some clues to distinguish
  - North west axis
  - Concordance of QRS complexes in praecordial leads
  - Left rabbit ear > right in V1 (very specific for VT)
  - Fusion & capture beats; AV dissociation
  - Brugada & Josephson's signs
  - Q wave in V6
  - Very broad complex
  - http://lifeinthefastlane.com/ecg-library/basics/vt_vs_svt/ for examples of above

# VT or SVT?

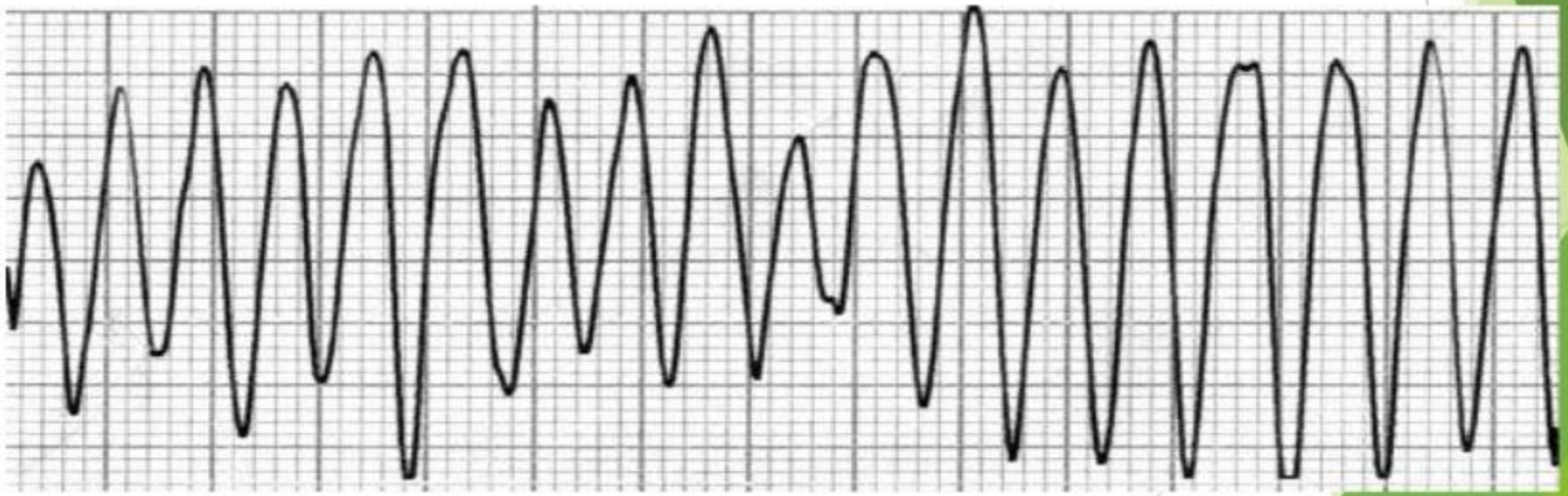

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
50 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld
MAC5K 005A
12SL™ v231

# VT or SVT?


10 mm/mV 25 mm/s Filter 35 Hz H 50 d 10 mm/mV
10 mm/mV
10 mm/mV
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhythm (II) 10 mm/mV

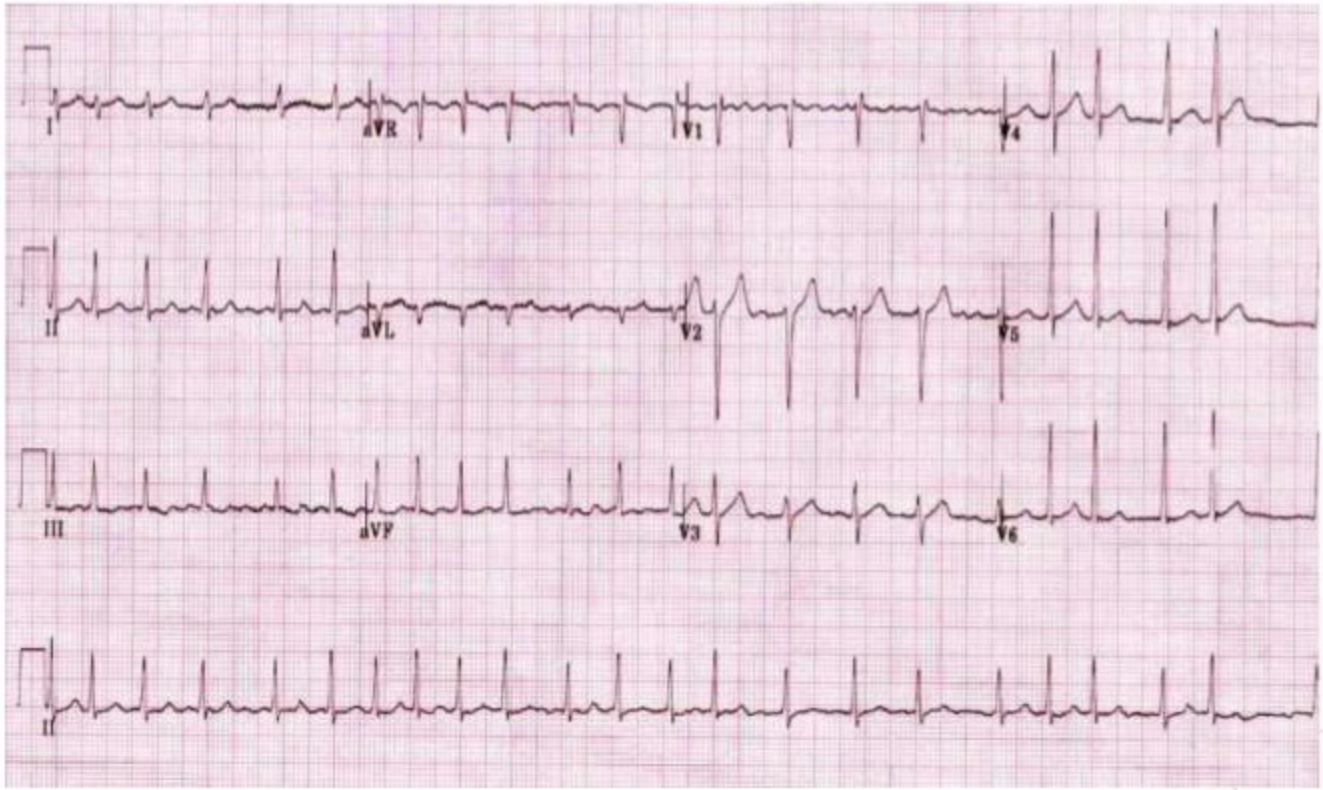
# Ventricular Flutter

- Regular sine wave, rate >200.
- Absence of P waves, QRS complex, T waves
- Lethal (deteriorates to VF) unless abruptly terminated.

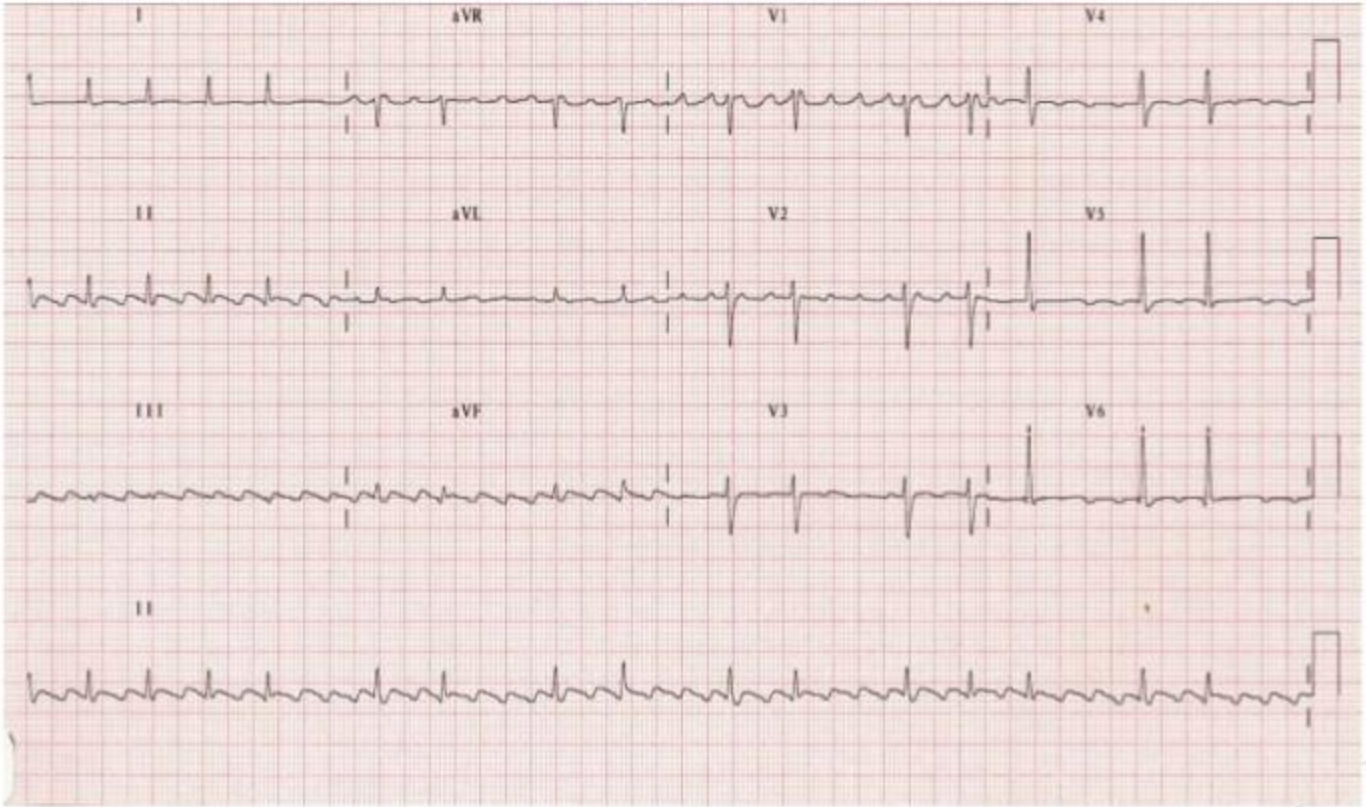


# Irregular & Narrow Complex

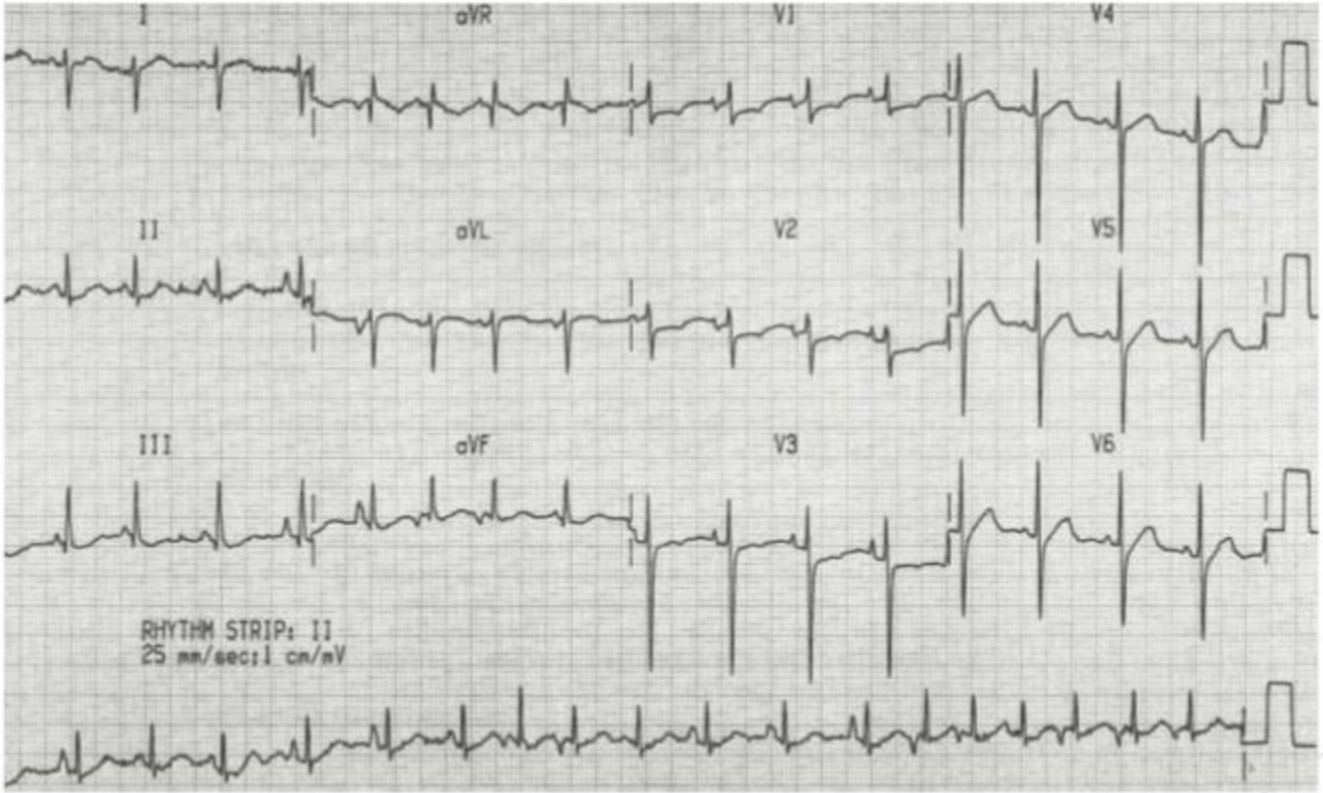
# Atrial Fibrillation



# Atrial Flutter, Variable Block



# Multi Focal Atrial Tachycardia

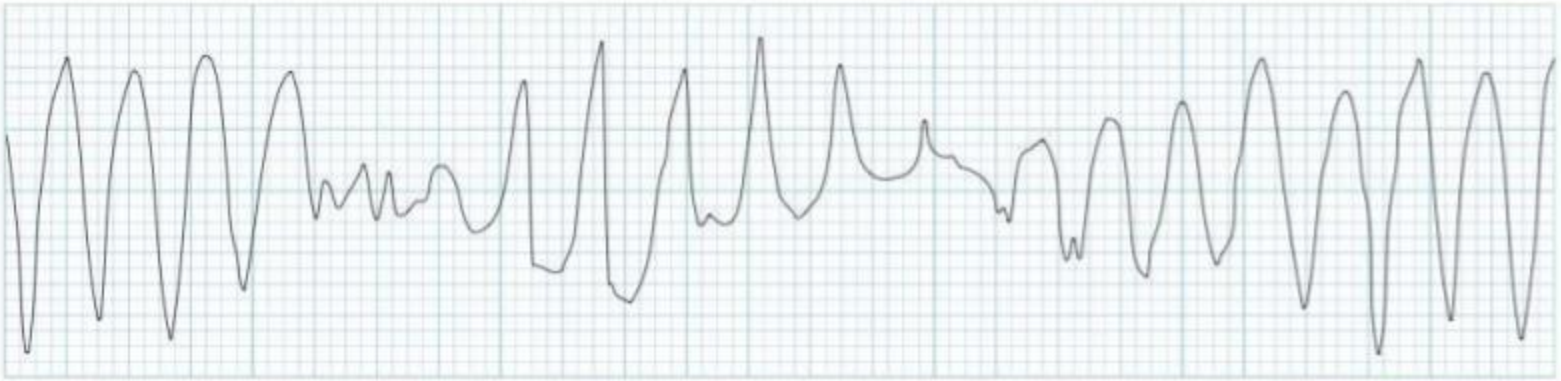



- Associated w severe respiratory disease/ hypoxia
- High mortality - survival at 1 yr is 20%
- Treat the underlying condition, not the tachycardia
- Must have 3 P wave morphologies
- Baseline is isoelectric!!!

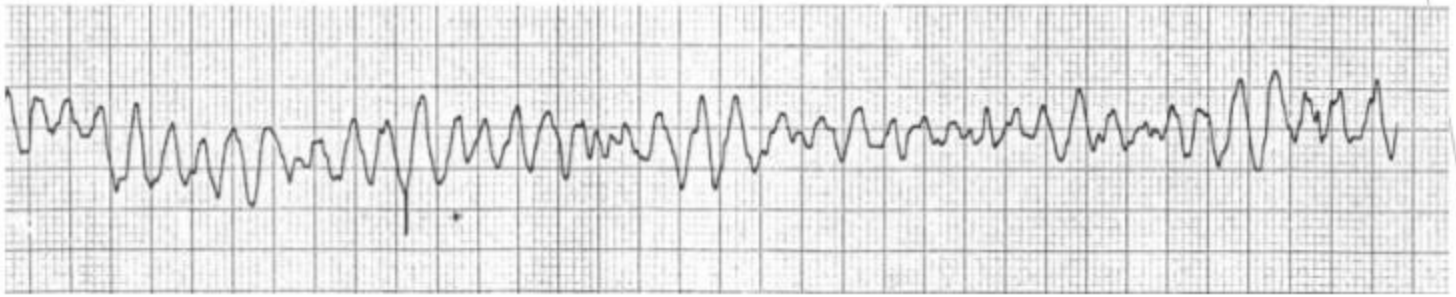
# Irregular & Broad Complex

# Polymorphic VT - torsades

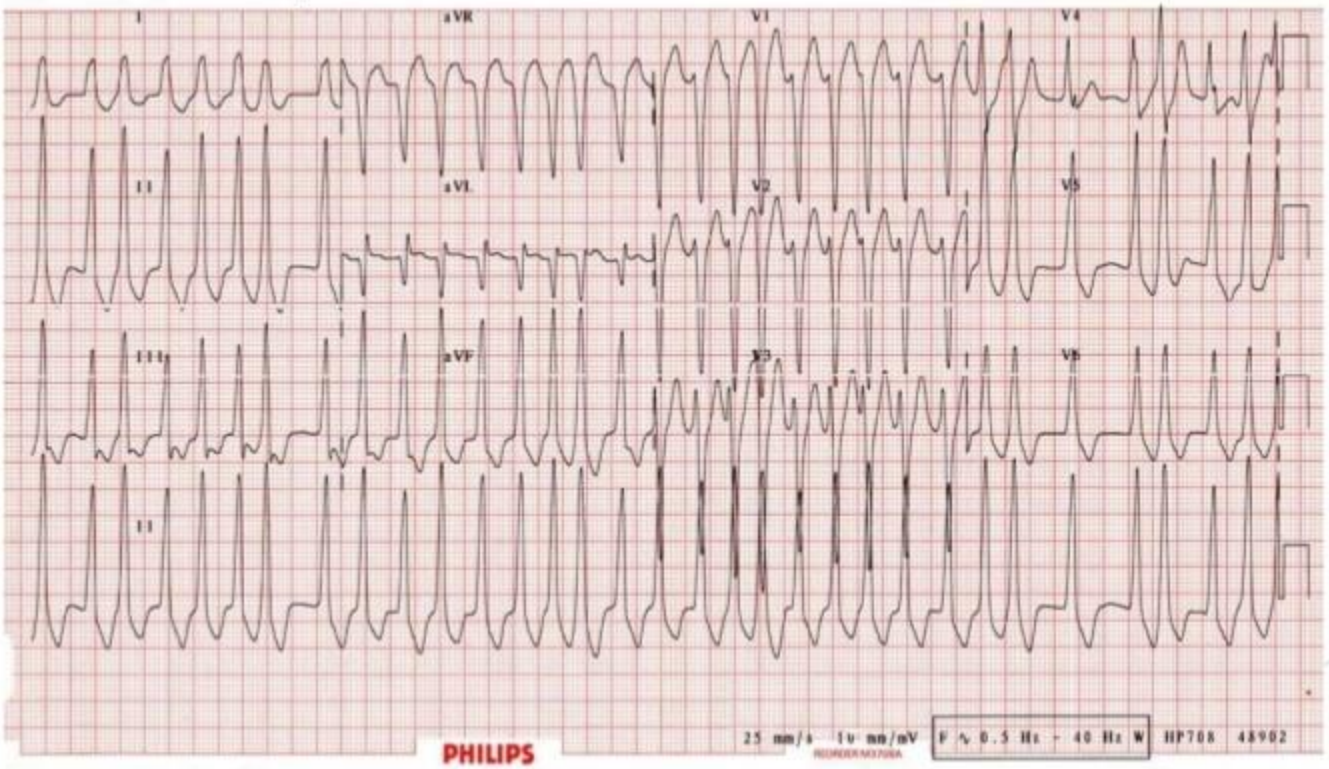
- Requires polymorphic appearance AND QT prolongation
- PVC causes ‘R on T’ phenomenon
- May be self terminating, or degrade into VF
- Treatment – magnesium, overdrive pacing, isoprenaline



# Ventricular Fibrillation



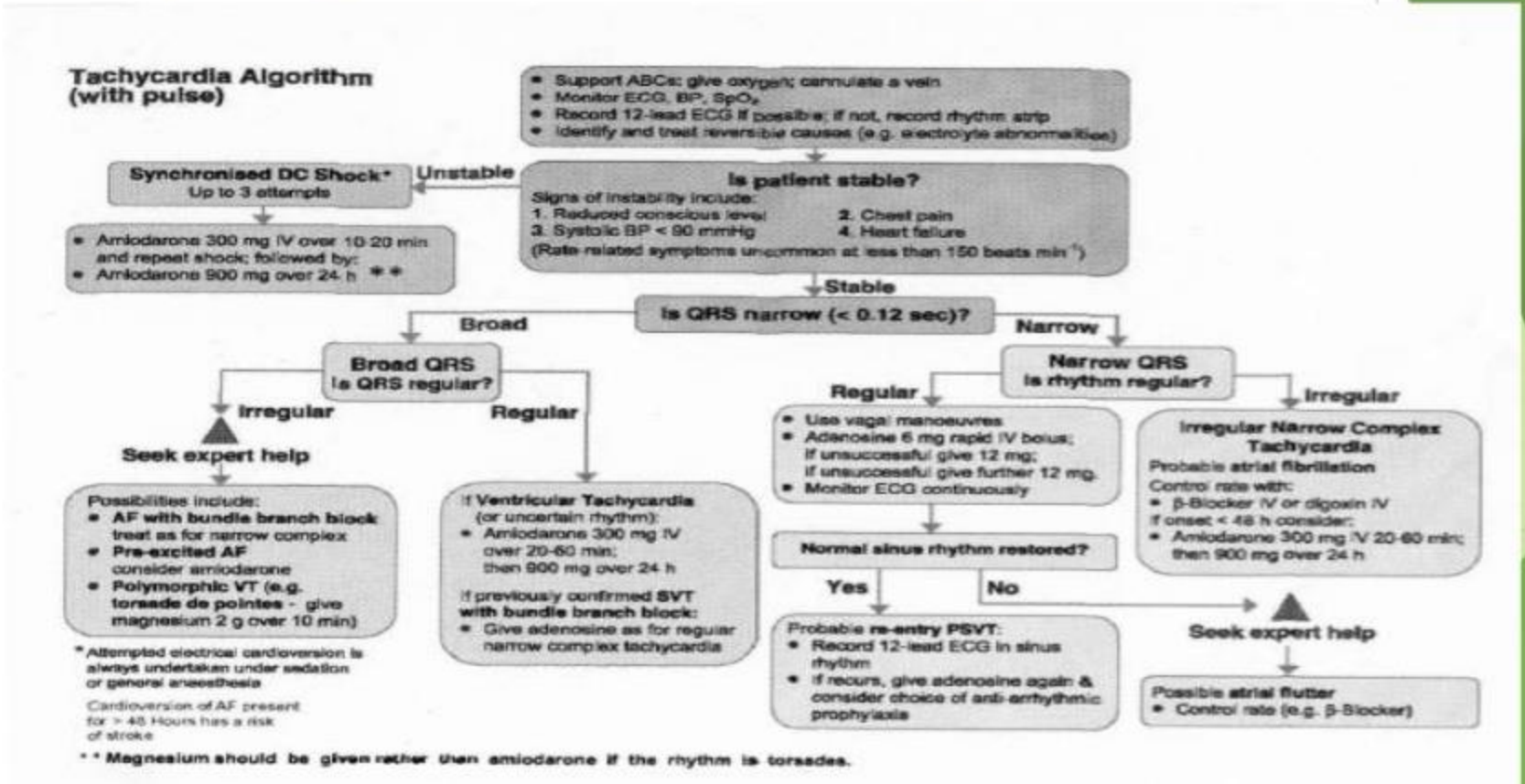
# AF / flutter in pre-excitation



# Other causes of broad complex tachys

- Don’t forget
  - Metabolic derangements e.g. hyperkalaemia
  - Toxidromes e.g. TCA poisoning
  - Pacemaker related

# Tachyarrhythmia Treatment Algorithm



# Summing it up…

- Tachyarrhythmias can be regular, irregular, narrow or broad complex
- May be the result of functional or anatomical re-entrant circuits, or increase automaticity
- Treatment may depend on underlying cause and on whether the patient is haemodynamically stable
- VT and SVT with aberrancy can be difficult to distinguish, when in doubt, cardioversion