Section 7

QT Abnormalities

Other Cardiac Conditions and EKG Abnormalities

Objectives

- At the conclusion of this presentation the participant will be able to
  - Outline a systematic approach to 12 lead ECG interpretation
  - Dysrhythmias
  - Demonstrate the process for determining axis
  - List criteria for LVH, RVH, RAE, LAE LBBB, RBBB, Bifasicular and trifasicular block, acute and chronic MI changes
  - **Define QTc significance and other EKG Abnormalities**

Causes of Regular, Wide Complex Tachycardia

- Ventricular Tachycardia
- SVT with preexisting BBB
- SVT with aberrant conduction

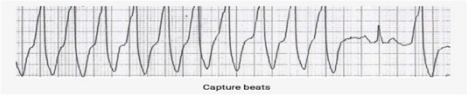
HIS DEBS

- H ypoxia
- I schemia
- S ympathomimetic disturbances
- D rugs
- E lectrolytes
- B rady
- S tretch

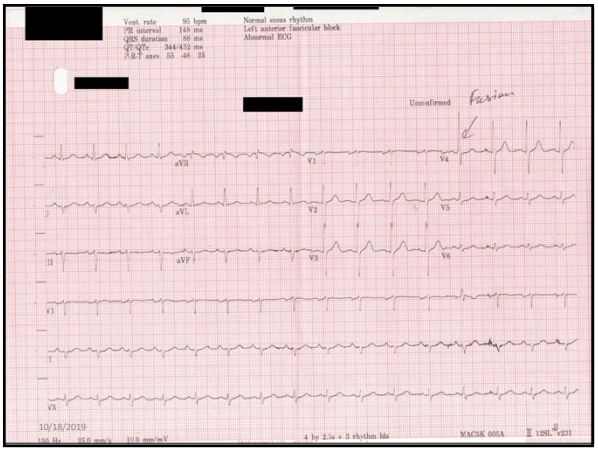
VT vs. SVT with aberrancy

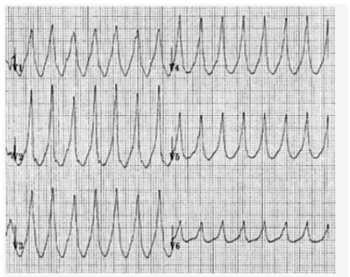
- IT is more likely VT if:
  - Absence of typical RBBB or LBBB
  - Extreme axis deviation (northwest axis)
  - Very broad complexes (> 160 ms)
  - Capture beats
  - Fusion beats
  - Positive or negative concordance throughout chest leads
  - RSR' complexes with a taller left rabbit ear. This is the most specific finding in favor of VT

Capture Beats

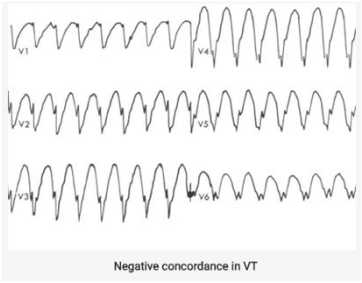


Fusion beats – the first of the narrower complexes is a fusion beat (the next two are capture beats)

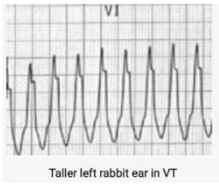




Positive concordance in VT

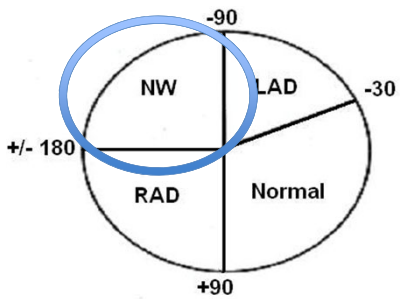


Negative concordance in VT



Taller left rabbit ear in VT

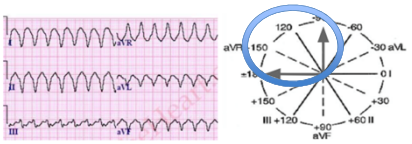
Northwest Axis



Indeterminate Axis or Northwest Axis

Indeterminate Axis

If the QRS is downward (negative) in lead I and downward (negative) in lead aVF, then the axis is indeterminate and sometimes referred to as "northwestern axis". This finding is uncommon and usually from ventricular rhythms, but can also be from paced rhythms, lead misplacement and certain congenital heart diseases.



Indeterminate Axis of the QRS Complex:
Negative in lead I and negative in lead aVF

SVT or AV nodal re-entry tachycardia (AVNRT)

- Classified based on site of origin (atria or AV node) or regularity (regular or irregular)
- QRS width not helpful and influenced by pre-existing BBB, Rate related aberrant conduction or accessary pathways

Classification of SVT by site of Origin

	Regular	Irregular
Atrial	ST Atrial Tach Atrial Flutter Inappropriate ST SN re-entrant tach	Atrial Fibrillation Atrial Flutter with variable block Multifocal atrial Tach
Atrioventricular	AV re-entry tach (AVRT) AV nodal re-entry Tach (AVNRT) Automatic Junctional tachycardia	

AVNRT

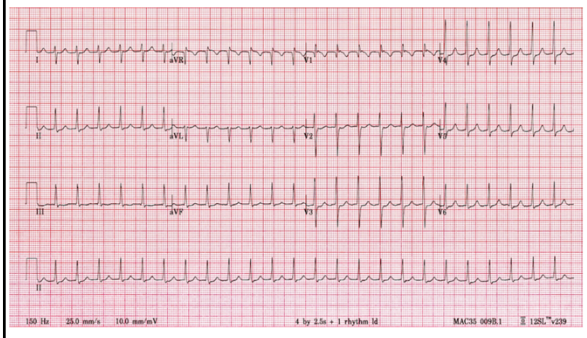
- Most common cause of palpitations in pts with structurally normal hearts
- Occurs spontaneously or upon provocation (caffeine, ETOH, Beta agonists, sympathomimetics (amphetamines)
- More common in women and may occur in young healthy patients
- Sudden onset of rapid, regular palpitations
- SOB
- Pts with CAD may c/o angina
- Tachy rate 140-220 bpm
- Generally well tolerated
- May cease spontaneously and abruptly

Typical ECG findings

- Regular tachy 140-280 bpm
- QRS complexes usually narrow (< 120 msec) unless pre-existing BBB
- ST-segment depression may be seen without CAD
- QRS alternans
- P waves if visible exhibit retrograde conduction with P-wave inversion in leads II, III, aVF
- P waves may be buried in the QRS

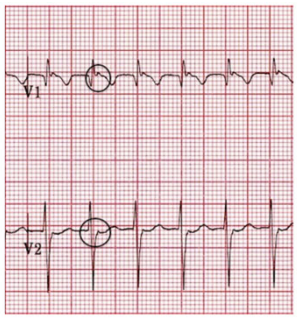
Slow –Fast AVNRT

Example 1a

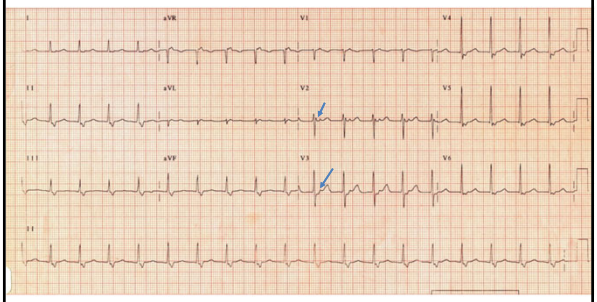


Typical AVNRT

- Narrow complex Tachycardia
- No visible P-waves
- There are pseudo R' waves in V1-2



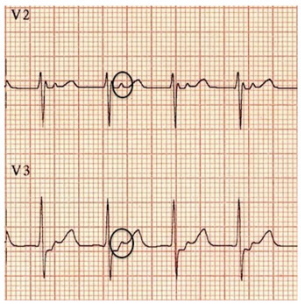
Pseudo R' waves in V1-2



Fast-Slow AVNRT

Narrow complex Tachycardia

Retrograde P waves are visible after each QRS complex



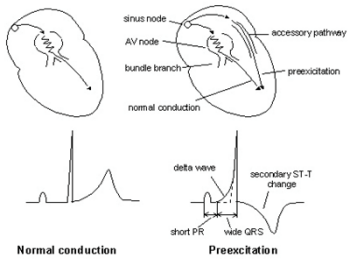
Retrograde P waves

Pre-Excitation & Accessory Pathways

- Activation of the ventricles due to impulse bypassing the AV node via an accessary pathway
- Abnormal conduction pathways
- Impulses conduct either antergrade towards the ventricle or retrograde, away or in both directions
- Majority conduct in both directions
- Reentry circuit involving accessary pathways termed Atrioventricular reentry tachycardias (AVRT)

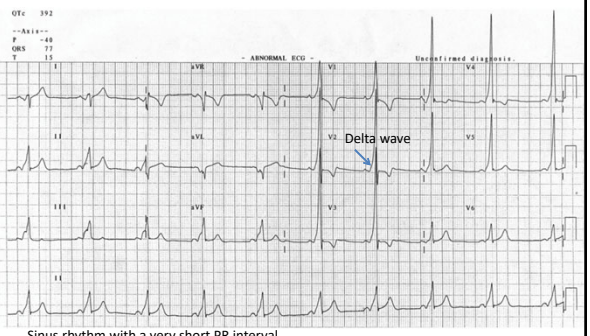
Wolf Parkinson White (WPW)

- PR interval < 120 ms
- Delta wave: slurring slow rise of initial portion of the QRS
- QRS prolongation > 110 ms
- ST segment and T wave discordant changes
- Pseudo-infraction pattern can be seen in up to 70% of patients (pseudo q waves, or prominent R wave in V1-V3 mimicking posterior infarction)



Other Pre-Excitation Syndromes

- Lown-Ganong-Levine (LGL) syndrome
  - Accessary pathway composed of *James Fibres*
  - ECG
    - PR interval < 120 ms
    - Normal QRS morphology
  - The term should not be used in the absence of paroxysmal tachycardia
  - Existence is disputed and may not exist

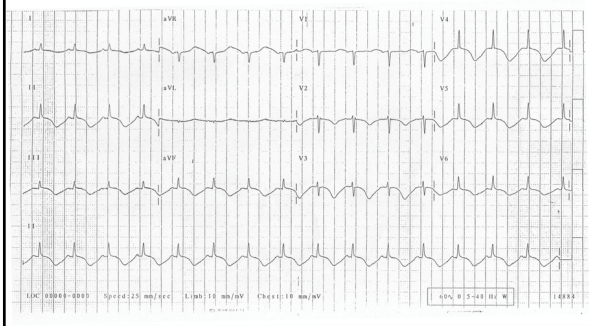


Sinus rhythm with a very short PR interval
Broad QRS with slurred upstroke (delta wave)
Dominant R wave V1
Tall R wave and inverted T wave in V1-3 mimicking RVH
Negative Delta wave in aVL (pseudo infarction pattern)

Common causes of QT Prolongation

- Drugs
  - Type IA, III Antiarrythmic
  - Tricyclic antidepressants
  - Phenothiazines
- Electrolyte disturbances
  - Hypokalelmia
  - Hypomagnesemia
  - Hypocalcaemia
- CNS disturbances
  - Stroke
  - ICB or Brainstem bleed
  - Coma

Example 2 – SAH



- Another example of cerebral T-waves with marked QT prolongation secondary to subarachnoid haemorrhage.

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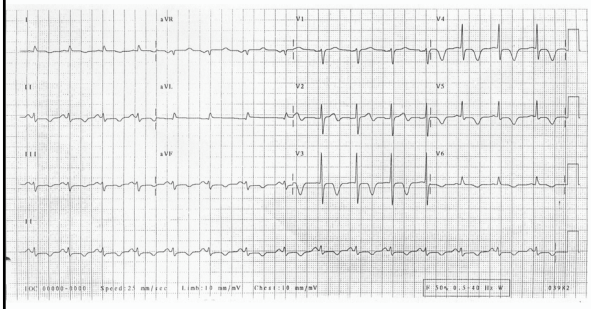
Example 3 – SAH



- Widespread T-wave inversions with slight ST depression secondary to subarachnoid haemorrhage.
- The QT interval is prolonged (greater than half the R-R interval).
- This ECG pattern could easily be mistaken for myocardial ischaemia as the T-wave morphology is very similar, although obviously the clinical picture would be very different (coma versus chest pain).

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Common causes of ST Depression

- Ischemia
- Strain
- Digitalis effect
- Hypokalemia/hypomagnesemia
- Rate related changes
- Any combination of the above

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Common causes of Tall R wave in V1

- WPW
- RBBB
- RVH
- Posterior MI
- Normal variant

Common causes of Nonspecific ST-T wave Abnormalities

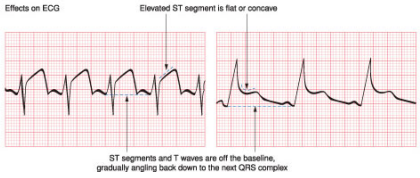
- Ischemia
- LVH
- Cardiomyopathy
- MVP
- Drug effect
- Lyte abnormalities
- CNS disorder
- Hyperventilation
- Severe medical illness
- Severe emotional stress
- Exercise
- Hypoxemia
- Acidosis
- Temp extremes
- Other causes

Other Cardiac Conditions

- Many conditions cause changes to the ECG
  - Electrolyte abnormality
  - Ischemia
  - Infarction
  - Inflammation
  - Medications

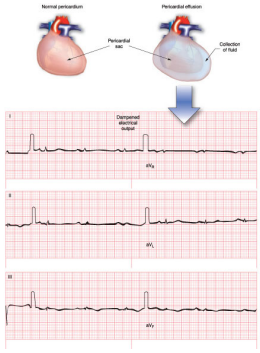
ECG Changes in Pericarditis

- T wave initially upright and elevated but then during recovery phase it inverts
- ST segment elevated and usually flat or concave



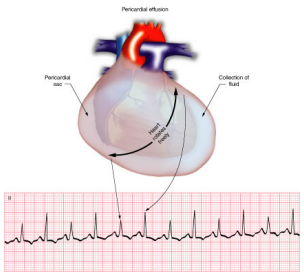
Pericardial Effusion

- Can occur with pericarditis
- Can cause low-voltage QRS complexes in all leads and electrical alternans



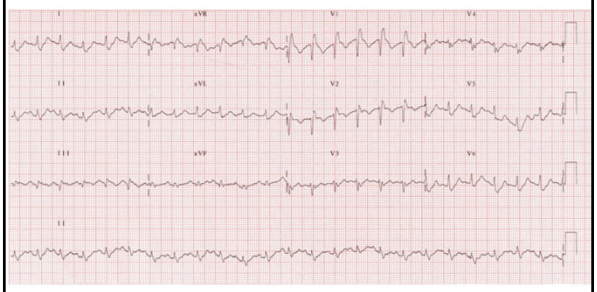
Electrical Alternans

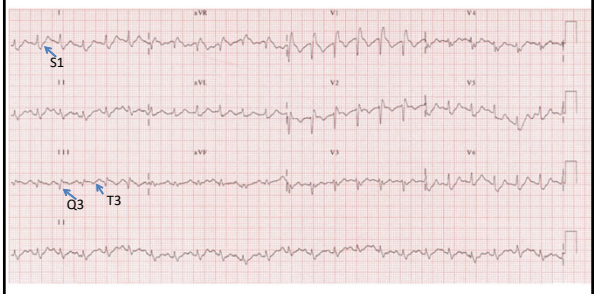
- QRS complexes change in height with each successive beat



Pulmonary Embolism

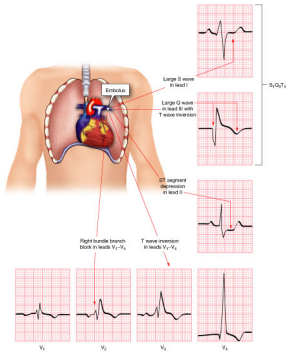
- Acute blockage of one of the pulmonary arteries
- Leads to obstruction of blood flow to the lung segment supplied by the artery
- Produces large S wave in lead I, deep Q wave in lead III, inverted T wave in lead III
  - Called the *S1 Q3 T3* pattern





Sinus Tach
RBBB
T-wave inversions in right precordial leads (V1-3) as well Lead III

Pulmonary Embolism



Electrolyte Imbalances

- Increases or decreases in potassium and calcium serum levels can have a profound effect on the ECG

Hyperkalemia

- Key characteristics include:
  - T wave peaking
  - Flattened P waves
  - 1st-degree AV heart block
  - Widened QRS complexes
  - Deepened S waves
  - Merging of S and T waves

Definitions

- Hyperkalaemia is defined as a potassium level > 5.5 mEq/L
- Moderate hyperkalaemia is a serum potassium > 6.0 mEq/L
- Severe hyperkalaemia is a serum potassium > 7.0 mE/L

Effects Of Hyperkalaemia On The ECG

Serum potassium > 5.5 mEq/L is associated with **repolarization abnormalities**:

- Peaked T waves (usually the earliest sign of hyperkalaemia)

Serum potassium > 6.5 mEq/L is associated with **progressive paralysis of the atria**:

- P wave widens and flattens
- PR segment lengthens
- P waves eventually disappear

Serum potassium > 7.0 mEq/L is associated with **conduction abnormalities** and **bradycardia**:

- Prolonged QRS interval with bizarre QRS morphology
- High-grade AV block with slow junctional and ventricular escape rhythms
- Any kind of conduction block (bundle branch blocks, fascicular blocks)
- Sinus bradycardia or slow AF
- Development of a sine wave appearance (a pre-terminal rhythm)

Serum potassium level of > 9.0 mEq/L causes **cardiac arrest** due to:

- Asystole
- Ventricular fibrillation
- PEA with bizarre, wide complex rhythm

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Hyperkalemia

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Suspect Hyperkalemia

- New bradycardia
- New AV block especially with CKD or ESRD taking ACE-I or potassium sparing meds

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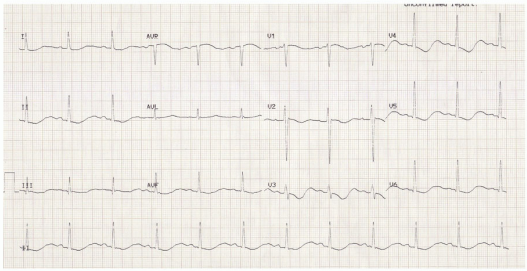
Hypokalemia

- Key ECG characteristics include:
  - ST segment depression
  - Flattening of the T wave
  - Appearance of U waves

Example 1



Hypokalaemia:

- ST depression.
- T wave inversion.
- Prominent U waves.
- Long QU interval.

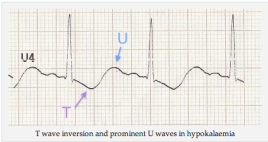
This patient had a serum K+ of 1.7

Handy Tips

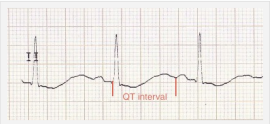
- Hypokalaemia is often associated with hypomagnesaemia, which increases the risk of malignant ventricular arrhythmias
- Check potassium and magnesium in any patient with an arrhythmia
- Top up the potassium to 4.0-4.5 mmol/l and the magnesium to > 1.0 mmol/l to stabilise the myocardium and protect against arrhythmias – this is standard practice in most CCUs and ICUs

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



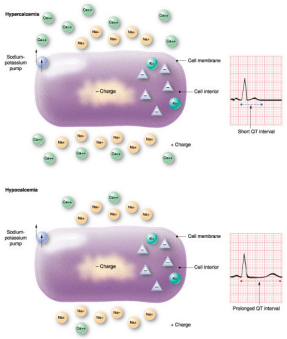
T wave inversion and prominent U waves in hypokalaemia



Apparent long QT interval with hypokalaemia (actually T-U fusion)

Hypocalcemia

- QT interval slightly prolonged



Definitions

- Normal serum corrected calcium = 2.2 – 2.6 mmol/L.
- Mild-moderate hypocalcaemia = 1.9 – 2.2 mmol/L.
- Severe hypocalcaemia = < 1.9 mmol/L.

Causes

- Hypoparathyroidism
- Vitamin D deficiency
- Acute pancreatitis
- Hyperphosphataemia
- Hypomagnesaemia
- Diuretics (frusemide)
- Pseudohypoparathyroidism
- Congenital disorders (e.g. DiGeorge syndrome)
- Critical illness (e.g. sepsis)
- Factitious (e.g. EDTA blood tube contamination)

Symptoms

- Neuromuscular excitability
- Carpopedal spasm
- Tetany
- Chvostek's sign
- Trousseau's sign
- Seizures

ECG Changes

- Hypocalcaemia causes QTc prolongation primarily by prolonging the ST segment.
- The T wave is typically left unchanged.
- Dysrhythmias are uncommon, although atrial fibrillation has been reported.
- Torsades de pointes may occur, but is much less common than with hypokalaemia or hypomagnesaemia.

Hypocalcaemia

Hypocalcaemia causing a long QTc (510ms)

Systematic approach

- Compare with old ECG
- Look at Rate
- Look at Rhythm
- Look at Axis
- Look at Hypertrophy
- Look at I's

  Intervals, ischemia, injury, infarct