ECG INTERPRETATION MANUAL

THE ADVANCED ABNORMAL ECG

Lancashire And South Cumbria
Cardiac Physiologist Training Manual
Junctional Rhythms are caused by disease of the Sino-Atrial node. When the SA node becomes diseased and blocks its output, the natural pacemaker of the heart is lost. Therefore the pacemaker function is taken over by the portion of the conductive system with the next highest intrinsic discharge rate.

I.E. ATRIO VENTRICULAR NODE
CONDUCTION SEQUENCE

The conduction begins in the AV node allowing conduction through both the Atria and the Ventricles.

1. Impulse through to the ventricles is normal with conduction through the Bundle Branches following the normal pathway.

2. Conduction through the atria is retrograde (opposite direction) from the AV node backwards.

3. This produces an inverted P-Wave where normally it is upright.

4. The P-Wave in V1 becomes pointed and positive (normally diphasic).

5. The speed of the retrograde conduction will affect the position of the P-Wave relative to the QRS complex on the ECG.

6. Which ever portion of the AV node is acting as the pacemaker will determine the speed and order of conduction through Atria/Ventricles.
HIGH AV NODAL RHYTHM

This is where the head of the AV node which is nearest to the atrial myocardium takes over the pacemaker function of the heart. This results in an inverted P-Wave preceding the QRS complex and a shortened PR Interval.

![Diagram of High AV Nodal Rhythm]

P-Wave (sinus) P-Wave (nodal) P-Wave (nodal)

MID AV NODAL RHYTHM

This is where the Mid portion of the AV node takes over the pacemaker function of the heart. This causes the Atria and the Ventricles to be depolarised simultaneously. This results in the inverted P-Wave being seen within the QRS complex therefore altering the appearance of the QRS complex. (NB there is no preceding P-Wave)

![Diagram of Mid AV Nodal Rhythm]
LOW AV NODAL RHYTHM

This is where the lowest portion of the AV node takes over the pacemaker function of the heart. This causes the ventricles to be depolarised before the atria are depolarised retrograde. This results in the inverted P-Wave being seen after each QRS complex.
THE HEMIBLOCK CONCEPT

NORMAL ANATOMY; The left bundle divides into two fascicles

Antero-Superior division — spreads anteriorly and superiorly over the sub-endocardium of the lateral wall of the left ventricle. It is long and thin with a single blood supply.

Postero-Inferior division — spreads inferiorly and posteriorly over the diaphragmatic surface of the left ventricle. It is shorter and thicker with a double blood supply.

Both divisions eventually join peripherally and form a closed conducting network which has the property of rapid conduction.
Factors affecting LBB conduction

(a) Interruption of the division by myocardial infarction (antero septal / antero lateral)

(b) Fibrosis – chronic coronary artery disease

(c) Cardiomyopathy

(d) Lev's disease – calcareous encroachment from the aortic valve and intraventricular septum (Affects the antero – superior division)

Normal conduction

In the antero – superior division its vector (axis) is directed inferiorly and to the right. (1)

In the postero – inferior division its vector (axis) is directed superiorly and to the left. (2)
Since conduction occurs together, this results in both forces modifying each others direction and results in a mean QRS vector which is directed downward and to the left. (3)

The Hemiblock concept is when conduction is delayed or interrupted in one of the divisions of the left bundle branch. It is termed a Hemiblock.

**LEFT ANTERIOR HEMIBLOCK**

Is an interruption of the antero – superior division. Conduction therefore takes place in the postero – inferior division, resulting in the QRS axis directed upwards and to the left. (left axis deviation).
LEFT POSTERIOR HEMIBLOCK

Is an interruption of the postero – inferior division. Conduction therefore takes place in the antero – superior division, resulting in the QRS axis directed downwards and to the right. (right axis deviation).
**ECG CRITERIA**

**Left Anterior Hemiblock**

(1) Left axis deviation (-30 to –90°)

(2) Deep S waves in leads II, III, AVF

**Left Posterior Hemiblock**

(1) Right axis deviation (+90 to +120°)

(2) Deep S waves in leads I, AVL and tall R waves in leads II, III and AVF

Other causes of axis deviation must be excluded before diagnosis of Hemiblock can be made.

(1) Inferior wall MI

(2) WPW type A

(3) Ventricular Hypertrophy (RV)

**Bifascicular block**; Left anterior Hemiblock + RBBB.
Left posterior Hemiblock + RBBB

**Trifascicular block**; Left anterior hemiblock + RBBB + 1° AV block.
Left posterior hemiblock + RBBB + 1° AV block.
PRE-EXCITATION SYNDROMES

Since the introduction of specialised electrophysiology techniques it is now possible to acknowledge the existence of additional electrically conducting pathways, known as ACCESSORY PATHWAYS.

(1) BUNDLE OF KENT

Connections between atrial and ventricular myocardium. They exist in the AV groove either left, right or septally.

The accessory pathway is called the Bundle of Kent and gives rise to the Wolfe Parkinson White syndrome.

In general terms two types of WPW exist although the position of the pathway can be specifically named.

(1) Left sided WPW – Type A
   Gives a positive QRS in lead V1

(2) Right sided WPW – Type B
   Gives a negative QRS in lead V1
ECG

(a) The impulse arises normally in the SA node.

Normal P wave

(b) The impulse then travels to the ventricular myocardium by both the accessory pathway and the normal conducting system, via the AV node.

Because there is no delay through the accessory pathway the impulse taking that route will result in premature depolarisation or ‘pre-excitation’ of that part of the ventricular myocardium.
A short or no PR interval is seen: no delay occurs at the pathway.

An initial slurring of the QRS complex is seen: premature depolarisation of the ventricle occurs slowly from myocardial cell to myocardial cell. This slurring of the complex is called a Delta wave.

(c) Normal conduction through the AV node, His bundle and Purkinje system then occurs.

Completion of ventricular depolarisation is therefore narrow and the terminal part of the QRS is normal.
Summary of the ECG findings

P wave normal

PR interval short

Delta wave

QRS – fusion beat
i.e.: initial part is a result of depolarisation through the accessory pathway. The final part is a result of depolarisation through the normal conducting system.

T wave: the direction is usually in the opposite direction to the main QRS deflection.

NB:- Q waves can be seen in the Inferior leads if the pathway is in the right postero septal region and therefore diagnosis of Inferior MI cannot be made.
Clinical Significance :-

In isolation the ECG appearance of WPW is not significant. However, patients are likely to get paroxysmal atrial tachycardias due to a re-entry mechanism.

Prognosis :-

Isolated ECG finding does not alter life expectancy. Recurrent atrial tachycardias not responding to drug therapy may be troublesome and dangerous and require surgical intervention.

If part or all of the ventricular myocardium is depolarized earlier because of the accessory pathway than it would have been through the normal conduction system it is known as “pre-excitation.”
2. MAHAIM FIBRES

Connection between distal AV node and a portion of ventricular myocardium.

**ECG**

P wave normal

PR interval normal (normal delay at the AV Node)

But pre-excitation of ventricular myocardium occurs down the accessory pathway producing a delta wave

T wave:- negative
3. LOWN – GANONG - LEVINE (LGL)

Connection between atrial myocardium and the His bundle.

ACCESSORY PATHWAY

ECG

P wave normal
PR interval short
QRS normal
INTRA AV NODAL PATHWAY

Circus movement within the AV node

The atria and ventricles are depolarised almost simultaneously.

ATRIA

VENTRICLES

Two pathways exist within the AV Node.
(1) SLOW pathway
(2) FAST pathway

Typically route taken during tachycardia is down the slow pathway and up the fast.

LONG PR - SHORT RP

Atypically the reverse may occur, with the impulse travelling down the fast and up the slow pathway.

LONG RP - SHORT PR
VENTRICULAR TACHYCARDIA
SUDDEN CARDIAC DEATH

UNDERLYING RHYTHMS - SCD

62% VT
8% VF (as primary rhythm)
13% Torsades de Pointes
17% Bradycardia

CLINICAL SUBSTRATES ASSOCIATED WITH VT/VF ARREST

80% CAD
10% Dilated Cardiomyopathy
10% Other
   HOCM
   Long QT
   Arrhythmogenic RV dysplasia
   Brugada Syndrome
   Repaired Tetralogy of Fallot
MONOMORPHIC VT

ECG CHARACTERISTICS

4/5 or more VE’s in rapid succession

Complexes have the same configuration (monomorphic)

Abnormal shape

Duration > 0.12s (mostly 0.14s)

Rhythm is regular (unless capture beats are seen)

Rate 120-250 bpm

Sustained VT
Duration > 30secs or if termination is required, in less than 30s by cardioversion/pacing due to severe hypotension.

Non Sustained VT
Ceases spontaneously in less then 30secs.
ATRIAL ACTIVITY

Identification of atrial activity excludes a tachycardia originating from the AV node or above and will diagnose VT.

Direct Evidence

P waves can be clearly seen independently from the ventricular complexes

Indirect evidence

capture / fusion beats

CAPTURE BEAT
occurs when SA node activity occurs at the right time to conduct via the AV node and gives a beat with normal morphology.

FUSION BEAT
occurs when simultaneous activation from atrial activity is transmitted through the AV node and depolarisation from the ventricular ectopic focus. Both contribute to ventricular depolarisation.

CAUSES (* MOST COMMON)

* Acute MI or ischaemia
  
  Past MI

* Dilated / Hypertrophic Cardiomyopathy

  Myocarditis

  Right Ventricular Dysplasia

  MVP

  Valvular heart disease

  Idiopathic
Mechanisms

**Re-entry**
presence of a potential circuit made up of 2 pathways of tissue differing in electrical characteristics.
Block of one pathway in the presence of an early impulse will set up a re-entry circuit.

**Enhanced automaticity**
damaged / diseased cells acquire enhanced automaticity and discharge spontaneously at a rate higher than the SA node.

**Initiation**
Usually an ectopic beat, ischaemia, ↑ sympathetic nervous system activity, electrolyte imbalance.
Distinctive types of monomorphic VT

**RVOT VT**

Arises from the right ventricular outflow tract

ECG complexes are similar to LBBB

The impulse spreads inferiorly from the site just below the pulmonary valve to give an inferior axis → RAD

Take care not to misinterpret :-

**SVT @ LBBB aberrance**

**WPW @ R sided pathway (broad complex tachycardia)**

75-85% non sustained

15-25% sustained but usually self terminating
Characteristics RVOT VT

- No identifiable structural heart disease
- usually benign
- slight female preponderance
- clusters during the day (probably due to exercise/emotion)
- usually asymptomatic
- usually non-compromising (rate dependent)
- exercise induced (rule out exercise induced VT due to ischaemia)
- increased occurrence in highly trained athletes! (?sternal trauma on RV?)

Treatment RVOT VT

Verapamil + adenosine sensitive (termination)
β blockade
Ca antagonists
RF ablation
Fascicular VT

- uncommon

- arises mostly from the posterior fascicle of the LBB (occasionally from the anterior fascicle)

- usually occurs in the young

- no identifiable structural heart disease

ECG characteristics

- posterior fascicle : \textbf{RBBB + LAD}

- anterior fascicle : \textbf{RBBB + RAD}

- usually slightly narrower complexes (due to the impulse arising from the specialised conducting system) i.e. short duration (0.12s)

Atypical RBBB → with loss of primary R wave + small Q wave is sometimes seen.
Arrhythmogenic Rv Dysplasia

Genetic – high incidence in Italy

Arises from RV with impaired function.

Sometimes only localised areas of impaired function are seen.

No / little LV impairment

**ECG characteristics**

- Rises from the RV hence LBBB pattern

- during sinus rhythm - T wave inversion V1→V3

Mostly seen in the young

Male preponderance

Often exercise induced

Brugada syndrome

Genetic

Brugada brothers, Electrophysiologists (Pedro and Joseph), Genetasiist (Ramon)

High incidence Thailand

Incomplete RBBB, ST elevation V1 – V3

VT
TREATMENT (MONOMORPHIC VT)

TERMINATION

1 Cardioversion
   if VT causes cardiac arrest or shock
   if anti arrhythmic drugs are ineffective or contraindicated

2 Anti arrhythmic drugs
   lignocaine (1st line)
   sotalol, disopyramide, flecaainide
      (-ve inotropes therefore avoid with poor LV function)
   amiodarone (2nd line) 24 hours – effective

verapamil for RVOT/Fascicular VT

3 Pacing
   to be considered if drugs are ineffective, multiple
   cardioversions are necessary or pacing wire in situ
   (overdrive pacing - burst few seconds, rate 10-30% faster than the VT rate)
PREVENTION OF RECURRENCE

1 Intravenous infusion
if a bolus terminates the VT and short term VT is expected to recur (acute MI) an infusion should be used to maintain blood levels of antiarrhythmic drug.

2 Oral drugs
if long term therapy is indicated (structural heart disease)
   if exercise provoked → beta blockers

   otherwise → amiodarone*, sotalol, flecainide, disopyramide
   (* most commonly used)

3 Pacing
if VT is due to brady / tachy syndrome

4 Catheter ablation
mostly for RVOT/Fascicular VT.
Can be successful for arrhythmogenic RV dysplasia or an ectopic focus at the site of an MI

5 Surgery
for past MI, revascularisation can be effective
excision/isolation of focus (but is high risk if ventricular function is poor)
POLYMORPHIC VT

Characterised by repeated progressive changes in QRS complex – complexes twist about the baseline.

QT interval during SR normal → acute MI, other causes myocardial damage.

Management is the same as for monomorphic VT.

QT interval during SR prolonged → Long QT syndrome → torsade de pointes tachycardia
TORSADE DE POINTES TACHYCARDIA

Polymorphic VT

QT interval prolonged during sinus rhythm

Prominent U waves

Causes

Bradycardia (SSS or AV block)

Anti arrhythmic drugs

Congenital prolongation QT interval (see later)

Hypokalaemia, hypomagnesaemia

Other drugs eg prenlyamine (like dobutamine), tricyclic antidepressants, erythromycin, thioridazine (anti-pyschotic)

Treatment

Reversal of cause

IV magnesium sulphate (even if serum mg is normal)

Pacing (overdrive sinus rate)
CONGENITAL PROLONGATION OF QT INTERVAL

Dominant gene – Romano Ward syndrome

Recessive gene – Jervell and Lange Nielson syndrome

**Characteristics**

Resting ECG – long or normal QT interval

Exercise ECG – does not shorten
- sometimes prolongs

Some positive signal averaged (Hi Res.) ECG

Catecholamine dependent (emotion/exercise)

**Treatment**

β blockade

pacing (for bradycardia if present or symptoms due to β blockade)

left cervical sympathectomy

Implantable cardioverter defibrillator
QT INTERVAL MEASUREMENTS

QT interval is measure of ventricular repolarisation from the onset of the QRS to the end of the T wave.

**Long QT Interval**

Either due to :-

Uniform prolongation of repolarisation throughout the myocardium

Or:-

Variation in the rate of repolarisation in differing regions of the myocardium (more likely to lead to arrhythmias)

**NB**

QT interval normally shortens with rate due to - an increase in rate itself

- an increase in sympathetic nerve system activity

If measuring the QT interval it should be corrected to the heart rate → QTc

\[
QTc = \frac{\text{measured Qt interval}}{\text{Sq root of cycle length}}
\]

Normal QTc less than 0.42 s