Systematic Approach to ECG

Adopting a systematic approach to ECG assessment is crucial in helping to develop your interpretation skills. It is important to standardise your approach for every ECG and practice, practice, practice to improve your speed and accuracy.

Below is a brief outline of one approach which may be helpful:

- Rate
- Rhythm
- Axis
- P wave and PR interval
- QRS
- ST segment
- T wave
- QT interval
- Additional waves

Rate
Rate is fairly easy to quantify given the ECG paper speed is standardised at 25mm/s.

- Rate = 300/number of large squares of RR interval or
- Rate = 1500/number of small squares of RR interval

If the rhythm is very irregular you should quote a range. If the atrial and ventricular rates differ, you should calculate both.
Bradycardia is defined as < 60 bpm. Tachycardia is defined as > 100bpm.

Rhythm
This is best appreciated by looking at the rhythm strip at the bottom of the ECG which is typically lead II.

Sinus Rhythm

Sinus Arrhythmia

1st degree AV block
2\textsuperscript{nd} degree AV block (type 1)

2\textsuperscript{nd} degree AV block (type 2)

3\textsuperscript{rd} degree AV block

Junctional rhythm

AVRT

Atrial fibrillation

Atrial flutter

Idioventricular rhythm
A rapid method to assess axis is to look at leads I & II. If the QRS in both of these leads are predominately positive, the axis is normal. If it is positive in lead I, but negative in lead II the axis is leftward. If it is negative in lead I the axis is rightward.

<table>
<thead>
<tr>
<th>QRS Deflection</th>
<th>Axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead I</td>
<td>Lead II</td>
</tr>
<tr>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Negative</td>
<td>Positive or Negative</td>
</tr>
</tbody>
</table>

It is important to appreciate what has caused the access deviation.

Causes of leftward deviation:
- LAFB
- LBBB
- LV hypertrophy
- Inferior MI
- WPW syndrome
Causes of rightward deviation:

- LPFB
- Lateral MI
- RV hypertrophy
- Lung disease
- Sodium channel blocker toxicity
- Normal variant in children or slim adults

**P wave**
The P wave is best examined in the inferior leads. Its duration should be < 0.12s and amplitude < 0.25mV. A normal P wave is upright through all leads except for V1, where it is biphasic and aVR where it is inverted.

**PR interval**
The normal PR interval is 0.12 to 0.2 seconds. A prolonged interval signifies a degree of AV blockade. A short interval suggests the presence of an accessory pathway and pre-excitation or can be seen in some junctional rhythms.

**QRS**
The normal QRS duration is less than 0.1s. Broader complexes are due to aberrant conduction or are ventricular in origin.

**LAFB**
- L axis
- Prominent R waves I, aVL
- Prominent S waves II, III & aVF
**LPFB**

- R axis
- Prominent S wave I, aVL
- Prominent R wave III (and often II & aVF)

**LBBB**

- QRS > 0.12s
- Leftward axis
- Prominent S wave V₁₋₃ usually with STE
- Prominent R wave with inverted T wave 1, aVL and V₆

**RBBB**

- QRS > 0.12s
- Normal or rightward axis
- RSR' in V₁
- Wide S wave in V₆ & I
- Deemed incomplete if 0.1 – 0.12s

**Q waves** are considered pathological if they are more than 25% of the depth of the QRS complex or if they are present in V₁ to V₃. Pathological Q waves usually represent myocardial infarction, but can be seen in HOCM.

**R wave progression** throughout the praecordial leads typically transitions at V3, such that there are small R waves in V₁₋₂ and large R waves in V₅₋₆. Poor R wave progression is a feature of ischaemia, LVH, right heart strain or imprecise lead placement, although it can be a normal variant.

**ST segment**

Normally, the ST segment is isoelectric. It represents the period between ventricular depolarisation and repolarisation.
Causes of ST elevation:
- Myocardial infarction
- Pericarditis
- BER
- LBBB
- Ventricular aneurysm
- Brugada syndrome
- Paced rhythm

Localisation of STE to specific territories can be helpful to confirm myocardial ischaemia.

<table>
<thead>
<tr>
<th>Localisation</th>
<th>ST elevation</th>
<th>Reciprocal changes</th>
<th>Coronary artery involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior MI</td>
<td>V₁ to V₆</td>
<td>None</td>
<td>LAD</td>
</tr>
<tr>
<td>Septal MI</td>
<td>V₁ to V₄</td>
<td>None</td>
<td>LAD</td>
</tr>
<tr>
<td>Lateral MI</td>
<td>I, aVL, V₅₋₆</td>
<td>II, III, aVF</td>
<td>Cx</td>
</tr>
<tr>
<td>Inferior MI</td>
<td>II, III, aVF</td>
<td>I, aVL</td>
<td>RCA (80%) Cx (20%)</td>
</tr>
<tr>
<td>Posterior MI</td>
<td>V₇₋₉</td>
<td>V₁₋₃</td>
<td>RCA or Cx</td>
</tr>
<tr>
<td>RV MI</td>
<td>V₁, V₄R</td>
<td>I, aVL</td>
<td>RCA</td>
</tr>
</tbody>
</table>

**T wave**
T waves represent ventricular depolarisation and should be upright in all leads except V₁ and aVR, although T wave inversion can also be a normal variant in lead III.

Causes of T wave inversion:
- Ischaemia
- LVH
- BBBs
- R heart strain
- HOCM
- Normal finding on paediatric ECG
- Persistent Juvenile T wave pattern

Hyperacute T waves with ischaemia
![Hyperacute T waves with ischaemia](image)

Peaked T waves with hyperkalaemia
![Peaked T waves with hyperkalaemia](image)
**QT interval**
The QT interval is measured from the start of the Q wave to the end of the T wave. It is affected by many things such as the heart rate, where the QT interval shortens as the heart beat increases. There are multiple formulas which correct the QT for heart rate, the most popular of these currently is Bazett’s formula;

\[ QTc = \frac{QT}{\sqrt{RR}} \]

A normal QTc is considered < 440ms for men and < 460ms for women.

A prolonged QT interval puts you at risk of Torsades. More recent research investigating the association between Torsades and a prolonged QT interval plots the QT data into clouds from which they developed the QT nomogram – above which a person is much more likely to develop Torsades. The nomogram roughly correlates with a QTc of 500ms.

For the purposes of ECG interpretation, calculate the QT interval and use Bazett’s formula to correct.

A quick way to determine if the QT is normal for rates of 60 to 100 bpm, can be to see if the QT interval is less than half the preceding RR interval.
Causes of long QT
- Congenital long QT syndromes
- Ischaemia
- Drugs/Toxins
- Hypokalaemia
- Hypocalcaemia
- Hypomagnesaemia
- Hypothermia

Additional waves

**U wave**
- The U wave is a small wave seen immediately after the T wave
- Seen in bradycardia, hypokalaemia, hypothermia, and following some antiarrhythmics

**Obsorne or J wave**
- The Osborne wave refers to a rounded positive deflection at the J point
- Seen classically in hypothermia but can be a normal variant or secondary to medications

**Delta wave**
- The delta wave is a slurred upstroke to the QRS seen in WPW

**Epsilon wave**
- The epsilon wave is a small blip buried in the end of the QRS complex seen in arrhythmogenic RV dysplasia
Special ECG scenarios

Benign Early Repolarisation

Characteristic findings:
1. Upward concave STE
2. Notching at J point
3. Large amplitude T waves

Hypothermia

Characteristic findings:
1. AF
2. Bradycardia
3. Prolonged QRS & QT
4. Obsborne waves

Pericarditis

Characteristic findings:
1. Widespread concave STE
2. Widespread PR depression
3. Reciprocal STD & PR elevation aVR

R heart strain

Characteristic findings:
1. STD V1-3
2. TWI V1-3
**Sodium channel blockade**

Characteristic findings:
1. Prolonged QRS
2. Terminal R wave 
   >3mm in aVR

**Paediatric ECG**

Characteristic findings:
1. Rightward axis due to dominance of RV
2. Tachycardia
3. TWI in V₁₋₃ “juvenile pattern”
4. Shorter PR & QRS intervals due to less cardiac mass
5. QTc 490 normal < 6months

**Brugada pattern**

Characteristic findings:
1. RBBB or incomplete RBBB
2. STE right praecordial leads
LVH

Characteristic findings:
1. Largest R + largest S waves in chest leads > 45mm
2. ST depression and TWI in V4-6
3. Leftward axis
4. LAE

HOCM

Characteristic findings:
1. LVH
2. Deep Q waves laterally & inferiorly
3. LAE

WPW

Characteristic Findings:
1. Short PR interval < 0.12s
2. Broad QRS > 0.1s
3. Delta wave
Other considerations:

VT v SVT with aberrancy
Practically this can be difficult to differentiate. The clinical state of the patient is paramount, if in doubt treat as VT.

Features of VT:
- Extreme axis
- Very broad complex >160ms
- AV dissociation
- Fusion beats
- Capture beats
- Concordance throughout chest leads (either all negative or all positive)
- RSR’ with a taller left peak (compared to RBBB with a taller right peak)

Performing a Right sided ECG
A right sided ECG is particularly important to identify an RV infarct. In essence it is a mirror image of the left sided ECG. V₄R is the most sensitive lead for infarction.

Performing a Posterior ECG
Again a posterior ECG is required to identify posterior involvement in a STEMI. It is suggested if there is ST depression anteriorly. The posterior leads are V₇, V₈ and V₉ which continue posteriorly from V₆.