

ECG interpretation

1 small square = 0.04s = 40ms
1 big square = 0.2s = 200ms

Demographics

- Patient name, DOB, any symptoms (e.g. chest pain)
- ECG date and time and which in series
- Check calibration
 - Paper speed – 25mm/s
 - 1mV calibration deflection (at start of trace) – 2 large squares in height

Rate and rhythm

Use rhythm strip

- **Rate:** 300 / number of large squares between R peaks **OR**, if irregular, total R waves on ECG x 6 (ECG is 10 seconds long)
 - sinus bradycardia <60 (physical fitness; hypothermia; hypothyroidism; SA node disease; B-blockers)
 - sinus tachycardia >100 (exercise/pain/anxiety; pregnancy; anaemia; PE; hypovolaemia; fever; thyrotoxicosis)
- **Rhythm**
 1. Regularity: mark 4 R peaks on plain piece of paper and move along trace to confirm (irregular may be: AF; ectopics; 2nd degree AV block)
 2. Sinus: look for a normal P wave before each QRS complex (no clear P waves and irregular QRS = AF; sawtooth baseline = atrial flutter; broad complex tachycardia with no p waves = VF or VT or rarely SVT with BBB/WPW; narrow complex tachycardia with abnormal or no p waves = supraventricular tachycardia)

Axis

Use leads I and II

- Short method: QRS complexes in leads I and II are normally both predominantly positive
 - If R waves point away from each other i.e. QRS predominantly positive in lead I and negative in lead II ('legs apart') there is **left axis deviation** (i.e. more electricity going to left due to: LV hypertrophy/strain; left anterior hemiblock; inferior MI; WPW; VT)
 - If R waves point towards each other ('legs together' - right!) there is **right axis deviation** (i.e. more electricity going to right due to: tall & thin body type; RV hypertrophy/strain e.g. in PE; left posterior hemiblock; lateral MI; WPW)

P wave

Use rhythm strip

- **Height** ≤2 small squares (increased in right atrial hypertrophy e.g. caused by pulmonary hypertension or tricuspid stenosis)
- **Morphology**
 - Bifid = P mitrale (left atrial hypertrophy e.g. caused by mitral stenosis)
 - Peaked = P pulmonale (right atrial hypertrophy)

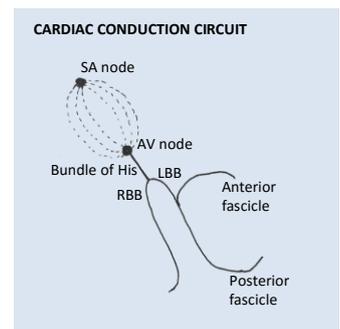
PR interval

Use rhythm strip

- **Length** 3-5 small squares
 - Decreased: accessory conduction pathway
 - Increased in AV block or 'heartblock'
 - 1st degree AV block: PR >5 small squares and regular
 - 2nd degree AV block
 - Mobitz type 1 (Wenkebach): PR progressively elongates until there is failure of conduction of an atrial beat (then the cycle repeats)
 - Mobitz type 2: constant normal PR with occasional dropped beats
 - 2nd degree AV block with 2:1/3:1/4:1 block: alternate conducted and non-conducted atrial beats (P:QRS)
 - 3rd degree (complete) AV block: complete dissociation between p waves and QRS complexes. Normal atrial beats which are not conducted to ventricles resulting in ventricles self-depolarising at a much slower rate 'ventricular escape rhythm'.

1st and 2nd AV block may be caused by: ↑vagal tone/athletes, coronary artery disease, myocarditis, acute rheumatic carditis, digoxin toxicity, or electrolyte disturbances.

3rd degree AV block is caused by fibrosis around Bundle of His (may be caused by ischaemia, congenital, idiopathic, aortic stenosis, or trauma) or block of both bundle branches.



QRS complex

Use chest leads

- **R wave progression** (QRS complexes should progress from mostly negative in V1 (i.e. dominant S) to mostly positive in V6 (i.e. dominant R)) – normally the 'transition point' (i.e. the lead where R and S are equal) is V3/4
 - 'Clockwise rotation' i.e. transition point after V4 (**right ventricle enlargement, usually caused by chronic lung disease**)
 - Dominant R wave in V1/2 (**right ventricular hypertrophy; posterior MI**)

Use rhythm strip

- **Length** <3 small squares
 - Increased
 - **RBBB**: QRS in V1 has **M** (RSR¹) pattern and QRS in V6 has **W** pattern – **MarroW** (**may be caused by: normal variant; atrial septal defect; PE**)
 - **LBBB**: QRS in V1 has **W** pattern and QRS in V6 has **M** (RSR¹) pattern – **WilliaM** (**may be caused by: ischaemic disease; acute MI; cardiomyopathy; hypertension; aortic stenosis**)
 - Notes: 1. The W pattern is often not fully developed; 2. The RSR¹ pattern may be seen with a normal QRS length – this is 'partial (incomplete) bundle branch block' and is of no clinical significance

Use V1 and V5/V6

- **Height** <4 big squares
 - R wave in V5 or V6 >5 big squares (**left ventricular hypertrophy, but can be normal in physically fit patients (look for other signs e.g. T wave inversion in lateral leads)**)
 - Dominant R wave in V1 (**right ventricular hypertrophy if there are other signs too e.g. T wave inversion in right chest leads (V1-V3) or right axis deviation**)

Check in all leads

- **Q wave** – note small Q waves (<1 small square wide and <2 small squares deep) are **normal in I, aVL and V6** (LV leads) due to septal depolarisation
 - Pathological Q waves (**established full thickness MI; previous full thickness MI**)

ST segment

Check in all leads

- **Elevation** ≥1 small square (**infarction – see table; pericarditis or tamponade if in every lead**)
- **Depression** ≥1 small square (**ischaemia; posterior infarction 'reciprocal change'**)
- **Morphology**
 - Saddled (**pericarditis; tamponade**)
 - Upward sloping (**normal variant**)
 - Downward sloping/'reverse tick' (**digoxin toxicity**)

ECG lead ST changes by infarct territory		
	Leads	Artery
Inferior MI	II, III, aVF	Right coronary
Anteroseptal MI	V1-V4	L.A.D.
Anterolateral MI	V4-V5, I, aVL	L.A.D. or left circumflex
Lateral MI	I, aVL ± V5-6	Left circumflex
Posterior MI	Dominant R wave V1-2, ST depression	Left circumflex or right coronary

T wave

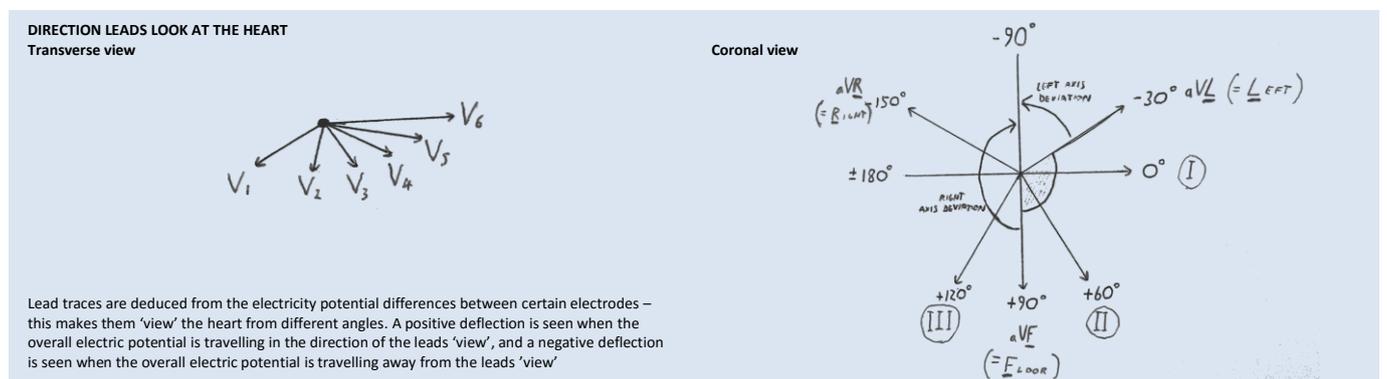
Check in all leads

- **Inversion** – note its **normal in III, aVR & V1** (right leads) due to the angle they look at the heart (and also in V2-3 in black people). It's almost always inverted in aVR.
 - Causes: **ischaemia/post-MI; right/left ventricular hypertrophy (right chest or lateral leads respectively); bundle branch block; digoxin treatment**
- **Morphology**
 - Tented (**hyperkalaemia**)
 - Flat (**hypokalaemia**)

Other things

Use rhythm strip

- **Corrected QT interval (QT_c)** <450ms – ECG machine should calculate
 - Increased – predisposes to polymorphic VT (**causes: congenital syndromes; anti-psychotics; sotalol/amiodarone; TCAs; erythromycin; hypokalaemia/hypomagnesaemia/hypocalcaemia**)
- **U waves** – can be normal or in hypokalaemia



Common ECG pathologies

Rhythm abnormalities

- **Supraventricular tachycardias**
 - **AF:** irregular without P waves
 - **Atrial flutter:** regular with saw-tooth baseline (fluttering p waves) – 2:1, 3:1 or 4:1 block
 - **Atrial tachycardia:** regular with abnormal P waves
 - **AV nodal re-entry (junctional) tachycardia:** regular with no P waves
 - **AV re-entry tachycardia:** regular with no P waves
- **VF:** no discernible P waves/QRS complexes (random wavy line) – NO PULSE!
- **VT:** broad complex tachycardia is VT until proven otherwise (organised wavy line)
- **Atrial ectopic:** narrow QRS ± preceding ectopic P wave (resets the P wave cycle)
- **Ventricular ectopic:** abnormal broad QRS at abnormal time (doesn't affect the SA node so next P wave occurs at predicted time)
- **Ventricular bigeminy (regular ventricular ectopics):** abnormal premature ventricular complexes after each sinus complex
- **Wolff-Parkinson-White (WPW) syndrome:** slurred upstroke into the QRS complex (delta wave), short PR interval, QRS complexes may be slightly broad, dominant R wave in V1 (because the accessory pathway is left-sided)
- **Brugada syndrome (cardiac sodium channelopathy):** RBBB with ST elevation V1-3

Perfusion abnormalities

- **Infarction:** ST elevation (first change), T wave inversion, pathological Q waves (signify full thickness MI and develop 8-12 hours after ST elevation if myocardium is not reperfused)
- **STEMI criteria:** ST elevation in >2 small squares in 2 adjacent chest leads, ST elevation > 1 small square in 2 adjacent limb leads, OR new LBBB
- **Ischaemia:** ST depression, new T-wave inversion
- **Posterior (wall of LV) infarction:** dominant R wave in V1/2 (like right ventricular hypertrophy but without other changes), ST depression. *Note: Q waves can only be seen by placing the chest leads on the patient's back.*
- **Previous infarcts:** T wave inversion (last weeks-months) and pathological Q waves (permanent)

Hypertrophy

- **Left ventricular hypertrophy** = R wave >5 big squares in V5/6, T wave inversion in lateral leads
Sokolow-Lyon voltage criteria: S depth in V1 + tallest R wave height in V5/6 = >7 big squares
- **Right ventricular hypertrophy** = dominant R wave in V1, T wave inversion in right chest leads (V1-3), right axis deviation
- **Hypertrophic cardiomyopathy** = left ventricular hypertrophy signs + dramatic T wave inversion in lateral leads (maximal in V4 rather than V6)

Fascicular blocks

- **Unifascicular block** – one of the 3 conduction paths after the bundle of His blocked (see conduction circuit image above)
 1. Right bundle branch i.e. RBBB
 2. Anterior fascicle of left bundle branch i.e. left anterior hemiblock = marked left axis deviation
 3. Posterior fascicle of left bundle branch i.e. left posterior hemiblock (rare) = marked right axis deviation
- **Bifascicular block** (i.e. RBBB & left anterior/posterior hemiblock) = RBBB + left/right axis deviation
- **Trifascicular block** (i.e. RBBB, left anterior hemiblock & left posterior hemiblock)
 - **Incomplete** = 3 possible patterns
 - Bifascicular block + 1st degree AV block ← *MOST COMMON PATTERN REFERRED TO AS "TRIFASCICULAR BLOCK"*
 - Bifascicular block + 2nd degree AV block
 - RBBB + alternating left anterior hemiblock/left posterior hemiblock
 - **Complete** = 3rd degree AV block + signs of bifascicular block

Metabolic

- **Hyperkalaemia:** low flat P waves, wide bizarre QRS, slurring into ST segment, tall tented T waves
- **Hypokalaemia:** small flattened T waves, prolonged PR, depressed ST, prominent U wave
- **Hypercalcaemia:** short QT
- **Hypocalcaemia:** prolonged QT

Other conditions

- **PE – possible changes:** tachycardia, RV strain (i.e. T-wave inversion in right chest and inferior leads, RBBB, right axis deviation), RA enlargement (i.e. P pulmonale), RV dilation (i.e. dominant R in V1)
S₁Q₃T₃ classical pattern (prominent S wave in lead I, and Q wave and inverted T wave in lead III) is rare
- **Pericarditis:** PR depression (specific), saddle-shaped ST interval