HOW TO READ AN ECG

Pathophysiology

Pacemaker Rates:
SAN 60-100
AVN 40-60
Ventricle 20-40

Areas of ECG

Calculating Rate

Horizontal scale: 1mm = 0.04s  5mm = 0.2s

Rate = 300 / big squares
1 line = 300  2 line = 150  3 line = 75  4 line = 60  5 line = 50  6 line = 42  7 line = 38

Or, count number of complexes in 6 seconds and x10
Or, count number complexes on strip and x6 (this is the method I used)
Calculating Axis

Use lead I and aVF to determine; if I+ and aVF-, use II

**Transitional lead:** where QRS is equally positive and negative; should occur at V3-4
- Transition zone at V1-2 = counterclockwise rotation
- Transition zone at V5-6 = clockwise rotation

**Normal axis** = -30 to +90; +ive in I and either II/aVF

**L axis deviation** = -90 to -30
- **Causes:** old people
  - LVH – most common
  - L anterior fascicular block – LAD must be present for LAFB to exist
  - Inferior MI – due to Q wave in inferior leads
  - LBBB – LAD suggests severe conducting system disease
  - Mechanical shifts of heart (eg. pneumothorax, emphysema)
  - Ectopic ventricular rhythms (eg. VT), pacing, hyperkalaemia, pregnancy, WPW (R sided accessory pathway), tricuspid atresia, ostium primum ASD

**R axis deviation** = +90 to +180
- **Causes:** Infants and children; normal young/thin adults
  - RVH (PE; COPD (+/- pul HTN))
  - L posterior fascicle block
  - Antero-lateral MI – due to Q wave in I
  - RBBB
  - Mechanical shifts of heart (eg. Pneumothorax)
  - Dextrocardia – inversion of P and QRS in lead I, QRS complexes decrease from V1-6
  - Limb electrode misplacement – L/R arm reversal; P, QRS and T waves inverted in I; V1-6 appear normal
  - Ectopic ventricular rhythms, WPW (L sided accessory pathway), ASD / VSD

**Extreme axis deviation** = +180 to -90
- Dextrocardia; COPD (emphysema); Pacing; hyperkalaemia; VT; Incorrect lead placement

**Anticlockwise rotation:** Electrical shift to R: RVH, WPW, post MI, L septal fascicular block
- Septal shift to R: HOCM

**Clockwise rotation:** IV conduction abnormalities 2Y to myocardial degeneration: RVHD
- Septal shift to L: dilated CM
- Heart shift to L: emphysema, tall thin person

**Poor R wave progression:** suggestive of infarction; tall R waves in V1-2 suggest Q waves of posterior infarction; dextrocardia

**Reverse R wave progression:** R waves getting smaller from V1-4 suggests infarction / precordial lead reversal
Incorrect Lead Placement

- **aVR** should always have inverted P, QRS and T – if not, incorrect lead placement
- **L arm / R arm reversal** = RAD
- **L arm / L leg reversal** = taller P wave in I than II
- **R arm / L leg reversal** = positive inflections in aVR, negative in all I, II and III
- **R leg / either arm reversal** = flat line appearance to one of I – III
- **Precordial reversal** = abnormality to R wave progression
- **Limb lead reversal** = P and QRS inverted in lead I; normal R wave progression therefore not dextrocardia

<table>
<thead>
<tr>
<th>Complexes</th>
<th>Cause: atrial depolarisation</th>
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<tbody>
<tr>
<td>P Wave</td>
<td>Negative in aVR (maybe in aVF, aVL); biphasic in V1 (and maybe V2, III)</td>
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<td>Normal axis 0 to +75 (if abnormal, ectopic atrial focus)</td>
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<tr>
<td></td>
<td>Duration: 0.12s Size: &lt;2.5mm</td>
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<td></td>
<td><strong>Abnormal:</strong> RAH / RAD / P pulmonale: large P wave in aVF, II, III, +ive in V1 (eg. pulmonary disease, congenital heart disease, incorrect lead placement, dextrocardia)</td>
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<td></td>
<td>P mitrale: in I and II; deeper negative aspect in V1; &gt;0.11s; more prominent in I, aVL, V5-6; most specific ECG sign of LAH, usually due to mitral stenosis</td>
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<tr>
<td>PR Interval</td>
<td><strong>Bialtrial abnormality:</strong> P wave wide and notched and large</td>
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<td>Retrograde P' wave: impulses from near AVN activate atria; P waves inverted in II, III, aVF; may be buried in QRS complex</td>
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<th>Cause: P + PR</th>
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<td><strong>Normal:</strong> duration: 0.12 – 0.2s (Increases with age; decreases as rate increases)</td>
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<tr>
<td><strong>Abnormal:</strong> Short: exercise tachycardia; WPW; low ectopic atrial rhythm, AV junctional rhythm; neonate</td>
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<td>Long: AV block, hyperkalameia, cardiomyopathy, digoxin, beta-blockers, hypothyroid, hypothermia, old</td>
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<tr>
<td>Elevation: myopericarditis (in aVR, V1), atrial infarction</td>
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<td>Depression: myopericarditis (mostly in II, V5-6), atrial infarction, exercise-induced sinus tachycardia</td>
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<th>Cause: depolarization heads L → R in ventricular septum</th>
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<td><strong>Normal:</strong> small in I, II, III (longer and deeper), aVL, aVR, V4, V5, V6</td>
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<tr>
<td>Duration: &lt;1 box wide Size: &lt;4 small boxes / &lt;25% height of R wave deep</td>
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<tr>
<td><strong>Abnormal:</strong> any in V1-3; suggest MI, ventricular enlargement, abnormal ventricular conduction</td>
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<th>Cause: depolarization of L+R ventricular myocardium, heads L and posterior</th>
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<td><strong>Normal:</strong> small R waves in V1-2; large R waves in V5-6; R wave progression</td>
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<th>Cause: depolarization goes posteriorly</th>
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<td><strong>Normal:</strong> S wave in anterior leads; usually largest in aVR and V2; get progressively smaller from V1-6</td>
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<th>R' Wave</th>
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<td>Any positive deflection that occurs following S wave</td>
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**J Point**

**Cause:** junction of terminal QRS and initial ST segment  
**Abnormal:** Osborn wave: in hypothermia; elevation of the J point; prominent in II, III, aVF, V5 and 6  
**Elevation:** hyperkalaemia

| Normal: Axis: | -30 to +90  
| Duration: | <0.12s  
| Size: | 10 – 30mm in precordial leads  
| Abnormal: | Increased amplitude: ventricular enlargement, athletes, normal variant, benign early repolarisation, hyperthyroidism  
| | Decreased amplitude: pericardial effusion, amyloidosis, myxoedema, nephrotic syndrome, anascara, pneumothorax, pleural effusion, restrictive cardiomyopathy, COPD  
| | Increased duration: ventricular enlargement, LBBB, RBBB, ventricular ectopic, pacing, cardiomyopathy, hyperkalaemia, pre-excitation, pericardial effusion, amyloidosis, myxoedema, nephrotic syndrome, anascara, pneumothorax, pleural effusion, restrictive cardiomyopathy, COPD, hypothermia  

**QRS Prolongers = Na channel blockers = delays phase 0 = depolarisation → VT, VF**  
Type Ia (procainamide, quinidine), Type Ic (flecainide)  
TCA’s (abnormal R sided R in aVR), Carbamazepine, Cocaine  
Quinine, chloroquine  
Phenothiazines (eg. Chlorprom, prochlorperazine)  
Antihistamines (diphenhydramine)  
Type IV (Diltiazem, verapamil), Propanolol  
LA’s, amantadine

**Investigation:** look for large R in aVR (>3mm), QRS >100 in II  
**Treatment:** Use NaHCO₃ if: QRS >100, persistent decreased BP despite IVF, significant arrhythmia, seizures

**T Wave**

**Normal:** should be in same direction as QRS  
**Size:** <10mm in precordial leads; may be flattened in aVF; inverted in aVR, III; variable in AVL and V1  
**Abnormal:** Prominent: ACS (within 30mins; if persist, may be post MI), hyperK, BER, myopericarditis, BBB, LVH  
| Inverted: ACS, post-MI, BBB, pericarditis, PE, LVH, digoxin, CNS injury, paced rhythm, intra-abdominal disorders, metabolic syndromes, toxic, pre-excitation; Wellen’s syndrome (deep T wave inversion of precordial leads)  

**ST Segment**

**Normal:** Rule of appropriate discordance: in BBB, ST segment / T wave is directed opposite to terminal portion / major vector of QRS complex; so in RBBB, STE seen in lateral leads; in LBBB, STE seen in R to mid-precordial and inferior leads; this rule also applies to LVH (STE in R to mid-precordial leads) and paced rhythm  
**Abnormal:** Elevated: ACS, Prinzmetal’s angina, MI, acute pericarditis, BER, LV aneurysm, LBBB, RBBB, LVH, paced rhythm, cardiomyopathy, myocarditis, hypothermia, hyperK, post-cardioversion, contusion, CNS injury, Brugada syndrome, pre-excitation  
| Depressed: ACS, ischaemia, NSTEMI, BBB, LVH, paced rhythm, digoxin, rate-related, metabolic syndromes, post-cardioversion, contusion  

For examples, see next page.
Fig 1: LBBB but 1Y ST/T wave changes suggesting MI: inverted T waves in II, III, aVF.

Fig 2: Ventricular paced rhythm with complete heart block (P waves march through with independent rate); ST elevation infero-laterally with reciprocal depression in V1-3.

Complexes (cntd)

**Normal:** duration: <0.44s (no more than half preceding RR interval if HR 60-100); if you want to calculate: QTc = QT (eg. 0.52) / sqRR (I never used this equation, but if you do want to, the square routes of RR’s are below for you to memorise)

- RR 5 boxes = 0.2 → square 0.44
- RR 10 boxes = 0.4 → square 0.63
- RR 15 boxes = 0.6 → square 0.77
- RR 20 boxes = 0.8 → square 0.9
- RR 25 boxes = 1 → square 1
- RR 30 boxes = 1.2 → square 1.1

**Abnormal:** Short: digoxin, hyperCa, hereditary (rare)

Long: long QT, CNS system disease (eg. Increased ICP, SAH)

- metabolic (hypoK, hypoCa, hypoMg, hypothyroid, hypothermia)
- Lange-Neilson (autosomal recessive, assoc with sensorineural deafness)
- Romans-Ward (autosomal dominant)

**QTc Prolongers = K channel blockers = delays phase 3 = repolarization → VT, TdP**

Type Ia (procainamide, quinidine) type Ic (flecainide)

- TCA’s, Carbamazepine, Cocaine
- Quinine, chloroquine
- Phenothiazines (eg. Chlorpromazine, prochlorperazine)
- Antihistamines (diphenhydramine, terfenadine)
- Type III (amiodarone, sotalol), Type IV (not verapamil / diltiazem)
- Sumatriptan, antipsychotics (Haloperidol, risperidone, quetiapine, droperidol), SSRI, methadone, lithium
- Erythromycin, clarithromycin, tetracyclines
- Organophosphates, omeprazole, ondansetron

**Treatment:** NaHCO₃, MgSO₄, K to 4.5-5, overdrive pacing, calcium
Complexes (cntd)

QT Interval

Fig 1: Hypercalcaemia with short ST and QT interval

Fig 2. Long QT with broad T waves with notching—increased risk of TdP, syncope, sudden death; consider for implantable defibrillator, beta-blockers

LVH
S in V1 + R in V5 >35mm

RVH
R in V1 + S in V5 >10mm

Benign Early Repolarisation

Epidemiology: 1-2% population; more common in males, athletes, 20-40yrs, blacks, 23-48% cocaine users with chest pain; more prominent at slow HR’s

J point: junction of QRS and ST segment; often notched; best seen in V4-V5

T waves: usually also tall and peaked

R waves: tall in L precordial leads; R shift of transition zone

QRS: Often notching of downstroke of QRS

ST segment: minimally raised; starting at elevated J point; upwardly concave especially initial part of ST; global (usually precordial, most obvious at V2-4); usually <2mm in precordial but can be up to 5mm; reciprocal ST depression may be seen in aVR if limb lead involved

Differential Diagnosis: pericarditis (but no PR depression and pericarditis doesn’t have large T waves), MI, LBBB, LVH, LV aneurysm, ventricular paced rhythm, high take off
**Athlete’s Heart**

- **ECG:** Sinus bradycardia, sinus arrhythmia, maybe pauses up to 2secs; 1st or 2nd degree heart block; increased R + S wave size; minimal ST elevation at J point (concave); peaked T waves (although may be biphasic or even inverted); deep Q waves in inferior leads; displaced apex beat; ejection systolic murmur; incomplete RBBB
- **CXR:** prominent pulmonary vasculature due to increased CO
- **Echo:** uniform hypertrophy and normal MV
- **Differential diagnosis:** HOCM, LVH, MI, BER, LV aneurysm, pericarditis

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**LV Aneurysm**

Persistent ST elevation (concave / convex) >2/52 following AMI, most commonly in precordial leads; usually associated with Q waves; small amplitude T waves in comparison to QRS; no reciprocal ST depression (unlike in STEMI)