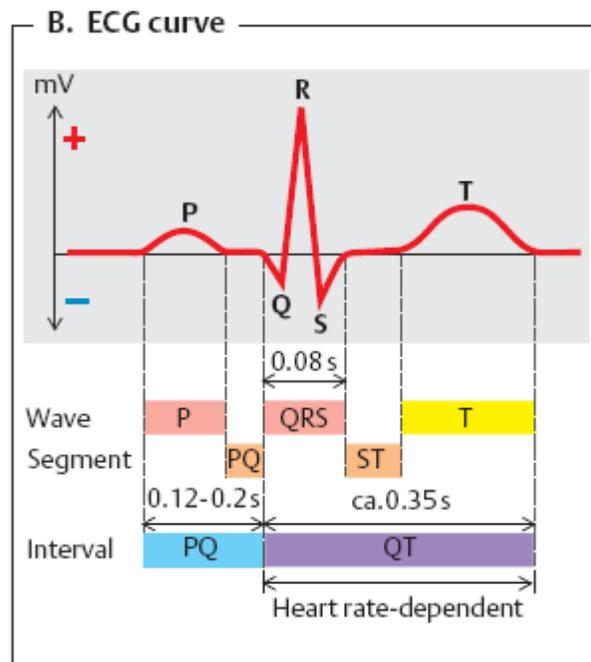


Electrocardiogram

How to read an ECG:

1. Look for calibration marks – speed & amplitude
2. Find the rhythm (sinus rhythm?)
3. Find the frequency (300,150,100,75,60 rule)
4. Find the electric axis of the heart – this is usually considered to be the electrical axis of the QRS complex
5. Evaluate wave characteristics; each wave has four characteristics: duration, amplitude, axis and morphology



To diagnose an ECG as being normal one has to exclude any abnormality.

1. Check the speed of the paper; it usually is 25 mm/s, so that one horizontal small square represents 0.04s, and one big square represents 0.2s.
Then check the amplitude: one vertical small square should represent 0.1 mV.
2. normal sinus rhythm
 - you can identify P waves, and there is a P wave for every cardiac cycle
 - each P wave is followed by a QRS
 - P waves should have normal duration, amplitude and axis
 - P waves should have identical aspects in the same lead
 - PQ interval should be constant (0.12-0.21s)
 - P wave rate 60 - 100 bpm with <10% variation
 - rate <60 = sinus bradycardia
 - rate >100 = sinus tachycardia
 - variation >10% = sinus arrhythmia
3. normal frequency: between 60 -100 bpm
4. normal QRS axis : between + 90 and -30 degrees. Look for the electrical axis in the frontal plane leads only.
5. normal wave characteristics

- normal P waves
- amplitude < 2.5 mm in lead II
- duration < 0.11 s in lead II
- axis: between +45 and +60 degrees
- morphology : rounded, simetrical, usually positive wave, except aVR
 - for abnormal P waves see right atrial hypertrophy, left atrial hypertrophy, atrial premature beat, hyperkalaemia
- normal PR interval (includes the p wave and PQ segment)
- duration 0.12 to 0.21 s (3 - 5 small squares)
 - for short PR segment consider Wolff-Parkinson-White syndrome
 - for long PR interval see first degree heart block and 'trifasicular' block
- normal QRS complex
- amplitude : the tallest R wave should be between 5- 15 mm tall.
 - hypovoltage < 5mm: obesity, pulmonary emphysema, pneumothorax, pericarditis.
 - hypervoltage >15mm: left ventricular hypertrophy
- duration < 0.12 s duration (3 small squares)
 - for abnormally wide QRS consider right or left bundle branch block, ventricular rhythm, hyperkalaemia, etc.
- axis: normal QRS axis range (+90 to -30 degrees); this implies that the QRS be mostly positive (upright) in leads II and I.
- morphology:
 - Frontal leads:
 - Normal q-waves reflect normal septal activation (beginning on the LV septum) in the lateral leads (DI, aVL, as well as in V5, V6 of the precordial leads). Normally they should not be present in other leads. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction, that do not meet the following criteria:
 - duration <0.04s
 - amplitude <25% the of the R wave.
 - morphology : negative, sharp, small wave, before the R wave, only in the lateral leads.
 - Precordial leads: there are three models of QRS complexes
 - V1, V2 – rS model (right model)
 - V3, V4 – RS model (transition leads)
 - V5, V6 – qRs model (left model)
 - Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.
 - In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.
 - The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
 - Small "septal" q-waves may be seen in leads V5 and V6.
- normal QT interval
- it is heart rate dependent
- calculate the corrected QT interval (QTc) by dividing the QT interval by the square root of the preceding R - R interval. Normal = 0.42 s.
- causes of long QT interval
 - myocardial infarction, myocarditis, diffuse myocardial disease
 - hypocalcaemia, hypothyrodism
 - subarachnoid haemorrhage, intracerebral haemorrhage

- drugs (e.g. sotalol, amiodarone)
- hereditary
- Romano Ward syndrome (autosomal dominant)
- Jervill + Lange Nielson syndrome (autosomal recessive) associated with sensorineural deafness
- normal ST segment
- no elevation or depression
 - causes of elevation include acute MI (e.g. anterior, inferior), left bundle branch block, normal variants (e.g. athletic heart, Edeiken pattern, high-take off), acute pericarditis
 - causes of depression include myocardial ischaemia, digoxin effect, ventricular hypertrophy, acute posterior MI, pulmonary embolus, left bundle branch block
- normal T wave
- duration: hypokalaemia increases the duration of the T wave
- amplitude: it is considered in relation to the R wave; ideally it should be $\frac{1}{3}$ R, but it is considered normal within the $\frac{1}{4}$ R – $\frac{1}{2}$ R interval.
 - causes of tall T waves include hyperkalaemia, hyperacute myocardial infarction and left bundle branch block
 - causes of small, flattened or inverted T waves are numerous and include ischaemia, age, race, hyperventilation, anxiety, drinking iced water, LVH, drugs (e.g. digoxin), pericarditis, PE, intraventricular conduction delay (e.g. RBBB) and electrolyte disturbance.
- axis: (+30 , +60 degrees)
- morphology: rounded, asymmetrical wave.
 - the first sign of ischemia can be a symmetrical T wave.
- normal U wave
- U wave amplitude is usually $< \frac{1}{3}$ T wave amplitude in same lead
- U wave direction is the same as T wave direction in that lead
- U waves are more prominent at slow heart rates and usually best seen in the right precordial leads.
- Origin of the U wave is thought to be related to afterdepolarizations which interrupt or follow repolarization.