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ECG Made EASIER

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ECG MADE EASIER

WHAT IS AN ECG?

The electrocardiogram (ECG) is a powerful tool which is both easy to use and applicable to numerous clinical scenarios. An ECG can be used to diagnose myocardial injury, identify one of many arrhythmias, highlight the acute effects of a large pulmonary embolus or the chronic effects of established hypertension, or simply provide reassurance to an executive having an annual medical checkup[1].

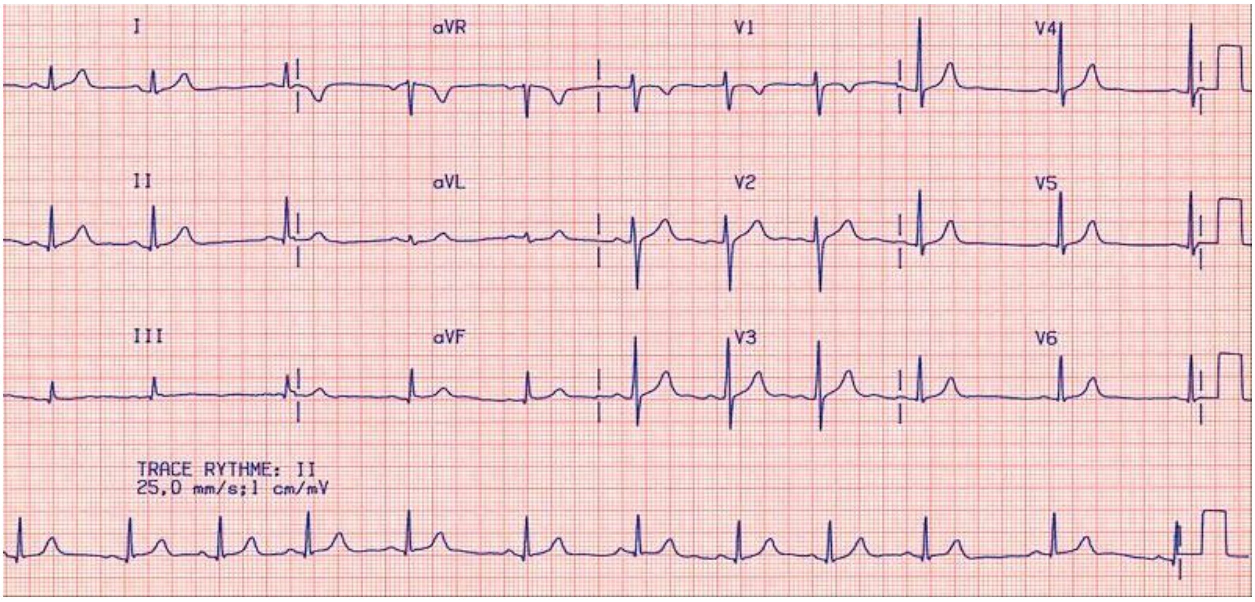


Figure 1: Normal ECG

An ECG is the primary monitor for diagnosis of cardiac conduction abnormalities and rhythm disturbances. An ECG is a tracing created with electrodes on the skin that amplify cardiac electrical potentials. The normal ECG tracing is a complex composed of three waveforms:

- ✓ P wave (atrial depolarization),
- ✓ QRS complex (ventricular depolarization), and
- ✓ T wave (ventricular repolarization)

The direction of the electrical signal relative to a ground electrode determines the direction of the deflection seen on the ECG. Positive signals are represented by deflections above the isoelectric line and negative signals are represented as deflections below the isoelectric line[2].

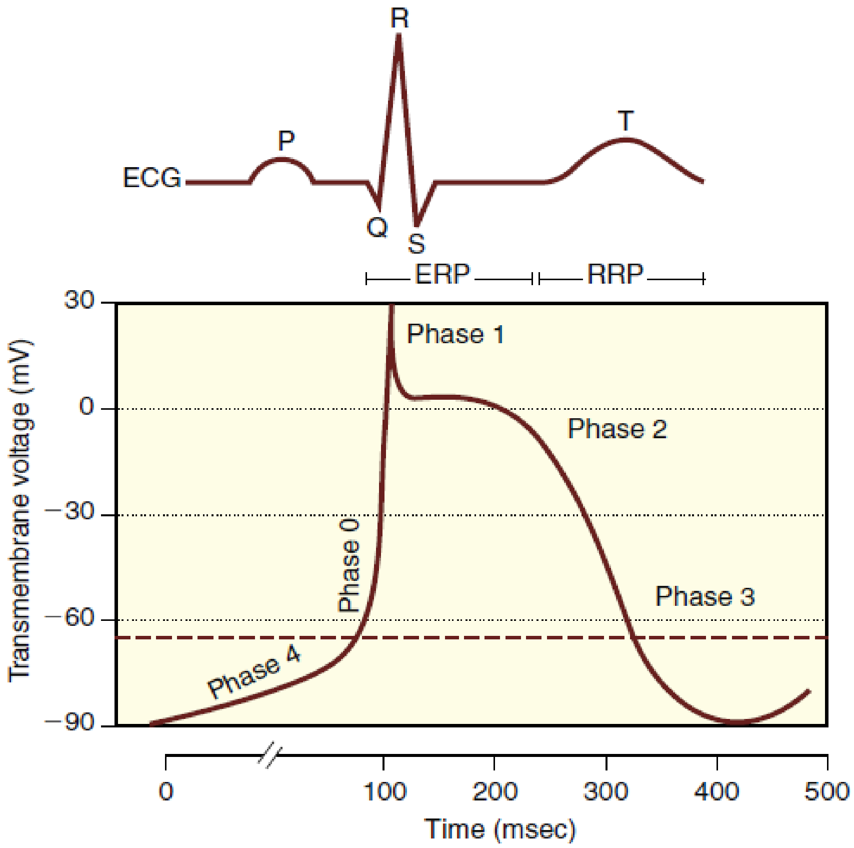
The PR interval is the time between atrial depolarization and initiation of ventricular depolarization. The QRS complex corresponds to the wave of depolarization moving downward from the AV node to the right and left ventricles.

The ST segment starts from the end of the S wave (end of ventricular contraction / depolarisation) and ends at the beginning of the T wave and represents the period from ventricular depolarization to the initiation of ventricular repolarization. Even though it is usually isoelectric, there may be a 1mm elevation without any cardiac abnormality. ST segment depression on the other hand is never normal.

The T wave and the QRS complex should both deflect in the same direction and the T wave amplitude should be a maximum of 5 mm in standard leads or 10 mm in precordial leads. Normal values for the QT interval should be corrected for the heart rate

ELECTROPHYSIOLOGY OF THE CONDUCTION SYSTEM [2]

In the resting state, the outside of a cardiac cell is positive relative to the inside (and vice versa). Impulses are conducted through the heart by progressive depolarization. Cardiac muscle cells have a resting membrane potential of -80 to -90 mV. The resting gradient is maintained by membrane bound $Na^{+}K^{+}$ATPase that amasses potassium intracellularly and moves sodium extracellularly. When the sodium and calcium channels open in response to neighbouring cell membrane charge shifts the membrane potential increases. When the membrane potential reaches + 20 mV, an action potential (or depolarization) occurs after which cells are refractory to subsequent action potentials for phases 1, 2 & 3 of the depolarization potential.



[2]Figure 2: Transmembrane action potential generated by an automatic cardiac cell and the relationship of this action potential to the ECG trace.

Phase 4 undergoes spontaneous depolarization from the resting membrane potential (-90 mV) until the threshold potential (broken line) is reached, whence depolarization (phase 0) occurs, corresponding to the QRS complex on the ECG.

Phases 1, 2 and 3 represent repolarisation, with phase 3 corresponding to the T wave on the ECG. The effective refractory period (ERP) is the time during which cardiac impulses cannot be conducted, regardless of the intensity of the stimulus (Phase 1).
During the relative refractory period (RRP) (Phases 2&3), a strong stimulus can initiate an action potential.

WHY DO AN ECG PRE-OPERATIVELY – IS IT NECESSARY?

ACC/AHA 2007 Recommendations for Preoperative Resting 12-Lead ECG[3]

Class I

1. Preoperative resting 12-lead ECG is recommended for patients with at least 1 clinical risk factor who are undergoing vascular surgical procedures. (Level of Evidence: B) [3]
2. Preoperative resting 12-lead ECG is recommended for patients with known CHD, peripheral arterial disease, or cerebrovascular disease who are undergoing intermediate-risk surgical procedures. (Level of Evidence: C) [3]

Class IIa

1. Preoperative resting 12-lead ECG is reasonable in persons with no clinical risk factors who are undergoing vascular surgical procedures. (Level of Evidence: B) [3]

Class IIb

1. Preoperative resting 12-lead ECG may be reasonable in patients with at least 1 clinical risk factor who are undergoing intermediate-risk operative procedures. (Level of Evidence: B) [3]

Class III

1. Preoperative and postoperative resting 12-lead ECGs are not indicated in asymptomatic persons undergoing low-risk surgical procedures. (Level of Evidence: B) [3]

The decision to perform a preoperative EGC should be based on the patient's history, co-morbidities and examination. Appropriate testing should be performed on all patients with signs or symptoms of cardiovascular disease, regardless of their preoperative status[4].

A normal ECG adds little to preoperative evaluation apart from providing a baseline for comparison postoperatively. Although abnormal tracings are quite common, few are likely to change management.

According to ACC/AHA guidelines[3], in major non-cardiac surgery, the presence of a pathological Q wave on the preoperative ECG (found in 17% of the population) was associated with an increased risk of major cardiac complications (myocardial infarction, pulmonary oedema, heart block, ventricular fibrillation or cardiac arrest). ECG abnormalities other than Q waves are minor predictors of complications and may incur costly evaluation (the yield of which is quite low) and delay necessary surgery.

A normal ECG does not exclude cardiac disease because the specificity of an abnormal ECG for predicting postoperative cardiac complications is only 26%[5]

Ideally, a 12-lead ECG should be obtained within 30 days of surgery, in patients with stable cardiac pathology in whom a preoperative ECG is indicated[3].

TECHNICAL ASPECTS OF AN ECG

- Patient name
- Date and time
- Quality
  - Baseline wandering
  - Interference
- Times and speeds
  - Standard sweep speed 25mm/sec
    - small square = 0.04 s (1mm)
    - large square = 0.2 s (5mm or 5 small squares)
  - Rhythm strip = 25cm or 10 seconds
- Standard voltage setting
  - = 1mV and should move the stylus vertically 10mm (2 large squares)

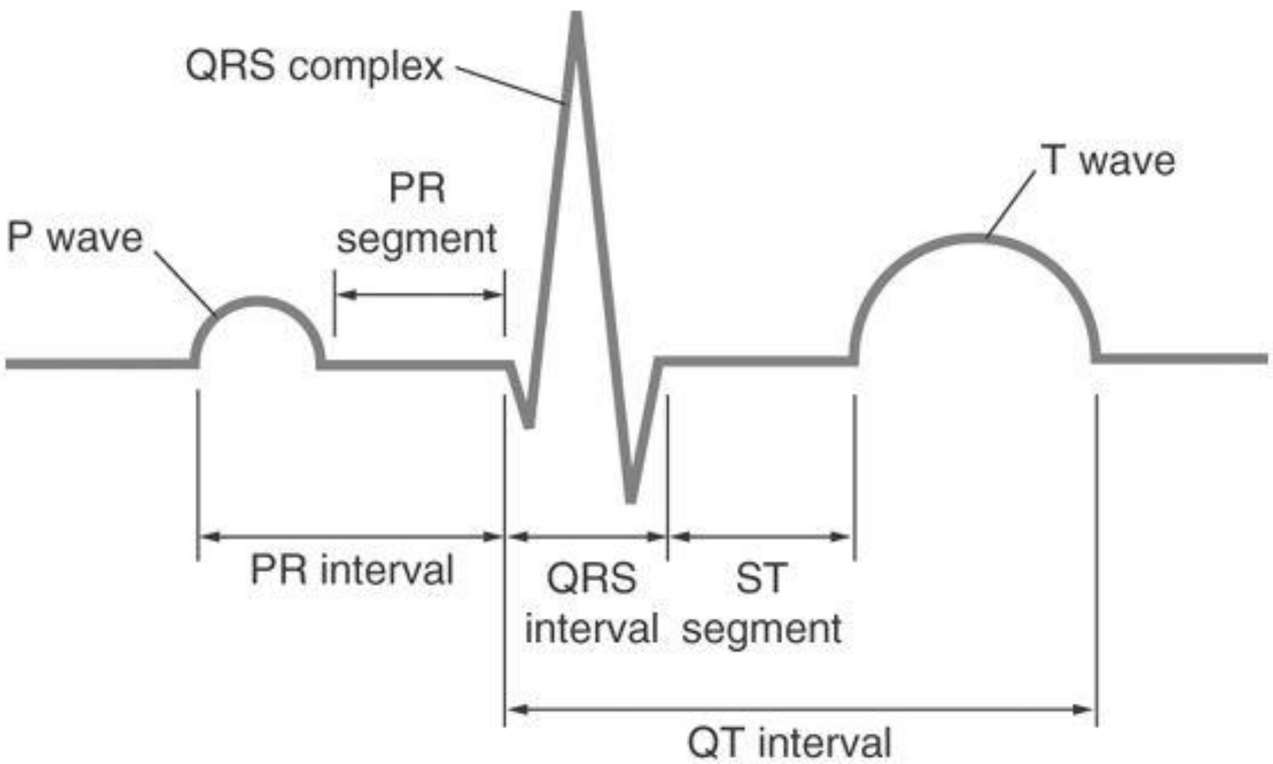


Figure 3: Normal Cardiac Cycle[1]

TEN STEP METHOD FOR READING ECGS

1. **Rate**
   - 300 / (R-R interval) (R-R interval in big squares)
     OR (for AF)
   - (# QRS complexes in rhythm strip) x6 (10 sec strip - speed 25mm/sec)

   Tips
   - Find an R wave that falls on one of the heavy lines
   - Tally the number of large squares before the next R wave

2. **Rhythm**
   - Always ask 4 questions[1]:
     - Are normal P waves present?
     - Are QRS complexes narrow or wide?
     - What is the relationship between P waves and QRS complexes?
     - Is rhythm regular or irregular?

   - Check if rhythm is sinus
     - P-waves must be normally shaped and constant
     - There must be a P-wave before every QRS complex
     - There must be a QRS complex after every P-wave
     - Rhythm should be regular (R-R intervals are a constant distance apart)
     - May be sinus tachycardia (>100) or sinus bradycardia (<60)
     - Sinus arrhythmia: >10% variation of heart rate with respiration

   - Card method
     - Place an index card above the first two R waves on the left side of the rhythm strip. Using a sharp pencil, mark on the index card above the two R waves. Measure from R wave to R wave across the rhythm strip, marking on the index card any variation in R wave regularity. If the rhythm varies by 0.12 second (3 small squares) or more between the shortest and longest R wave variation marked on the index card, the rhythm is irregular. If the rhythm doesn't vary or varies by less than 0.12 second, the rhythm is considered regular.

3. **Axis**

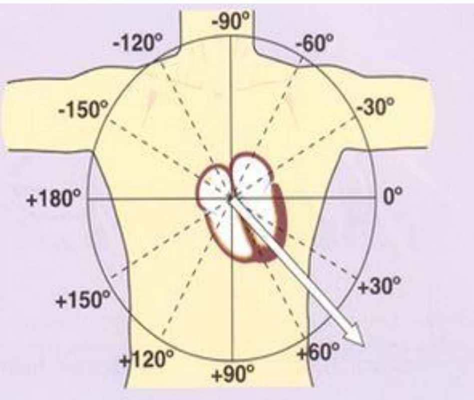


Figure 4: Calculation of Axis[6]

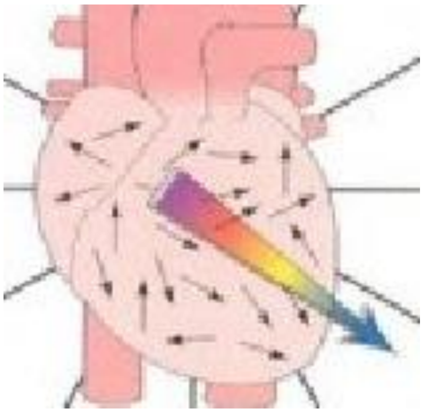


Figure 5: Average movement of depolarization[7]

The axis is the direction of the mean electrical vector[6] representing the average movement of depolarization (direction of current flow), which spreads through the heart to stimulate the myocardium to contract.

The usual direction of ventricular depolarization is downward and to the patient's left which is the direction of the mean QRS vector.

- Axis is calculated using the following:

a. Rule of thumb

	Lead I	Lead aVF	Vectors
Normal axis	Positive	Positive	-30° to +90˚
Left axis deviation	Positive	Negative	-30° to +90˚
Right axis deviation	Negative	Positive	+90° to +180˚
Extreme Right axis deviation	Negative	Negative	+180° to -90˚

b. Standard vector diagram

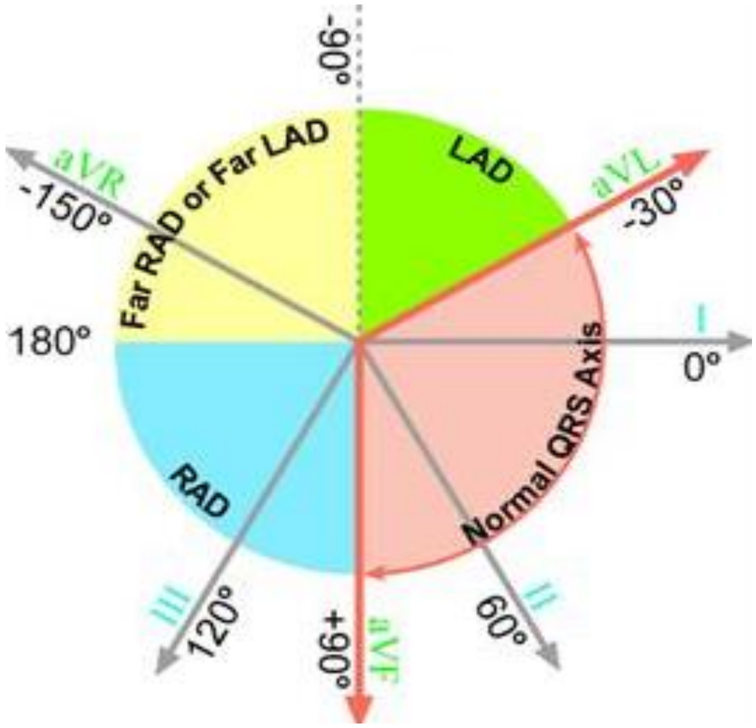


Figure 6: Standard vector diagram to calculate QRS axis[8]

c. To more accurately calculate the axis, first identify the axis quadrant. The axis lies at 90° to the limb lead in that quadrant where the QRS is the most isoelectric.

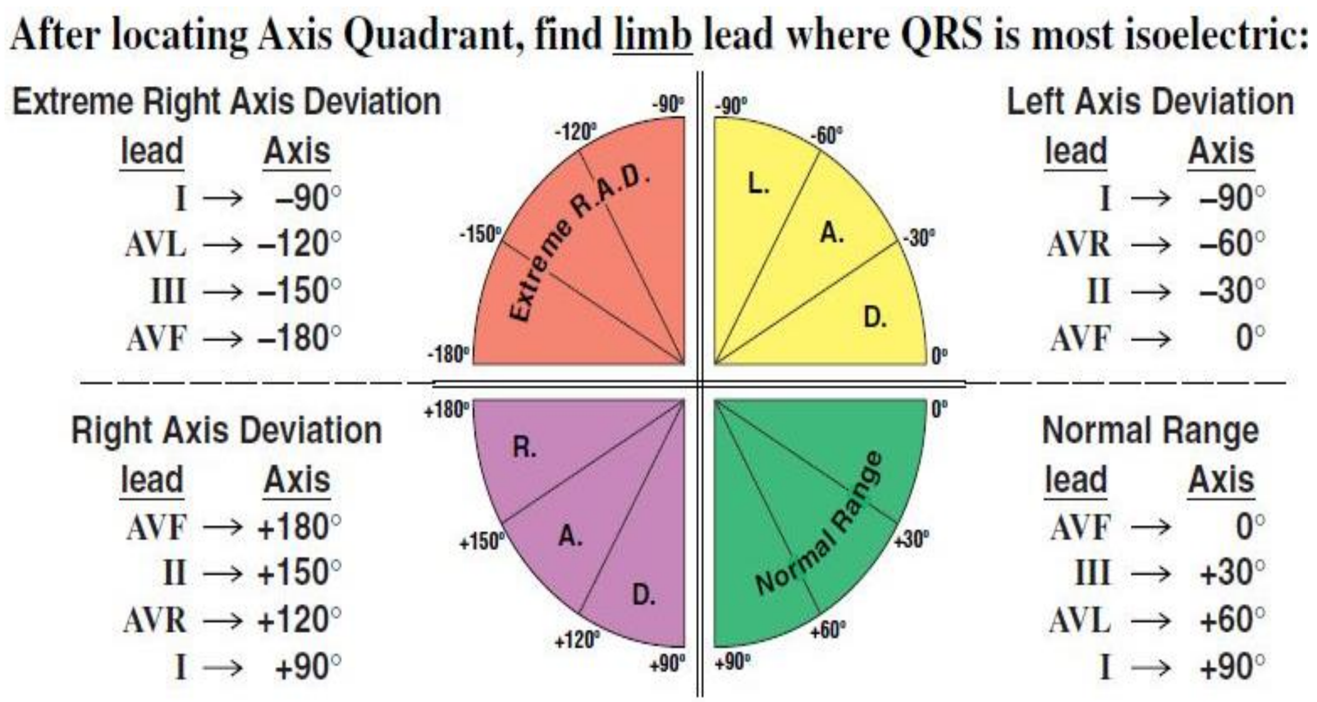


Figure 7: Methodology for locating the axis more precisely[6]

- Mean QRS Vector tends to point:
  - Toward ventricular hypertrophy
  - Away from myocardial infarction
- Axis deviation = frontal plane
- Axis rotation = horizontal plane

- Causes of LAD (Left Axis Deviation)[11]
  - Inferior MI
  - Left anterior hemiblock
  - WPW Syndrome
  - VT from LV focus

- Causes of RAD (Right Axis Deviation)[12]
  - Right ventricular hypertrophy
  - Pulmonary embolization
  - Anterior lateral MI
  - Left posterior hemiblock
  - WPW Syndrome

4. P waves (Atria)

- Morphology
  - P waves should all have the same shape as they normally all originate from the SA node. Varying shape implies different areas of origin e.g. ectopic beats
  - Normal height ≤ 2.5mm
  - Normal width ≤ 0.11sec

5. PR interval

- Time taken for the AV node to depolarize
- From start of P wave to start of QRS complex
- Normally 0.12-0.2 sec (3 -5 small squares)
- PR interval <0.12sec – abnormally rapid AV node conduction down an accessory pathway eg. Wolf-Parkinson-White Syndrome (see later), Lawn-Ganong-Levine syndrome or nodal rhythm. Look for slurring of the upstroke (delta waves)

6. Q waves

- Initial negative deflection of the QRS complex – may or may not be present
- Normal occurrence in septal leads (V_3 and V_4)
- Significant Q waves – > 0.04sec (1 small block) wide and 25% of the height of the subsequent R wave

7. QRS Complexes

- Examine QRS complex for size / height, shape and duration
- Normal width / duration ≤0.12 sec (3 small squares). Longer duration indicates conduction delays
- Look for R wave progression with increasing size from V_1 to V_6
- At V_3 – V_4 usually get transition from dominant negative to dominant positive
- Various measurements of QRS will indicate either right or left ventricular hypertrophy

8. T waves

- Should be upright in leads V_{2-5}
- May be flat or inverted in V_1 or V_2 – However if upright in V_1 must also be upright in V_2
- Height should be 1/8 – 2/3 of preceding R-wave
- Abnormal if inverted in leads I, II, V_{4-6}
- **U-wave**: if present should be <25% of preceding T-wave

9. ST Segment

- Should not deviate from iso-electric line by >1mm
- Elevation >1mm implies infarction
- Depression >0.5mm implies ischaemia

10. QT interval

- From the beginning of the QRS complex to the termination of the T-wave
- Usually < 400msec
- Varies inversely with rate
- Corrected QT interval [(QT interval in sec)/√R-R' interval]
- Normal corrected QT interval 0.35-0.42sec (< 420msec)

MORE COMMON ECG ABNORMALITIES

Hypertrophy and Enlargement [1]

The ECG can reveal whether a certain ventricular or atrial chamber is hypertrophied or enlarged in conditions such as valvular diseases, sustained hypertension, and inherited cardiac muscle disorders.

Hypertrophy refers to an increase in muscle mass and is usually caused by pressure overload.
Enlargement refers to dilatation of a particular chamber and is usually caused by volume overload.
Enlargement and hypertrophy often co-exist.

Three changes can occur on a wave of the ECG when a chamber hypertrophies or enlarges:

a. Increase duration of wave: chamber can take longer to depolarize.
b. Increase amplitude of wave: chamber can generate more current and thus a larger voltage
c. Mean electrical vector / axis may shift: larger percentage of the total electrical current can move through the expanded chamber.

<u>ATRIAL HYPERTROPHY</u> (look at leads II and V_1)

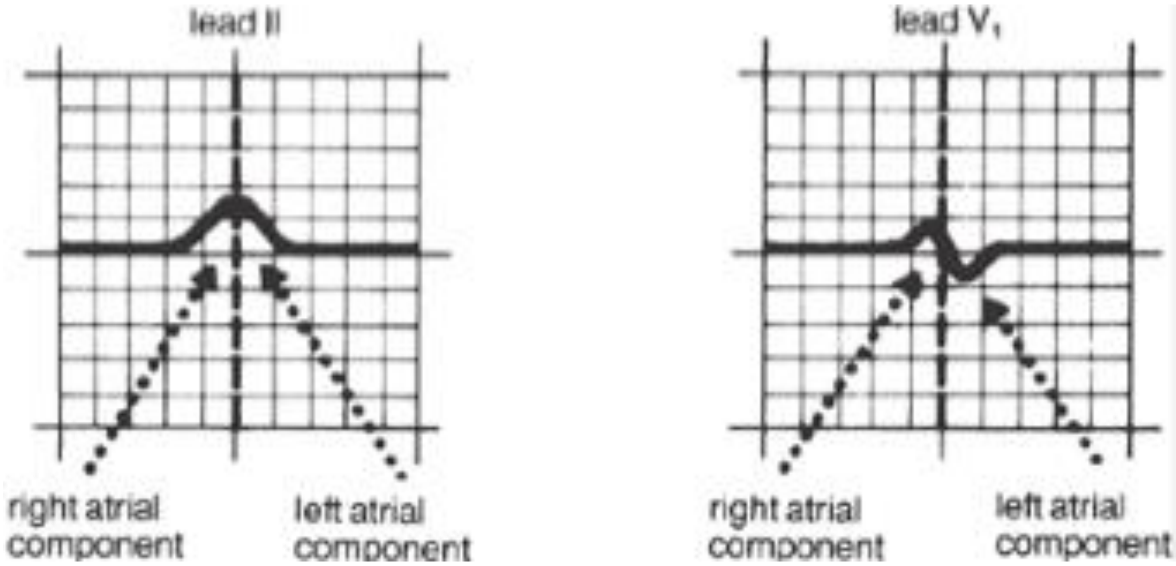


Figure 8: Normal p waves in lead II and lead V_1[1]

Right Atrial Hypertrophy – p pulmonale (increased height p wave)

- P wave height >2.5mm in II, III, aVF
- Referred to as p-pulmonale because caused by severe lung pathology

Left Atrial Hypertrophy – p mitrale (increased duration p wave)

- P wave duration longer than 0.12 s (3 small blocks) in II
- Bifid p wave in I, II, aVF, aVL
- Biphasic P wave in V1 (negative component area > than that of positive component)

VENTRICULAR HYPERTROPHY

Right Ventricular Hypertrophy (RVH)

- Dominant R in V_1 (R, Rs, RR', qR or qRs) + frontal plane axis > $+90^o$
- Persistent S wave V6
- RAD
- Strain pattern of ST segment depression and T-wave flattening / inversion strongly suggests hypertrophy (V_{1-3})

Causes of a dominant R wave in V_1:

- ✓ RVH
- ✓ RBBB
- ✓ Dextrocardia
- ✓ Duchenne's muscular dystrophy
- ✓ Variants of WPW
- ✓ Dextrocardia
- ✓ Incorrect lead placement

Left Ventricular Hypertrophy (LVH)

There are numerous criteria used for diagnosing LVH. The more criteria that are positive, the greater the reliability of ECG diagnosis of LVH.

- Sum of S wave in V_1 (or V_2) + R wave in V_5 (or V_6) = >35 mm (Sokolow's criteria) *
- S wave in V_1, V_2, V_3 = > 30 mm
- R wave in V_6 = > 26 mm
- R wave in V_6 = > 18 mm
- R wave in aVL = > 13 mm *
- R wave in aVF = > 20 mm
- R wave in I = > 14 mm
- Strain pattern of ST segment depression and T-wave flattening / inversion strongly suggests hypertrophy (V_{4-6})

- ST segment depression, T wave flattening / inversion in leads facing left ventricle (V_5, V_6)
- May see LAD

Abnormalities of Rhythm

There are numerous ways to classify abnormalities of rhythm. A simple classification can be found in the book "Rapid Interpretation of EKG's" by Dr D Dubin[6].

1. Irregular Rhythms
    a. Sinus arrhythmia – irregular rhythm that varies with respiration. Identical p waves.
    b. Wandering pacemaker & Multifocal atrial tacchycardia
    c. Atrial fibrillation - irregular baseline and no recognizable P-waves, QRS complexes are irregularly irregular

2. Escape Rhythms
    a. Atrial, junctional, ventricular - with progressively decreasing rate: 60-80/min > 40-60/min > 20-40/min

3. Premature beats – from an irritable automaticity focus may suddenly discharge and produce a:
    a. Premature atrial beat
    b. Premature junctional beat
    c. Premature ventricular contraction – may be: multiple, multifocal, in runs, or coupled with normal cycles

4. Tacchyarrhythmias
    a. Paroxysmal (sudden) tachycardia (150-250/min)
        i. Supraventricular tachycardia (SVT)
        ii. Ventricular tachycardia
    b. Flutter (250-350bpm)
        i. Atrial flutter – 'saw tooth' pattern. Many flutter waves needed to produce a ventricular response
        ii. Ventricular flutter – rapid series of smooth sine waves
            - ✓ Torsades de Pointes
    c. Fibrillation
        i. Atrial fibrillation
        ii. Ventricular fibrillation

5. Conduction blocks
    a. AV block – delay / prevent atrial impulses reaching ventricles
        i. 1^o AV block
            - ✓ Consistently prolonged PR interval >0.2 s (> 1 large block)
        ii. 2^o AV block
            - ✓ Wenckebach (Type I): progressive lengthening of PR interval until a P wave is not followed by a QRS. Cycle then repeats
            - ✓ Mobitz (Type II): Regular, punctual P waves but some not followed by a QRS. Can be quantified ie. 2:1, 3:1
        iii. 3^o (complete) AV block
            - ✓ Complete atrioventricular dissociation. P-waves and QRS complexes are completely independent of each other.

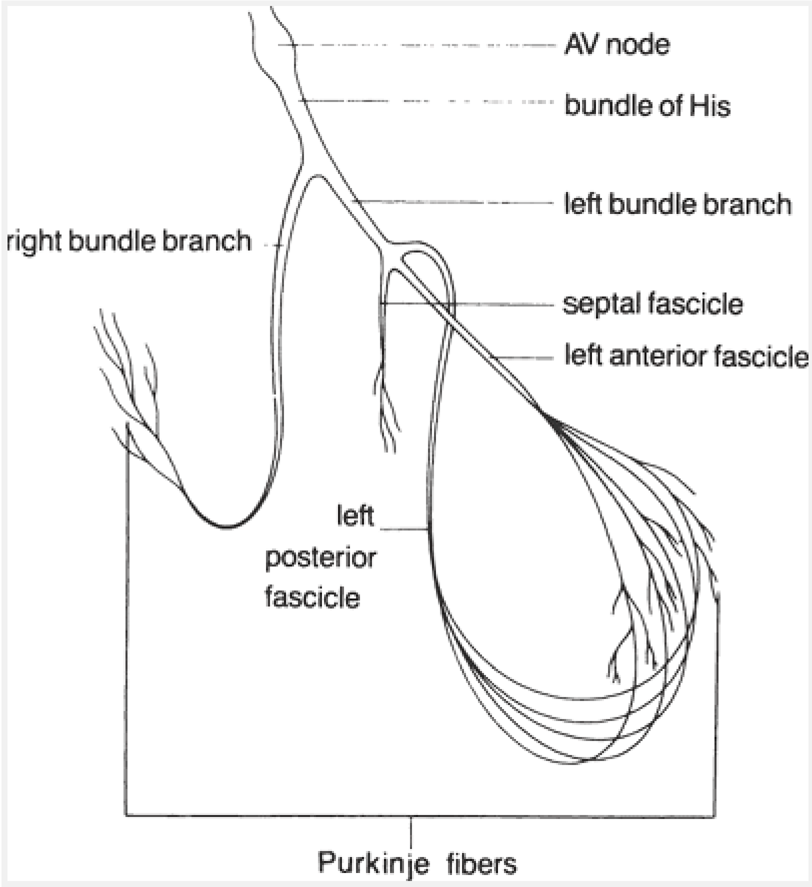


Figure 9: Anatomy of the ventricular bundle branches[1]

b. Bundle Branch Block
   i. RBBB
      - ✓ QRS > 0.12 s plus,
      - ✓ 2nd R wave in V_1 = rSR
      - ✓ Reciprocal deep S waves in I, aVL, V_5, V_6
      - ✓ T-wave inversion leads V_1-V_3
      - ✓ MaRRoW
      - ✓ Normal criteria for QRS complexes and for frontal plane axis apply
   ii. LBBB
      - ✓ QRS > 0.12 s plus,
      - ✓ Large T waves in opposite direction to main complex
      - ✓ Wide R in V_6 (+/- notched)
      - ✓ Absent Q wave in V_5, V_6,
      - ✓ WiLLiaM
      - ✓ Normal criteria for QRS complexes, ST segments and T waves do NOT apply thus beware of making incorrect diagnosis of myocardial infarction or LVH
      - ✓ Normal criterion for frontal plane axis does apply

c. Hemiblock (Essentially normal QRS duration)
   i. LAHB (Left Anterior Hemiblock)
      - ✓ LAD more negative than - 30^o +
      - ✓ Small Q wave in V_1 and aVL
      - ✓ Small R waves are visible in II, III and aVF (ie, absence of Q wave evidence of inferior infarction)
      - ✓ LAHB and Inferior MI are the two commonest causes of LAD

ii. LPHB (Left Posterior Hemiblock)
    - ✓ Usually associated with inferior infarction
    - ✓ RAD
    - ✓ Deep or wide S wave in I
    - ✓ Q wave in III

d. Bifasicular block
   i. RBBB and left anterior hemiblock
      - ✓ LAD (-30^o to +90^o)

   ii. RBBB and left posterior hemiblock
      - ✓ RAD
         - RBBB and left axis deviation

e. Trifasicular block
   - ✓ bifasicular block and first degree heart block

PRE-EXCITATION SYNDROMES

Wolff-Parkinson-White Syndrome (accessory conduction pathways)

- Accessory Bundle of Kent causes ventricular pre-excitation and paroxysmal tacchycardia
  - ✓ PR interval less than 0.12 seconds
  - ✓ Wide QRS complexes: > 0.12 s
  - ✓ Delta wave seen in some leads ie. Slurring of initial 0.03-0.05 s of QRS complex - creates impression of a 'shortened' PR interval and lengthened QRS
- In the presence of ventricular pre-excitation the normal criteria for QRS complexes, ST segments and T waves do not apply
- Do not make incorrect diagnosis of LBBB or MI

The pneumonic **HIS DEBS** can be used to identify treatable, precipitating arrhythmogenic factors:

H – Hypoxia
I – Ischaemia & Irritability
S – Sympathetic stimulation
D – Drugs
E – Electrolyte disturbances
B – Bradycardia
S – Stretch (enlargement / hypertrophy)

Myocardial Ischaemia / Infarction

As blood flow to the heart is compromised, the ECG evolves from an ischaemic pattern to that of injury and necrosis. The following 3 aspects of the ECG can be used to assess myocardial injury.

1. Q waves

- Significant Q wave is > 1mm wide (0.04 s duration) OR
- > 1/3 amplitude of QRS complex
- Old Q waves persist so must assess ST segments and T waves to determine if acute

2. T waves

- Inverted T wave of ishaemia is symmetrical (left half and right half are mirror images).
- Asymmetrical T wave inversion implies other pathology eg. LVH
- Normally T wave is upright when QRS is upright, and vice versa.

3. ST segments

- ST segment changes imply an acute process
- Elevation >1mm implies infarction (associated with significant Q waves)
- A tiny 'non-Q wave infarction' can occur with significant ST segment elevation but without associated Q waves
- Depression >0.5mm implies ischaemia or can represent 'subendocardial' infarction

- Myocardial infarction
  - T wave becomes abnormally tall and ST segment begins to rise within a few hours
  - Within 24 hrs T-waves invert and ST elevation begins to decrease
  - Q waves occur within a few days, usually persist but may resolve in 10% of patients
  - T wave inversion may or may not persist
  - ST elevation rarely persists but may remain elevated in ventricular aneurysm

Infarction location / Coronary Vessel Involvement:

- Posterior
  - Right coronary artery
  - Tall R waves with ST depression in V_1 & V_2
  - Mirror image changes in V_{1-2}
- Lateral
  - Circumflex coronary artery
  - Q waves in I & aVL
- Inferior
  - Right or Left coronary artery
  - Q waves in II, III, aVF
- Anterior
  - Anterior descending coronary artery
  - Q waves in V_{1-4}
- Anterolateral
  - V_{4-6}, I, aVL

Electrolyte Disturbances

Because the electrical events of the heart are dependent on electrolytes, it follows that various electrolyte disorders can affect cardiac conduction and even lead to sudden death if untreated.

1. Potassium (K^{+})
   a. HyperK^{+}
      - Progressive changes in the ECG that can end with ventricular fibrillation and death
      - Likened to tying a piece of string to the T wave and gradually pulling up
      - T waves peak, PR interval prolonged, P waves gradually flatten and disappear
      - QRS widens and combines with the T wave eventually producing ventricular fibrillation
   b. HypoK^{+}
      - ST segment depression
      - Flattening of the T wave
      - U wave

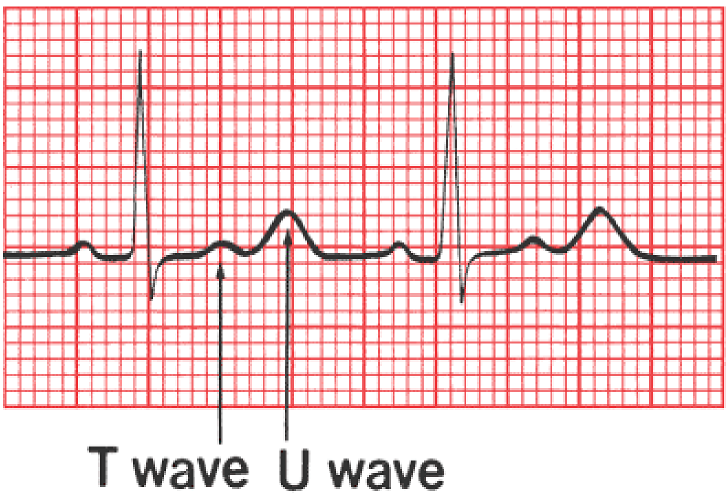


Figure 10: U wave[1]

2. Calcium (Ca^{2+})
   a. HyperCa^{2+}
      - Increased Ca^{2+} accelerates both ventricular depolarization and repolarisation – manifests as
      - shortened QT interval
   b. HypoCa^{2+}
      - Prolonged QT interval

Miscellaneous

1. Hypothermia[1]
   - ECG changes occur when body temperature drops below 30^{o}C.
   - Everything slows leading to sinus bradycardia and prolongation of all the segments and intervals
   - J wave (Osborne wave) is an abrupt ST segment elevation at the J point with an equally rapid drop to baseline

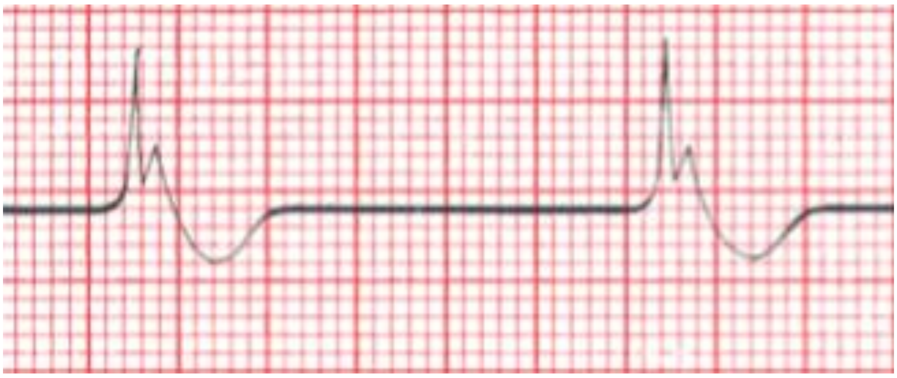


Figure 11: J wave of hypothermia[1]

2. Pulmonary Embolus (PE)[1]
   - Right ventricular hypertrophy with repolarization changes, due to acute right ventricular dilatation[1]
   - RBBB[1]
   - A large S wave in lead I, a deep Q wave in lead III and an inverted T wave in lead III is called the S1Q3T3 pattern[1]
   - Sinus tachycardia and atrial fibrillation are not uncommon[1]

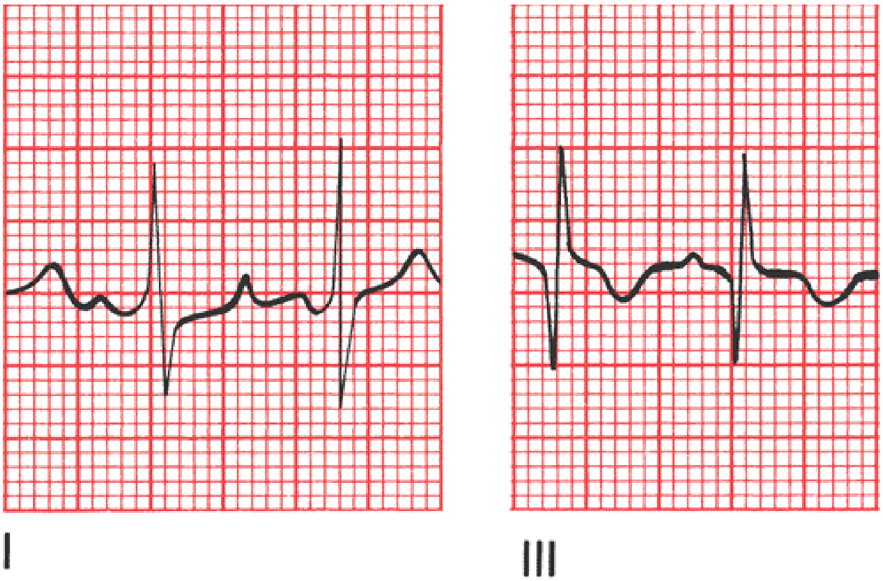


Figure 12: S1Q3 pattern of a massive PE

3. COPD
   - Low voltage
   - Right axis deviation
   - Poor R wave progression in precordial leads
   - If progression to cor pulmonale and right sided heart failure:
     - P pulmonale & RVH with repolarisation abnormalities

TABLE WITH NORMAL VALUES

PARAMETER	NORMAL VALUES
Time = horizontal axis	One small square = 0.04 s
	One large square = 0.2 s (5 small squares)
Standard sweep speed	25mm/s – strip length 25cm or 10 s
Voltage = vertical axis	One small square = 0.1 mV
	One large square = 0.5 mV (5 small squares)
Standard voltage setting	10 mm = 1 mV
Rhythm	Each p-wave followed by a QRS
	Rate 60 – 100 bpm
	<10% variation with respiration
QRS axis	-30˚ to +90˚
P wave	Height <2.5 mm in lead II
	Width <0.11 s in lead II
PR interval	0.12 – 0.2 s (3 – 5 small squares)
QRS complex	< 0.12 s (3 small squares)
QT interval	< 0.42 s (corrected QTc)
	QTc = QT interval in sec / √R-R’ interval
ST segment	< 1mm elevation / depression
T wave	Upright and asymmetrical (except AVR)
U wave	Not usually seen. Upright

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