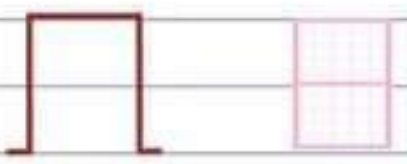
ECG Interpretation Notes

1. 12-lead vs rhythm strip, rate (normal 25 mm/s). Check calibration: (5mm wide, 10mm high = 1mV)



2. Rate: Normal 60 – 100/ min method: 300/RR interval (large squares) or number of QRS complexes x 6 (if paper speed is 25mm/s)

3. Rhythm: Check pattern – regular/ regularly irregular/ irregularly irregular.

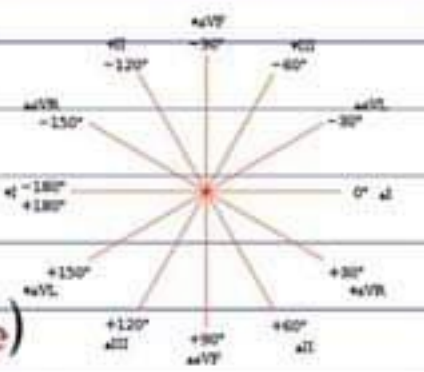
4. Axis

Normal QRS axis -30 to 90

Left Axis Diviation (LAD)

Right Axis Diviation (RAD)

NW axis Rotation (clockwise/ anticlockwise)



5. P wave: Present? (esp check II and V1) Amplitude + duration: normal ? <3mm high and wide (LAE/RAE/both) Contour: normal — inverted aVR, biphasic V1, upright I, II, aVF, V2 -V6

PR interval + PR segment duration 0.12- 0.2s normal/ short/ long/ varying heart blocks: 1st, 2nd type I and II, 3rd degree.

ECG Interpretation Notes continue.....

6. QRS complex: Duration: 60-110msec normal/ wide; R wave peak time Amplitude: normal/ large voltage/ low voltage/ alternans Morphology: notched/ RBBB/ LBBB

Q wave: Normal: <25% R in I, aVL, aVF, V4,5,6.
Pathological: V2,3 >0.02s, other >0.03s + >1mm deep

R wave Transition: normal V3-4, early: R>S in V1/2 Poor R Wave Progression: R <3mm V3

7. ST Segment Displacement: elevation/ depression (J point vs TP interval) Contour: horizontal / upsloping / downsloping

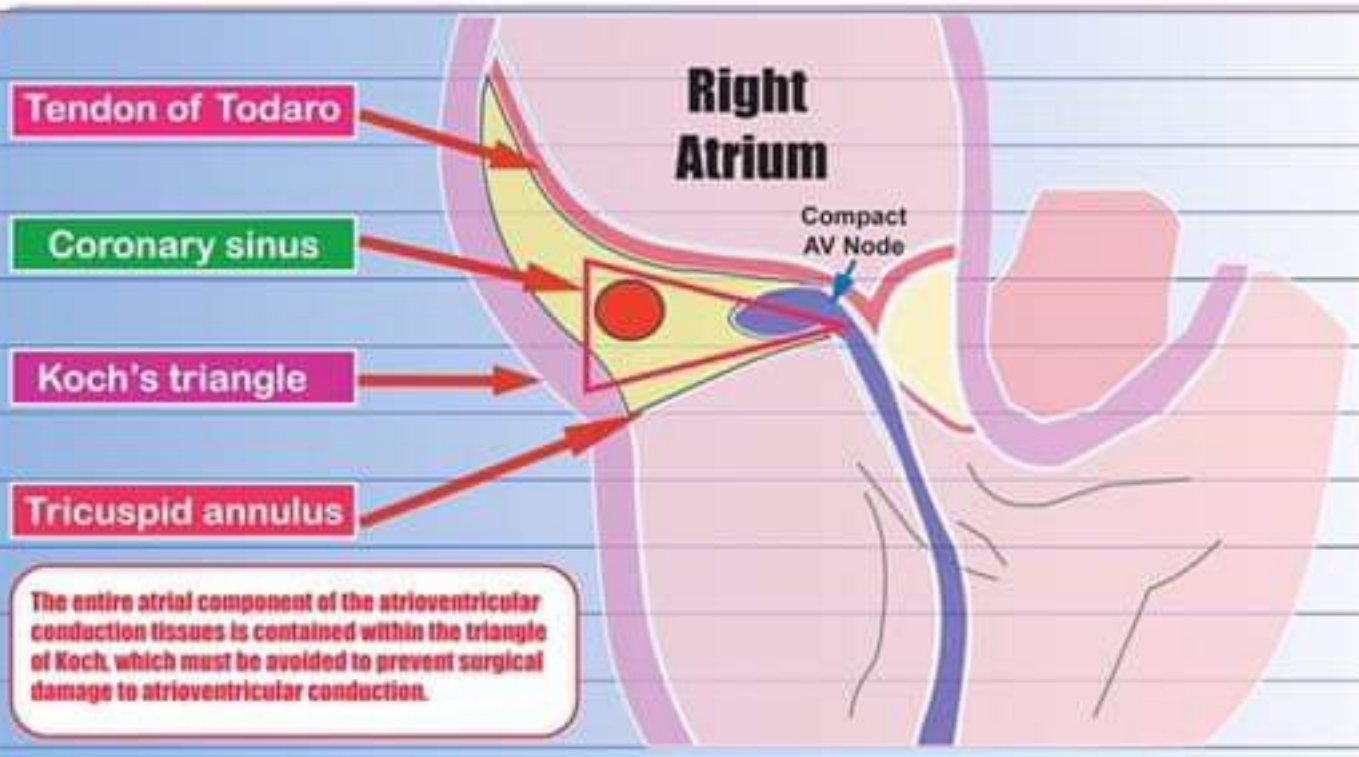
T wave
Amplitude: normal <2/3 R/ peaked/ inversion/ alternans

8. QT Interval: Duration: normal 390-450 or 460msec / long or short/

QT dispersion: Method, 3+ QRS in 3+ leads, QTc formulae, caveats

9. U wave normal 10% T or <1.5mm/ prominent/ inversion/ alternans

Koch's Triangle



Koch's triangle is an anatomical area located in the superficial paraseptal endocardium of the right atrium, which its angles are **coronary sinus** orifice, **tendon of Todaro** and atrioventricular node. Also the elements anatomically near to it, are the insertion of the **tricuspid valve,** membranous septum, and the Eustachian ridge. This triangle ends at the site of the **coronary sinus** orifice inferiorly and, continuous with the sub-Eustachian pouch. **The tendon of Todaro** forms the hypotenuse of the triangle and the base is formed by the CSO and the vestibule of the right atrium. Variations in the size of **Koch's triangle** are common among people with different age and gender. The Atrioventricular node or the AV node is located at the apex of this triangle which depicts its anatomical importance .

Coagulation cascade made easy

The intrinsic Pathway

After initiation, each step in the intrinsic pathway is catalysed by the product of the preceding step: factor XII, also termed Hageman factor, is converted to XIIa on contact with negatively-charged surfaces e.g. that of subendothelial collagen. For activation to its enzymatic form, it requires high molecular weight kininogen and prekallikrein.

factor XIIa converts factor XI to XIa
factor XIa converts factor IX converted to IXa
factor IXa converts factor X to Xa; And to do this it requires:
calcium ions
factor VIIIa

12 → 11 → 9 —8→

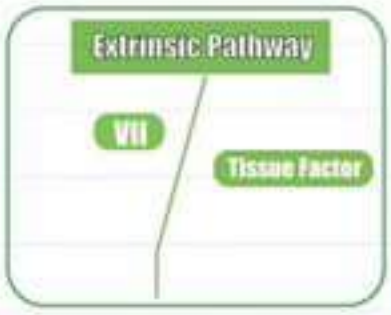
a negatively charged surface; in vivo, this is the surface of platelets so localizing the cascade factor Xa then activates the common pathway of coagulation

The extrinsic Pathway

In the extrinsic pathway of blood coagulation, a tissue factor is produced after injury. Likely sources include endothelial cells and the surface cells of atheromatous plaques. The factor is held within the plasma membrane.

The tissue factor interacts with factor VIIa, calcium ions and phospholipid to activate factor X to Xa. Factor Xa then acts on prothrombin according to the common pathway of coagulation.

Factor XIIa indirectly facilitates the extrinsic pathway by converting factor VII to VIIa.



The Common Pathway

The common pathway of coagulation starts with the conversion of factor X to Xa; in the intrinsic pathway this is caused by factor IXa, in the extrinsic pathway this is caused by tissue factor.

Factor Xa requires its own co-factors for activity. These include calcium ions, circulating factor V and the negatively-charged platelet surface for localisation. It is then able to cleave prothrombin (factor II) to thrombin (factor IIa).

10 —5→ 2 (Thrombin) → 1 (Fibrinogen)

Coagulation cascade continued...

Blood coagulation occurs through a series of reactions by a group of substances. These substances are thirteen clotting factors which are as follows:

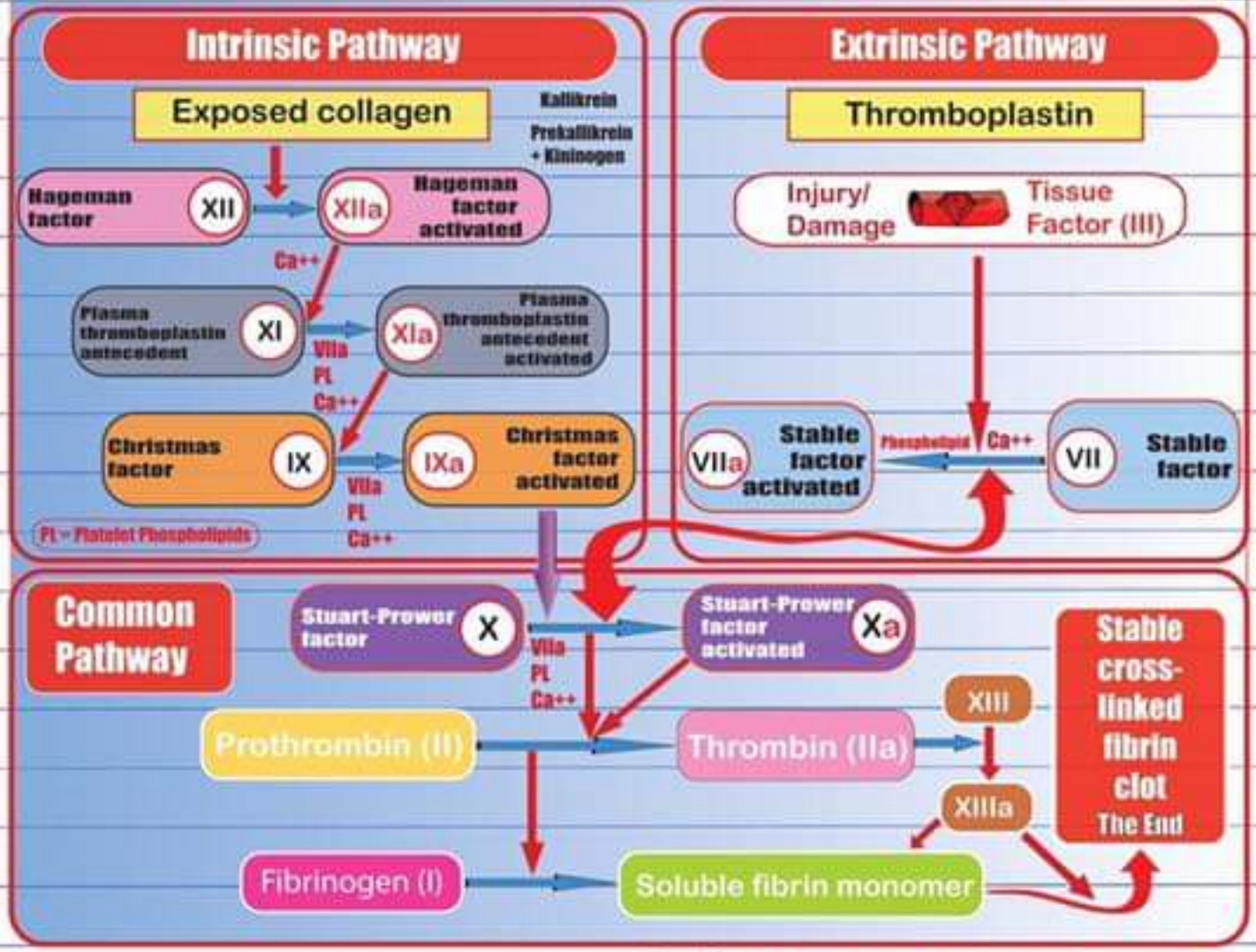
Clotting Factors	Substances	Source
Factor 1 (I)	Fibrinogen	Liver
Factor 2 (II)	Prothrombin	Liver
Factor 3 (III)	Thromboplastin (Tissue factor)	Platelets and endothelium
Factor 4 (IV)	Calcium ions	Bone and GIT
Factor 5 (V)	Labile factor (Proaccelerin)	Liver and platelets
Factor 6 (VI)	-	-
Factor 7 (VII)	Stable factor	Liver
Factor 8 (VIII)	Antihemophilic factor	Platelets and endothelium
Factor 9 (IX)	Christmas factor (plasma thromboplastin)	Liver
Factor 10 (X)	Stuart-Prower factor	Liver
Factor 11 (XI)	Plasma thromboplastin antecedent	Liver
Factor 12 (XII)	Hageman factor	Liver
Factor 13 (XIII)	Fibrin-stabilizing factor	

Easy Memo

Collagen
Platelets Active

12a → 11a → 9a + 8a → Tissue factor → 7a + 10a + 5a → 2a → 1a + 13a → Stabilized fibrin clot

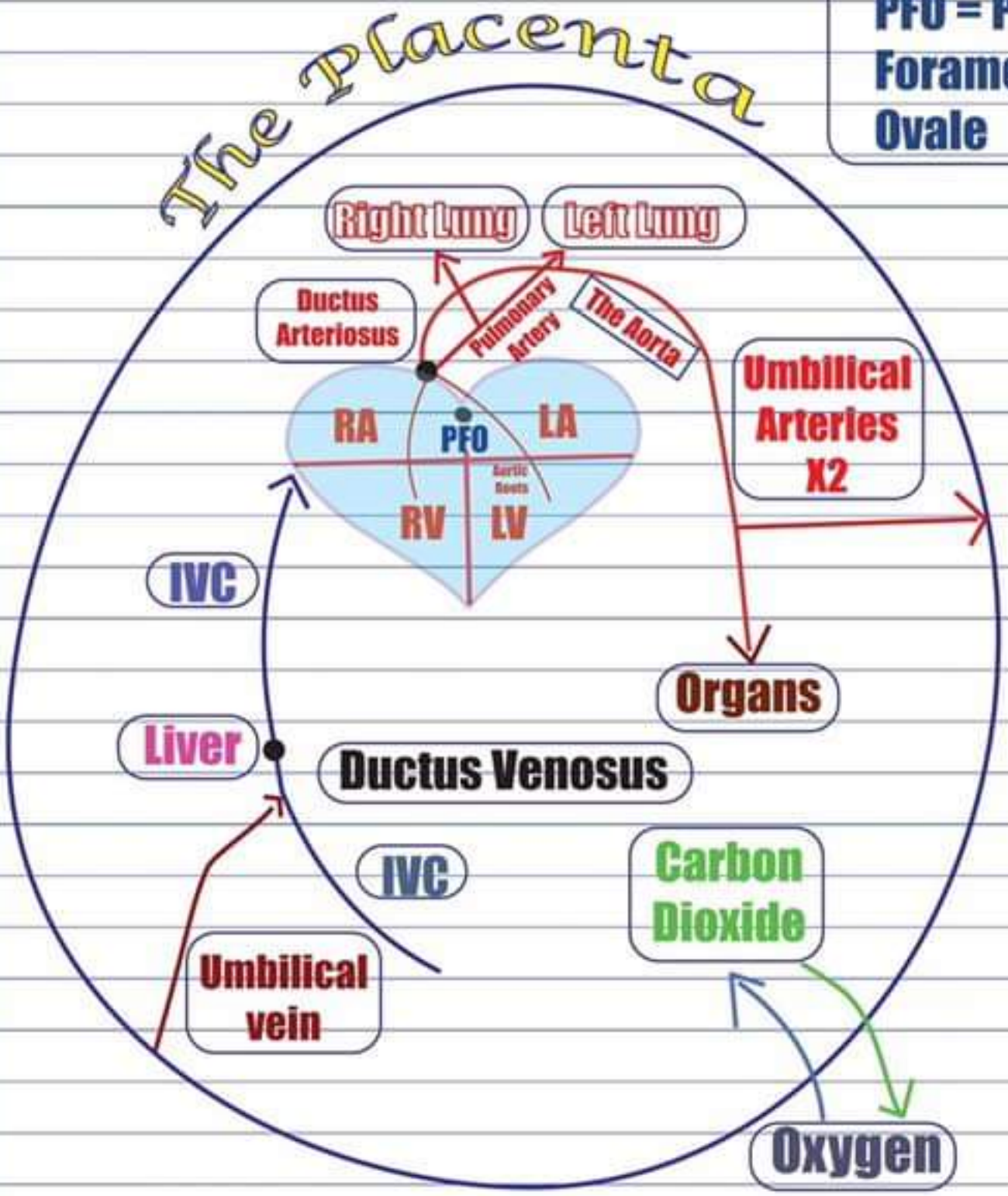
Diagram: 3 pathways of clot formation



Fetal Circulation Made Easy

the fetal circulation is the circulatory system of a fetus. The term usually encompasses the entire fetoplacental circulation, which includes the umbilical cord and the blood vessels within the placenta that carry fetal blood.

PFO = Patent Foramen Ovale



Umbilical Vein + 2 Umbilcal Arteries = Umbilical Cord

Fetal Circulation Made Easy Continue:

The fetal (prenatal) circulation works differently from normal postnatal circulation, mainly because the lungs are not in use. Instead, the fetus obtains oxygen and nutrients from the mother through the placenta and the umbilical cord. The advent of breathing and the severance of the umbilical cord prompt various neuroendocrine changes that shortly transform fetal circulation into postnatal circulation.

Physiology

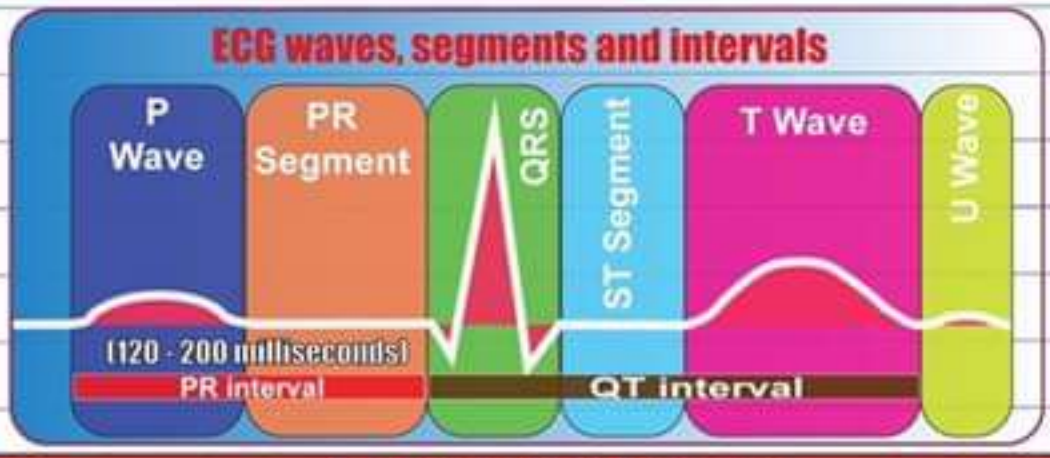
Blood from the placenta is carried to the fetus by the umbilical vein. In humans, less than a third of this enters the fetal ductus venosus and is carried to the inferior vena cava, while the rest enters the liver proper from the inferior border of the liver. The branch of the umbilical vein that supplies the right lobe of the liver first joins with the portal vein. The blood then moves to the right atrium of the heart. In the fetus, there is an opening between the right and left atrium (the foramen ovale), and most of the blood flows through this hole directly into the left atrium from the right atrium, thus bypassing pulmonary circulation. The continuation of this blood flow is into the left ventricle, and from there it is pumped through the aorta into the body. Some of the blood moves from the aorta through the internal iliac arteries to the umbilical arteries, and re-enters the placenta, where carbon dioxide and other waste products from the fetus are taken up and enter the maternal circulation. Some of the blood entering the right atrium does not pass directly to the left atrium through the foramen ovale, but enters the right ventricle and is pumped into the pulmonary artery. In the fetus, there is a special connection between the pulmonary artery and the aorta, called the ductus arteriosus, which directs most of this blood away from the lungs (which are not being used for respiration at this point as the fetus is suspended in amniotic fluid).

The core concept behind fetal circulation is that fetal hemoglobin (HbF) has a higher affinity for oxygen than does adult hemoglobin, which allows a diffusion of oxygen from the mother's circulatory system to the fetus.

Miss P Wavey

Introduction To Heart Blocks

Which type of AV block is it?



Evaluate 'PR' Interval

Miss PR

CONSTANT (PRI >0.2 secs)

VARIABLE

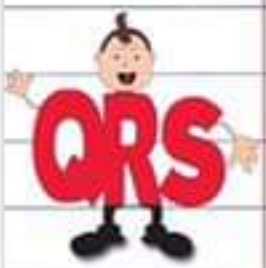
Evaluate 'P' QRS RATIO

Common ratio: 2:1, 3:1 and 4:1

Evaluate 'RR' Interval

1 'P' per QRS	>1 'P' per QRS	VARIABLE	CONSTANT
1st Degree	2nd Degree Type 2 (Mobitz)	2nd Degree Type 1 (Wenckebach)	3rd Degree
If the R is far from P, then you have a FIRST DEGREE	If some Ps don't get through, then you have MOBITZ II	Longer, longer, longer drop! Then you have a WENCKEBACH	If the Ps and Qs don't agree, then you have a THIRD DEGREE

How to try to remember these AV blocks



Mr QRS

1st Degree	2nd Degree Type 2 (Mobitz)	2nd Degree Type 1 (Wenckebach)	3rd Degree
All P waves conduct	More P waves then QRS complexes	More P waves then QRS complexes	More P waves then QRS complexes
PRI constant >0.20 secs	PR Interval is constant	PR Interval gets longer and longer	No PRI; P to P constant
All present ✓	QRS dropped periodically	QRS dropped periodically	QRS to QRS constant

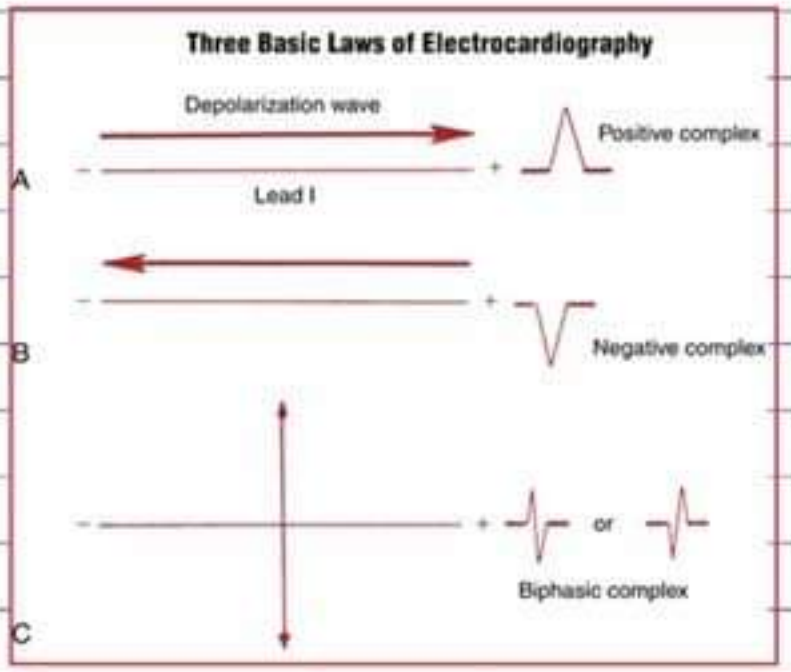
Introduction To Heart Blocks continued...

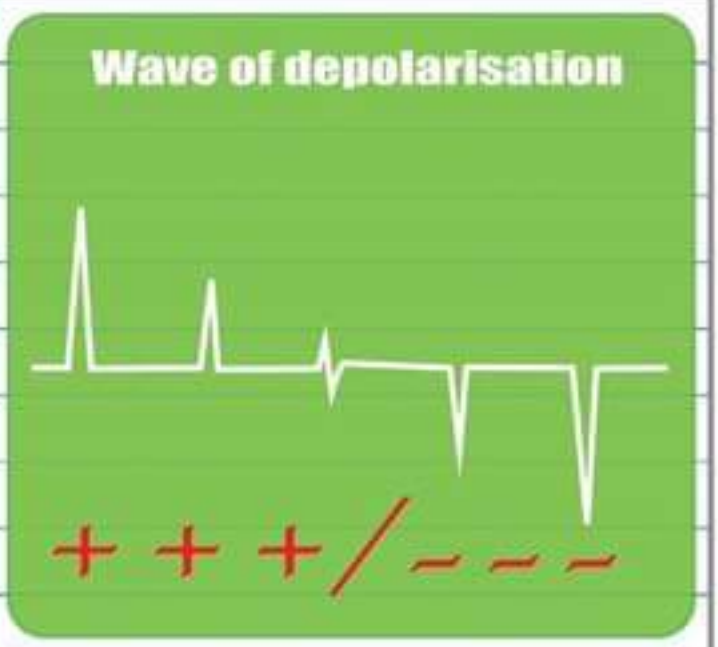
Heart block is a disease or inherited condition that causes a fault within the heart's natural pacemaker due to some kind of obstruction (or "block") in the electrical conduction system of the heart. Despite the severe-sounding name, heart block may often cause no symptoms at all in some cases, or occasional missed heartbeats in other cases (which can cause lightheadedness, syncope (fainting), and palpitations), or may require an artificial pacemaker to be implanted, depending upon exactly where in the heart conduction is being impaired and how significantly it is affected.

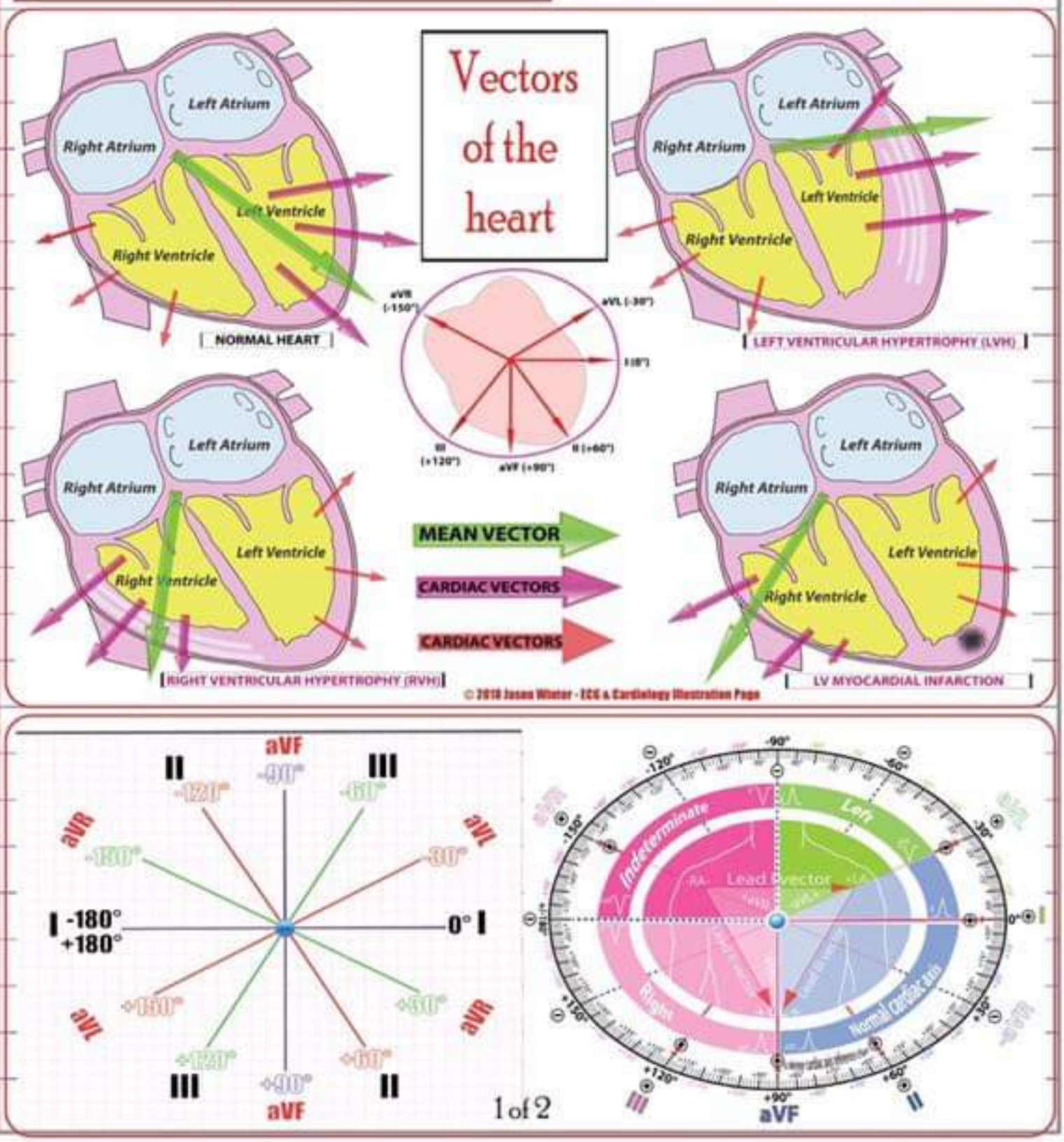
In severe cases where the heart's ability to control and trigger heartbeats may be completely ineffective or unreliable, heart block can usually be treated by inserting an artificial pacemaker, a medical device that provides correct electrical impulses to trigger heart beats, compensating for the natural pacemaker's unreliability. Therefore, heart block frequently has no effects, or mild and occasional effects, and is not life-threatening in the vast majority of cases, and is usually treatable in more serious cases.

The human heart uses electrical signals to maintain and initiate the regular heart beat in a living person; incorrect conduction can lead to mild or serious symptoms depending upon the location of the blockage and how severely conduction is being blocked. Conduction is initiated by the sinoatrial node ("sinus node" or "SA node"), and then travels to the atrioventricular node ("AV node") which also contains a secondary "pacemaker" that acts as a backup for the SA nodes, then to the bundle of His and then via the bundle branches to the point of the apex of the fascicular branches (shown in the diagram on the right). Blockages are therefore classified based on where the blockage occurs - namely the SA node ("Sinoatrial block"), AV node ("AV block" or AVB), and at or below the bundle of His ("Intra-Hisian" or "Infra-Hisian block" - respectively). Infra-Hisian blocks may occur at the left or right bundle branches ("bundle branch block") or the fascicles of the left bundle branch ("fascicular block" or "Hemiblock"). SA and AV node blocks are each divided into three degrees, with second degree blocks being divided into two types (written either "type I or II" or "type 1 or 2"). The term "Wenckebach block" is also used for second degree type 1 blocks of either the SA or AV node; in addition second degree blocks type 1 and 2 are also sometimes known as "Mobitz 1" and "Mobitz 2". Clinically speaking, the blocks tend to have more serious potential the closer they are to the 'end' of the electrical path (the muscles of the heart regulated by the heartbeat), and less serious effects the closer they are to the 'start' (at the SA node), because the potential disruption becomes greater as more of the 'path' is 'blocked' from its 'end' point. Therefore, most of the important heart blocks are AV nodal blocks and infra-Hisian blocks. SA blocks are usually of lesser clinical significance, since in the event of SA block, the AV node contains a secondary pacemaker which would still maintain a heart rate of around 40 - 60 beats per minute, sufficient for consciousness and much of daily life in the majority of individuals.

The Cardiac QRS Axis



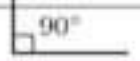


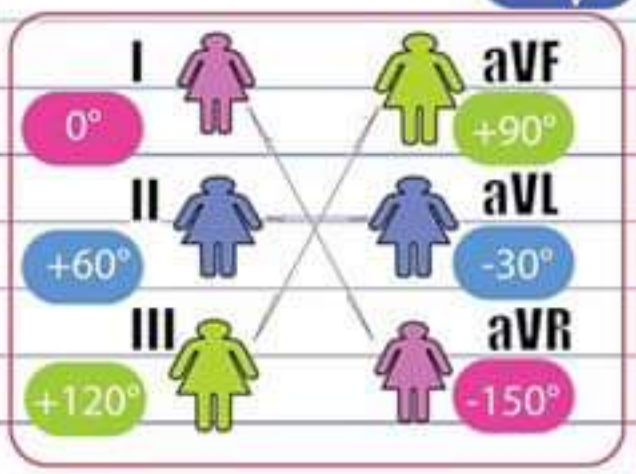


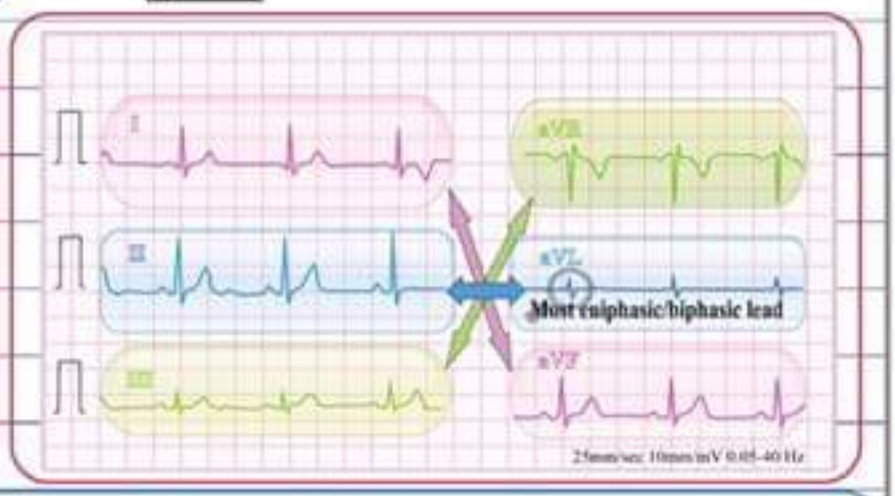
The Cardiac QRS Axis continued..... (Working out the cardiac axis):

STEP-1: Find the most euiphasic/biphasic limb lead or (isoelectric lead).

STEP-2: Look at the perpendicular lead running at 90° to this.



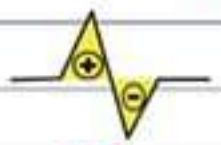




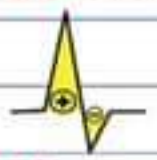
The Perpendicular ECG Lead Sisters (Always 90° apart from each other)

STEP-3: Is the impulse mostly +ve? (moving towards this lead) or -ve (moving away from this lead)?

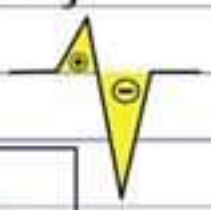
STEP-4: Is the euiphasic lead truely euiphasic? YES - you have found your cardiac axis.



STEP-5: Slightly more +ve? - move the axis 15° towards the euiphasic lead.



STEP-6: Slighty more -ve? - move the axis 15° away.



Now you have found your cardiac axis to the nearest 15° :)

Posterior Wall Myocardial Infarction

Posterior Wall ECG Lead Placement

Posterior ECG lead placement

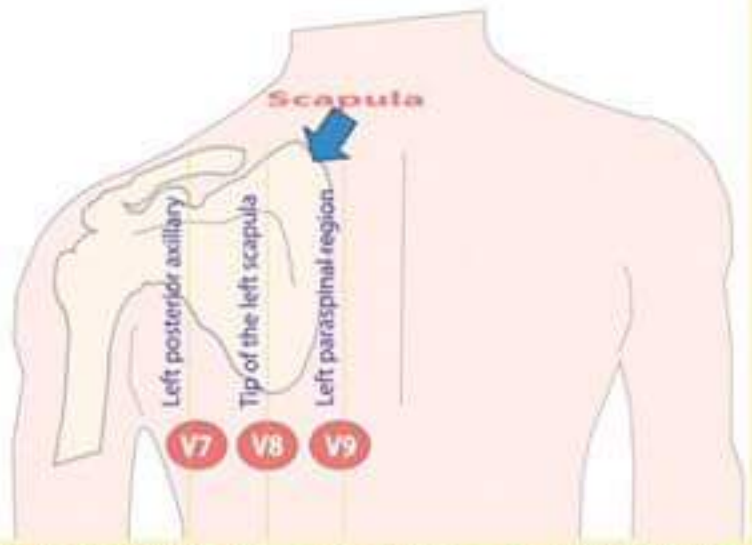


Figure 1 - Shows correct positioning of posterior leads

Posterior MI is suggestive by the following changes in V1-V3:

*Horizontal ST depression
*Tall, broad R waves (>30ms)
*Upright T waves
*Dominant R wave (R/S ratio >1) in V2

V1-V3 Should remain unchanged from standard 12-lead ECG

V7

Left posterior axillary line: in the same horizontal plane as V4-V6

V4 becomes V7

V8

Tip of the left midscapula: in the same horizonal plane as V7-V9

V5 becomes V8

V9

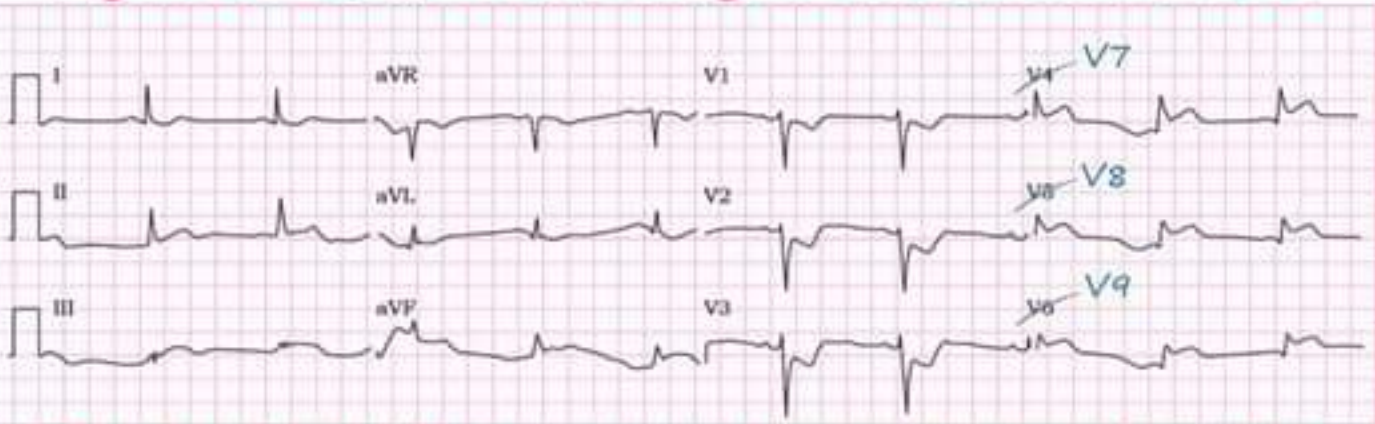
Left paraspinal region: in the same horizonal plane as V4-V6

V6 becomes V9

Please note that V6 is a good reference point for the horizontal placement of the posterior electrobes V7-9.

If you don't have access to to a 15 or 18 lead ECG machine, then leave V1-3 in their normal position and use V4-6, these leads will then become V7-9.

Figure 2 - Correct labeling of the Posterior ECG



*Note that only 0.5 mm of ST elevation is required to make the diagnosis of posterior MI

TYPES OF QRS COMPLEXES

R 	The first (and only) wave positive and thus an R wave.
Rs 	The first wave positive is large and positive (R), followed by a small negative wave (s).
rS 	Initially a small positive wave (r), followed by a large negative wave (S).
qRs 	The first wave is negative and small (q), followed by a large positive wave (R), & small negative (s).
QR 	Initially a large negative (Q), then a large positive wave (R).
QS 	A single negative wave is called a QS-complex.
Qr 	A large negative (Q), followed by a small positive wave (r).
rsR 	The negative wave manages to pass the baseline, and is therefore qualified as an s wave.
qR 	Initially a small negative wave (q), followed by a large positive wave (R).
R 	Notching on the upstroke of the R wave.
rR 	The negative deflection does not manage to pass the baseline, therefore can qualify as an s wave.

Examples of fragmented QRS-complexes.

Right Ventricular Wall Infarction

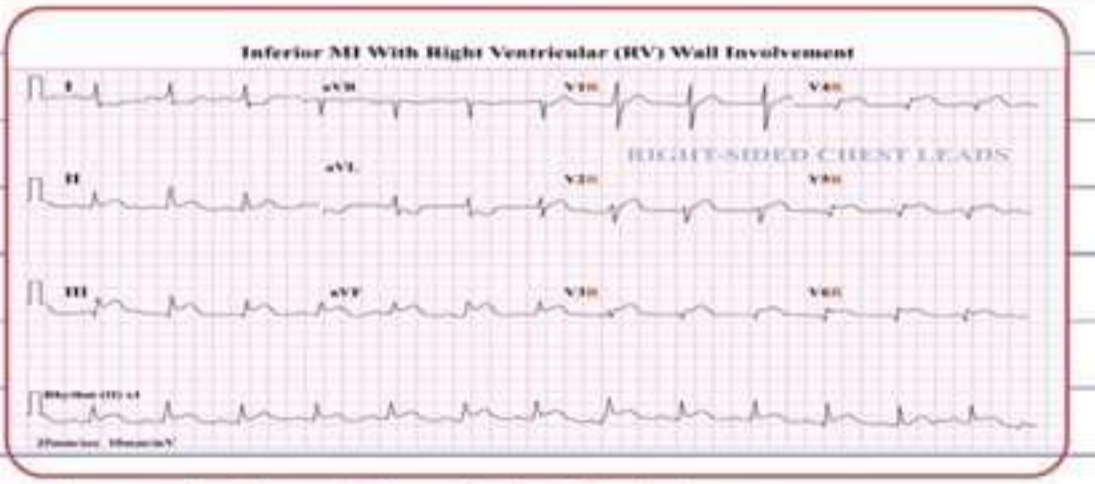
Clinical Significance

Right ventricular infarction complicates up to 40% of inferior STEMIs. Isolated RV infarction is extremely uncommon. Patients with RV infarction are very preload sensitive (due to poor RV contractility) and can develop severe hypotension in response to nitrates or other preload-reducing agents. Hypotension in right ventricular infarction is treated with fluid loading, and nitrates are contraindicated. The ECG changes of RV infarction are subtle and easily missed!

How to spot right ventricular infarction

*The first step to spotting RV infarction is to suspect it... in all patients with inferior STEMI!

In patients presenting with inferior STEMI, right ventricular infarction is suggested by the presence of: ST elevation in V1 – the only standard ECG lead that looks directly at the right ventricle. ST elevation in lead III > lead II – because lead III is more "rightward facing" than lead II and hence more sensitive to the injury current produced by the right ventricle. Other useful tips for spotting right ventricular MI: ST elevation in V1 > V2. ST elevation in V1 + ST depression in V2 (= highly specific for RV MI). Isoelectric ST segment in V1 with marked ST depression in V2. Right ventricular infarction is confirmed by the presence of ST elevation in the right-sided leads (V3R-V6R).



https://lifeinthefastlane.com/ecg-library/right-ventricular-infarction/

Right-sided leads

There are several different approaches to recording a right-sided ECG:

A complete set of right-sided leads is obtained by placing leads V1-6 in a mirror-image position on the right side of the chest (see diagram, below). It may be simpler to leave V1 and V2 in their usual positions and just transfer leads V3-6 to the right side of the chest (i.e. V3R to V6R). The most useful lead is V4R, which is obtained by placing the V4 electrode in the 5th right intercostal space in the midclavicular line. ST elevation in V4R has a sensitivity of 88%, specificity of 78% and diagnostic accuracy of 83% in the diagnosis of RV MI.

ECG Basics - A ECG right-sided chest leads

Right Ventricular Wall Infarction

Right-sided ECG lead placement

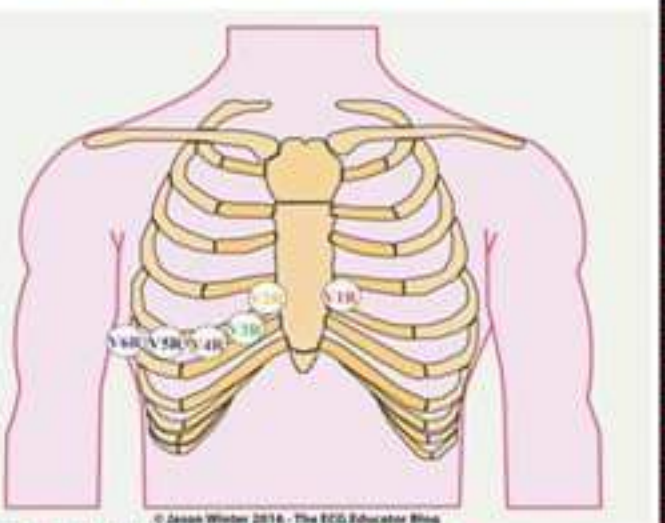


Figure 1 - The Right-sided correct positioning of ECG leads

Always double check your lead placement to confirmed your in the correct anatomical spaces.

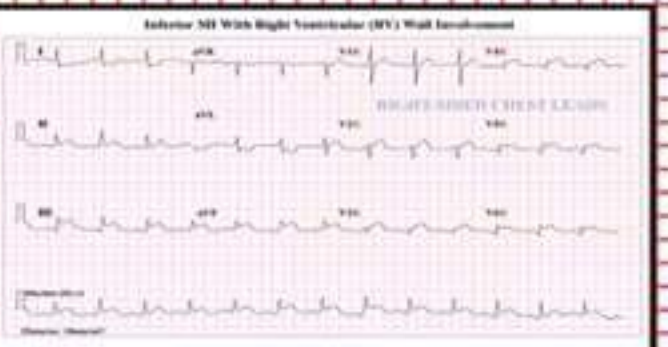


Figure 2 - Correct labeling of the Right-sided ECG

A complete set of right-sided leads is obtained by placing leads V1-6 in a mirror-image position on the right side of the chest (see Fig-1 diagram).

It may be simpler to leave V1 and V2 in their usual positions and just transfer leads V3-6 to the right side of the chest (i.e. V3R to V6R).

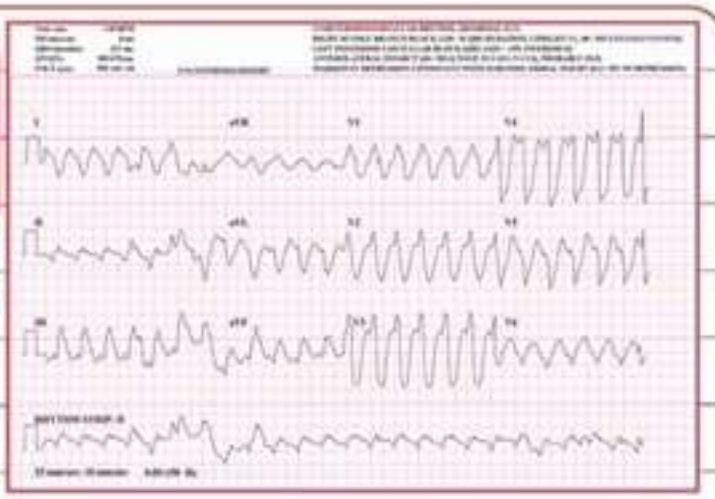
***The most useful lead is V4R**

https://lifeinthefastlane.com/ecg-library/right-ventricular-infarction/

Ventricular Tachycardia (VT) Signs

Signs of Ventricular Tachycardia

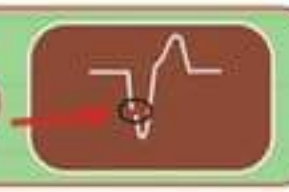
Very broad complexes (>160ms)



A fusion beat - Dressler beat occurs when sinus and ventricular electrical impulses coincides at the same time to produce a hybrid complex/beat. If it acts upon the ventricular chambers it is called a ventricular fusion beat.

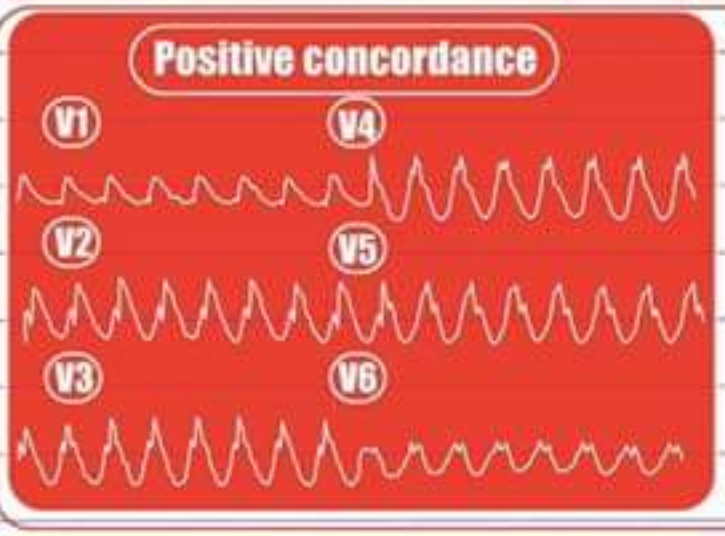
A capture beat occurs from the production of a ventricular complex by a supraventricular source in the cardiac cycle after atrioventricular (AV) dissociation, for the atria to regain control of the ventricles.

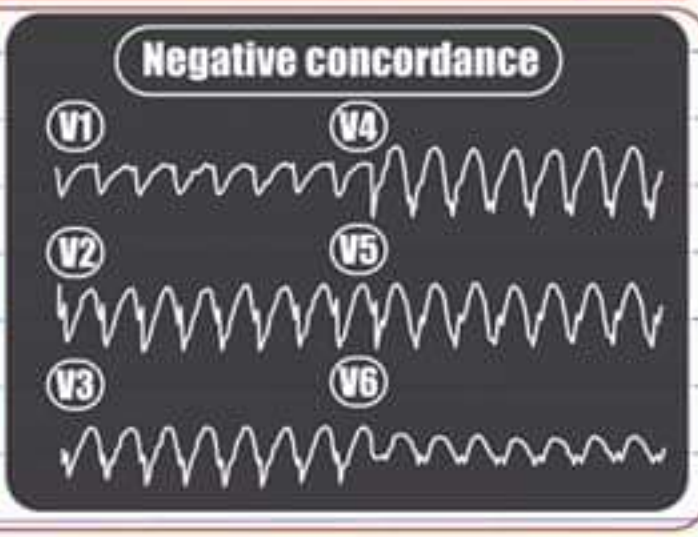
Josephson's sign – Notching near the nadir of the S-wave.



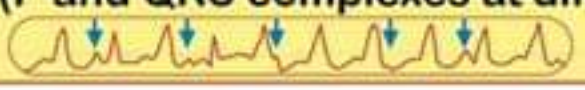
Brugada's sign – Is the distance from the onset of the QRS complex to the nadir of the S-wave is >100ms.

Extreme axis deviation ("northwest axis") — QRS is positive in aVR and negative in I + aVF

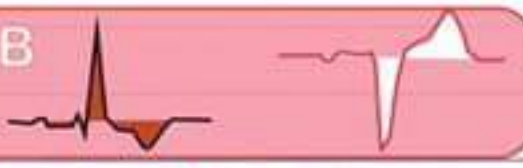




AV dissociation (P and QRS complexes at different rates).



Absence of typical RBBB or LBBB morphology.



WELLS Score (PE)

		Score
• Clinical signs and symptoms compatible with DVT	Yes ☐ No ☐	3
• PE judged to be the most likely diagnosis	Yes ☐ No ☐	3
• Surgery or bedridden for more than 3 days during past 4 weeks	Yes ☐ No ☐	1.5
• Previous documented DVT or PE	Yes ☐ No ☐	1.5
• Heart rate > 100 min	Yes ☐ No ☐	1.5
• Hemoptysis	Yes ☐ No ☐	1
• Active cancer (treatment ongoing or within previous 6 months or palliative treatment)	Yes ☐ No ☐	1

< 4 points unlikely PE

> 4 points likely PE

Score

< 2: LOW pretest probability	2 - 6: MODERATE pretest probability	> 6: HIGH pretest probability

WELLS Score (DVT)

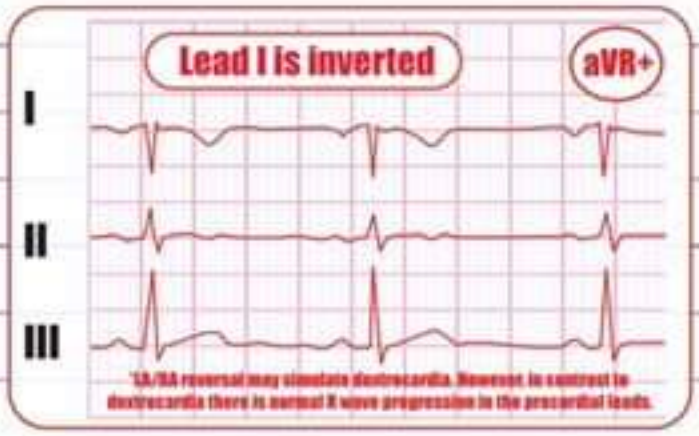
Clinical feature	Score
• Active cancer (treatment ongoing or within previous 6 months or palliative treatment)	Yes ☐ No ☐ +1
• Paralysis, paresis, or recent plaster immobilization of lower extremities	Yes ☐ No ☐ +1
• Recently bedridden for 3 days or more, or major surgery within previous 12 weeks requiring general or regional anesthesia	Yes ☐ No ☐ +1
• Localized tenderness along the distribution of the deep venous system	Yes ☐ No ☐ +1
• Entire leg swollen	Yes ☐ No ☐ +1
• Calf swelling > 3 cm compared to asymptomatic leg (measuring 10 cm below tibial tuberosity	Yes ☐ No ☐ +1
• Pitting oedema confined to the symptomatic leg	Yes ☐ No ☐ +1
• Nonvaricose collateral superficial veins	Yes ☐ No ☐ +1
• Previously documented DVT	Yes ☐ No ☐ +1
• Alternative diagnosis at least as likely as DVT	Yes ☐ No ☐ -2

< 0: LOW pretest probability	1 or 2: MODERATE pretest probability	> 3: HIGH pretest probability	Score

ECG Limb Lead Reversal Errors

Q

LA↔RA lead revarsal



* If there is P wave inversion in lead I, check for LA↔RA reversal.

* Also, If the other complexes in lead I appear inverted, check for LA↔RA reversal.

*Leads II and III switch places.
*Leads aVL and aVR switch places.
*Lead aVF remains unchanged.

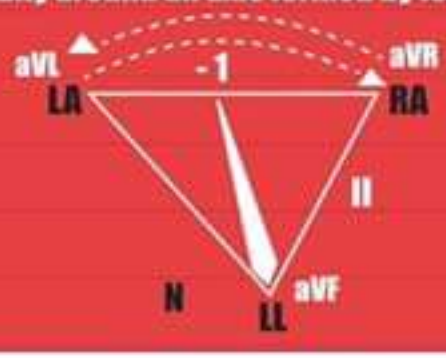
Quick notes to spotting LA/RA reversal:

Lead I is completely inverted (P wave, QRS complex and T wave).

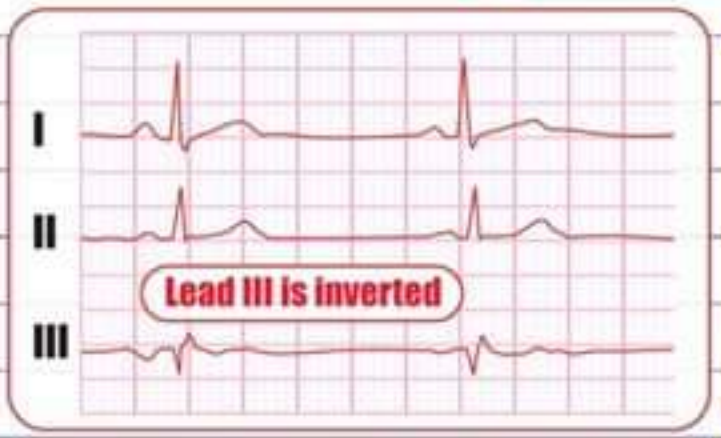
Lead aVR often becomes positive.

There may be marked right axis deviation.

With reversal of the LA and RA electrobes, Einthoven's triangle flips 180 degrees horizontally around an axis formed by lead aVF.



LA↔LL lead revarsal



* If P waves or QRS complexes appear inverted in lead III, check for LA↔LL reversal.

* However, be aware that physiologic left axis deviation can cause a negative QRS in lead III.

* If P wave is larger in lead I than in lead II, check for LA↔LL reversal.

This has the following effects on the ECG:

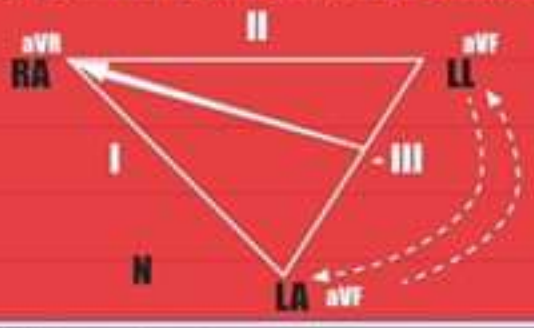
Lead III becomes inverted.

Leads I and II switch places.

Leads aVL and aVF switch places.

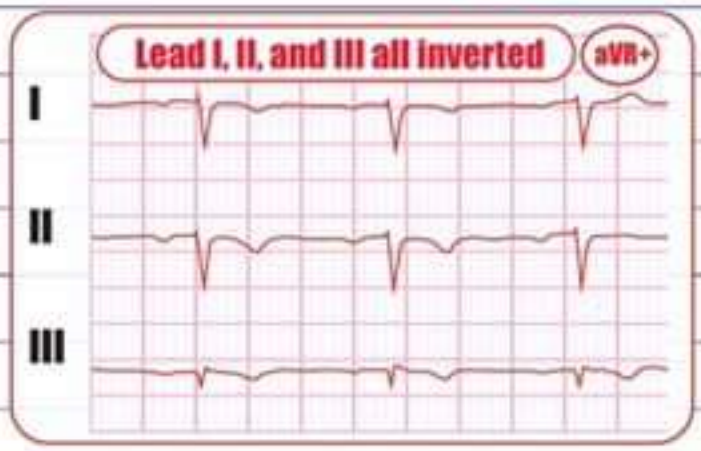
Lead aVR remains unchanged.

With reversal of the LA and LL electrobes, Einthoven's triangle rotates 180 degrees vertically around an axis formed by lead aVR.



ECG Leads Errors continue...

RA ↔ LL lead reversals



This has the following effects on the ECG:

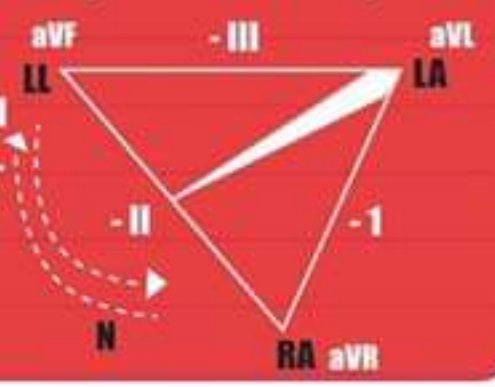
* Lead II becomes inverted.
* Leads I and III become inverted and switch places.
* Leads aVR and aVF switch places.
* Lead aVL is unchanged.

Quick guide to spotting RA/LL reversal:

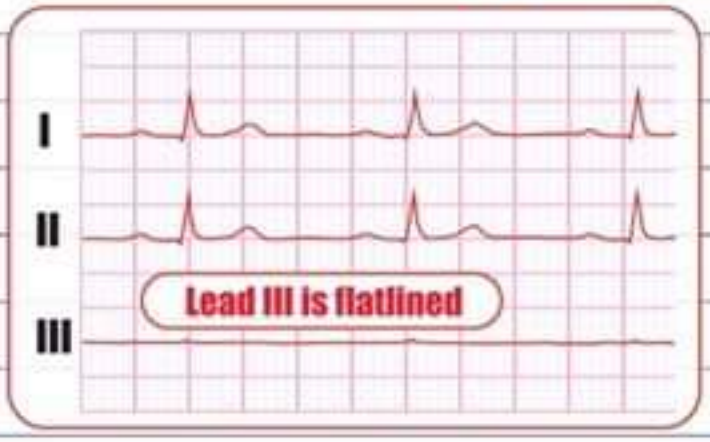
Leads I, II, III and aVF are all completely inverted (P wave, QRS complex and T wave).

Lead aVR is upright.

With reversal of the RA and LL electrobes, Einthoven's triangle rotates 180 degrees vertically around an axis formed by lead aVL.



LA ↔ RL lead reversals



LA/RL(N) lead reversal has the following ECG features:

* Lead I becomes identical to lead II.
* Lead II is unchanged.
* Lead III records a flat line (zero potential).
* Lead aVR approximates to an inverted lead II.
* Leads aVL and aVF become identical.

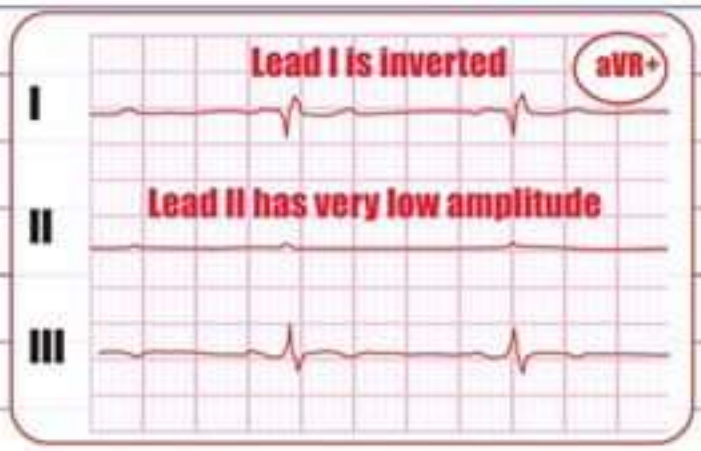
Quick guide to spotting LA/RL(N) reversal

Lead III is a flat line.

With reversal of the LA and RL(N) electrodes, Einthoven's triangle collapses to very thin "slice" with the RA electrode at its apex. The LA and LL electrodes now record almost identical voltages, making the difference between them negligible (i.e. lead III = zero). Lead aVR runs within this thin slice, facing approximately opposite to lead II. The displacement of the neutral electrode renders leads aVL and aVF mathematically identical, but appears different to the baseline ECG).

ECG Leads Errors continue...

RA ↔ RL lead reversals



RA/RL(N) lead reversal has the following ECG features:

* Lead I becomes an inverted lead III.
* Lead II records a flat line (zero potential).
* Lead III is unchanged.
* Lead aVL approximates an inverted lead III.
* Leads aVR and aVF become identical.
* As the neutral electrode has been moved.

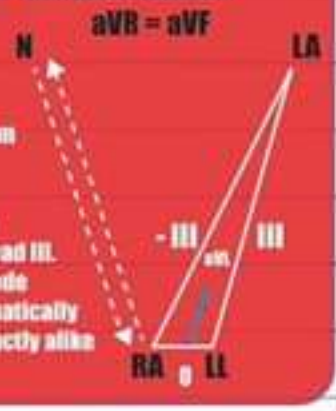
Quick guide to spotting RA/RL(N) reversal:

Lead II is a flat line.

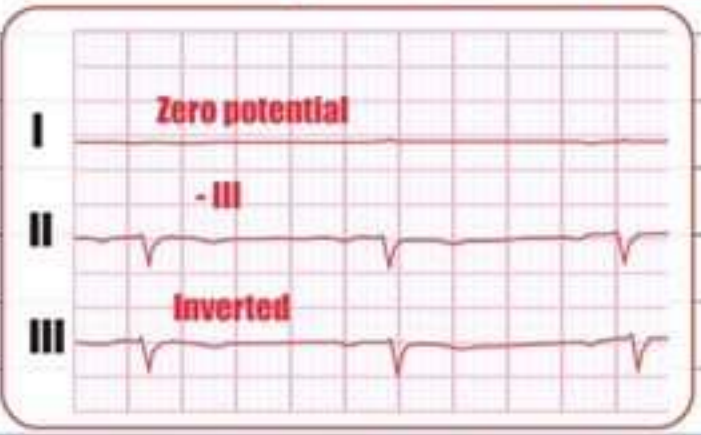
With reversal of the RA and RL(N) electrodes: Einthoven's triangle collapses to very thin "slice" with the LA electrode at its apex.

The RA and LL electrodes now record almost identical voltages, making the difference between them negligible (i.e. lead II = zero).

Lead aVL runs within this thin slice, facing approximately opposite to lead III. Displacement of the neutral electrode renders leads aVR and aVF mathematically identical, such that they appear exactly alike (but different to the baseline ECG).



Bilateral Arm-Leg Reversal (LA-LL plus RA-RL)



Bilateral arm-leg reversal has the following ECG features:

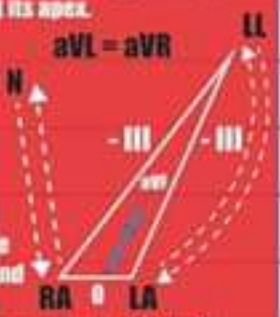
* Lead I records a flat line (zero potential).
* Lead II approximates an inverted lead III.
* Lead III is inverted.
* aVR and aVL become identical.
* aVF looks like negative lead III.

Quick Guide To Spotting Bilateral Arm-Leg Reversal

Lead I is a flat line.

If the electrodes on each arm are swopped with their corresponding leg electrode (LA with LL, RA with RL), Einthoven's triangle collapses to a very thin slice with the LL electrode at its apex.

The RA and LA electrodes (now sitting on adjacent feet) record almost identical voltages, which makes the difference between them negligible (i.e. lead I = zero). Leads II, III and aVF all become identical (equivalent to inverted lead III), as they are all now measuring the voltage difference between the left arm and the legs. The displacement of the neutral electrode renders leads aVL and aVR mathematically identical, such that they ppear exactly alike (but different to the baseline ECG).



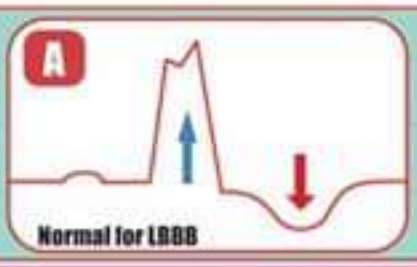
Sgarbossa criteria

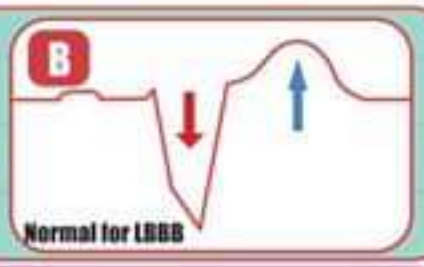
MI Diagnosis in LBBB or paced rhythm

Sgarbossa's criteria are a set of electrocardiographic findings generally used to identify acute myocardial infarction in the presence of a left bundle branch block (LBBB) or a ventricular paced rhythm. This was first described by Elena B Sgarbossa in 1996.

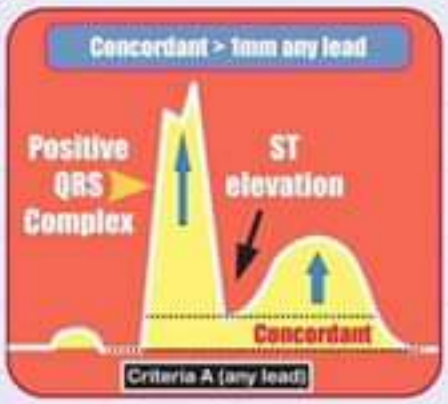
Rules of appropriate discordance in LBBB

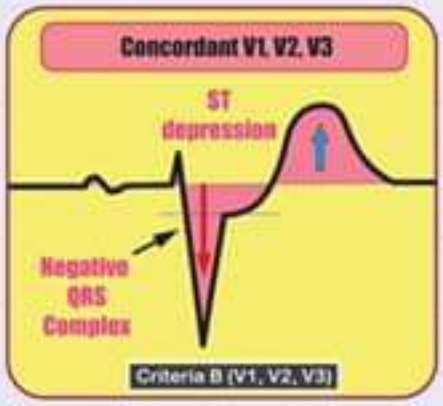
Normal finding with bundle branch block is appropriate T wave discordance. In other words, the T wave will be deflected opposite the terminal deflection of the QRS complex.





Excessive Discordant/concordant T wave and ST Segments in LBBB





The original three criteria used to diagnose infarction in patients with LBBB are:

Sgarbossa Criteria for diagnosis of MI in LBBB

Concordant ST elevation	Concordant ST depression	Excessively discordant ST elevation
> 1mm in leads with a positive QRS complex	> 1 mm in V1-V3	> 5 mm in leads with a -ve QRS complex
(score 5)	(score 3)	(score 2)

These criteria are specific, but not sensitive for myocardial infarction. A total score of ≥ 3 is reported to have a specificity of 90% for diagnosing myocardial infarction.

Smith modified Sgarbossa rule:

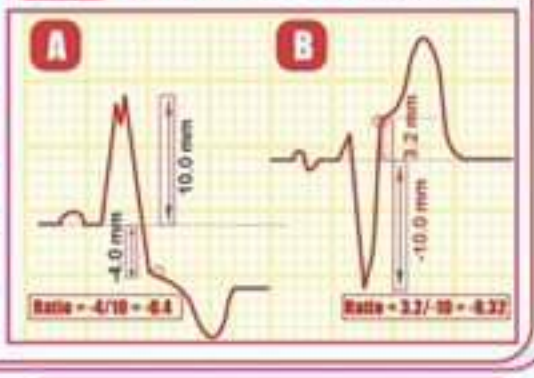
Criterion 3

NOTE: the Modified Sgarbossa Criteria below (which changes the third criteria) does not use the points system. It is positive if any criteria are met.

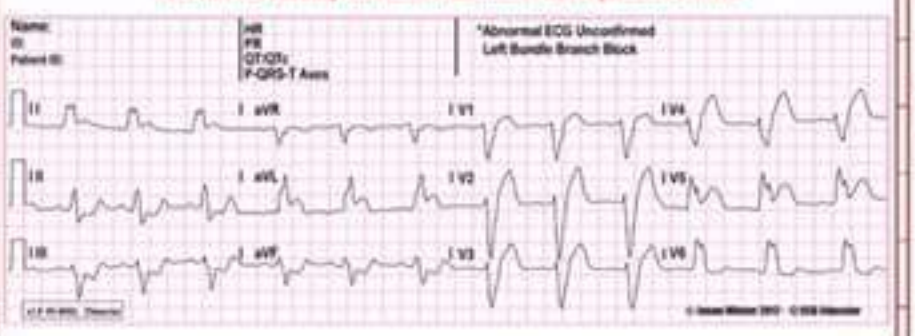
a) at least 1 mm discordant ST segment elevation

b) ST/S ratio > 25% (> 20% is still quite specific and more sensitive)

ST/S Ratio Ratio of ST segment elevation measure at the J point to the R or S wave, whichever was most prominent



Does This ECG Showing Left Bundle Branch Block Meet Sgarbossa Criteria?

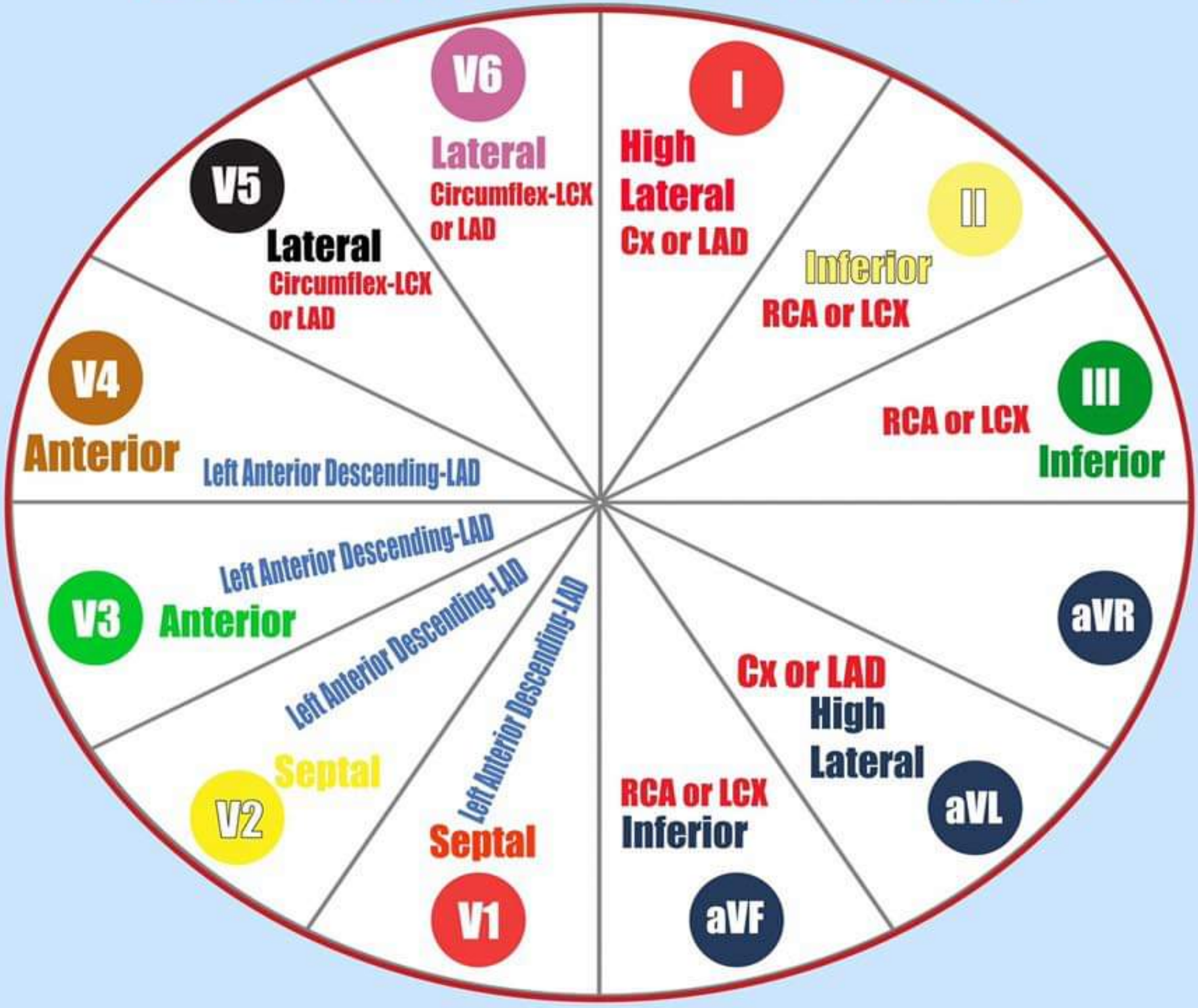


References

1. Meyers HP, Limkakeng AT, Jaffa EJ, Patel A, Theiling BJ, Bazzie SR, Stewart T, Zhuang C, Pers VK, Smith SW. Validation of the Modified Sgarbossa Rule for Diagnosis of STEMI in the Presence of Left Bundle Branch Block. American Heart Journal 178(6):1255-1264, December 2016

2. Smith SW, Dodd KW, Henrikson B, Henry TD, Pearce LA. Diagnosis of Acute Myocardial Infarction in the Presence of Left Bundle Branch Block using the ST Elevation to S-Wave Ratio in a Modified Sgarbossa Rule. Annals of Emergency Medicine 2012; 60(12):766-776

ECG Leads and Artery Localization MEMO



Mnemonic for Kawasaki Disease – "CREAM"

Kawasaki disease (mucocutaneous lymph node syndrome) is a rare childhood disease that causes inflammation in the walls of small-sized and medium sized arteries.

It is most often seen in children under the age of 5 of Asian descent. It is somewhat more common in boys than girls.

Small and medium vessel vasculitis

There is no known cause for this disease and it is not contagious. There is no single test for Kawasaki disease and in order to diagnose it, you must look for symptoms.

C: Conjunctivitis (non-exudative)

R: Rash (polymorphous non-vesicular)

E: Edema (or erythema of hands or feet)

A: Adenopathy (cervical, often unilateral)

M: Mucosal involvement (erythema or fissures, strawberry tongue)

Complications:
- *Coronary artery aneurysm
- *Myocarditis

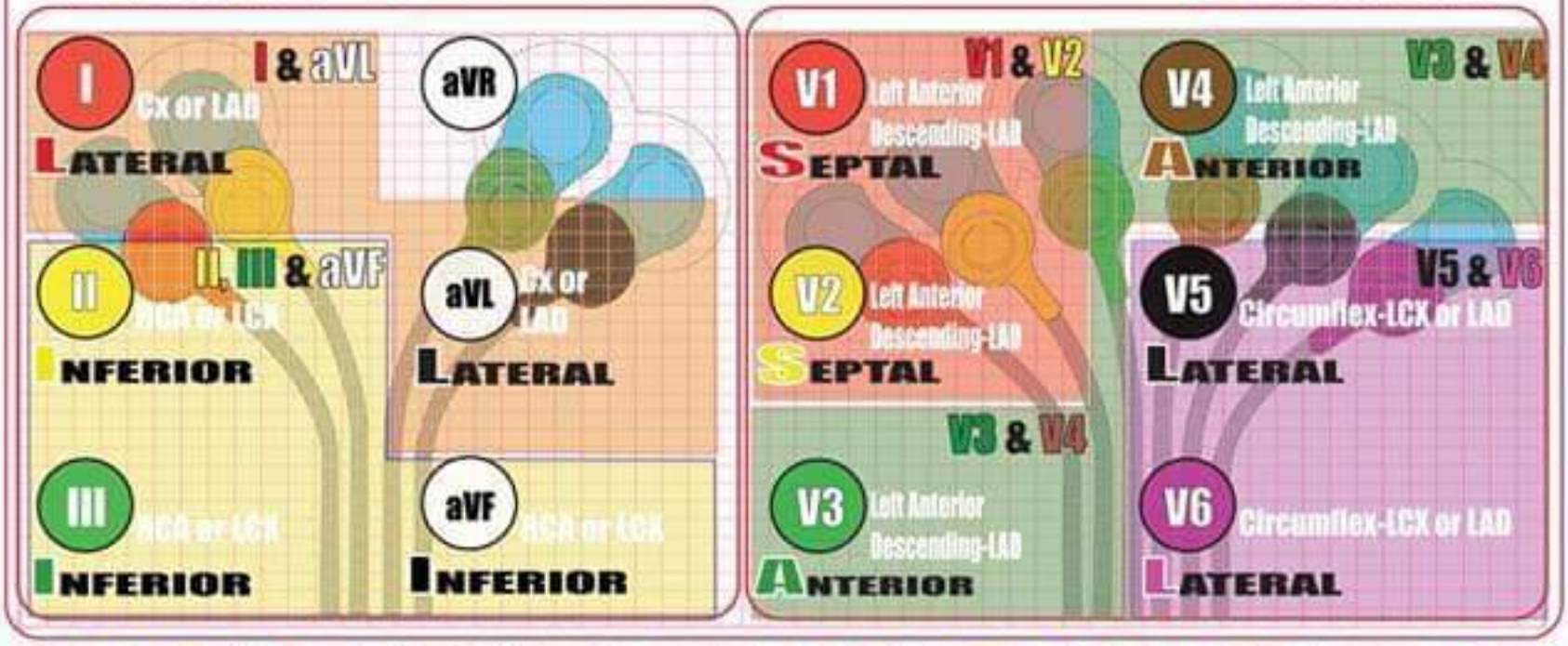
Treatment:
- *High dose ASA
- *IVIG

ECG 12-Lead Memo

Limb leads

12-Lead ECG Mneumonic (LISA - L)

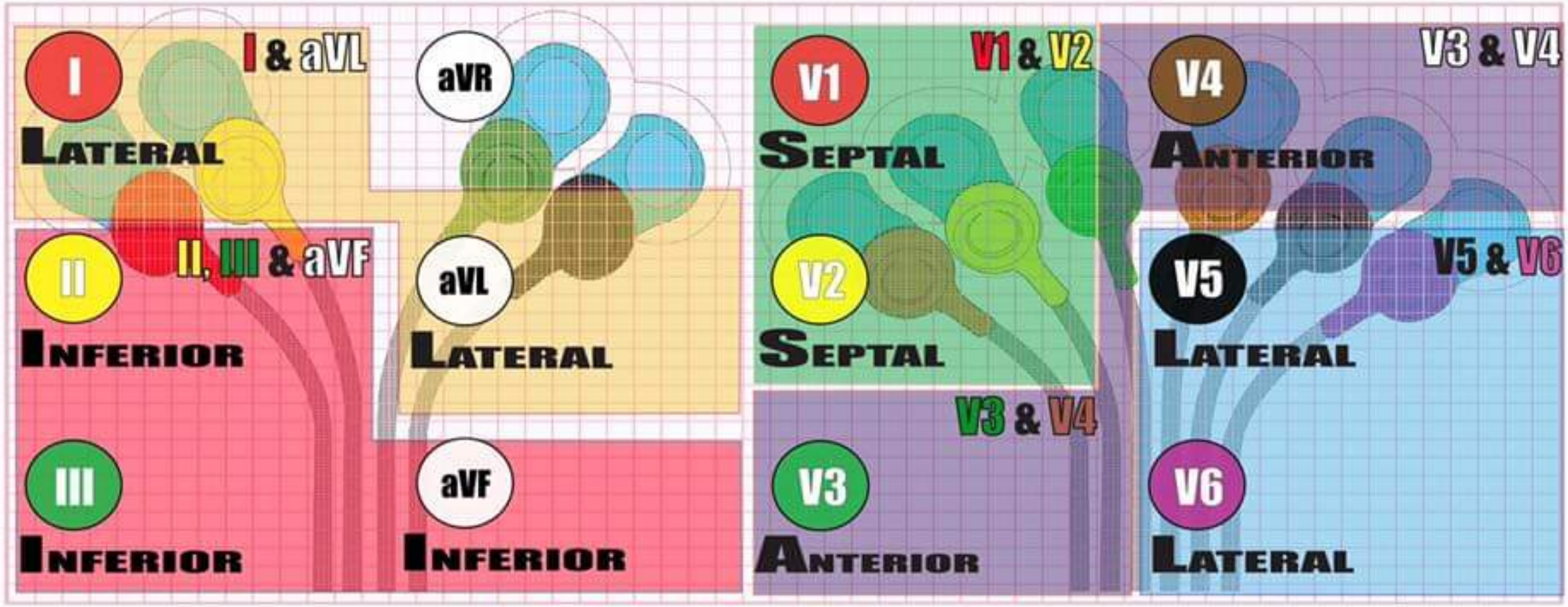
Chest leads



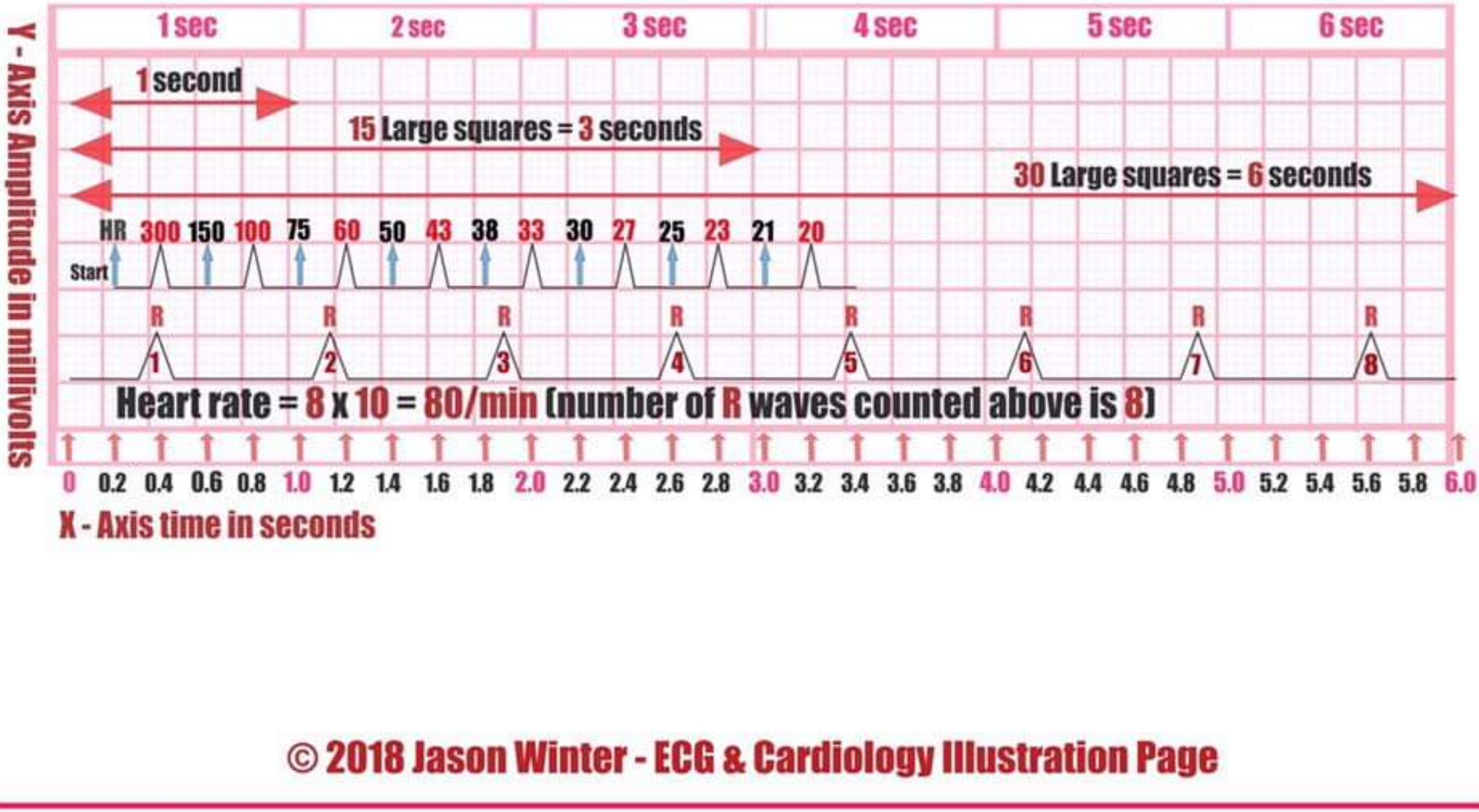
I LATERAL | II INFERIOR | III INFERIOR | aVR | aVL LATERAL | aVF INFERIOR | V1 SEPTAL | V2 SEPTAL | V3 ANTERIOR | V4 ANTERIOR | V5 LATERAL | V6 LATERAL

Electrode Placement		12 lead MI Locations	Vertical Leads/Frontal Plane
I	Right Arm (RA) RA (-) to LA (+)	HIGH LATERAL	Bipolar limb leads VERTICAL LEADS/FRONTAL PLANE
II	Left Arm (LA) RA (-) to LL (+)	INFERIOR	Bipolar limb leads VERTICAL LEADS/FRONTAL PLANE
III	Left Leg (LL) LA (-) to LL (+)	INFERIOR	Bipolar limb leads VERTICAL LEADS/FRONTAL PLANE
aVR		RIGHT ATRIUM	Augmented unipolar limb leads VERTICAL LEADS/FRONTAL PLANE
aVL	LA (+) to [RA & LL] (-)	HIGH LATERAL	Augmented unipolar limb leads VERTICAL LEADS/FRONTAL PLANE
aVF	LL (+) to [RA & LA] (-)	INFERIOR	Augmented unipolar limb leads VERTICAL LEADS/FRONTAL PLANE
V1	4th ICS, Right sternal border	SEPTAL/ANTERIOR	Unipolar (+) chest leads HORIZONTAL PLANE
V2	4th ICS, Left sternal border	SEPTAL/ANTERIOR	Unipolar (+) chest leads HORIZONTAL PLANE
V3	Halfway between V2 & V4	ANTERIOR	Unipolar (+) chest leads HORIZONTAL PLANE
V4	5th ICS, mid-clavicular line	ANTERIOR	Unipolar (+) chest leads HORIZONTAL PLANE
V5	Horizontally even with V4, in the left anterior axillary line	LATERAL	Unipolar (+) chest leads HORIZONTAL PLANE
V6	Horizontally even with V4 and V5 in the midaxillary line	LATERAL	Unipolar (+) chest leads HORIZONTAL PLANE

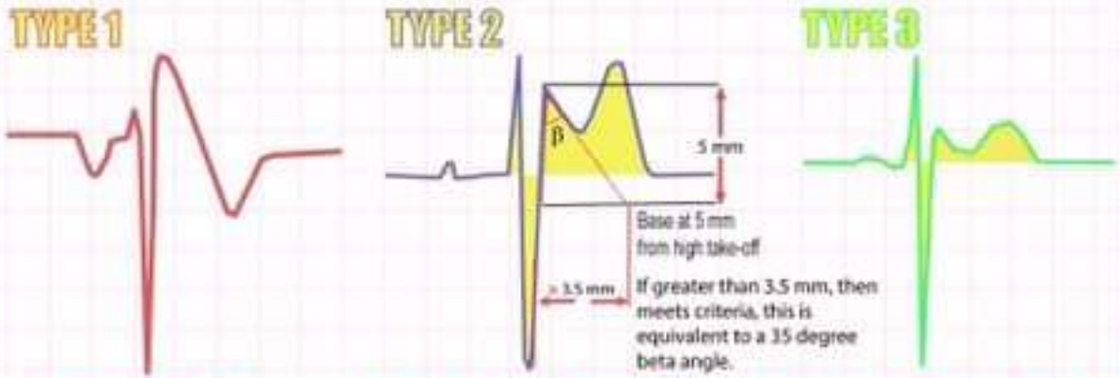
12-Lead ECG Mneumonic (LISA - L)



BASIC ELECTROCARDIOLOGY



Brugada Syndrome



Coved type STE **Saddle - back type STE** **Saddle - back type STE**

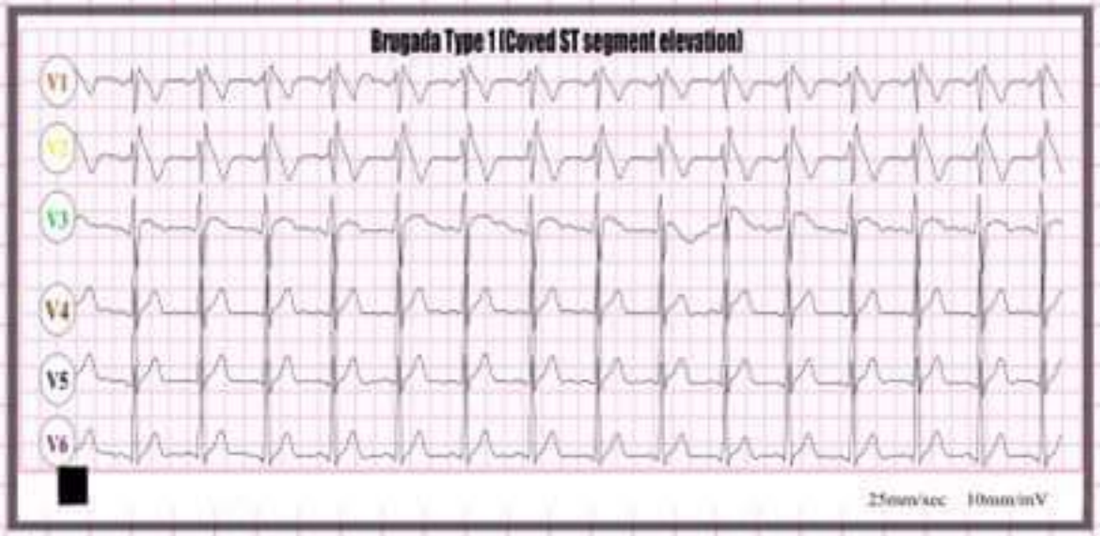
ECG pattern in Brugada syndrome. type 1 ST segment elevation, either spontaneously present or induced with the sodium channel-blocker challenge test, is considered diagnostic. Type 2 and 3 may lead to suspicion, but the drug challenge is required for diagnosis.

Brugada syndrome has three different ECG patterns:

Type 1 has a coved type ST elevation with at least 2 mm (0.2 mV) J-point elevation and a gradually descending ST segment followed by a negative T-wave.

Type 2 has a saddle-back pattern with a least 2 mm J-point elevation and at least 1 mm ST elevation with a positive or biphasic T-wave. Type 2 pattern can occasionally be seen in healthy subjects.

Type 3 has either a coved (type 1 like) or a saddle-back (type 2 like) pattern, with less than 2 mm J-point elevation and less than 1 mm ST elevation. Type 3 pattern is not rare in healthy subjects.



First described by Spanish cardiologists the Brugada brothers in 1992 by Dr. Pedro Brugada, Ramon and Josep Brugada.

Brugada syndrome is an hereditary disease that is associated with high risk of sudden cardiac death. It is characterized by typical ECG abnormalities: RBBB type ST segment elevation in the precordial leads (V1 - V3).

Brugada syndrome have been shown to be associated with mutations in the SCN5A gene that encodes for a sodium ion channel in the cell membranes of the muscle cells of the heart (the myocytes); this is often referred to as a sodium channelopathy.

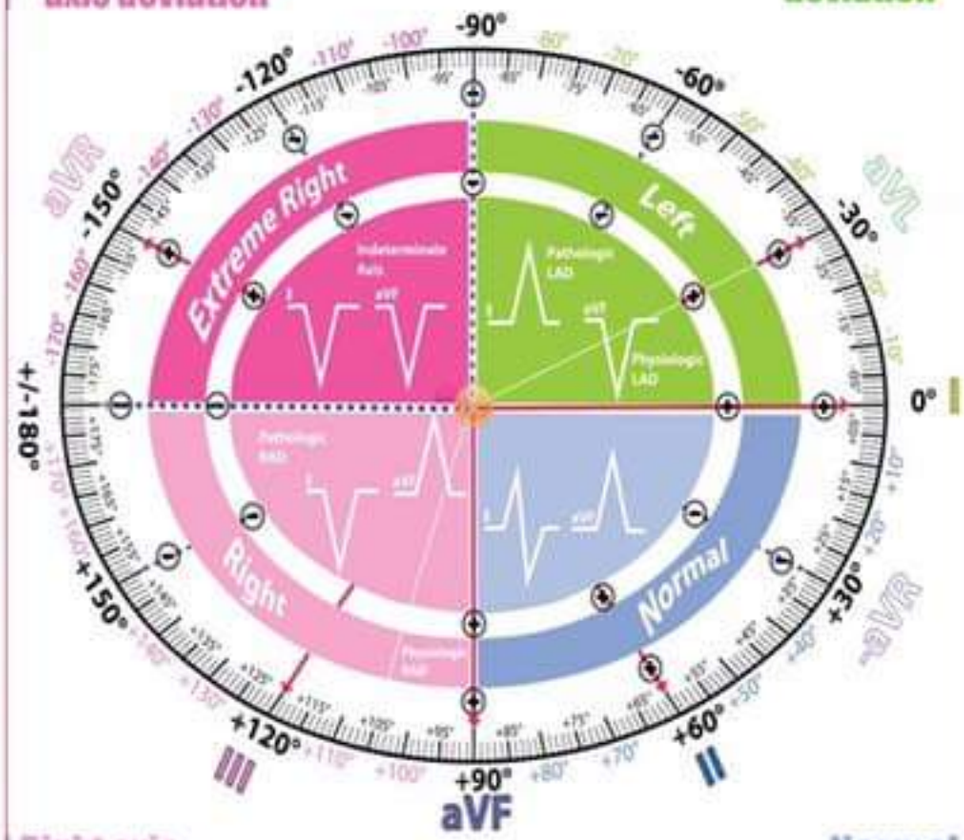
Seconds

0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0 1.1 1.2 1.3 1.4 1.5 1.6 1.7 1.8 1.9 2.0 2.1 2.2 2.3 2.4 2.5 2.6 2.7 2.8 2.9 3.0 3.1 3.2 3.3 3.4 3.5 3.6 3.7 3.8 3.9 4.0

Cardiac Axis

Extreme right axis deviation

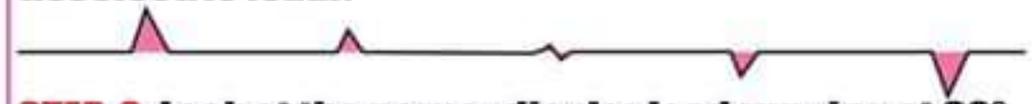
Left axis deviation



Right axis deviation

Normal axis

STEP-1: Find the most euiphasic/biphasic limb lead or (isoelectric lead).

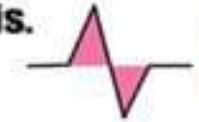


STEP-2: Look at the perpendicular lead running at 90° to this.

STEP-3: Is the impulse mostly +ve? (moving towards this lead) or -ve (moving away from this lead)?

Positive Complex ⊕ ⟷ Negative Complex ⊖

STEP-4: Is the euiphasic lead truely euiphasic? YES - you have found your cardiac axis.



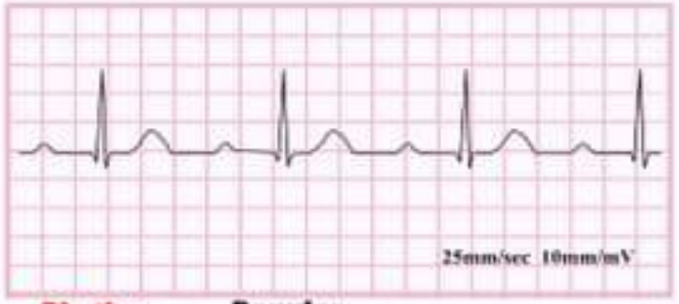
NO?

STEP-5: Slightly more +ve? - move the axis 15° towards the euiphasic lead.

STEP-6: Slighty more -ve? - move the axis → 15°away. Now you have found your cardiac axis to the nearest 15°

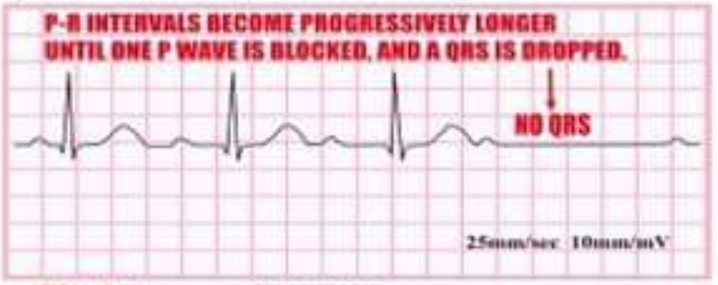
Heart Blocks

First Degree AV Block



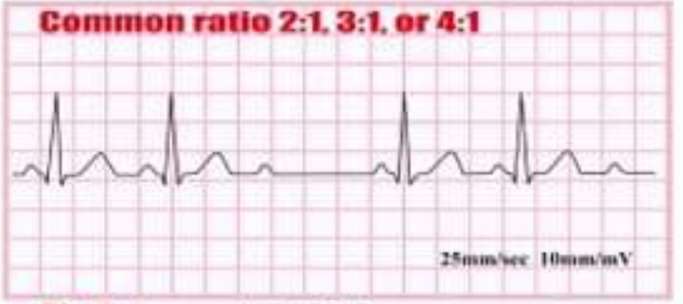
Rhythm: Regular
PR interval: Prolonged >0.20 sec
P Wave: Normal
QRS: <0.12

Second Degree AV Block - Type 1 (aka Mobitz 1, Wenckebach):



Rhythm: Irregular
PR interval: Increasingly Prolonged
P Wave: Normal
QRS: <0.12

Second Degree AV Block - Mobitz Type 2

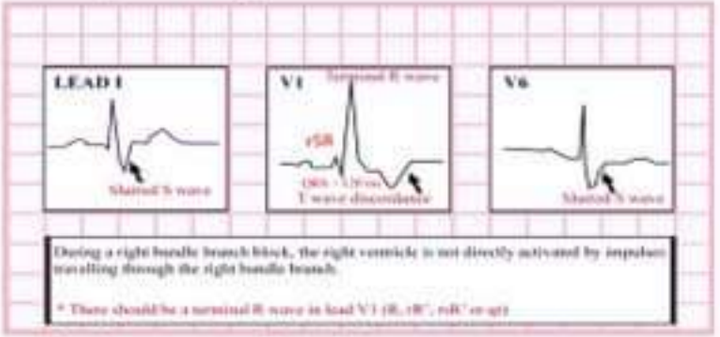


Rhythm: Irregular
PR interval: Normal (more P waves then QRS)
P Wave: Normal
QRS: Usually wide >0.10

3rd Degree AV Block

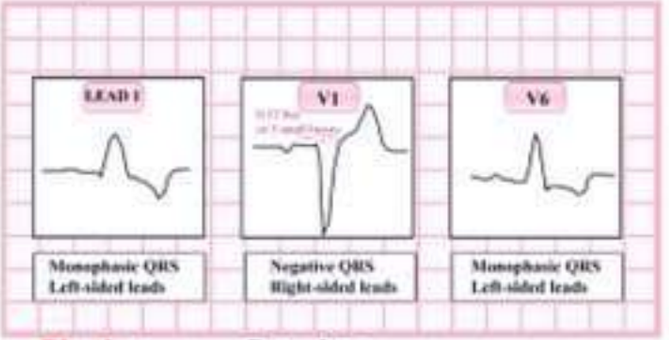
Rhythm: Regular
PR interval: None
P Wave: Normal does not relate to QRS
QRS: Normal or wide

Complete Right Bundle Branch Block



Rhythm: Regular
PR interval: Normal
P Wave: Normal
QRS: Wide >0.12

Complete Left Bundle Branch Block

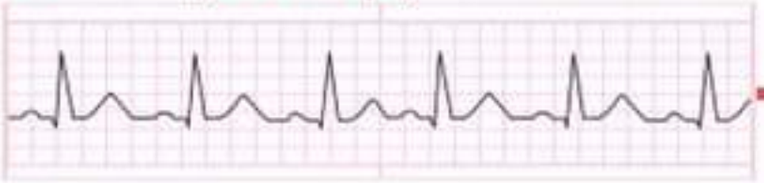


Rhythm: Regular
PR interval: Normal
P Wave: Normal
QRS: Wide >0.12

Changes on ECG in Hyperkalemia

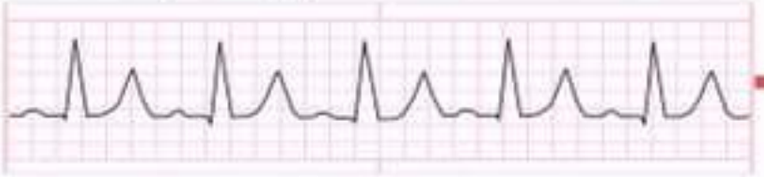
Serum potassium levels

A. normal (3.5 - 5.5 mEq/L)



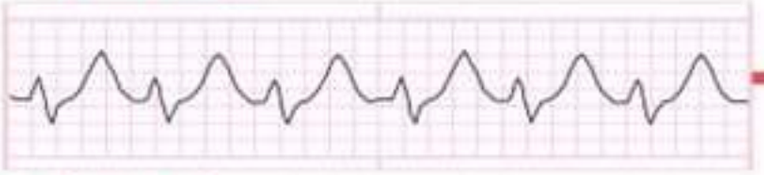
- Normal intervals and complexes

B. about (7.0 mEq/L)



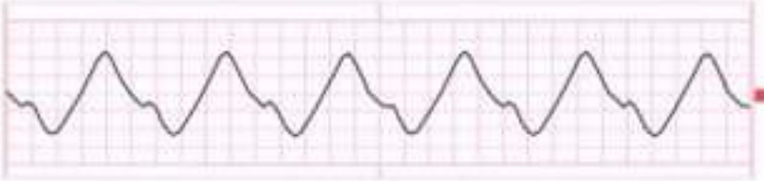
- Peaked T waves (usually the earliest sign of hyperkalaemia)
- P wave widens and flattens, PR segment lengthens, P waves disappear.

C. (8.0 - 9.0 mEq/L)



- Prolonged QRS interval with bizarre morphology and development of sine wave
- Can cause any kind of conduction blocks.

D. (>10 mEq/L)



- Sine wave (a pre-terminal rhythm). Wide, bizarre QRS. causes: Asystole, VF, PEA with bizarre wide complex rhythm.

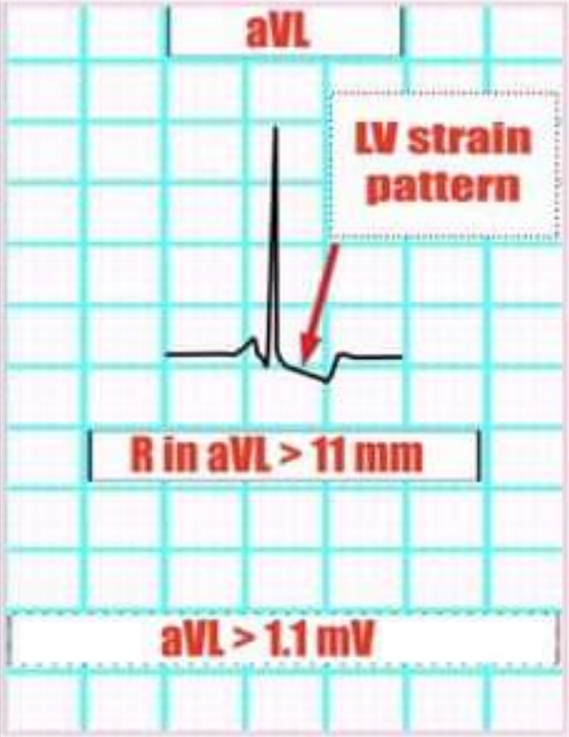
What is Hyperkalemia?

Hyperkalemia is elevated potassium (k+). Potassium is vital for regulating the normal electrical activity of the heart. Increased extracellular potassium reduces myocardial excitability, with depression of both pacemaking and conducting tissues.

The progressively worsening of hyperkalaemia leads to suppression of impulse generation by the SA node and reduced conduction by the AV node and His-Purkinje system, resulting in bradycardia with junctional & ventricular escape rhythms and conduction blocks and ultimately cardiac arrest.

LVH by voltage criteria - (Sokolow-Lyon Index)

Limb Leads



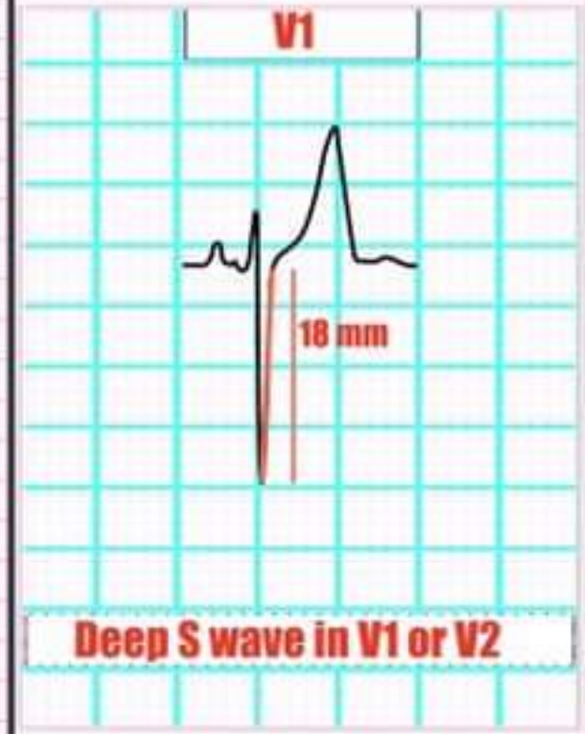
Leftward shift in frontal Axis -30°

I ⊕

II/aVF ⊖

LAD is associated with LVH

Right-sided Leads



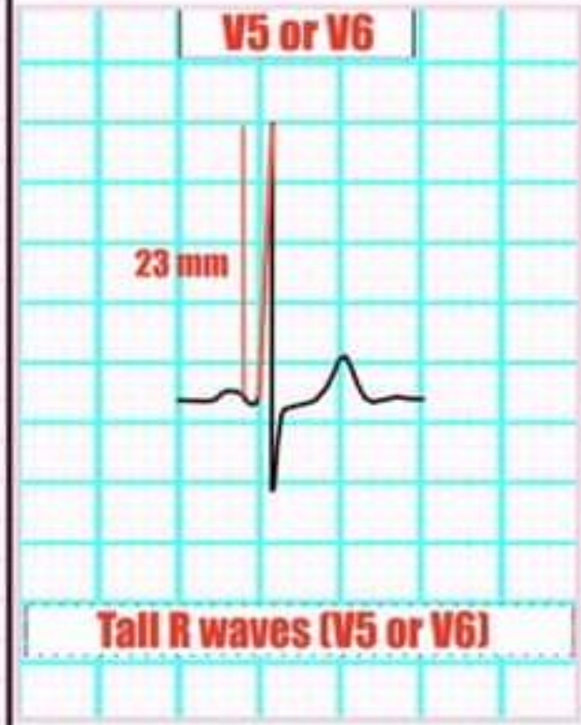
The S wave in V1 is deep

In this example above we measure the S wave in V1 at 18 mm.

Add the S wave in V1 plus the R wave in V5 or V6. If the sum is > 35 mm, then LVH is present.

> 35 mm is significant

Left-sided chest Leads



The R wave in V5 and V6 is high

In this example above we measure the R wave in V5 at 23 mm.

So we add both measurements together from V1 & V5:

S(V1) + R(V5) = 41mm (>35 mm)

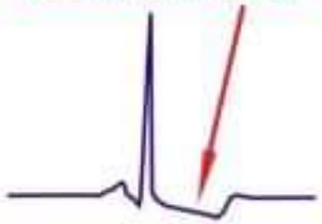
* This meets the criteria for LVH

Causes to Consider

Pressure Overload
Aortic Stenosis
Systemic Hypertension

Volume Overload
Congestive Heart Failure
Aortic Regurgitation

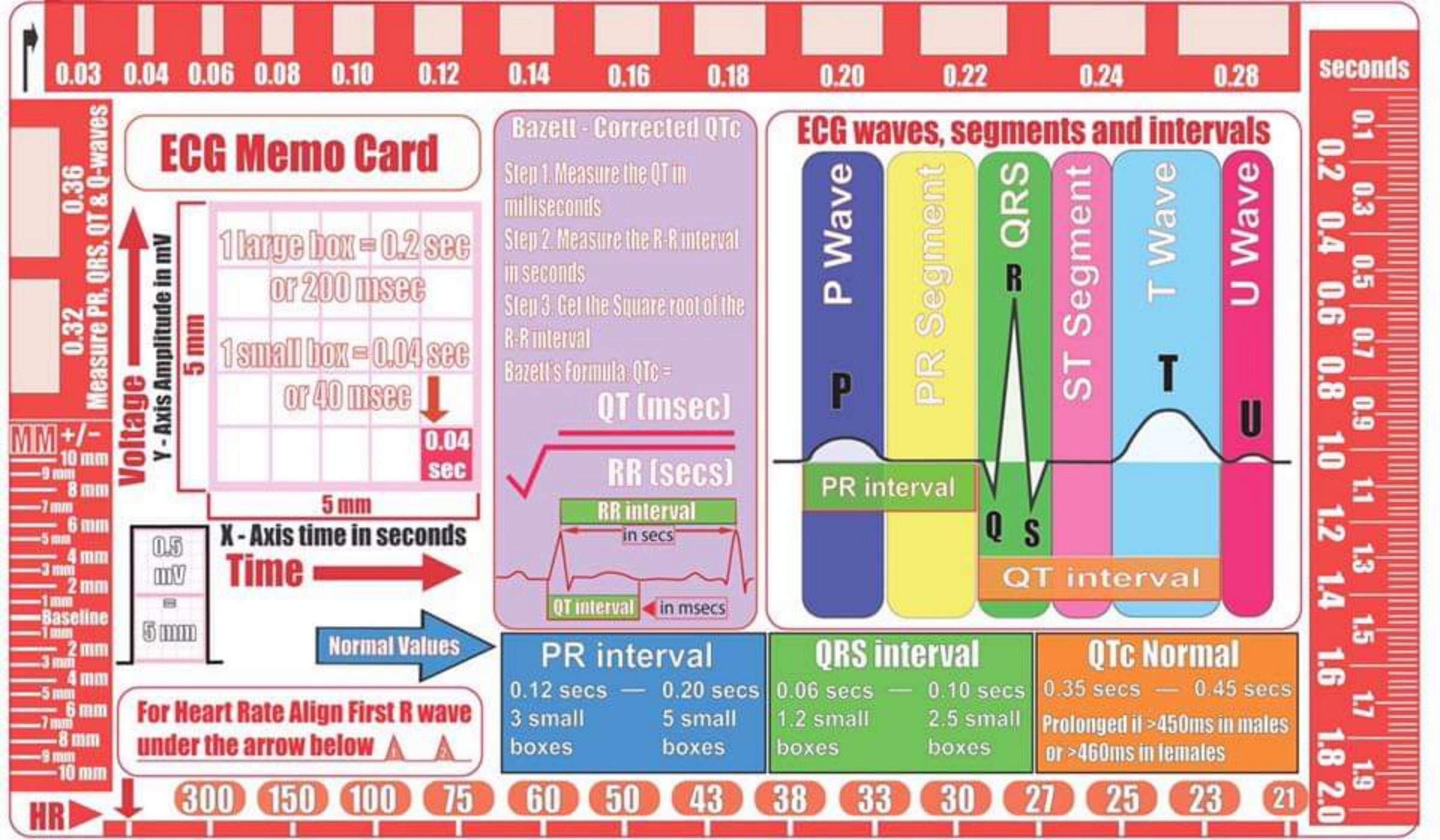
ST depression and T wave inversion (TWI) can occur in leads with prominent R waves.

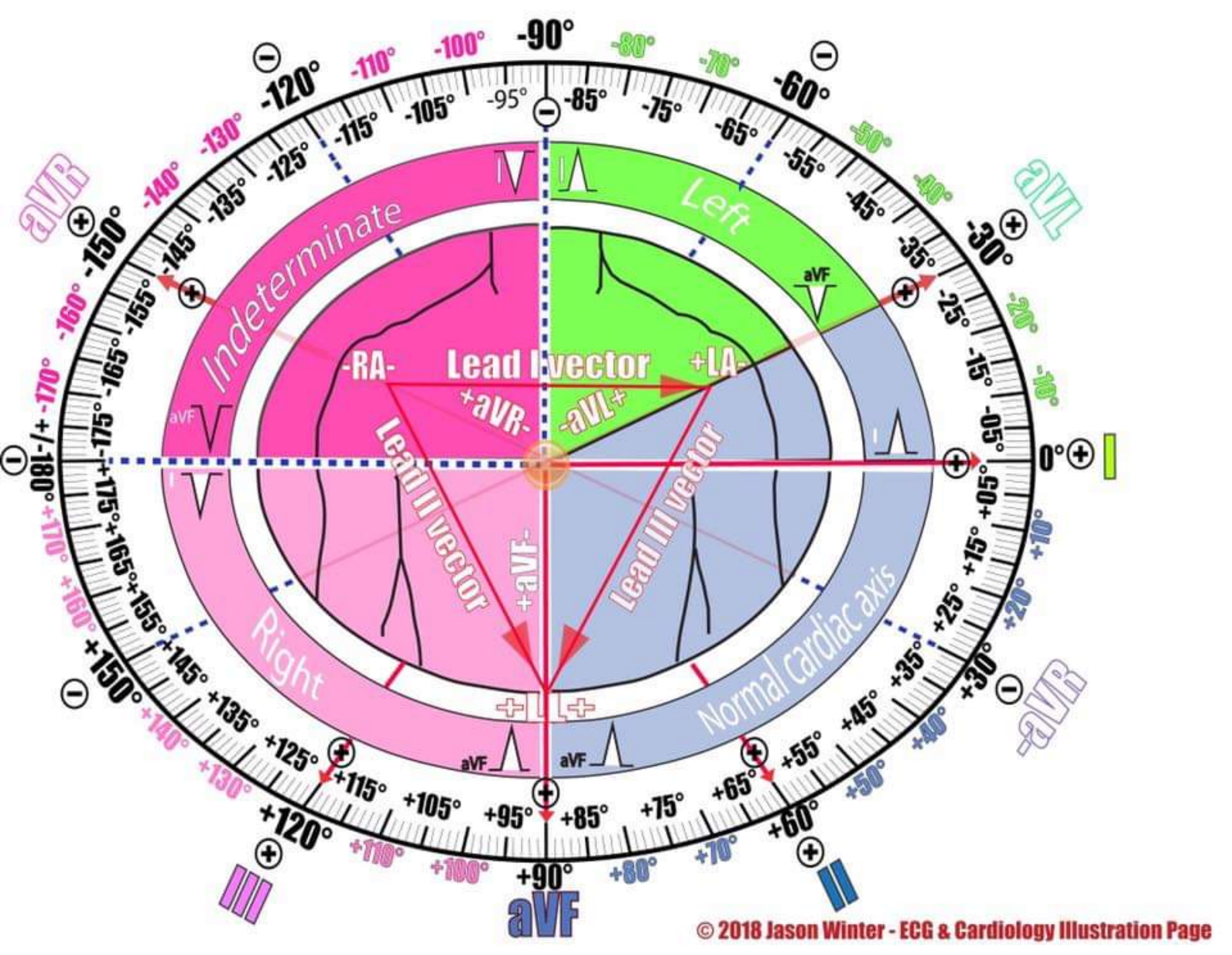


The Sokolow-Lyon index

S in V1 + R in V5 or V6 (whichever is larger) > 35 mm or 3.5 mV = (7 large squares).

or R in aVL > 11 mm





QT Interval: How to measure it

Bazett - Corrected QTc

· **Normal QTc values:** QTc is prolonged if > 440ms in males or > 460ms in females.
> 500ms is associated with increased risk of torsades de pointes. QTc is abnormally short if < 350ms.

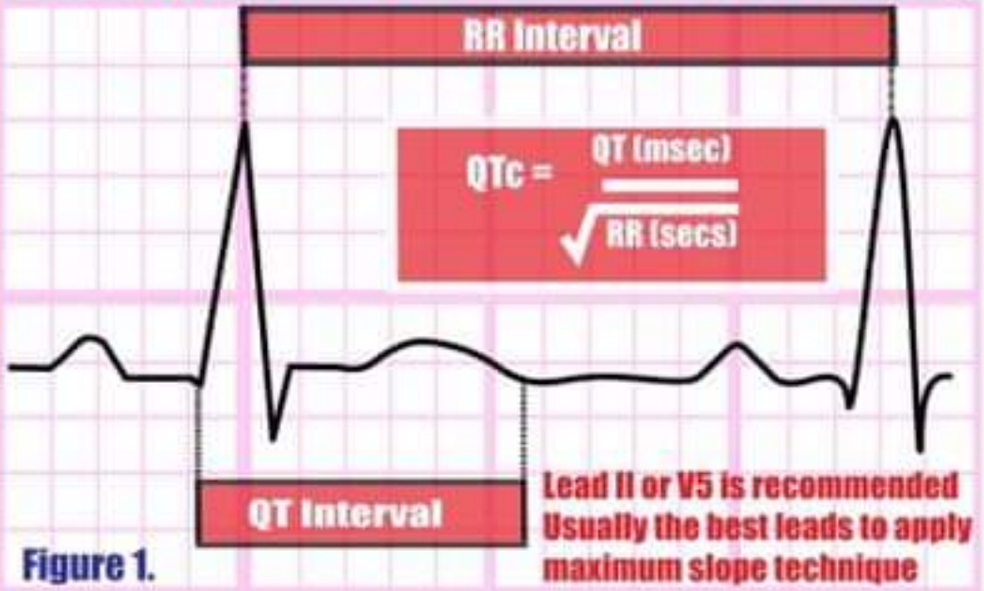


Figure 1.

Lead II or V5 is recommended
Usually the best leads to apply maximum slope technique

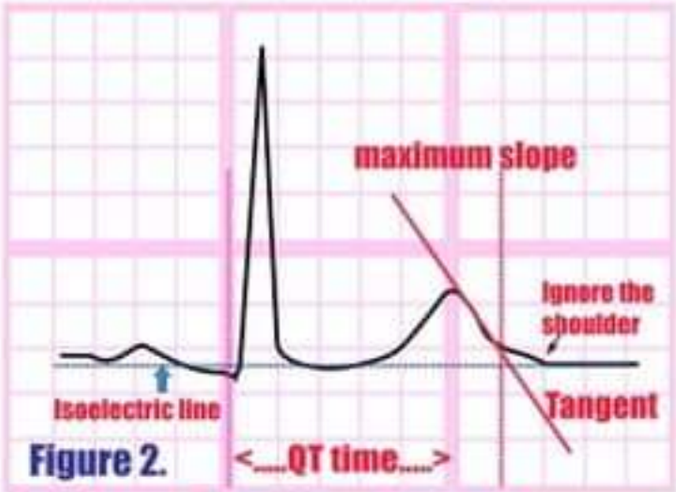


Figure 2.

- Large U waves (> 1mm) that are fused to the T wave should be included in the measurement.
- Smaller U waves and those separate from the T wave should be excluded.

Measuring the QTc

Step 1. Measure the QT in milliseconds

Step 2. Measure the R-R interval in seconds

Step 3. Get the Square root of the R-R interval

Bezett's Formula: $QTc = \frac{QT\ interval}{\sqrt{RR\ (Seconds)}}$

***Abnormally long and short QT intervals have been shown to be associated with an risk for life-threatening ventricular arrhythmia and sudden cardiac death.**

* The QT shortens at faster rates and lenthens at slower rates

* The corrected QT interval (QTc) estimates the QT interval at a heart rate of 60 bpm, this allows comparison of QT values over time at different heart rates and improves detection of arrhythmias.

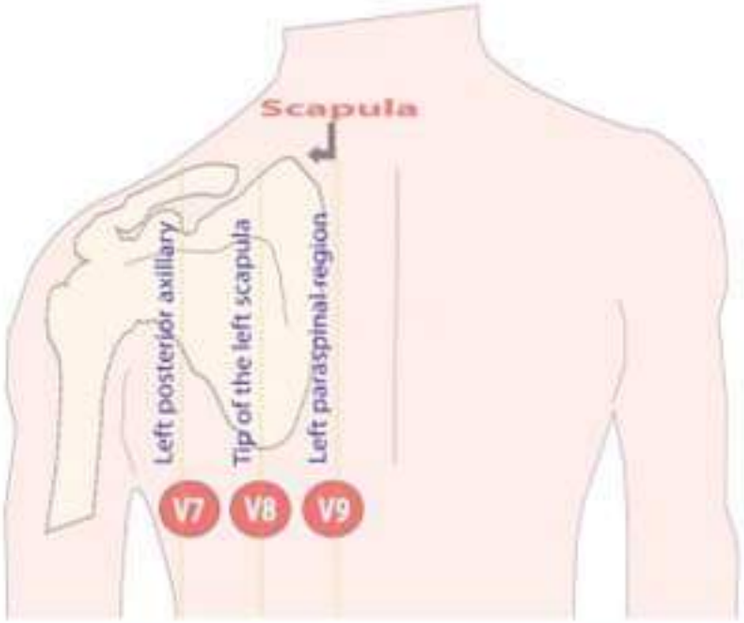
QTc = Corrected QT interval
R-R interval = Time from two consecutive R waves
QT interval = Q wave to end of T wave

ECG Basics - Posterior ECG leads

ST elevation greater then 0.5 mm in leads V7-9 is diagnostic of a Posterior wall MI

Figure 1

Posterior ECG lead placement



V7
Left posterior axillary line: in the same horizontal plane as V4-V6

V8
Tip of the left midscapula: in the same horizontal plane as V7-V9

V9
Left paraspinal region: in the same horizontal plane as V4-V6

V1-V3
Should remain unchanged from standard 12-lead ECG

Posterior MI is suggestive by the following changes in V1-V3:

- Horizontal ST depression
- Tall, broad R waves (>30ms)
- Upright T waves
- Dominant R wave (R/S ratio >1) in V2

Please note that V6 is a good reference point for the horizontal placement of the posterior electrodes V7-9.

If you don't have access to a 15 or 18 lead ECG machine, then leave V1-3 in their normal position and use V4-6, these leads will then become V7-9.

12-Lead acute Posterior wall MI

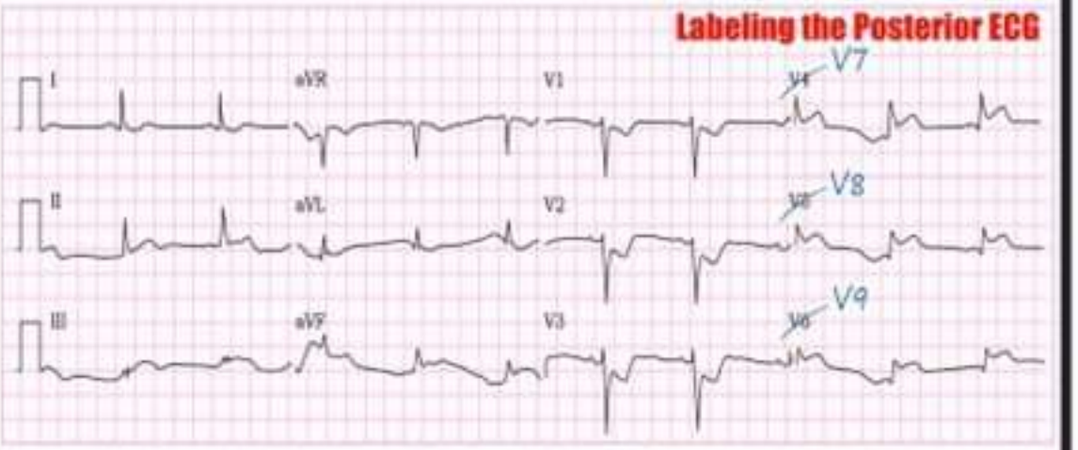


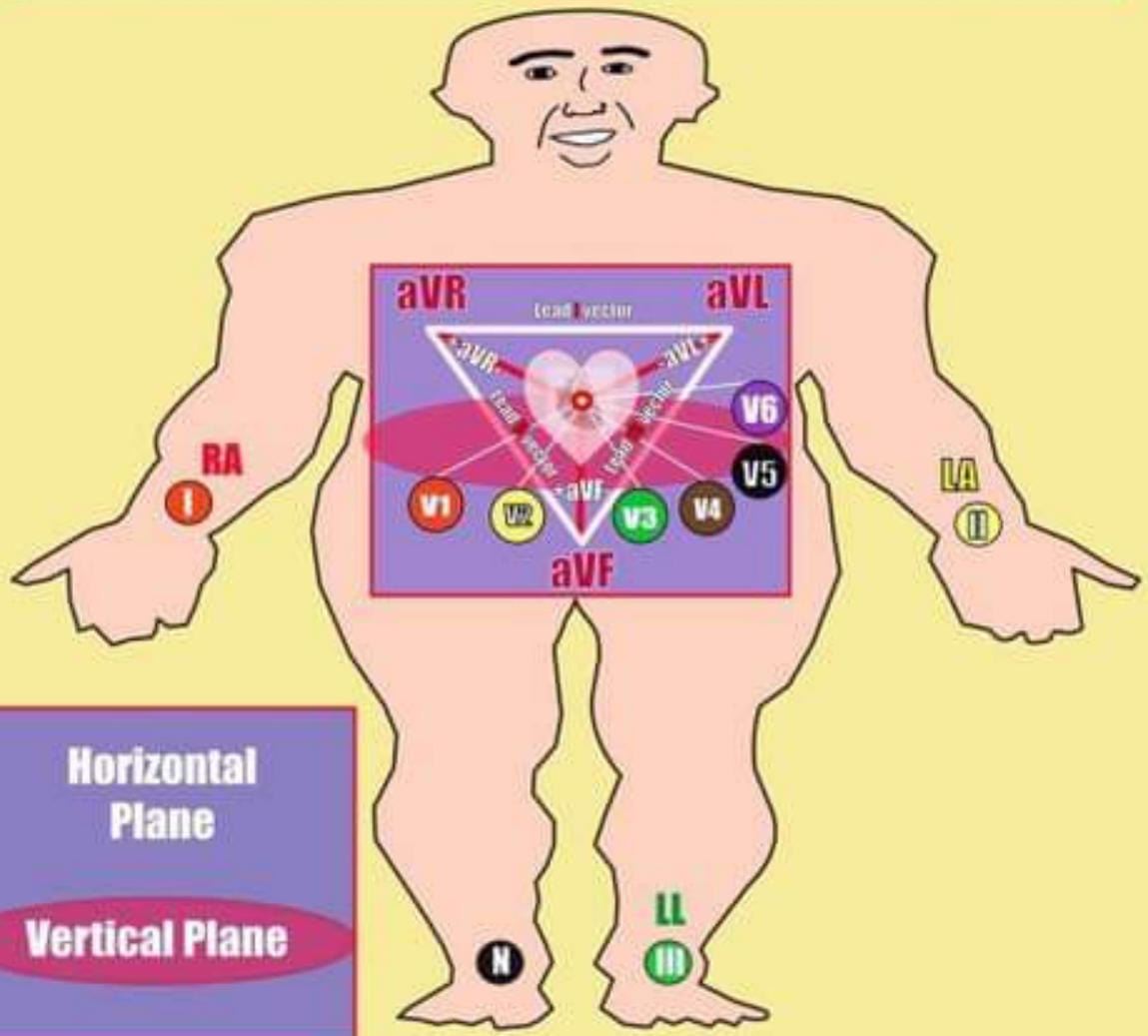
Figure 2 - Correct labeling of the Posterior ECG

Tips for recording Posterior Electrocardiograms (ECG's)

- Arm and leg electrodes remain unchanaged
- Lead cable V4 connects to V7
- Lead cable V5 connects to V8
- Lead cable V6 connects to V9
- REMEMBER to handwrite POSTERIOR LEADS on the ECG.
- Relabel V4-V6 on the printout V7-9 (Figure-2).
- Findings may be very subtle, only 0.5 mm.
- Always double check your lead placement to confirmed your in the correct anatomical spaces.

ECG Vertical & Horizontal Leads

ECG Leads Perspective Views



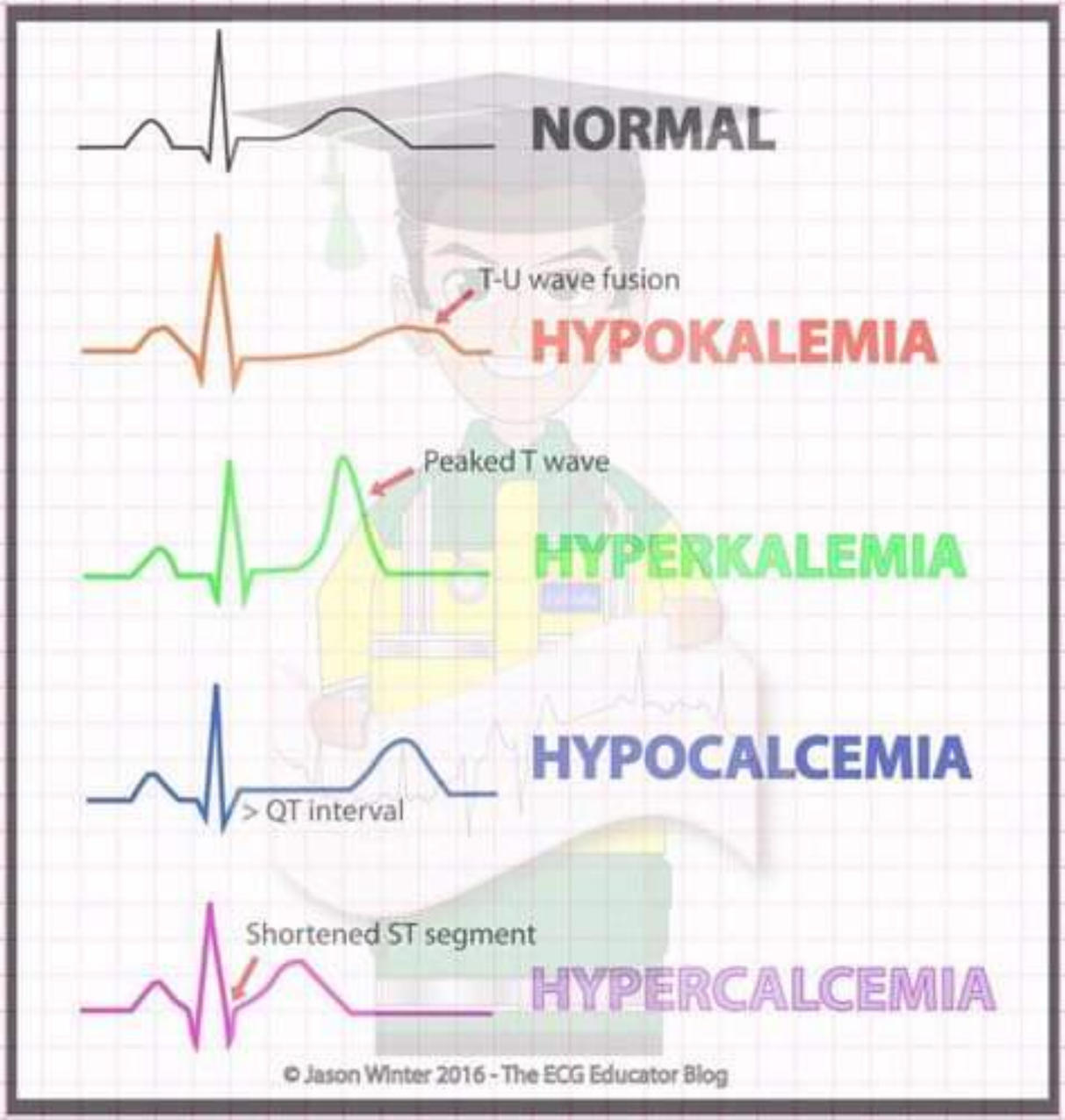
VERTICAL LEADS/FRONTAL PLANE

I	HIGH LATERAL (Right Arm (RA))	aVR	RIGHT ATRIUM
II	INFERIOR (Left Arm (LA))	aVL	HIGH LATERAL
III	INFERIOR (Left Leg (LL))	aVF	INFERIOR

HORIZONTAL PLANE

V1 SEPTAL/ANTERIOR (4th ICS, Right sternal border)	V2 SEPTAL/ANTERIOR	V3 ANTERIOR (Halfway between V2 & V4)
V4 ANTERIOR (5th ICS, mid-clavicular line)	V5 LATERAL (Halfway between V4 & V5)	V6 LATERAL (Mid-axillary line, even with V5)

Electrolyte effects on the ECG



HYPOKALEMIA (low blood potassium):

* > Amplitude and width of P wave
* ST depression and flattening or inversion of the T wave
* A U-wave may be visible
* Long QT interval due to T-U fusion = (long QU interval)

HYPERKALEMIA (high blood potassium):

* QRS widening
* fusion of QRS-T
* Tall tented T waves
* low amplitude & wide P-waves, due to slowing of conduction,
* P waves will eventually disappear

HYPOCALCEMIA (low blood calcium):

* Prolongation of the QT interval
* Prominent U-wave
* T wave flattening and inversion
* Narrowing of the QRS complex
* Prolonged ST and ST-depression
* Reduced PR interval

HYPERCALCEMIA (high blood calcium):

* Wide QRS
* Disappearearance of p waves
* Tall slightly broad peaking T waves
* Shortening of the QT interval
* Osborn waves (J waves) sometimes seen in severe hypercalcaemia