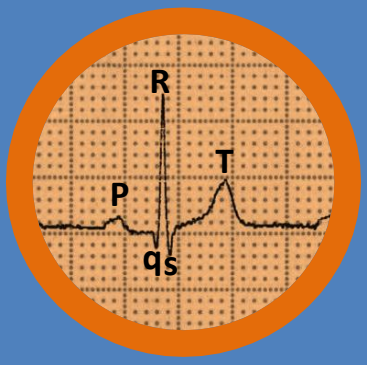

ECG Review : Some momentous articles

Series 6

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

A dance of ideas where
Thoughts brightly glow .....

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A dance of ideas where
Thought brightly glow

Knowledge and skill in the field of electrocardiography are constantly changing with the new researches and understanding.

With humble words I wish to say that some momentous articles of my write-up are being covered within this book.. It is only a step towards the vast ocean of knowledge. I may be excused for any error or omission.

With thanks and regards


DEDICATED
TO
FELLOW COLLEAGUES
Who are vibrant source of knowledge

Index

RECORDING OF ECG ON 12-LEAD SYSTEM : A FUNDAMENTAL APPROACH

RECORDING OF ECG ON 12-LEAD SYSTEM - A FUNDAMENTAL APPROACH

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OUTLINE

Introduction

The recording of ECG is a technical phenomenon by which the flow of current from the negative pole towards the positive pole is picked up due to the prevailing potential difference in between two poles of a lead with a bright reply onto the graph through the language of P-QRS-T.

ECG recording on 12-lead system

- ☐ **Hexaxial lead system :**.
  - ○ A six pointed electrical vertical system – The three bipolar arms lead I , II , III and three augmented unipolar leads aVR , aVL , aVF constitute hexaxial lead system
  - ○ The basic facts leading to the formation of hexaxial lead system
- ☐ **Precordial lead system :**
  - ○ Horizontal electrical field consisting of chest leads V1-V6
  - ○ How does it help in recording the electrical changes

Some pertinent points while placing the 12-leads over the body

A broader convas of 12-lead system to gain insight the electrical changes in toto at a glance

- ☐ The combined mental picture of 12-lead system
- ☐ The graphing of electrical axis

Take Home Message

References

Recording of ECG on 12-lead system - A fundamental appraoch

A Narrative Review

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'O Heart' , thou sends the electrical signal from within to outskirts of thou biospherical territory. The ECG transforms thou electrical impulses into measurable waves , converting each depolarization and repolarization into a visible pattern as a precise recording of thou electrical activity through 12-lead system .

- ☐ **Hexaxial lead system for recording the electrical flow over the vertical plane through the arrangement of limb leads (I , II ,III) with augmented unipolar leads (aVR , aVL and aVF)**
- ☐ **Precordial lead system is spreadover through six leads (V1-V6) placed over the chest in a specific semicircular manner.**

The 12-lead system is a boon in recording the electrical activity of the heart.

1. Introduction

Each cardiac impulse innovates and dissipates an electric field which wraps the heart from within and out. The physical principle states that every electrical field is coupled with a magnetic field as well and this association creates an electromagnetic force around the heart. The heart is having the largest rhythmic electromagnetic field which is widespread over the body with its expanding flame even outside its sphere.

By 12-lead ECG , this cardiac electrical activity is picked up from both the vertical and horizontal planes. The limb leads are used to detect the electrical current over the vertical plane and the chest leads are used to detect the electrical changes over the horizontal plane.

The recording of ECG is a technical phenomenon by which the flow of current from the negative pole towards the positive pole is picked up due to the prevailing potential difference in between two poles of a lead with a bright reply onto the graph through the language of P-QRS-T.

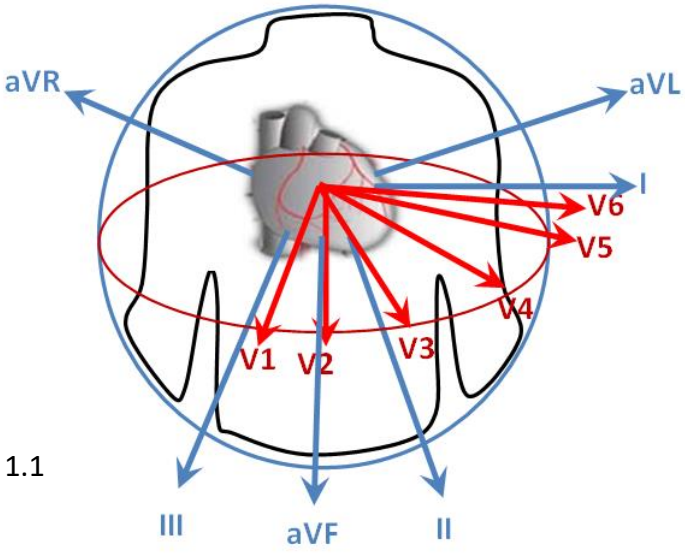


Fig. 1.1

2. ECG recording on 12-lead system

- ☐ **Hexaxial lead system**
- ☐ **Precordial lead system**

Hexaxial lead system

The hexaxial lead system expands the overview of the heart's electrical field over the vertical plane, creating a six pointed perspective system which captures the electrical signals from different angles , transforming the complex current into measurable waves. It makes also easier to understand at which direction the cardiac vector is projected.

The basic facts leading to the formation of 'Hexaxial lead system' :

- **Einthoven's triangle** is the "Soul-essential" for the purpose.
  Einthoven Triangle is an imaginary geometry based three limb leads triangle , being formed by the two shoulders (left and right) and the left leg
  This is an inverted electrical triangle with the heart lying at the centre.
- **Augmented Unipolar Limb leads : Dr. Emanuel Goldberger**
  Unipolar limb leads were named as augmented unipolar limb leads – aVL, aVR, aVF (aV denotes augmented vector and L, R, F used for different concerned limbs).
- **The concept of the central terminal : Goldberger's indifferent electrode**
  Since the recorded signals from these unipolar leads were small , Goldberger devised a special central terminal to augment these leads by 50% - by averaging only two of the limb electrodes and excluding the one being measured. **This is used as an indifferent electrode (negative electrode) to record the potential difference in respect to unipolar limb leads.**

The understanding of stepwise construction of hexaxial lead system :

STEP 1 Leads I , II , III represent bipolar limb system , while aVL, aVR , aVF represent augmented unipolar limb lead system

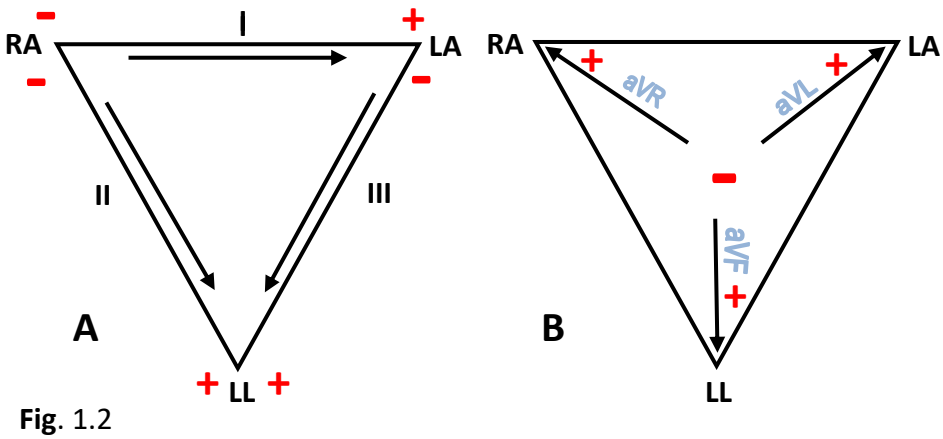
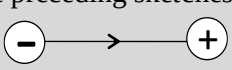


Fig. 1.2

The flow of cardiac current on the vertical plane is picked up through the negative electrode to be directed towards the positive electrode due to the potential difference in between these two poles , as illustrated in the preceding sketches.



Lead aVR

Being placed at its extreme opposite end , the lead aVR usually records all the waves as negative (P-QRS-T).

STEP 2

By rearranging the augmented unipolar leads (aVR, aVL, aVF) with those of limb leads of Einthoven's triangle (I, II ,III) , as per the following sketch :

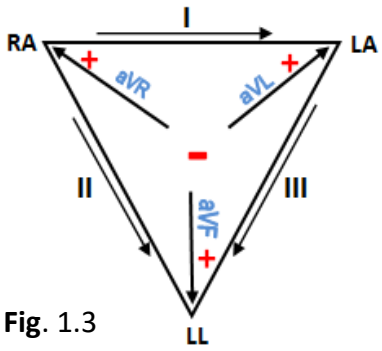


Fig. 1.3

Leads	Negative Electrode	Positive Electrode
Bipolar I.	Right Arm	Left Arm
II.	Right Arm	Left Leg
III.	Left Arm	Left Leg
Unipolar aVR	Central Terminal	Right Arm
aVL		Left Arm
aVF		Left Leg

STEP 3

The three bipolar arms leads I ,II ,III are imagined to pass parallel through the centre of the triangle and the augmented unipolar leads aVR , aVL , aVF are remaining as such , being projected from the centre to the respective corners of the triangle – thus , the 'Hexaxial Lead System' came into existence (the heart is conceptually considered to lie at the centre)

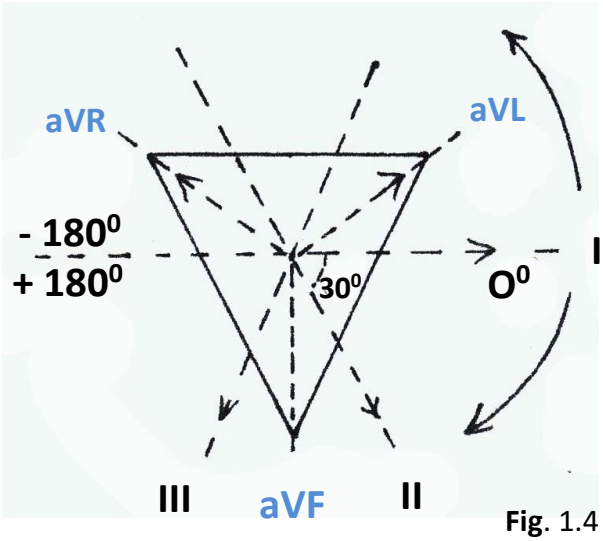
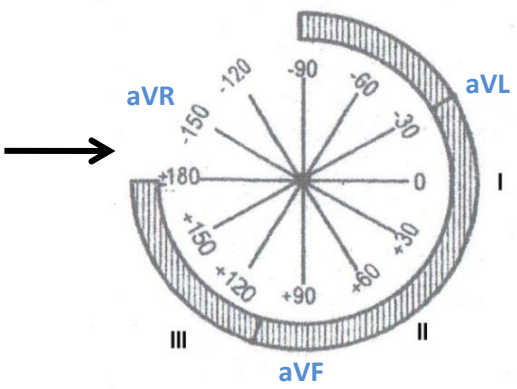


Fig. 1.4



Hexaxial Lead System

By convention the leads are arranged degreewise around a circle as shown above. Lead I is located at 0^o being used as the reference lead. Positive designation gets increased by 30^0 increments in a clockwise direction to $+180^0$ and negative designation gets increased by the same increment (30^0) in a counterclockwise direction to -180^0(please see the above diagram). Thus, so oriented negative and positive nomenclature of the various leads are used by convention for the mathematical determination of the electrical axis. The positive and negative degrees are nothing to do with the positive or negative pole of the leads. This is to be mentioned here that this system came into existence because of its conventional and

constant usage in clinical practice. This inscription of 'Hexaxial lead system' is very much helpful in the determination of the electrical axis.

Precordial lead system

The precordial lead system expands the overview of the heart's electrical field over the horizontal plane , it records all the net electrical events from the selected site , as per the placement of the precordial leads V1 to V6. This system records electrical potential from the underneath myocardium , as such illustrated below :

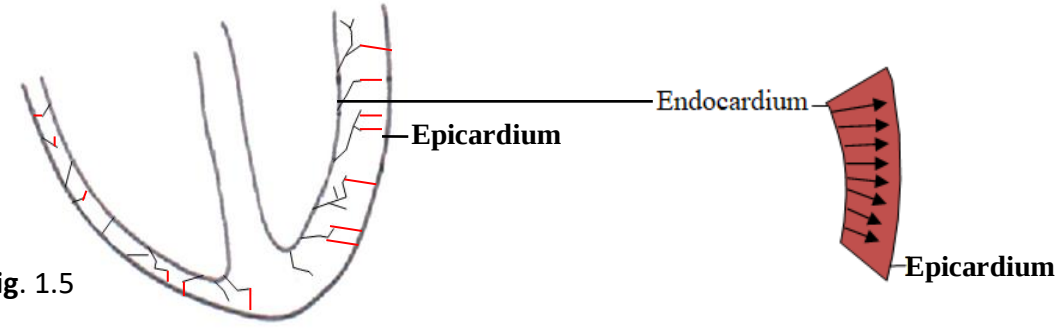


Fig. 1.5

The ventricular wall with Purkinje-myocytes network

Magnified view of ventricular wall (transmural)

How does it help in recording the electrical changes

- Waves morphology as per sequential activation of the Ventricles from V1 to V6
- The left ventricle is thicker than that of right ventricle , therefore the net flow is towards the left ventricle.
- Positive deflection towards the flow of current , equiphasic or no deflection at transitional zone and –ve deflection when away from the flow of current.
- Septal wave - the normal left-to-right depolarization of the interventricular septum
  r (V1-V2) / q (V5-V6) ; +ve R-wave over V5-V6 /-ve S-wave over V1-V2

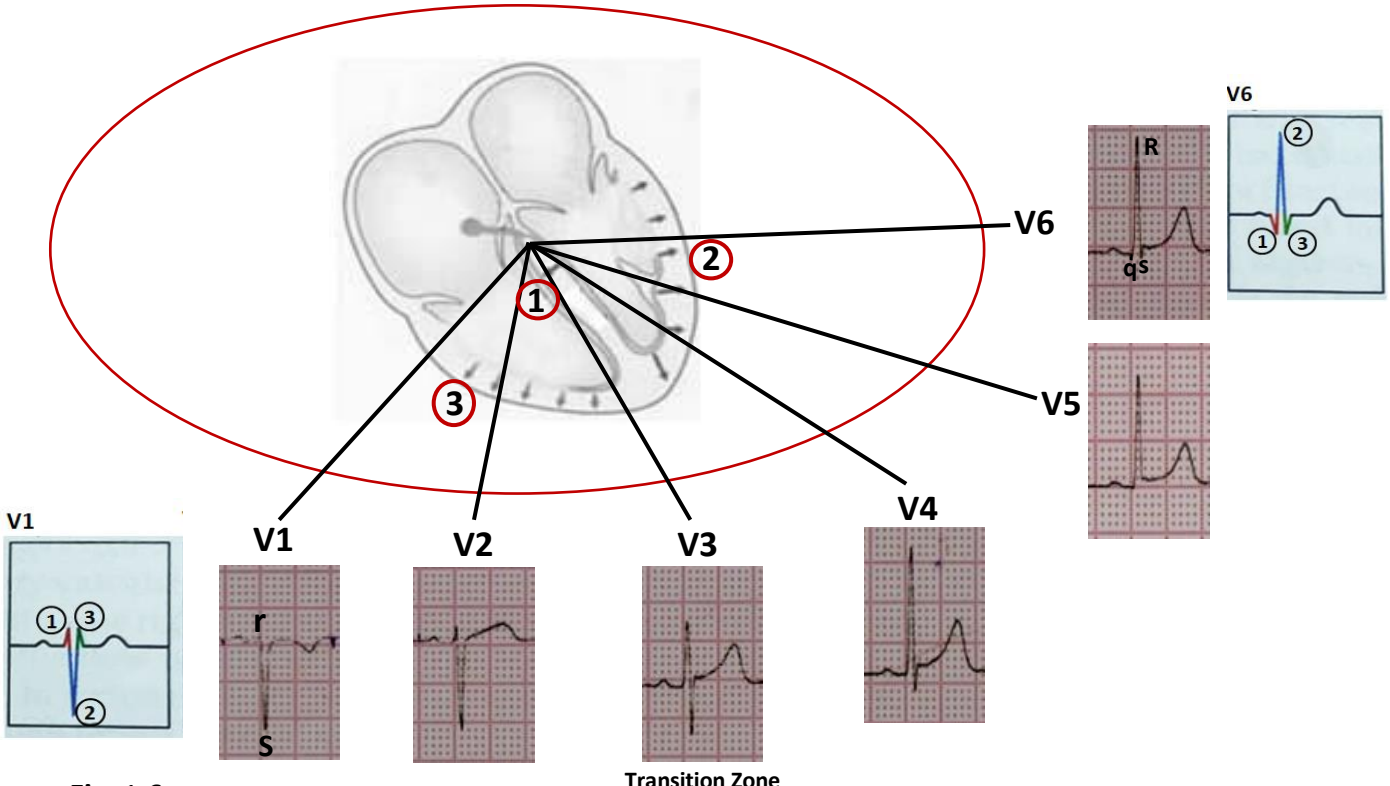
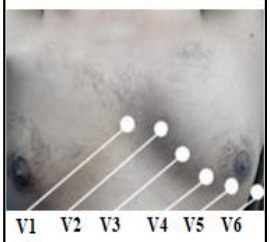
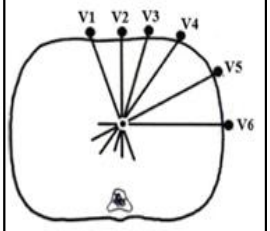
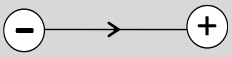


Fig. 1.6

Placement of precordial leads over the chest

Precordial   	V1 V2 V3 V4 V5 V6 **(V stands for Voltage)**	***Wilson central terminal** (This is different from that of Goldberger's central terminal)	4th Right intercostal space parasternal 4th left intercostal space parasternal Midway between V2 and V4 5th intercostal space at left midclavicular line Level with V4 at left anterior axiliary line Level with V5 at left mid axiliary line

***Wilson central terminal** is created by adjoining the potential from the right arm (RA) , left arm (LA) , and left leg (LL) electrodes. This terminal represents the average of the unipolar limbs potential near about 0 (zero). This indifferent terminal with negative potential records the potential different in respect to inidvidual precordial lead.



3. Some pertinent points while placing the 12- leads over the body

Hexaxial Lead system

☐ The individual limb electrode as guided by its printed colour is placed over the distal part of the corresponding right arm and left arm , just proximal to the wrist joint and over the right leg and left leg , just proximal to the ankle joint –taking special care to place the electrode over the bony prominence , not over the muscular part to have the smooth recording of ECG tracings.

IEC(International Electrotechnical Commission)

Inscription	Colour
RA (Right arm)	**Red**
LA (Left arm)	**Yellow**
RL (Right leg)	**Black**
LL (Left leg)	**Green**

- In case of any difficulty in placing the limb electrode over any limb, say in case of amputed or short arm , the exploring electrode may be placed over any point of the corresponding limb .
  It would be worthwhile to mention here that :

  > As per *LeoSchamroth – An Introduction to ELECTROCARDIOGRAPHY - EIGHTH ADAPTED EDITION : P-6 (‘ With distances greater than 15 cm from the heart, the decrement in the intensity of the electrical field is hardly noticeable. Consequently, all electrodes placed at a distance greater than 15 cm from the heart may, in an electrical sense, be considered to be equidistant from the heart ’).

- Right leg electrode works as neutral electrode , while this electrode does not directly contribute to the ECG waveforms tracings – it works as a neutral lead just to complete the electrical circuit.
  - Grounding and thus minimizing noise interference from the environment
  - Enhancing the quality and stability of the ECG tracings by allowing uninterrupted flow through the exploring electrodes.
- In nutshell, the limb leads work as augmented unipolar arm leads – aVR , aVL and aVF and the remaining right leg electrode works as a neutral lead-**N**.

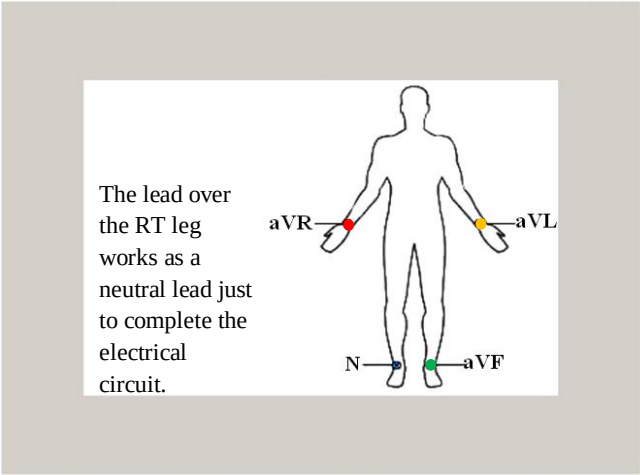


Fig. 1.7

The internal arrangement of bipolar leads I , II , III is facilitated through the internal adjustment within the ECG machine – one should not bother about this , it is automatically adjusted to record the ECG tracings through these bipolar leads. Only is needed to place the corresponding electrodes over the limbs as discussed with.

Precordial Lead system

- The correct placement of the precordial leads needs the correct counting of intercostal spaces – the fingers are moved downward across the upper part of the manubrium till the horizontal bony ridge (Angle of Louis) is felt , the intercostal space just below is 2^{nd} one and accordingly 4^{th} and 5^{th} intercostal spaces are counted.

Just follow the followings points for the purpose :

- Start at the second intercostal space
- Run your finger downward across the next ribs till the needed intercostal space is felt.

Common mistake in ECG precordial lead placement is placing V1 and V2 too high and placing V5 and V6 too medially.

☐ The placing of the individual precordial electrode is also guided by its printed colour over V1 , V2 and so on.

IEC(International Electrotechnical Commission)

Inscription	Colour
Lead V1	**Red**
Lead V2	**Yellow**
Lead V3	**Green**
Lead V4	**Brown**
Lead V5	**Black**
Lead V6	**Purple**

Please refer to P- 6 for placing of precordial leads over the chest

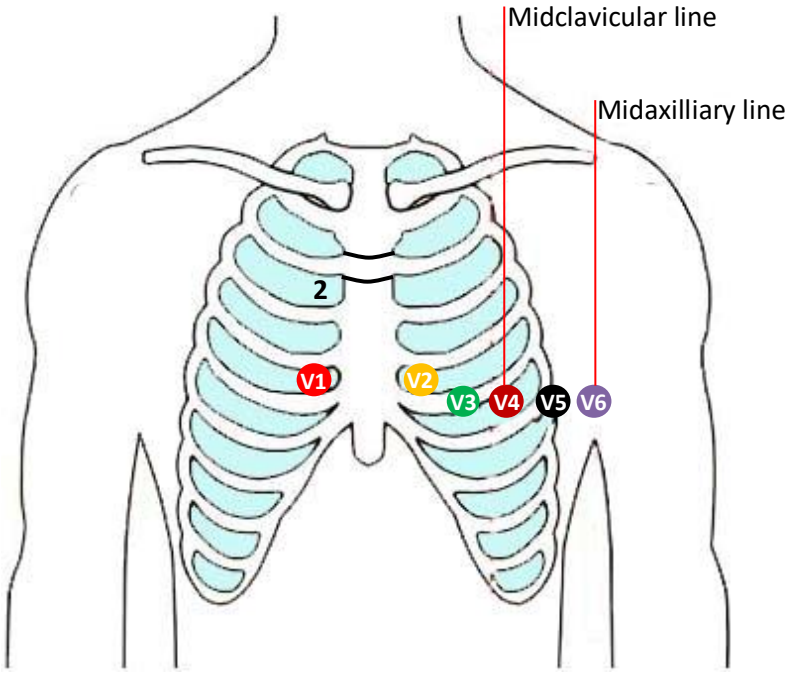


Fig. 1.8

4. A broader convas of 12-lead system to gain insight the electrical changes in toto at a glance

☐ It would be beneficial here to have the combined mental picture of 12-lead system

Classification of ECG based electrical changes , as reflected on 12-lead system

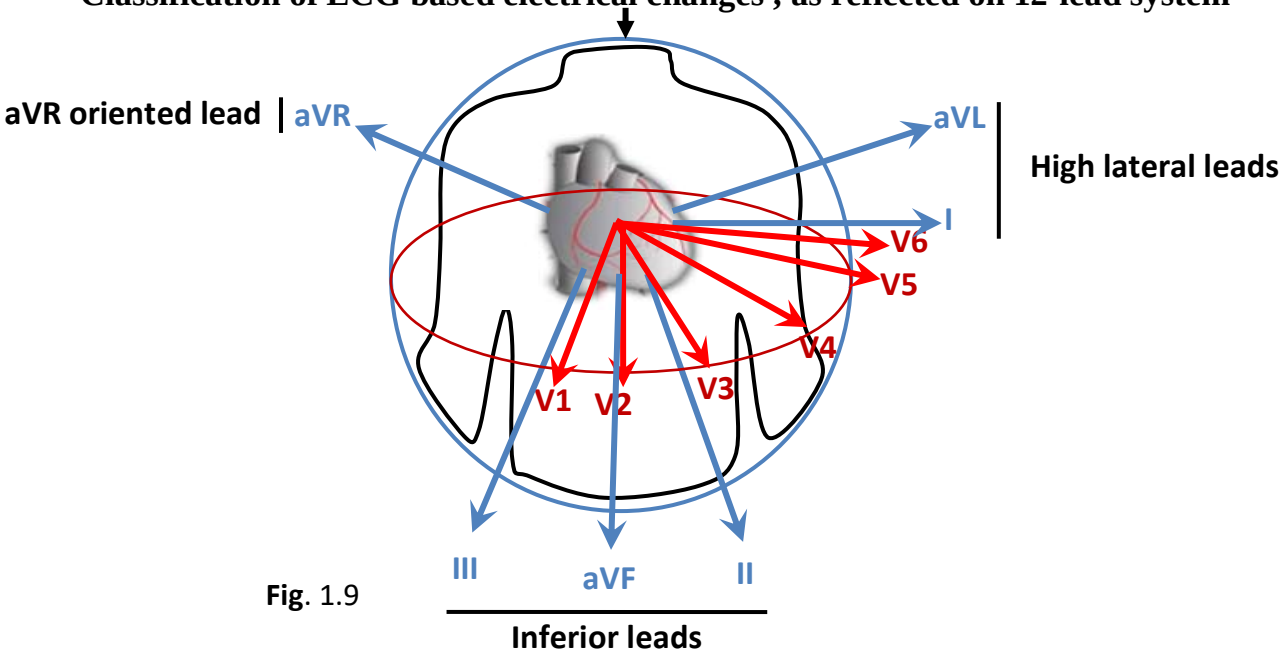


Fig. 1.9

Right ventricular epicardial complex - V1 , V2

Transistional zone - V3 , V4

Left ventricular epicardial complex - V5, V6, aVL, I

aVR oriented lead

This is possible to record the electrical activity from the right arm – a lead (aVR) looking opposite to the direction of electrical vector – a very useful discovery.

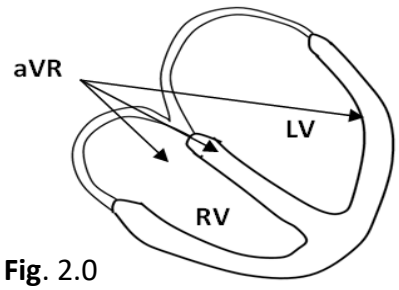


Fig. 2.0

This aVR lead gives a useful information viewing the right upper side of the heart such as outflow tract of the right ventricle and the basal part of the septum. This lead also gives reciprocal information from the lateral side of the left ventricle.

☐ **The entire frontal hexaxial system is divided into four quadrants for the purpose of graphing the electrical axis.**

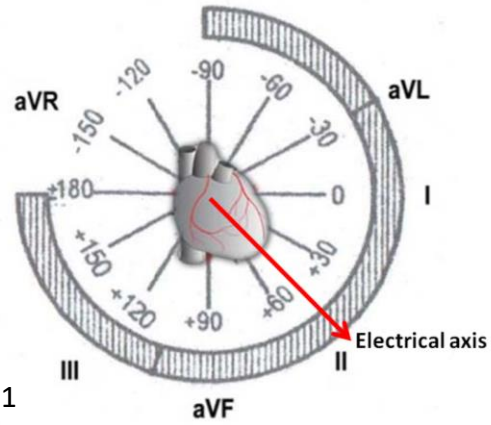


Fig. 2.1

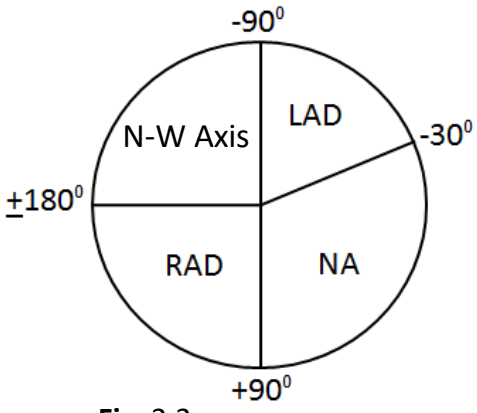


Fig. 2.2

QUADRANT DETERMINATION

Look at QRS complex in lead I and II		
QRS Positive in leads I and II	Normal Axis	-30^0 to $+90^0$
QRS complex is positive in lead I but negative in lead II	Left Axis Deviation	-30^0 to -90^0
QRS Negative in lead I but positive in lead II	Right Axis Deviation	$+90^0$ to $+180^0$

A predominantly positive QRS complex in aVR associated with a predominantly or wholly negative deflection in lead I and aVF- **North -west Axis** - 90^0 to -180^0

Indeterminate QRS axis is not equivalent to north-west axis. This terminology is used if QRS complex is equiphasic almost in all limb leads , then QRS axis cannot be truly plotted.

☐ **The basic principle of electrical axis determination**

The axis of the ECG denotes the major direction of the overall electrical activity of the heart over the vertical plane. The following concept would be very useful while calculating the electrical axis of the heart.

✓ Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive deflection , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection (with reference to hexaxial lead system)

☐ **The accurate localization of electrical axis**

- One can reach to the nearest multiple of 30 degree as per hexagonal lead system, the electrical axis would be in midway of both the equally positive deflections. And if one deflection is having more net positivity , the axis should be considered to be tilted towards that lead by the range of 10^0.

- Then it becomes easier to graph the mean electrical axis of any particular deflection with reasonable accuracy with an error of approximately 10^0 (within a few seconds by the process of inspection , but it comes by gradual practice while interpreting the ECGs).

✓ **And for this purpose , the hexaxial lead system must be kept always either in mind or on the paper, as illustrated below :**

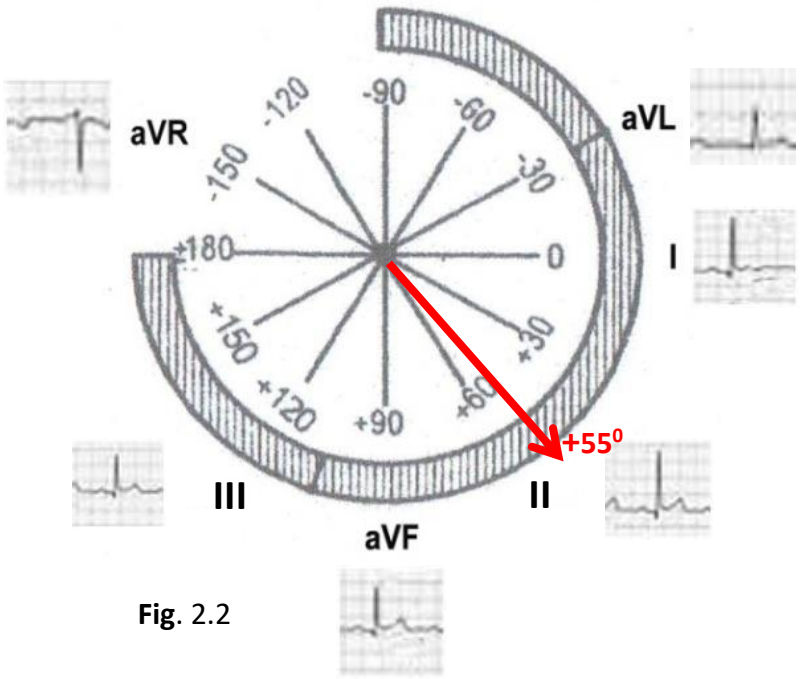


Fig. 2.2

If all the limb leads (except aVR) are showing predominantly positive waves as in this ECG , the axis lies in between $+30^0$ to $+60^0$. For the purpose the negative wave over aVR is plotted against the opposite pole at $+30^0$ as a presumptive positive deflection. The R in lead II is taller as compared to plotted positive wave of aVR at $+30^0$ and so the QRS axis is approximately at $+55^0$.

NB : The normal frontal QRS axis is at - 30^0 to $+90^0$

The atrial axis P and the T wave axis can also be determined by following the same principle (P axis : 0^0 to $+75^0$). The axis of the T wave should be accessed in relation to that of the QRS complex (the angle between QRS axis and T wave axis , QRS-T angle normally does not exceed 45^0-60^0 on the frontal plane) .

5. Take Home Message

- ☐ The recording of ECG is a technical phenomenon by which the flow of current from the negative pole towards the positive pole is picked up due to the prevailing potential difference in between two poles of a lead with a bright reply onto the graph through the language of P-QRS-T.
- ☐ ECG recording on 12-lead system
  - Hexaxial lead system
  - Precordial lead system
- ☐ The hexaxial lead system expands the overview of the heart's electrical field over the vertical plane, creating a six pointed perspective system which captures the electrical signals from different angles , transforming the complex current into

measurable waves. It makes also easier to understand at which direction the cardiac vector is projected.
The three bipolar arm leads I , II , III and the augmented unipolar leads aVR , aVL , aVF constitute the hexaxial lead system

- The precordial lead system expands the overview of the heart's electrical field over the horizontal plane , it records all the net electrical events from the selected site , as per the placement of the precordial leads V1 to V6. This system records electrical potential from the underneath myocardium.
- Right leg electrode works as neutral electrode , while this electrode does not directly contribute to the ECG waveforms tracings – it works as a neutral lead just to complete the electrical circuit.
  - Grounding and thus minimizing noise interference from the environment
  - Enhancing the quality and stability of the ECG tracings by allowing uninterrupted flow through the exploring electrodes.
- It would be beneficial here to have the combined mental picture of 12-lead system , as discussed on Page 9
- The axis of the ECG denotes the major direction of the overall electrical activity of the heart over the vertical plane. The following concept would be very useful while calculating the electrical axis of the heart.
  Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive deflection , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection (with reference to hexaxial lead system)
- Normal Electrical Axis

  The normal frontal QRS axis is at - 30^0 to $+90^0$
  The atrial axis P and the T wave axis can also be determined by following the same principle (P axis : 0^0 to $+75^0$). The axis of the T wave should be accessed in relation to that of the QRS complex (the angle between QRS axis and T wave axis , QRS-T angle normally does not exceed 45^0-60^0 on the frontal plane) .
- With precordial lead system
  - Waves morphology as per sequential activation of the Ventricles from V1 to V6
  - The left ventricle is thicker than that of right ventricle , therefore the net flow is towards the left ventricle.
  - Positive deflection towards the flow of current , equiphasic or no deflection at transitional zone and –ve deflection when away from the flow of current.
- Though this part as below is dealt here at the end but it should be rather touched in the beginning :
  - Voltage standardization is 1 mV = 10 mm (10 small squares deflection vertically on applying 1 mV current)
    Deviations from standardization by 1 mV = 5 mm (half standard) or 1 mV = 20 mm (double standard) may be used in cases where waveforms are unusually large or small.

- Speed of recording
  The standard paper runs at 25 mm / second.
  Speed with 50 mm / second is used to 'stretch out' the ECG for detailed analysis of rapid arrhythmias or closely spaced waveforms.

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MAKING SENSE THROUGH SERIES OF ECG SIGNS IN ACUTE MI IN THE PRESENCE OF LBBB

MAKING SENSE THROUGH SERIES OF ECG SIGNS IN ACUTE MI IN THE PRESENCE OF LBBB

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OUTLINE

Introduction
To make the diagnosis of occlusion myocardial infarction (OMI) in patients exhibiting left bundle branch block (LBBB) places a challenging diagnostic task to the attending clinician.

Consideration of ST-elevation in context with LBBB and occlusion MI

The basic electrophysiology in combined scenario of acute MI with LBBB
Practice has now emerged to access for concordant ST elevation/depression ± excessive discordant ST segment elevation indicative of myocardial infarction in the presence of LBBB

Outline of different diagnostic criteria for acute MI in the presence of LBBB

- ☐ The origianal Sgarbossa criteria
- ☐ The Smith-modified Sgarbossa criteria
- ☐ Barcelona criteria
- ☐ Chapman's sign
- ☐ Cabrera's sign

Stepwise interpretation of ECG having acute MI in the presence of LBBB

Illustration by ECGs

Take Home Message

References

Making Sense Through series of ECG signs in acute MI in the presence of LBBB

A Narrative Review

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When the heart stumbles with Acute MI under the shadowy imprint of LBBB, it becomes difficult to catch the real culprit red-handed. Really to say that there are many lacunae on the ECG that may hide the diagnostic clues, making it difficult to unearth the true image

- ☐ **The disruption of normal ventricular conduction in LBBB alters repolarization pattern , making a challenging task to diagnose occlusion myocardial infarction (OMI) in its presence.**
- ☐ **Timely diagnosis of occlusion MI in the presence of LBBB with precision opens the gateway to a better patient outcome**

There had been some researchers who had tried their best to curtain off the mist of this ignorance – to save the suffering humanity from this dilemma.

1. Introduction (Keynotes)

- To make the diagnosis of occlusion myocardial infarction (OMI) in patients exhibiting left bundle branch block (LBBB) places a challenging diagnostic task to the attending clinician.
- This diagnostic dilemma happens so due to the repolarization abnormality – encountered in both LBBB and acute occlusion MI
  - ECG with LBBB normally have a distinct ST elevation mainly over the right sided precordial leads
  - The occlusion MI , traditionally called STEMI is also having a particular pattern of ST elevation on ECG , as per involvement of coronary territory
- The presence of LBBB in a patient with symptoms suggestive of CAD had been conventionally considered as an ECG equivalent to ST-segment elevation MI and accordingly the 2017 European Society of Cardiology guidelines still recommend emergent reperfusion in such cases. However , increasing clinical evidence suggests that LBBB is a major cause of false activation of the cath lab. In view of this evidence there runs an opinion that LBBB should not be considered as a diagnostic norm of AMI in isolation.
- These facts put a diagnostic mist to the attending clinician what to do under the prevailing circumstances and so acute MI in the presence of LBBB may not receive timely repurfusion therapy with potential consequences to the suffering patient.

2. Consideration of ST-elevation in context with LBBB and occlusion MI

What about the ST elevation ?

- **LBBB** causes ST elevation over the right sided precordial leads - due to the fact that left ventricular repoarization occurs earlier than that of right ventricular repolarization , keeping the right one somewhat non-synchronized to that of left one. That's why , while left sided ventricular leads exhibit ST depression , right sided precordial leads show somewhat ST elevation.
- **Occlusion MI** (=STEMI for the purpose) in isolation causes epicardial injury , the axis of repolarization is directed towards the injured epicardial surface resulting in ST elevation over the leads dictated as per the involvement of coronary territory.

Sequence of conduction in LBBB

- ☐ Conduction delay / block means impulses travelling first via the right bundle branch (as indicated by **red arrow 2**) to reach the right ventricle.
- ☐ Septum is activated from right-to-left (as indicated by **red arrow 1**)
- ☐ Finally depolarization vector is directed towards the lateral leads (as indicated by **red arrow 3**)

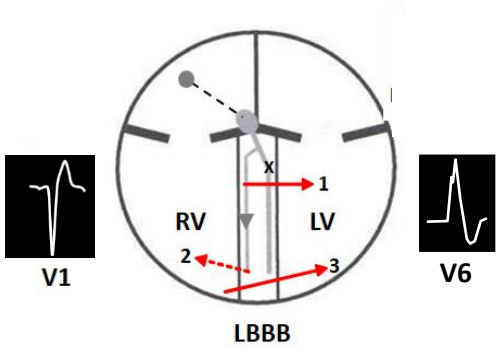


Fig. 1.1

To be noted that impulses from right ventricle (RV) pass somewhat slowly through the interconnecting myocardial mass to the left ventricle (LV) with consequent widening of depolarization wave (QRS) – this is known as **'abnormal depolarization'** in context with LBBB (since this occurs through the abnormal path of depolarization).

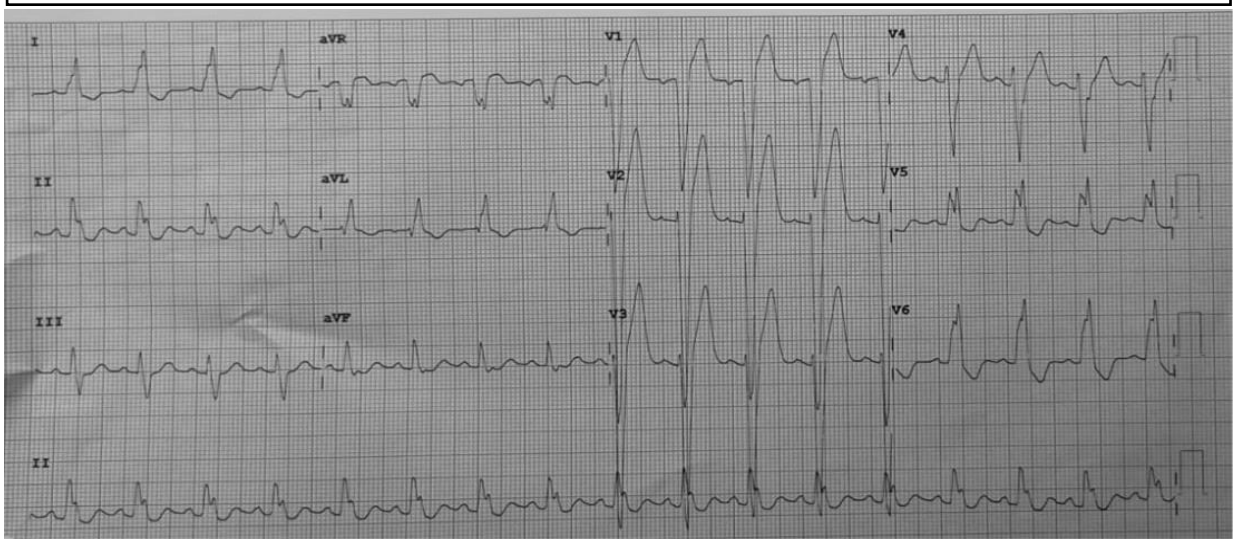
Here it would be worthwhile to mention about **'appropriate discordance'**

- ➢ Appropriate discordance means that abnormal depolarisation is followed by abnormal repolarisation, which appears discordant to the preceding QRS complex , as illustrated above with V1 and V6 leads.
- ➢ Lateral leads with tall, broad R waves will often have associated ST-segment depression and T-wave inversion, and those with deep S waves over the rt sided precordial leads can have a somewhat amount of ST elevation that does not indicate ischemia (generally accepted as normal < 25% of the size of the preceding S wave)

However , this appropriate discordance can mask the signs of associated acute MI either by much ST depression or excessive ST elevation. This scenario may keep the clinicians in diagnostic dilemma.

ECG diagnostic criteria in LBBB (essential to know)

- QRS duration ≥ 120 ms
- Dominant S wave in V1
- Broad monomorphic R wave in lateral leads (I , aVL , V5-V6)
- Absence of Q waves in lateral leads
- Prolonged R wave peak time > 60 ms in leads V5-6



3. The basic electrophysiology in combined scenario of acute MI with LBBB

The purpose of writing this article is to sort out the way how ST segment behaves in acute MI with LBBB.

The electrophysiological rule governing appropriate discordance in LBBB has been well discussed in the previous page. It is also well known fact that the occlusion MI , traditionally called STEMI is also having a particular pattern of ST elevation on ECG , as dictated by the site of coronary involvement. The combined impact of both the situations creates on ECG a concordant ± excessive discordant ST segment changes indicative of myocardial infarction.

The basic electrophysiological concept is outlined below :

Practice has now emerged to access for concordant ST elevation/depression ± excessive discordant ST segment elevation indicative of myocardial infarction in the presence of LBBB

This practical outline had been adopted by some researchers – as to diagnose acute MI in the presence of LBBB , initially by Dr. Elena Sgarbossa in 1996 and further laid down with some modification by Dr. Stephen Smith in 2012.

And the history rolls over – Barcelona et al added their additional observation by adopting a new algorithm possibly having the highest sensitivity and specificity – discordant ST deviation ≥ 1 mm in any lead where the R or S is ≤ 6 mm – proved to be very useful index with additional MI where the QRS is having somewhat low amplitude.

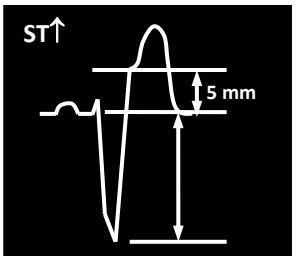
4. Outline of different diagnostic criteria for acute MI in the presence of LBBB

☐ **The original Sgarbossa criteria**

This original criteria included the three clues to diagnose infarction in patient with LBBB :

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

A cumulative score of 3 or more provides a diagnostic criteria for the purpose , having sensitivity (36%) and specificity (90%).



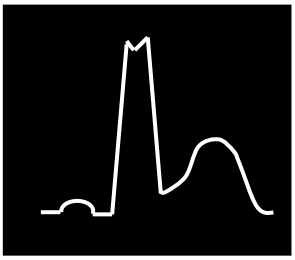
MI with LBBB

ST↓
More pronounced ST depression (delayed conduction with LBBB , more exaggerated in the presence of MI , anywhere on ECG)

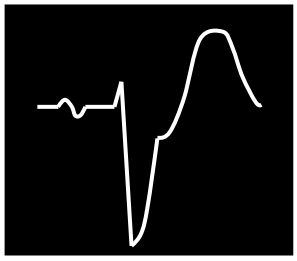
Fig. 1.2

ST-segment depression and elevation are having reverse relationship with each other. More ST depression on one side would be recorded as more ST elevation over the opposite side. Here this has been mentioned to clarify the mechanism of **excessive discordant ST elevation** (discordant pattern of QRS/ST in MI with LBBB)

NB : Concordant ST elevation or depression is not the characteristic of alone LBBB – this occurs usually in the presence of additional MI. The smallest unit of 1 mm is used as a denominator as > 1 mm



Concordant ST elevation > 1mm in any lead with a positive QRS complex



Concordant ST depression > 1 mm in V1-V3 with negative QRS complex

Fig. 1.3

☐ **The Smith-modified Sgarbossa criteria**

- **Concordant ST elevation** ≥ 1 mm in ≥ 1 lead
- **Concordant ST depression** ≥ 1 mm in ≥ 1 lead of V1-V3
- ***Proportionally*** **excessive discordant STE** in ≥ 1 lead anywhere with ≥ 1 mm STE, as defined by ≥ 25% of the depth of the preceding S-wave

Stephen W.Smith modified the third diagnostic element of original Sgarbossa criteria , mentioned above as the propotionally excessive discordant STE. This yields more sensitive (80%) and specific (90%) criteria for OMI diagnosis – all ECG findings are evaluated better with propotional excessive discordant ST elevation , discarding many false positives and false negatives of the original absolute 5 mm criterion.

☐ **Barcelona criteria**

✓ The best performance with the highest sensitivity was obtained by this algorithm , as stated below :

- Concordant ST elevation in any lead ≥ 1mm
- Concordant ST depression in ***any*** *lead*
- Discordant ST deviation ≥ 1mm in any lead where the R or S is ≤ 6mm

➢ As with the prior 2 criteria, criteria 1 is the same. Criteria 2 is expanded to all leads (not just V1-3). Criteria 3 is a bit unique.

➢ For criteria 3 , one is looking at low voltage.

➢ Barcelona criteria is having the sensitivity and specificity 93% and 94% respectively

---

☐ **Chapman's sign**

- The presence of a notch in the ascending limb in leads I , aVL or V6 is called Chapman's sign
- This sign is attributed to abnormal / delayed conduction through the LBBB , which is further aggravated in the presence of damaged cardiac muscle.
- It is one of the criteria that is also used to diagnose acute MI in the presence of LBBB
- This sign may also be seen in some patient of dilated cardiomyopathy or any cause of left ventricular dilatation with dysfunction.
- This has a low sensitivity , but specificity of about 90%.

☐ **Cabrera's sign**

- This sign indicates notching in the ascending limb of the S-wave in leads V3, V4 and V5. The notching must be at least 40 ms in duration which has a low sensitivity for MI around 27%.
- The same reasoning as that of Chapman's sign – abnormal conduction of electrical signal through the damaged myocardium in the presence of LBBB has been attributed to this sign.

5. Stepwise interpretation of ECG having acute MI in the presence of LBBB

Most importantly , one must understand that no ECG rule would likely ever identify AMI in either with LBBB or normal conduction , and therefore it becomes essential to seek the maximum potential of the ECG to identify occlusion MI.

The following steps are adopted while interpreting the ECG with acute MI in the presence of LBBB :

Step 1 : Evaluation of symptoms of acute MI

Step 2 : To identify LBBB , as diagnostic criteria has been laid down on page 3

Step 3 : To consider the implementation of different criteria , as innumerated below (Nutshell) :

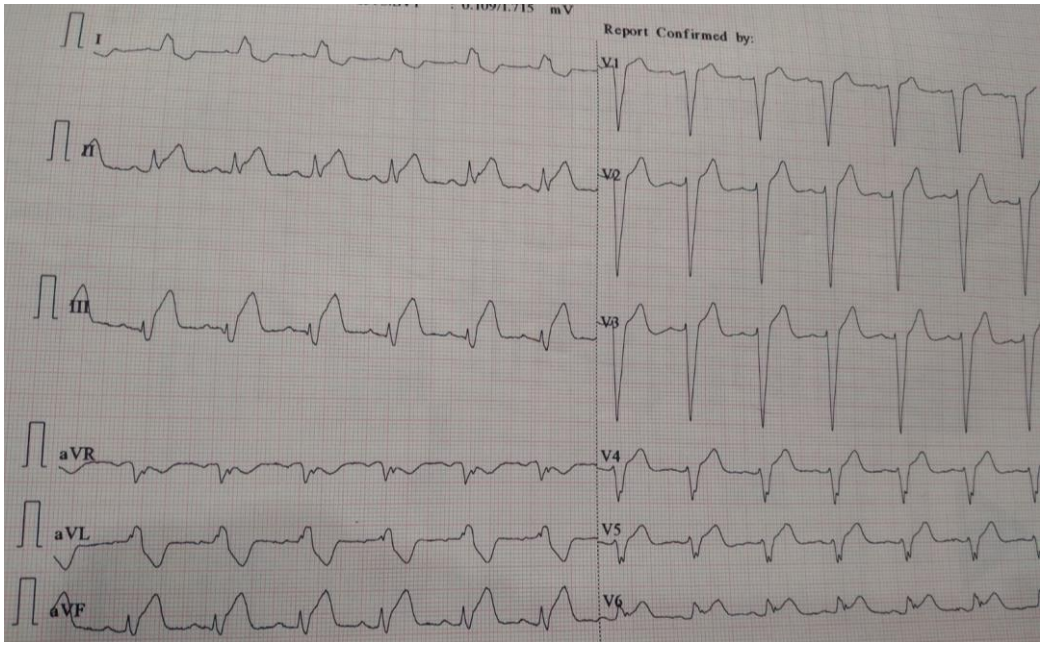
- **Concordant ST elevation** ≥ 1 mm in ≥ 1 lead
- **Concordant ST depression** ≥ 1 mm in ≥ 1 lead of V1-V3
  or
  Concordant ST depression in ***any*** *lead*
- ***Proportionally*** **excessive discordant STE** in ≥ 1 lead anywhere with ≥ 1 mm STE, as defined by ≥ 25% of the depth of the preceding S-wave
- Discordant ST deviation ≥ 1mm in any lead where the R or S is ≤ 6mm
- To see the presence of Chapman's sign and Cabrera's sign in the concerned leads if present.

These laid down points covers the maximum of the criteria , as mentioned above to be utilized for the purpose.

6. Illustration by ECGs

☐ **ECG 1**

61 years male - non-diabetic , non-hypertensive presents with severe chest pain and sweating having BP 150/90 mmHg , pulse 80 bpm



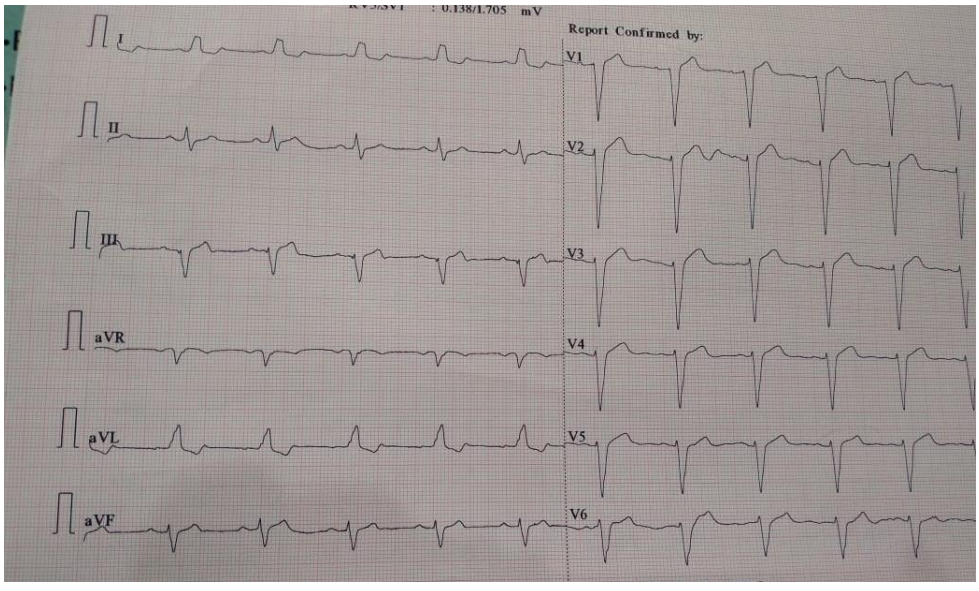
Source : GLOBAL HEART RHYTHM FORUM put by Dr. R.K Gupta , Senior Consultant Physician , Yamunanagar , Haryana on dated 04.10.2024

ECG findings : In the background of complete LBBB

- Concordant ST elevation ≥ 1 mm in lead II and aVF
- Concordant ST depression = 1 mm – border line in lead aVR
- Barcelona sign positive in lead V5 (discordant ST elevation of 1 mm and in V5 with S wave <6 mm)
- Cabrera's sign positive in lead V4 with a notch on the ascending limb of the S-wave with duration of 40 ms

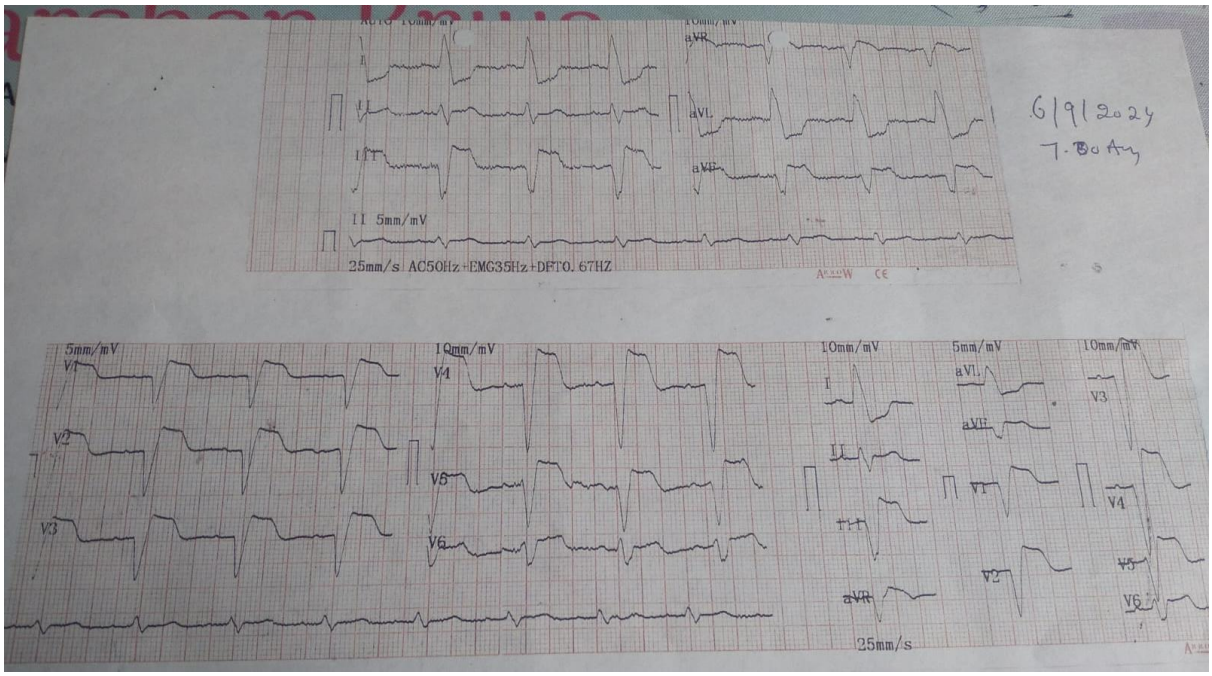
His CAG shows occlusion in RCA and LAD.

On thrombolysis the following is the ECG of the same patient (with pre-existing LBBB)



☐ **ECG 2**

60 years female presenting with chest pain



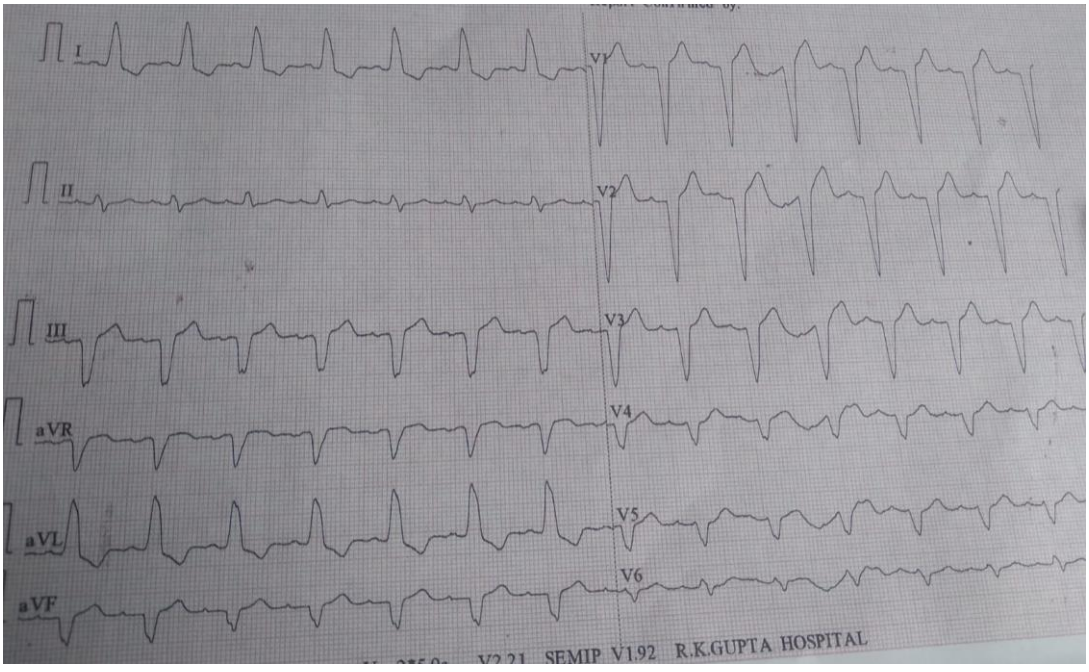
Source : GLOBAL HEART RHYTHM FORUM put by Dr. R.K Gupta , Senior Consultant Physician , Yamunanagar , Haryana on dated 09.10.2024

ECG findings :

Chest leads

- The Smith modified Sgarbossa criteria is positive from V1 to V6 (propotionally discordant STE) in these leads with ≥ 1 mm , as defined > 25% of the depth of the preceding S-wave
- Barcelona criteria positive in lead V6 (also positive over aVR and aVF)

Post-thrombolysis ECG of the same patient



7. Take Home Message

- ☐ Both the Sgarbossa and Smith criteria are approved for use to diagnose AMI in LBBB
- ☐ The Barcelona criteria are new (published in 2020)
- ☐ Unique part of the Barcelona criteria includes applying concordant depression to any lead , and the application of discordant ST elevation ≥ 1 mm in any lead where the R or S is ≤ 6 mm
- ☐ The Barcelona criteria have been derived and internally validated with promising results. However, it awaits external validation.
- ☐ As always, when applying any clinical rules, always consider the patient's pretest probability.
- ☐ No single criteria is suffice enough to diagnose acute MI in the presence of LBBB

Wackers et al. correlated ECG changes in LBBB with localization of the infarct by thallium scintigraphy. The most useful ECG criteria were:

- Serial ECG changes — 67 percent sensitivity
- ST segment elevation — 54 percent sensitivity
- Abnormal Q waves — 31 percent sensitivity
- Cabrera's sign — 27 percent sensitivity, 47 percent for anteroseptal MI
- Initial positivity in V1 with a Q wave in V6 — 20 percent sensitivity but 100 percent specificity for anteroseptal MI

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PRECORDIAL SWIRL SIGN : A NEW POINTER TOWARDS PROXIMAL LAD OCCLUSION MI

PRECORDIAL SWIRL SIGN – A NEW POINTER TOWARDS PROXIMAL LAD OCCLUSION MI

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OUTLINE

Precordial Swirl Sign – A new pointer towards proximal LAD Occlusion MI

A Narrative Review

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A culprit – if does the offence in disguise , it would be be a 'Herculean task' to get him identified.

This is also true with occlusion MI – a new term to denote a near or total occlusion with insufficient coronary circulation , to be reflected on ECG.

- ☐ **Epicardial current of injury so produced over the subepicardial zone in occlusion MI might inscribe ST elevation , somewhat different in distribution from that of classical STEMI.**
- ☐ **This 'Precordial Swirl Sign' is recognized on ECG by ST elevation in leads V1 and aVR, with reciprocal ST depression in leads V5 and V6.**

This is a dire emergency to realise this concept , otherwise this occlusion MI might be missed , it may end the life of the patient.

1. Introduction

There are certain occlusion myocardial infarction patterns which don't show the classical pattern of STEMI and therefore , by keeping only STEMI concept in mind one may be misdirected in arriving to the diagnosis of acute coronary artery occlusion. STEMI equivalents on ECG represent coronary occlusion in the absence of the traditional STE criteria but presenting itself in disguise form. The absence of classical ST elevation is not a reassuring sign that there is no major coronary event by occlusion. It becomes very essential to recognise this pattern within the time schedule to treat this condition by reperfusion therapy like PCI . ACCF (American College of Cardiology Foundation) and AHA (American Heart Association) guidelines recommend first medical contact to PCI device time (door to balloon time) of less than 90 minutes.

Classical STEMI is diagnosed as a group of symptoms consistent with acute coronary syndrome in the presence of new ST-segment elevation at J-point in at least two anatomical continuous leads of $\geq$ 2 mm in a male or $\geq$ 1.5 mm in a female over leads V2-V3 and/or at least 1 mm in other continuous leads or limb leads , in the absence of bundle branch block , LVH , or other non - MI ST-segment elevation situations.

There exist some STEMI equivalent patterns but in disguise on ECG what is now known as occlusive MI – a challenging situation to be diagnosed , otherwise a significant part of the left ventricle may be jeopardized with a poor outcome.

In 2018 , Meyers , Weingart and Smith put a new concept of 'occlusion myocardial infarction' (OMI) in the place of STEMI equivalent – a more reliable paradigm for detecting acute coronary occlusion.

Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.

2. What is precordial Swirl sign ?

In the month of october 15 , 2022 , Drs. Smith and Meyers induced a very useful sign to illustrate very proximal LAD myocardial occlusion (proximal to the first septal perforating branch – **S1** , as illustrated below by sketch 1.1) :

- ☐ This occlusion MI is the resultant of **septal ischemia**
- ☐ This entity is recognized on ECG by ST elevation in leads V1 and aVR. There is associated reciprocal ST depression in leads V5 and V6.

3. Electrophysiological mechanism – a consideration

This 'Precordial Swirl Sign' denotes septal ischemia and therefore, the following facts should be considered to get proper insight :

Blood supply of interventricular septum

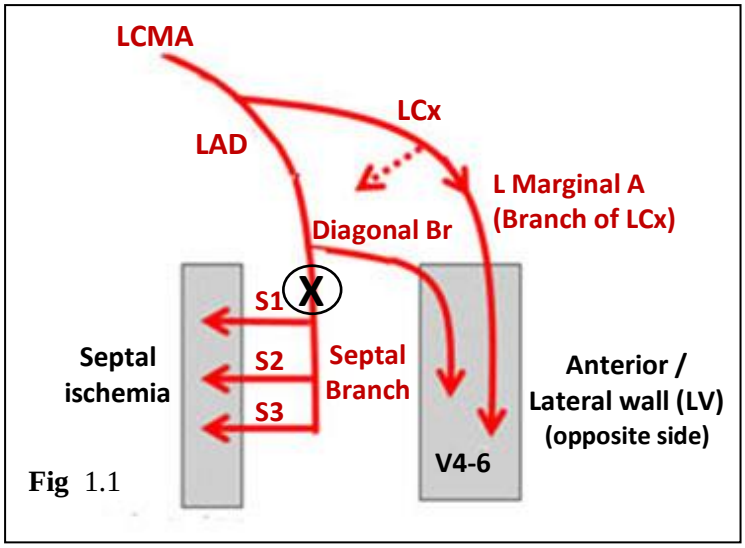


Fig 1.1

The site of lesion in precordial swirl sign is proximal to 1st septal perforating branch (**S1**) of LAD, as illustrated above.

The anterior 2/3rd interventricular septum is supplied by the anterior interventricular artery , which consists of septal branches (S1, S2, S3) arising from left anterior descending artery – LAD . (The remaining posterior 1/3rd of the interventricular septum is supplied by the posterior interventicular artery , a branch of right coronary artery). Thus , the maximum part of interventricular septum is jeopardized by the occlusion of LAD proximal to its 1st perforating branch – **S1** , resulting in **septal ischemia.**

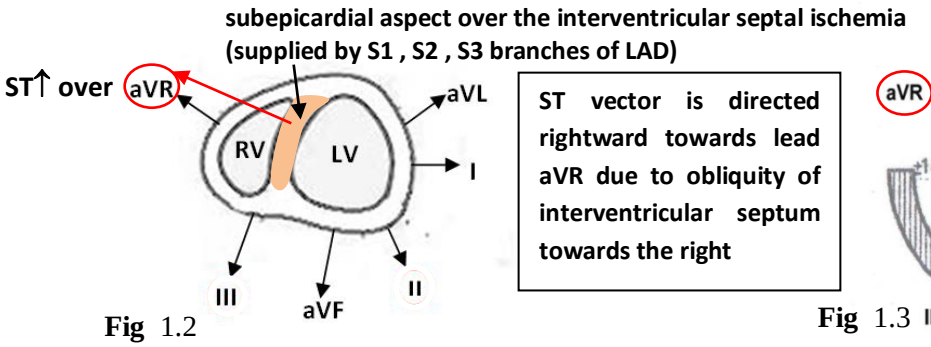
The concept of current of injury

The current of injury met with myocardial ischemia is defined as jeopardized myocardial tissue with the loss of the power of transferring the ions across the membrane during repolarization. This injured zone is considered to be on the minus side of the voltage , compared to the remaining healthy myocardium in vicinity. Therefore , the current of injury is reflected on ECG by ST segment elevation over the concerned leads within the range of 90^0, as governed by the following rule :

Concept of Rule of 90^0

Any exploring lead placed within the range of 90^0 in respect to resultant average ST vector records ST elevation , at 90^0 no deflection and beyond 90^0 ST depression.

A concept of ST elevation with reciprocal ST depression in collaboration with precordial Swirl sign



ST vector is directed rightward towards lead aVR due to obliquity of interventricular septum towards the right

Fig 1.2

VERTICAL PLANE

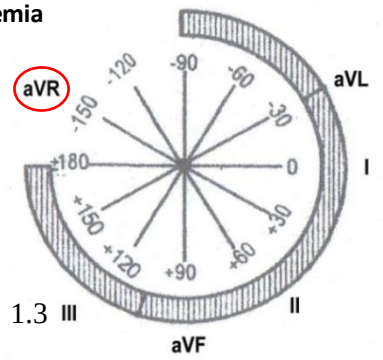


Fig 1.3

HEXAXIAL LEAD SYSTEM

V1 is in alliance with interventricular septal injury (within the range of 90^0) → ST elevation therein

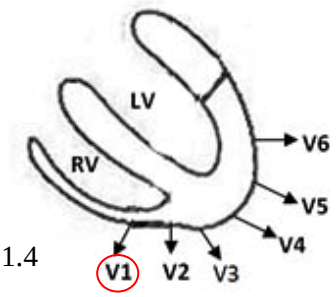
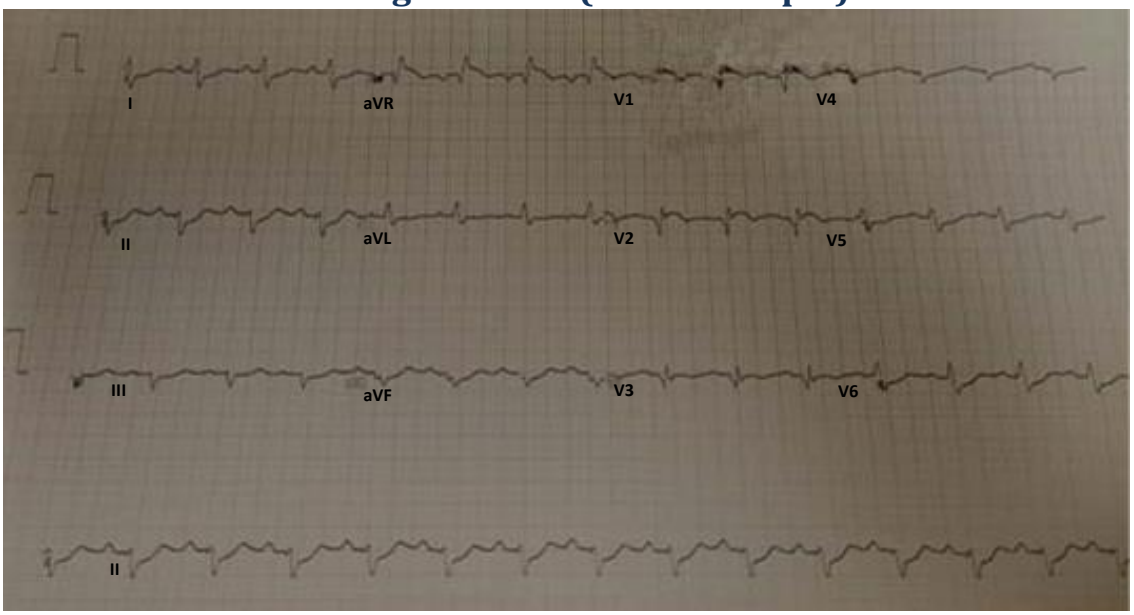


Fig 1.4

HORIZONTAL PLANE

- Since here the subepicardial injury is proximal to interventricular septal branch S1 of LAD, the maximum part of interventricular septum is jeopardized with the resultant vector pointing towards lead aVR with ST elevation therein.
- Since V1 lies in alliance with interventricular septum vector , it also records the ST elevation.
- Since precordial leads V5-V6 are facing the contralateral region over the opposite side, these leads record reciprocal ST depression. (The same is true with the inferior leads II, III & aVF ,as well).

4. The 'Precordial Swirl Sign' on ECG (as an example)



Source : Global heart rhythm Forum , Dated -24.06.2024

Findings on ECG :

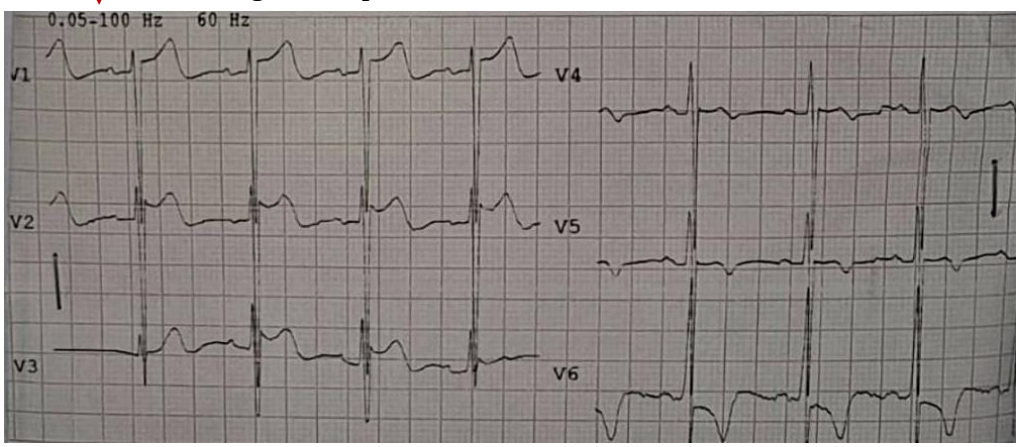
- ☐ ST elevation in aVR (frontal QRS axis at -150^0) with reciprocal ST depression over inferior leads II , III and aVF.
- ☐ ST elevation over V1-2 with reciprocal ST depression in leads V5 and V6.
- ☐ Low voltage (possibly due to associated occlusion MI)

Discussion :

- On this ECG, there is ST elevation in V1, (V2) with reciprocal ST depression in V5 and V6.
- The frontal QRS axis at -150^0 with ST elevation in aVR with reciprocal ST depression in inferior leads II , III and aVF.
  This is attributed to the septal ischmeia due to the occlusion of LAD proximal to its 1st perforating branch (S1) - there is additional involvement of lead V2 with ST elevation due to its possibie proximity to V1.

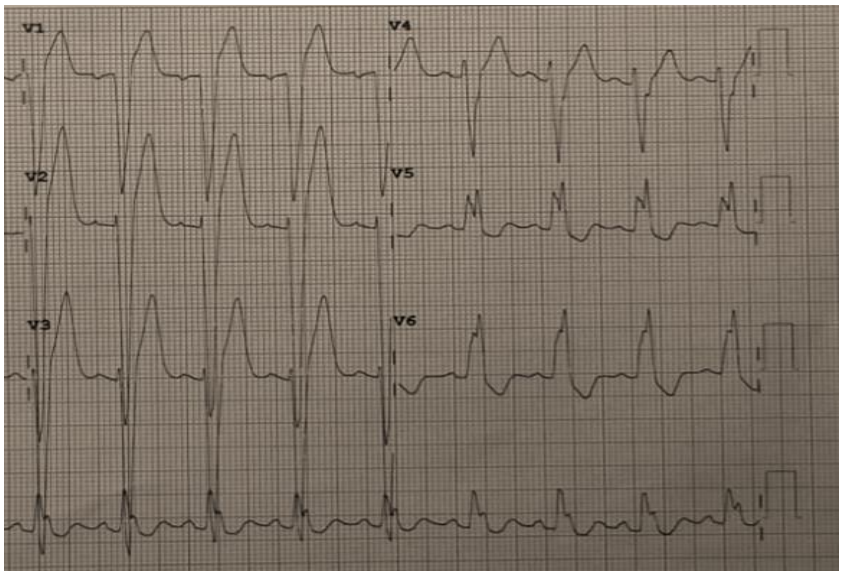
5. A mindful attention to the assoicated LVH , LBBB , etc to exclude the false impression of precordial swirl sign

- ○ There may be the presence of ST depression in leads V5-V6 with contralateral ST elevation in V1 in cases of LVH and LBBB. Therefore , before commiting precordial swirl sign to be positive , it become essential to exclude these conditions.



Hypertensive patient

This is ECG dictum that discordant ST elevation over right precordial leads V1-3 , specially with up concavity in the presence of ECG evidences in favour of LVH should be treated as a part of LVH itself , not as Precordial Swirl Sign.



Left bundle branch block pattern is characterized by the wide notched QRS complexes in lead V5-V6 with secondary repolarization abnormality in the form of ST segment depression and T wave inversion. **The opposite pattern of a wide S wave with upsloping ST and upright tall T is seen in right precordial leads.**
This fact must be kept in mind while interpreting Precordial Swirl Sign.

In both of these two ECGs tracings only from precordial leads have been illustrated.

6. Take Home Message

- ☐ Epicardial current of injury so produced over the subepicardial zone in occlusion MI might inscribe ST elevation , somewhat different in distribution from that of classical STEMI.
- ☐ In the month of october 15 , 2022 , Drs. Smith and Meyers induced a very useful sign to illustrate very proximal LAD myocardial occlusion (proximal to the first septal perforating branch – **S1**)
  This occlusion MI is the resultant of **septal ischemia**
- ☐ **'Precordial Swirl Sign' :**
  - This entity is recognized on ECG by ST elevation in leads V1 and aVR. There is associated reciprocal ST depression in leads V5 and V6.
- ☐ **Reasoning** : ST vector is directed rightward towards lead aVR due to obliquity of interventricular septum towards the right – this fact is responsible for ST elevation in lead aVR. Since V1 lies in alliance with interventricular septum vector , it also records the ST elevation therein.
  Since precordial leads V5-V6 are facing the contralateral region over the opposite side, these leads record reciprocal ST depression.
- ☐ Precordial Swirl Sign is never diagnosed in the presence of LVH and LBBB. There may be the presence of ST depression in leads V5-V6 with contralateral ST elevation in V1 in cases of LVH and LBBB. Therefore , before commiting precordial swirl sign to be positive , it become essential to exclude these conditions

7. Acknowledgement

My appreciation with thanks to Dr. Awais Shafi (from Pakistan) for the ECG tracing of 'Precordial Swirl Sign' , posted on page 30.

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LET US WAKE UP TO THIS NEW POSSIBILITY : OCCLUSION MI (OMI) IN DISGUISE (STEMI EQUIVALENT)

LET US WAKE UP TO THIS NEW POSSIBILITY - OCCLUSION MI (OMI) IN DISGUISE (STEMI EQUIVALENT)

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OUTLINE

Introduction

Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.

A basic concept while interpreting 'occlusion myocardial infarction'

Electrocardiographic changes – a spy-key to open the door of 'occlusion MI'

A working classification of 'occlusion MI' based on ECG changes

- ☐ **Alteration in T-wave**
  - ○ Hyperacute T-wave (HATW)
  - ○ Peaked tall T-wave (de Winter pattern)
  - ○ T-wave morphology in Wellens' syndrome
- ☐ **Alteration in ST segment**
  - ○ ST elevation in aVR
  - ○ ST depression in aVL
  - ○ Posterior MI (reciprocal changes in anterior leads V1-V3)
- ☐ **As Bundle Branch Block pattern**
  - ○ New bifascicular block
  - ○ New LBBB with OMI

Take Home Message

References

Let us wake up to this new possibility – Occlusion MI (OMI) in disguise (STEMI equivalent)

A Narrative Review

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A culprit – if does the offence in disguise , it would be a 'Herculean task' to get him identified . One has to adopt the policy of identifying such a situation as the police does in searching out the thief by his footprint impressions on the soil – even the smell of his clothes is utilized for the purpose. There are so other means , as well.

- ☐ **This is so true also with STEMI equivalent ECGs – being manifested without ST elevation pattern and might be so ignored as Non-STEMI.**
- ☐ **These are actually 'occlusion MI' in disguise. Missing of such alarming pathological entities means depriving the patient from the life saving procedure like PCI.**

Clinicians have been trying their best to search out ECGs imprints of such occlusion MI. **Knowledge works up for the knowledge** – paving the diagnostic approach to such a situation.

1. Introduction

Electrocardiogram (ECG) is considered as one of the most useful diagnostic tools for the identification of ST-segment elevation myocardial infarction. STEMI is diagnosed as a group of symptoms consistent with acute coronary syndrome in the presence of new ST-segment elevation at J-point in at least two anatomical continuous leads of $\geq$ 2 mm in a male or $\geq$ 1.5 mm in a female over leads V2-V3 and/or at least 1 mm in other continuous leads or limb leads , in the absence of bundle branch block , LVH , or other non acute MI ST-segment elevation situations. There exists some STEMI equivalent patterns but in disguise on ECG – a challenging situation to be diagnosed , otherwise a significant part of the left ventricle may be jeopardized with a poor outcome.

There are certain occlusion myocardial infarction patterns which don't show this classical pattern of STEMI , as mentioned above and therefore , by keeping only STEMI concept in mind one may be misguided in arriving to the diagnosis of acute coronary artery occlusion. STEMI equivalents on ECG represent coronary occlusion in the absence of the traditional STE criteria but presenting itself in disguise forms. The absence of ST elevation is not a reassuring sign that there is no major coronary event by occlusion. It becomes very essential to recognise this pattern within the time schedule to treat this condition by reperfusion therapy like PCI . ACCF (American College of Cardiology Foundation) / AHA (American Heart Association) guidelines recommend first medical contact to PCI device time (door to balloon time) of less than 90 minutes.

In 2018 , Meyers , Weingart and Smith put a new concept of 'occlusion myocardial infarction' (OMI) in the place of STEMI equivalent – a more reliable paradigm for detecting acute coronary occlusion. The following pitfalls have been observed by using the term STEMI equivalent :

- The chance of missing the diagnosis of acute coronary occlusion remains upto 30% .
- Accordingly , there is a lapse of time in salvaging the involved injured tissue by reperfusion therapy and so making this situation further grave by increased morbidity and mortality.
- Sometimes benign ST elevation on ECG is wrongly diagnosed as STEMI with unwarranted catheterization , exposing the concerned patients to the associated risks of coronary dissection ± perforation , arterial punctures with bleeding and contrast associated nephropathy.

Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.

2. A basic concept while interpreting 'occlusion myocardial infarction'

The following points are to be considered in this context :

- **The current of injury** met with myocardial ischemia/infarction is defined as jeopardized myocardial tissue with the loss of the power of transferring the ions across the membrane during repolarization. This injured zone is considered to be on the minus side of the voltage , compared to the healthy myocardium in vicinity. The current of injury is reflected on ECG either by ST segment elevation or depression depending upon the site of injury.

- **Voltage** is the measure of energy available to transport electrons. This requires a gradient (more energy on one side than the other) – the flow continues until the circuit reaches a common potential (equal charge) with no longer flow of current.

- **The direction of the flow of current** would depend upon the site of tissue with current of injury, if situated towards the exploring electrodes , it causes a positive deflection and negative deflection if otherwise.
  It is the site of current of injury (subepicardial , subendocardial or transmural) which dictates the direction of the flow of current.

NB : Due to the automatic shifting of the TP segment to the baseline by the ECG machine during the diastolic phase , one is not in a position to observe any effect on the TP segment – though there is potential difference in between injured negative zone and healthy zone of TP segment.

The current of injury associated with subendocardial myocardium has been illustrated as below :

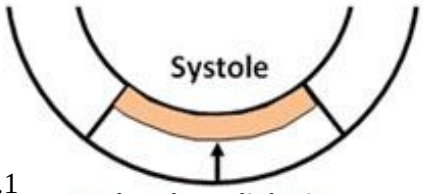


Fig 1.1

- The current of injury is having a potential gradient at the 'electrical border' in between the injured and normal myocardium.
- The current of injury will travel from normal myocardium having higher voltage (+) towards the lower voltage (-) of injured myocardium.
- Thus , the zone of subendocardial injury is accompanied by ST segment depression.

Lead aVR imparts a useful information in context with occlusion MI (its vector is directed away from left ventricular depolarization).
Lead aVL is the only true reciprocal lead to the inferior wall – the only lead facing the superior part of the left ventricle.

❖ **Alteration in T' wave might be observed in some specific conditions consistent with occlusion MI**

Normally , there is a outflow of K+ ions from intracellular compartment to extracellular compartment during the phase 3 , interrupting the isoelectric ST segment due to the newly created potential difference in between intracellular and extracellular compartments – the resultant situation causes the genesis of upright T wave.

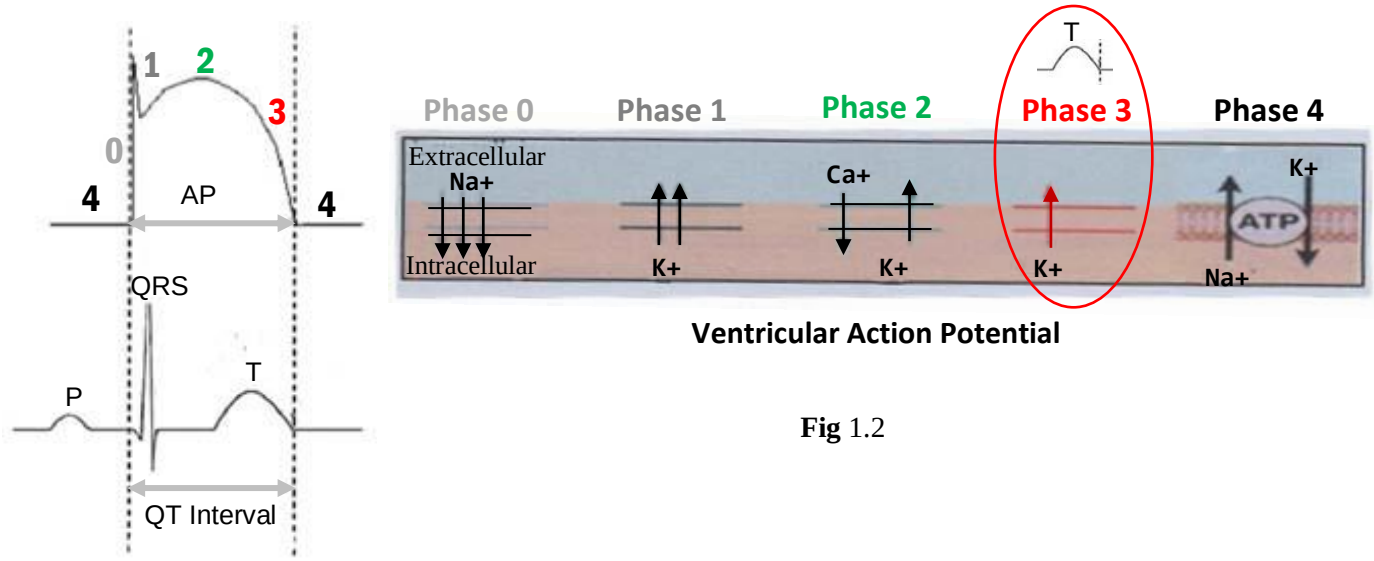


Fig 1.2

❖ **Bundle branch block pattern**

Might be encountered in context with occlusion MI

- New bifascicular block (RBBB with LAFB)
- New LBBB with OMI

3. Electrocardiographic changes – a spy-key to open the door of 'occlusion MI'

☐ Alteration in T-wave

- **Hyperacute T-wave (HATW)** : This is postulated that there is hyperpermeability related K+ ions transfer from intracellular compartment to extracellular compartment over the infarct area resulting in hyperacute T wave. Hyperacute T-wave with ≥ in two contiguous leads may be the first sign of a developing infarct, often preceding any STE. Hyperacute T-waves appear broad-based , often generally more symmetrical than the normal T-wave.

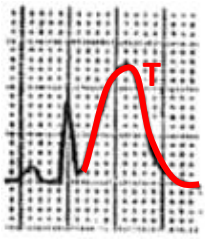


Fig 1.3

Dr Stephen W. Smith suggests that if any single T wave /QRS complex ratio in V1-4 is greater than 0.36, it represents acute MI, not subacute or old.

✓(A new upright T-wave in V1>V6 is considered as loss of precordial T-wave balance especially in favour of coronary insufficiency).

- **Peaked tall T-wave in 'de Winter pattern' (dWp)**

A regional subendocardial ischemia with preservation of a small ring of subepicardial tissue with alteration in T-wave morphology.

Peaked tall T-wave is the expression of the shorter time duration between subendocardial and subpericardial repolarization. There is a shorter distance in between the subendcoardial zone of ischemia and preserved peripheral rim of subpericardial healthy tissue with a positive voltage gradient between endo-epicardium.

- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression > 1mm at the J point in the precordial leads
- Absence of ST elevation in the precordial leads
- Reciprocal ST segment elevation (0.5mm – 1mm) in aVR

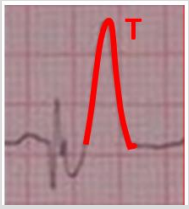


Fig 1.4

- **Transmural ischemia-reperfusion injury with change in T-morphology in Wellens' syndrome**

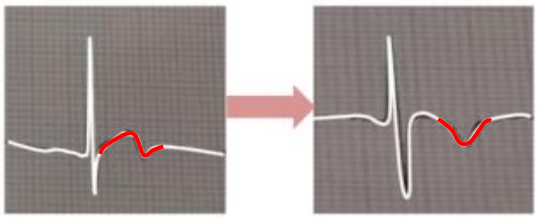
  - A temporary but significant obstruction of the LAD coronary artery , commonly caused by the rupture of an atherosclerosis plaque with the subsequent thrombolysis before complete myocardial infarct sets in.
  - The next phase is transmural ischemia-reperfusion injury leading to myocardial oedema.

Biphasic T-wave (with initial postivity and terminal negativity) is followed by deeply inverted T-wave , mainly in chest leads V2 and V3.

Reasoning :

- Myocardial ischemia – reperfusion injury results in local myocardial oedema which can change the direction of ongoing repolarization current. Due to the delay in passing the current through this oedematous myocardium – the current flows uniformly through the opposite direction i.e. from subendocardium to subepicardium. This produces symmetrical deep T-wave inversion.

- In the initial stage of partial oedematous myocardium the first half of the wave with initial positivity and the terminal part with negativity.



Biphasic T-wave (the initial positive and terminal negativity)

Deeply inverted and symmetrical T-wave

Fig 1.5 **Type A Wellens' Syndrome** **Type B Wellens' Syndrome**

☐ **Alteration in ST segment**

- **ST elevation in aVR**
  Lead aVR was previously supposed to have little diagnostic significance – since its vector is directed away from left ventricular depolarization. ST elevation > 1mm in lead aVR has been shown to be 80% sensitive and 93% specific for left main coronary or triple vessel disease in patients suffering from symptoms attributed to CAD.

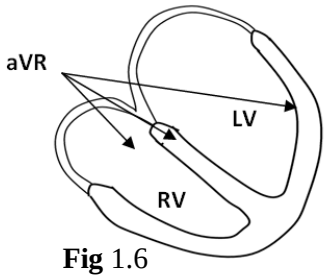


Fig 1.6

This aVR lead imparts a useful information viewing the right upper side of the heart such as outflow tract of the right ventricle and the basal part of the septum. This lead also gives reciprocal information from the lateral side of the left ventricular wall.

The Stipulated mechanism of STE in aVR has been proposed as the reciprocal changes to lateral left ventricular wall , or more specifically at the basal portion of interventricular septum , or that of the outer flow tract of the right ventricle (RVOT).
It is always rewarding to look at aVR in every case of occlusion myocardial infarction to be on safer side.

- **To look at aVL lead**

  aVL is the only true reciprocal lead to the inferior wall , since it is the only lead facing the superior part of the left ventricle.

  Coexisting presence of ST depression in aVL has been found to be highly sensitive for inferior STEMI. This has been described as reciprocal changes in aVL occurring in most patients with inferior wall MI , and **it may be the only early pointer of an impending MI.**

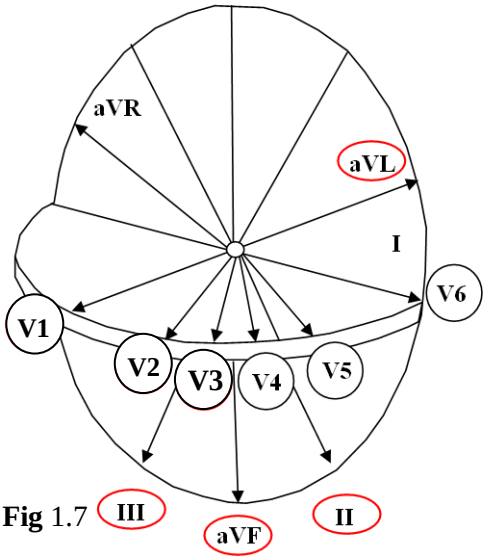


Fig 1.7

Vertical and horizontal planes are almost placed at right angle to each other.

Lead aVL and inferior leads (II , III and aVF) are placed at more than 90^0 to each other.

- **Reciprocal changes with ST depression , Tall R wave with prominent positive T wave in context with posterior MI**

☐ **As Bundle Branch Block pattern**

- **New RBBB and left LAFB**

  It had been demonstrated that the proximal LAD septal perforators (S1,S2,S3) perfuse the right bundle branch and the anterior fascicle of the left bundle branch is also supplied by proximal LAD. This new bifascicular block may be the only pointer for OMI.

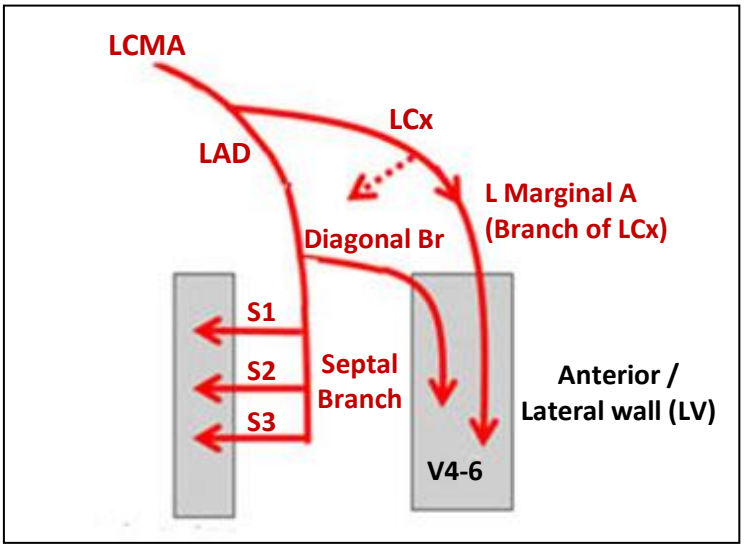


Fig 1.8

- **New LBBB with occlusion myocardial infarction**

Careful workup for the symptomatic patients of suspected ACS with new LBBB is the need of the hour.

The concept of LBBB

With the morphology of LBBB , the entire process of ventricular activation is oriented towards the left chest leads – the upper part of the left interventricular septum gets depolarized in the reverse manner from right to left , and the consequent stimulation of the left ventricle keeps this as electrically predominant ventricle.

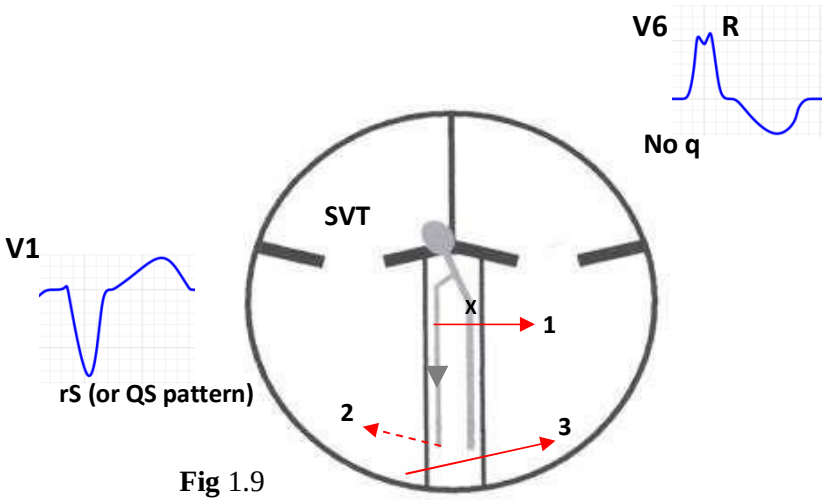


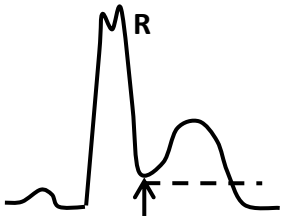
Fig 1.9

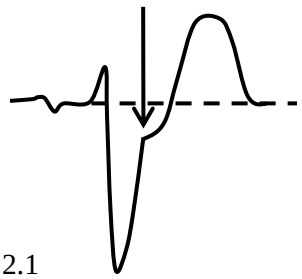
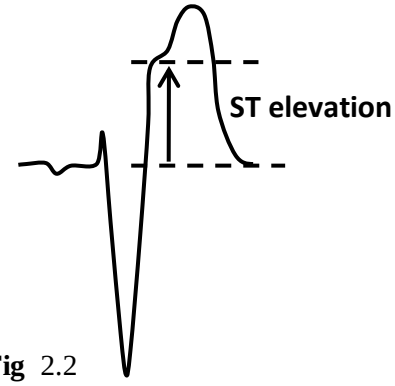
Sequence: **(1)** septal depolarization from right to the left ventricle **(2)** the next as shown to the right ventricle **(3)** Consequently from the right ventricle to the left ventricle.

(The left ventricle is still electrically predominant with LBBB and therefore , produces greater voltages than the right ventricle)

Smith-Modified Sgarbossa Criteria : Occlusion MI in the presence of LBBB

ST elevation MI if superimposed upon the pattern of left bundle branch block (LBBB) brings about some fundamental changes in the morphology of repolarization pattern. Thus, new pattern of repolarization sets in with the elevation of ST segment with somewhat altered amplitude and even with ST depression, as depicted in the following table (concordant ST elevation /depression or excessive discordant ST elevation).

Changes during repolarization	**Illustration by concerned sketches**
1. Concordant ST elevation ≥1mm in ≥ 1 lead (with a positive QRS complex)	 **Fig** 2.0 **Concordant ST elevation**

2. Concordant ST depression ≥1 mm in ≥ 1 lead of V1-V3	**Concordant ST depression**  **Fig** 2.1
3. Proportionally excessive discordant STE in ≥ 1 lead anywhere with ≥1 mm STE , as defined by ≥ 25% of the depth of the preceding S-wave.	 **Fig** 2.2

NB : The Smith-Modified Sgarbossa criteria improves diagnostic accuracy for occlusion MI in the presence of LBBB. The definition of excessive discordant is put in a specific manner , as discussed above in row 3.

✓This modified criteria is more specific to diagnose MI with LBBB , compared to original sgarbossa criteria (any one above mentioned criteria is suffice to diagnose MI in the presence of LBBB).

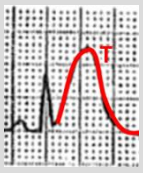
---

4. A working classification of 'occlusion MI' based on ECG changes

☐ **Alteration in T-wave**

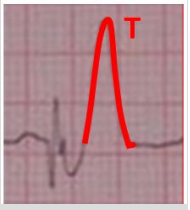
- Hyperacute T-wave (HATW)

There is no universal accepted definition of HATW. Dr Stephen W. Smith suggests that if any single T wave /QRS complex ratio in V1-4 is greater than 0.36, it represents acute MI, not subacute or old.



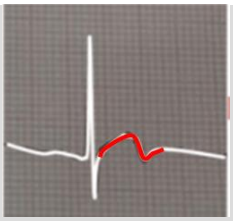
- Peaked tall T-wave (de Winter pattern)

- Tall prominent symmetrical T waves in the precordial leads
- Upsloping ST segment depression > 1mm at the J point in the precordial leads
- Absence of ST elevation in the precordial leads
- Reciprocal ST segment elevation (0.5mm – 1mm) in aVR

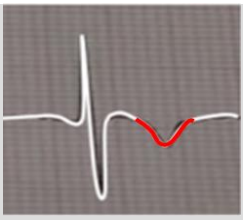


- T-wave morphology in Wellens' syndrome

Type A Wellens' syndrome
Biphasic T-wave (the initial positivity and terminal negativity)



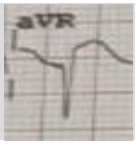
Type B Wellens' syndrome
Deeply inverted and symmetrical T-wave



☐ **Alteration in ST segment**

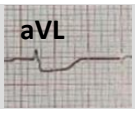
- ST elevation in aVR

More recent publications perceive this ECG pattern as consistent with left coronary main artery subtotal occlusion or complete occlusion with well-developed collateral circulation.



- ST depression in aVL

Any amount of ST depression in aVL even with subtle ST elevation with two contiguous inferior leads is highly sensitive for inferior OMI.

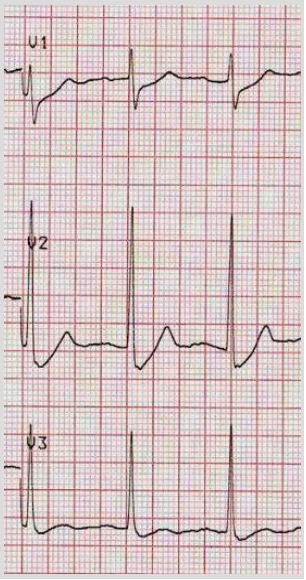


- Posterior MI (reciprocal changes in anterior leads V1-V3)

Reciprocal ECG changes (mirror image changes of MI of the posterior wall) : ST depression , tall R-waves and prominent positive T-waves (V1-3) should be considered a posterior OMI until proven otherwise.

Posterior MI is a result of a reduction in blood flow to a dorsal, infra-atrial portion of the left ventricle , which is supplied by the posterior descending artery (PDA), a branch of RCA in 70% of the population, the LCx in 20%, or of both in the remaining 10%. Isolated posterior MI is most commonly observed due to acute occlusion of the RCA**.**

(R=S in V1 also means as dominant R in V1)



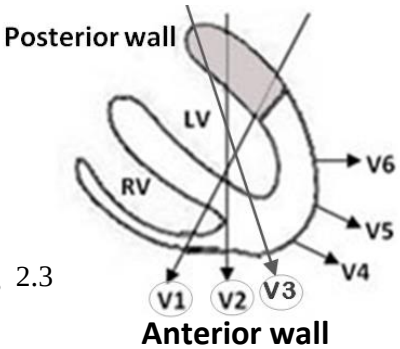


Fig 2.3

The precordial leads V1-3 oriented to the anterior wall , reflecting the inverse - mirror image changes of MI of the posterior wall

☐ **As Bundle Branch Block pattern**

- New bifascicular block

New RBBB with LAFB is highly suggestive of proximal LAD occlusion. One should raise the suspicion for OMI, and carefully look for this bifasicular block pattern.

- OMI in the presence of LBBB

ST elevation MI if superimposed upon the pattern of left bundle branch block (LBBB) brings about some fundamental changes in the morphology of repolarization pattern. Thus, new pattern of repolarization sets in with the elevation of ST segment with somewhat altered amplitude and even with ST depression (for details please see Page No. 40-41)

5. Take Home Message

- Occlusion myocardial infarction (OMI) is a new emerging concept of ACS representing a near or total occlusion with insufficient coronary circulation resulting in acute myocardial infarction - to be diagnosed in the absence of traditional ST elevation on ECG remains a 'Herculean task'.
- OMI should be suspected strongly in the presence of the clinical picture suggestive of ACS if associated with alteration in T-wave , ST-segment or bundle branch block pattern as discussed in the preceding pages. There may be difficulties in diagnosing OMI in patients with LBBB. The diagnosis can be facilitated by utilizing Smith-Modified Sgarbosa criteria.
- These occlusion myocardial infarction patterns can occur in patients with active, intermittent, or no chest pain at all. Observation of these patients may reveal the pattern consistent with OMI on 12-lead ECG.
- Symptoms like unexplained breathlessness , weakness, nausea/vomiting or indigestion may warrants the clinicians to peep through more in depth for the changes consistent with occlusion MI on ECG.

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TERMINAL QRS DISTORTION SIGN (TQRSD) : A RED ALERT ON ECG

TERMINAL QRS DISTORTION SIGN (TQRSD) : A RED ALERT ON ECG

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OUTLINE

Introduction

The terminal QRS distortion is accessed on ECG by the loss of both S wave and J wave with uplifting of the remaining R wave above its baseline by > 50% , at least with the precordial leads V2 / V3.

Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion

Electrophysiology of 'Terminal QRS distortion'

- When the myocardium is at the critical risk with ischemia , cells in the affected area (supplied by proximal LAD in this context) may loose somewhat oxygen with ATP , which disrupts the ability to maintain its action potential integrity.
- The electrical signal cannot travel smoothly through the distrupted area :
  Loss of both S wave and J wave → uplifting of the remaining R wave above its baseline.

How to proceed to detect this sign on ECG

A special attention to leads V2 and V3 to see the evidence of terminal QRS distortion sign

Illustration by ECGs

Distinction between STEMI and Terminal QRS Distortion Sign

Both are not the same

Take Home Message

References

Terminal QRS Distortion Sign (TQRSD) : A red alert on ECG

A Narrative Review

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This terminal QRS distortion sign , at times noticed during the evolution of acute anterior STEMI at least over leads V2/V3 in the presence of ongoing anginal chest pain , places a storm warning –this altered waveform bears silent witness to hidden, imminent danger—a testament to the heart's vulnerability towards a rapidly evolving STEMI.

- ☐ **Terminal QRS distortion is defined as loss of both S wave and J wave with simultaneous uplifting of the remaining R-wave above its baseline.**
- ☐ **Its presence in the precordial leads has been strongly correlated with a rapidly progressive STEMI in the territory of proximal LAD occlusion , marking an extensive area of myocardium at risk with an urgent need for revascularization.**

This sign is very much specific (100%) for proximal LAD occlusion and whispers to the ears of attending physician to have urgent reperfusion therapy with improved outcome.

1. Introduction (Keypoints)

- Sometimes during the evolution of acute Anterior STEMI (ST Elevation Myocardial Infarction) on 12-lead ECG, one can witness the terminal S-wave beginning to rise above the baseline , seen at least with leads V2/V3. **When the terminal S-wave rises above the baseline then it is technically considered no longer S-wave**. This phenomenon of terminal QRS distortion is almost 100% specific for rapidly evolving STEMI in the territory of proximal anterior descending artery.

In association with the loss of S-wave as discussed above , the remaining R-wave may take on as a qR configuration , as an imprint of ongoing myocardial insult with its uplifting by with >50% above its baseline :

A concept of evolving 'terminal QRS distortion' , as illustrated below :

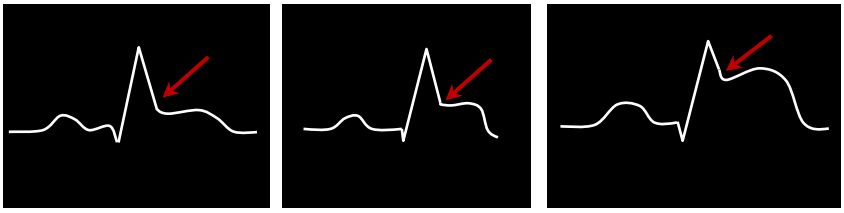

Fig. 1.1

It suggests that the ischemia process is advanced and on the verse of becoming a full transmural Q wave infarction

- ‘Terminal QRS distortion increased all-cause mortality by 81%. Our study suggests that terminal QRS distortion is an important tool to assess the risk in patients with STEMI’ .

Ref :
Terminal QRS Distortion in ST Elevation Myocardial Infarction as a Prediction of Mortality: Systematic Review and Meta-Analysis , 2019
Narut Prasitlumkum et al.
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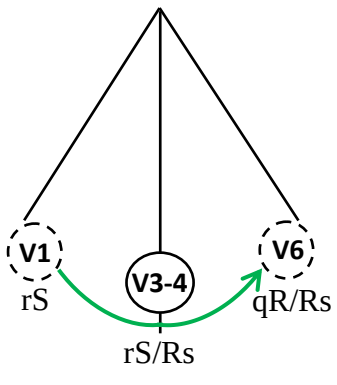
- Rapid and reliable diagnosis of terminal QRS distortion sign is critical for the urgent initiation of life-saving reperfusion therapy. Patients with symptoms suggestive of ongoing myocardial ischemia in the presence of TQRSD sign on ECG usually need urgent reperfusion therapy, as timely honoured reperfusion therapy reduces morbidity and mortality of patient with STEMI. A higher mortality rate has been observed with increasing time to primary percutaneous intervention.

- Since terminal QRS distortion is associated with up concavity , it might be confused with benign early repolarization syndrome (ERS) but this should be always remembered that this sign is never observed in benign ERS. Its rapid diagnosis with simultaneous exclusion of ERS is beneficial to the patient , as it may lead to earlier reperfusion therapy with improved outcome.
  Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion

2. Electrophysiology of ‘terminal QRS distortion’

- This would be better to witness how normally QRS complex progresses over the precordial leads from V1-V6 so that the mechanism of QRS distortion over V2-V3 is properly analysed.



Normal R wave progression

Normally the height of R wave becomes progressively taller from lead V1 through V6

- V1-V2 : R wave of low amplitude and S wave of more depth (R/S <1)
- V3-V4 (Transition zone) rS/Rs
- Onwards R wave amplitude is gradually more compared to S wave → qRs/Rs in leads V5-V6.

Fig. 1.2

- As illustrated in Fig. 1.2 in the preceding page there is one pertinent point that needs attention – the dominance of the voltages across the precordial leads fluctuates from right to the left as increasing amplitude of R-wave with decreasing amplitude of S-wave , considering the site on the precordium where R and S waves approximate each other as the transition zone. It is truly to state that the transition zone of normal R-wave progressive pattern lies in between V3 and V4. This R-wave progression gets interrupted at V2-V3 – how ? see below :

- Leads V2-V3 play a crucial role in the ECG assessment due to its location and sensitivity to ischemic changes in context with proximal LAD occlusion. This is to be mentioned here that expected changes over V2-V3 appear irrespective of the site of proximal LAD occlusion :
  - Proximal to the diagonal branch (D1)
  - In between the first diagonal (D1) and first septal (S1) branches

- **The following pertinent steps are enumerated and discussed in brief to visualize a concept of 'terminal QRS distortion' sign :**
  - When the myocardium is at the critical level of ischemia, cells in the affected area (supplied by proximal LAD in this context) may loose somewhat oxygen with ATP , which disrupts the ability to maintain its action potential integrity.
  - The electrical signal cannot travel smoothly through the distrupted area :
    Loss of terminal S wave and J wave both → the lifting of the reamining R wave above its baseline

The events as illustrated above contribute to the genesis of terminal QRS distortion on ECG. This sign is very much specific (100%) for proximl LAD occlusion , forecasting its rapid evolution to full blown transmural Q wave infarction

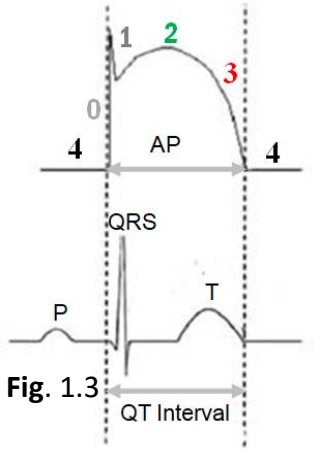
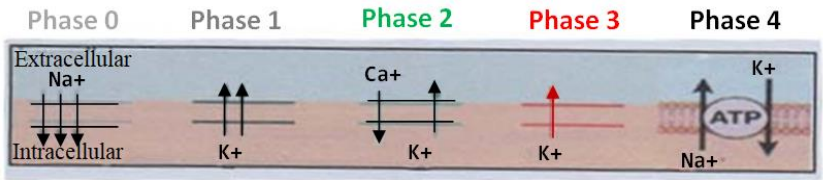


Fig. 1.3



Phase 1 (Nomally phase 1 dictates the genesis of J-point).

Since there is a disruption in the integrity of cardiac membrane , there is a loss of both S wave and J wave with uplifting of the remaining R wave above its baseline.

Now it becomes easier to grasp the definition of terminal QRS distortion – loss of both S wave and J wave with uplifting of the remaining R wave above its baseline by > 50% , at least present over the precordial leads V2/V3.

3. How to proceed to detect this sign on ECG

QRS distortion is an electrocardiographic (ECG) sign of severe ongoing critical myocardial ischemia. One should proceed to evaluate the association between the degree of QRS distortion and myocardium at risk.

- At this juncture , it is advisable to review all 12-leads on ECG with an aim to detect evidence of ongoing anginal chest pain ± the evidence of STEMI elsewhere on ECG
- A special attention to leads V2 and V3 to see the evidence of QRS distortion sign as already discussed
- And the clinician should always adhere to a strict regiment for each patient under such prevailing circumstances :
    - Clinical correlation
    - Comparision with old 12-lead ECG tracings and old medical records.
    - Serial 12-lead ECG tracings, cardiac markers as needed.

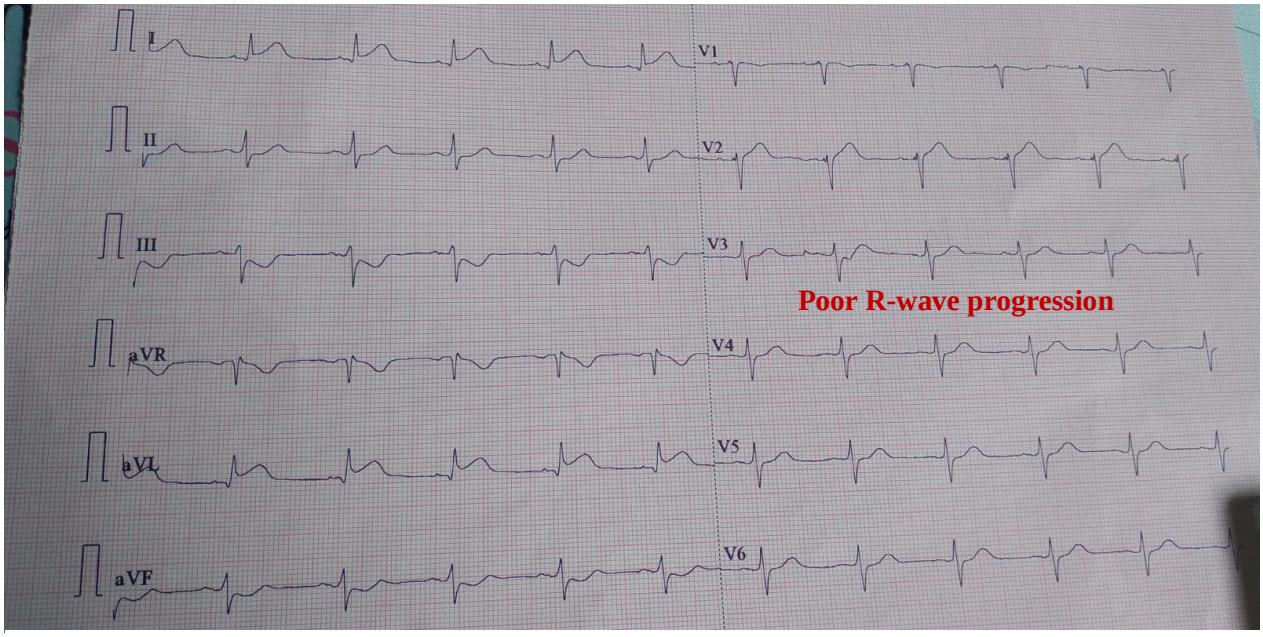
4. Illustration by ECGs

There are **three ECGs recorded in succession one after the other to show the serial changes :**

History : The case was presented by Dr. R.K. Gupta , Senior Consultant Physician , Yamunanagar , Haryana (On Global Heart Rhythm Forum dated 01.11.2024)

56 years old female presented with chest pain (Non-diabetic, Non-hypertensive with BP 130/80 mmHg , Heart rate 80 bpm , hemodynamically stable)

ECG 1

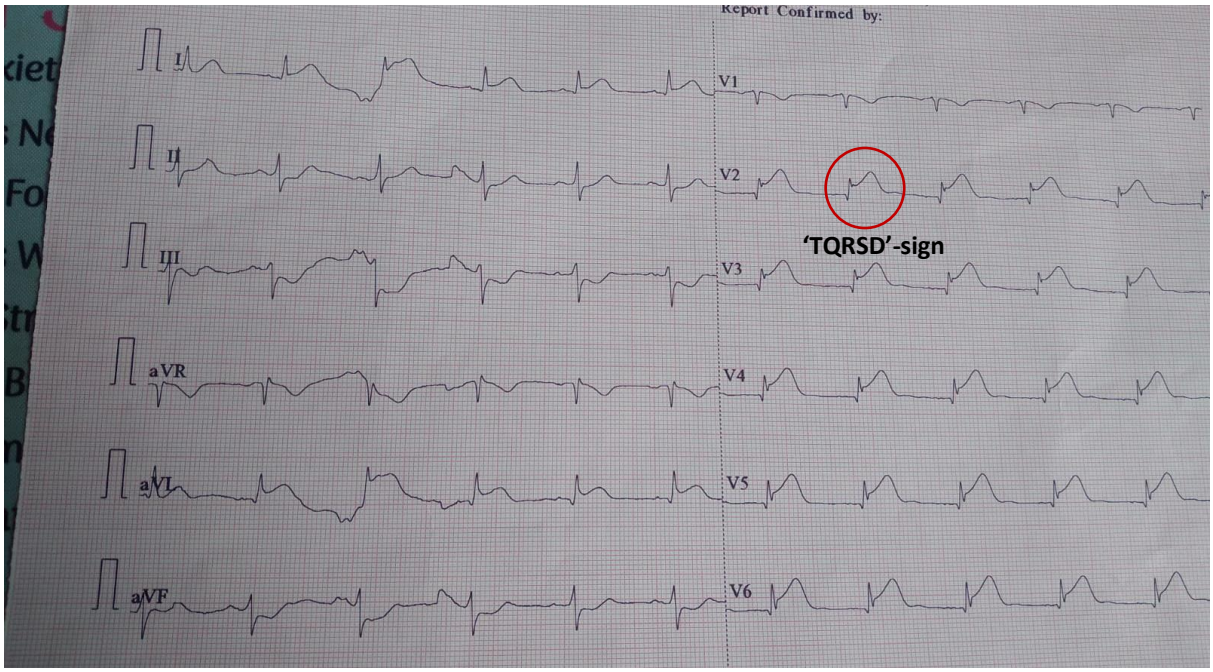


Source : Dr. R.K. Gupta , Senior Consultant Physician , Yamunanagar , Haryana

Findings : ST elevation in leads I and aVL with reciprocal ST depression in inferior leads (maximum over lead III) , associated ST elevation also over V2= suggestive of High lateral STEMI

See also poor R-wave progression in lead V3.

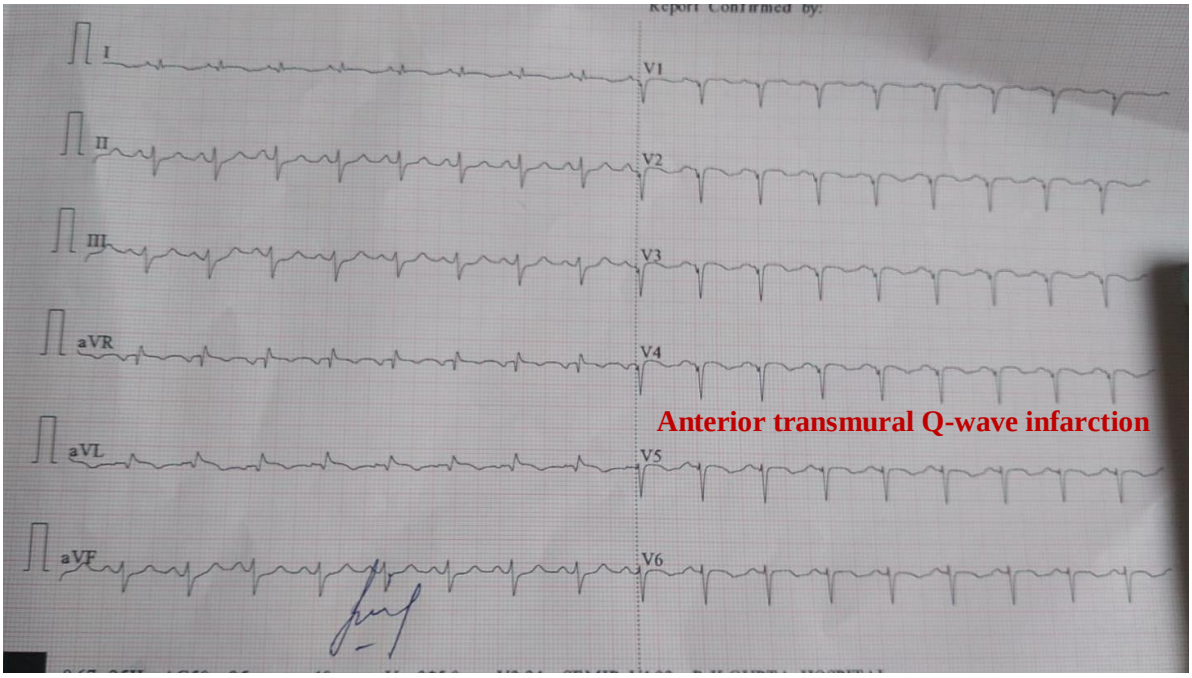
ECG 2 : The next immediate ECG



Source : The same as mentioned with ECG 1

Findings : In addition the presence of terminal QRS distortion sign – as qR with uplifting of the remaining R-wave, extending from V2-V6.

ECG 3 :Post PTCA ECG

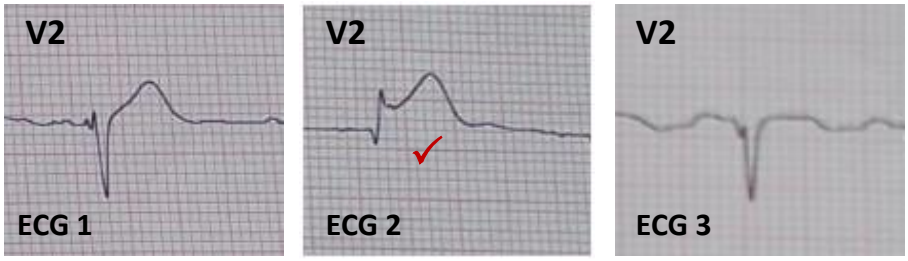


Source : The same as mentioned with ECG 1

Findings : Evidence of STEMI in lead I and aVL plus full blown anterior transmural Q-wave infarction (obvious over V1 to V4) indicating further worsening of ECG changes within the same territory of LAD occlusion.

(Echocardiography LVEF 35-40% , CART Proximal LAD 90% occlusion)

Discussion on these trio-ECGs recorded in succession one after the other :
(Lead V2 represents as the lead to denote the concerned ECG changes in the corresponding ECGs)



History of ongoing chest pain with High lateral STEMI on ECG

'TQRSD'-sign (qR with J point uplifting)

Full bloom Q-wave (Full blown transmural Q wave infarction)

- ECG 2 shows the presence of terminal QRS distortion sign (TQRSD) on the precordial leads , a forerunner of transmural myocardium infarction.
- ECG 3 illustrates the phenomenon of full blown anterior transmural Q-wave infarction.

NB : Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific for proximal LAD occlusion

5. Distinction in between STEMI and Terminal QRS Distortion Sign

STEMI and Terminal QRS Distortion Sign are not the same , though they may seem similar at first glance by morphology. Here there is a breakdown of the differences in between these two , as mentioned below :

STEMI indicates acute complete occlusion of a concerned coronary artery from the very start. Here ST elevation is due to epicardial injury with overlying complete coronary occlusion (the current of injury is in the same direction as that of exploring electrode , that's why , there is ST elevation in the concerned leads).

The Terminal QRS Distortion Sign indicates a somewhat damaged platform due to critically ischemic myocardium which sends electrical signal on ECG as distortion of terminal QRS complex with loss of both the S wave and J wave with uplifting of the remaining R-wave above its baseline by >50% , at least present over the preordial leads V2/V3. TQRSD sign is considered an electrical manifestation of critical myocardial ischemia – it means that the final part of the QRS complex is no longer following the usual conduction pattern of electrical flow through the heart muscle. This gets reflected as a consequence of ongoing coronary insufficiency , still having no complete coronary artery occlusion , but it rather indicates hidden ,

imminent danger to the heart' s vulnerability towards the rapidly evolving STEMI. It is very specific (100%) to proximal LAD occlusion.

STEMI	Terminal QRS distortion sign
• Acute complete occlusion of a coronary artery from the very start	A consequence of ongoing critical myocardial ischemia – leading to transmural Q-wave infarction within a short time
• This reflects epicardial injury , recorded on ECG as ST elevation	It is altered electrical manifestation of underlying myocardial insult as distorted terminal QRS complex – loss of both S wave and J wave with uplifting of the remaining R wave above its baseline
• It may occur with any branch of the coronary circulation	It is specific (100%) to proximal LAD occlusion marking an extensive area of myocardium at risk.

6. Take Home Message

- ☐ The terminal QRS distortion is accessed on ECG by the loss of both the S wave and J wave with uplifting of the remaining R wave above its baseline by > 50% , at least present over the precordial leads V2/V3.
  (In association with the loss of S-wave, the remaining R-wave may take the configuration as qR , mirroring the imprint of still ongoing myocardial ischemia)
- ☐ Its presence in the precordial leads has been strongly correlated with a rapidly progressive STEMI in the territory of proximal LAD occlusion , marking an extensive area of myocardium at risk with an urgent need for revascularization.
- ☐ Terminal QRS distortion increases all-cause mortality by 81%. One study suggests that terminal QRS distortion is an important tool to assess the risk in patients with STEMI.
- ☐ A timely honoured urgent reperfusion therapy may reduce morbidity and mortality both in such a situation.
- ☐ Since terminal QRS distortion is associated with up concavity , it might be confused with benign early repolarization syndrome (ERS) . Always to keep in mind that terminal QRS distortion (TQRSD) is 100% specific to proximal LAD occlusion
- ☐ Electrophysiology : When the myocardium is at the critical stage of ischemia , cells in the affected area (supplied by proximal LAD in this context) loose somewhat oxygen with ATP , which disrupts the ability to maintain its action potential integrity. The electrical signal cannot travel smoothly through the distrupted area :

Loss of both S wave and J wave with uplifting of the remaining R wave above its basseline

- ☐ It should be rather a rule of thumb that every ECG with ongoing chest pain ± any evidence of STEMI suggestive of proximal LAD occlusion should be analysed for the presence of terminal QRS distortion sign so that urgent reperfusion therapy might be instituted to prevent the further catastrophe.

Terminal QRS distortion suggests that the critically ischemic myocardium is more likely to further evolve into the full-thickness transmural Q-wave infarction within a short time if not rapidly managed by urgent reperfusion therapy , it may lead to poorer outcomes.

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ATRIAL TACHYCARDIA ON ECG

ATRIAL TACHYCARDIA ON ECG

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OUTLINE

Introduction
Atrial tachycardia (AT) is a form of supraventricular tachycardia (SVT) originating from a single ectopic focus within the atria but outside of the sinus node – since it is having a single point of firing outside the natural pacemaker, it is also known as Focal atrial tachycardia (FAT)

Electrophysiology

A. Abnormal automaticity
B. Reentry (Micro-reentrant circuit)
C. Triggered activity

What causes atrial tachycardia ? (Risk factors)
Atrial tachycardia occurs most commonly in elderly group of patients and those with other types of structural cardiac disorders, at times it may occasionally appear in children , younger generation and those with healthy hearts.

Evaluation by ECG

Complications of atrial tachycardia

Diagnosing atrial tachycardia

Illustration by ECGs
ECG 1 : 80 years old female with urosepsis and encephalopathy
ECG 2 : "Incessant" Atrial Tachycardia induced cardiomyopathy

Take Home Message

References

Atrial tachycardia on ECG

A Narrative Review

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Understanding atrial tachycardia needs unthreading the intricate electrical pathways of the heart, when even a single misfiring cell can create a cascade of rapid rhythms, reminding us of the delicate balance between order and chaos in human physiology.

Truly to say , the heart is electrophysiologically well protected but with certain points of breaking in , the frantic verse of cardiac arrhythmias may peep through.

- ☐ **Normally the beating of the heart begins with an electrical impulse arising from the sinus node , a single point in the heart's right atrium (natural pacemaker of the heart)**
- ☐ **In atrial tachycardia , a specific area within the atria acts as an ectopic pacemaker , producing rapid run of electrical impulses that overrride the sinoatrial (SA) node activity**

This narration of atrial tachycardia dictates how the order breaking in occurs with the impulses from SA node and is being intruded and replaced by a rapid run of beats arising from the ectopic atrial tissue.

1. Introduction (Keypoints)

- Atrial tachycardia (AT) is a form of supraventricular tachycardia (SVT) originating from a **single** ectopic focus within the atria but outside of the sinus node – since it is having a single point of firing outside the natural pacemaker, it is also known as Focal atrial tachycardia (FAT).

- It is characterized by rapid and regular heart rate , often exceeding 100 beats/min (usually 100 to 250 bpm). But with critical atrial rate , there may occur a physiological response in the form of variable AV conduction

- Accordingly ventricular conduction can be :
  - Irregular or irregularly irregular in the setting of variable AV conduction
  - Regular if 1 to 1, 2 to 1, or 4 to 1 AV conduction

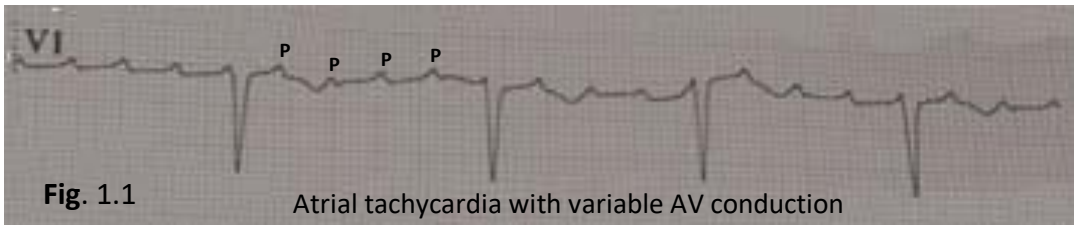


Fig. 1.1 Atrial tachycardia with variable AV conduction

- There is usually a distinct isoelectic baseline in between two atrial activities (unlike atrial flutter which is having uninterrupted saw-tooth appearance)

- Rhythm may be paroxysmal , usually lasting for a brief period which starts and stops spontaneously :
  - May demonstrate an increase in the rate at initiation ("warm up")
  - May demonstrate a decrease in the rate at termination ("cool down")

  If the episode continues and is sustained , it is called **persistent AT**.
- Focal tachycardia typically occurs in the setting of increased metabolic demand and stress, like infection, hypoxia, etc.
- Atrial tachycardia occurs most commonly in elderly group of patients and those with other types of structural cardiac disorders, at times it may occasionally appear in children , younger generation and those with healthy hearts.

2. Electrophysiology

The electrophysiology of AT refers to the study and understanding of the electrical mechanism underlying this arrhythmia. It involves analyzing how electrical impulses from ectopic atrial tissue are generated , conducted , and how they deviate from normal patterns of SA node impulses.

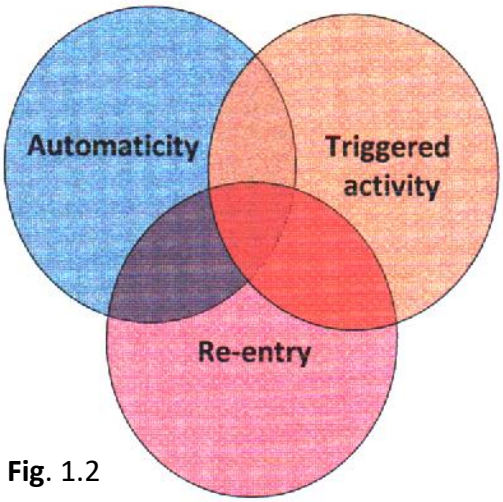


Fig. 1.2

The SA node is having the highest capability of discharging impulses and therefore , this is usually responsible for pacing the heart while the other subsidiary pacemakers remain dormant. In case of Focal atrial tahcycardia the initiating impulse arises from any or a combination of the three mechanisms: abnormal automaticity, reentry or as triggered activity , making these mechanisms dominant over the SA node.

A Abnormal automaticity

Normally during phase 4 of atrial action potential, the cardiac myocyte membrane remains polarized and the inwardly rectifying potassium channel is responsible for maintaining baseline resting potential .

But in focal atrial tachycardia abnormal automaticity occurs as a result of accelerated phase 4 with rapid upsloping , leading to spontaneously discharging cells. In other words the phase 4 is not having true resting membrane potential (RMP) due to the fact that it shows a continuous automatic activation in a rising semi-curve manner.

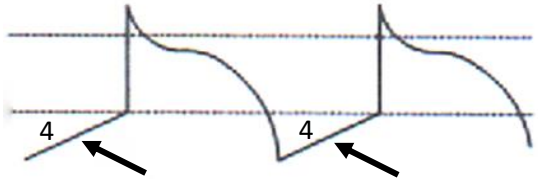


Fig. 1.3

B Reentry

If reentrant mechanism is involved in Focal atrial tachycardia , it involves a micro-rentrant circuit unlike atrial flutter whih involves macro-rentrant mechanism. This circuit is considered to have involvement of adjacent atrial tissue as susbstrates with different electrophysiological properties , that's why , this circuit has different phases of atrial tissue refractoriness being chased by its recovery but at a slower conduction – hence , there exists usually a distinct isoelectric line in between two atrial activities (unlike atrial flutter which is having uninterrupted saw-tooth appearance)

NB : Localized fibrotic areas in the atrial tissue can act as an anchor for miro re-entrant circuits by providing regions of conduction block or slow conduction .

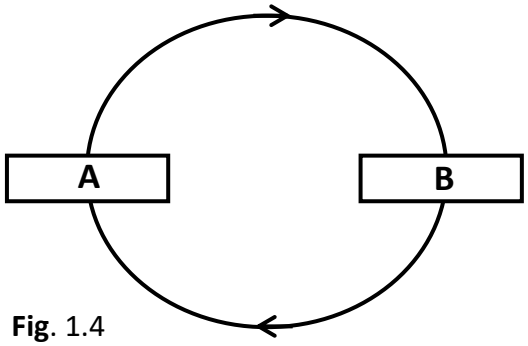


Fig. 1.4

Reentrant circuit involves adjacent atrial tissue as substrates having different electrophysiological properties , say for example ; as illustrated above , atrial tissue type A and B are having different conduction velocities and rates of repolarization. Due to these differences , the impulse travels around the circuit when one tissue type substrate is recovering from its slow refractive state , then the impulse is circulated over the next tissue substrate when it is no longer in refractory state.

C Triggered activity

Here the vulnerable repolarization period for triggered activity is at the start of phase 4 of the action potential whereupon the incoming atrial premature beat is sufficient enough in its strength to bring the membrane to its thershold potential – a series of spontaneous bursting of atrial beats occur as triggered activities leading to atrial tacychycardia , but not so rapidly – having isoelectric gap in betwwen two such triggered activities.

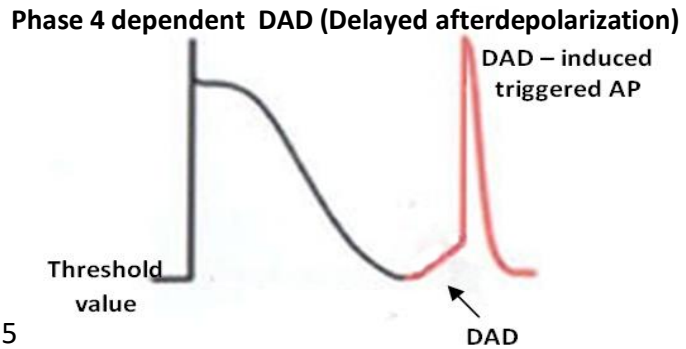


Fig. 1.5

During DADs , calcium intracellular load triggers the sodium-calcium exchanger (the intrusion of sodium ions inside the cells) , which can generate depolarizing currents strong enough to reach the threshold and initiate other action potentials in series resulting in atrial tachycardia.

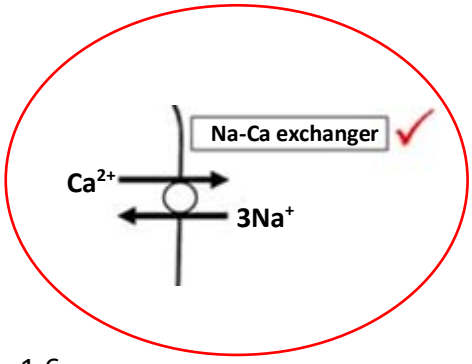


Fig. 1.6

➢ Some basic facts with abnormal automaticity , reentrant mechanism and triggered activity are as follows :

Abnormal automaticity	Reentrant circuit	Triggered activity
☐ Warm –up and cool-down phenomenon	Sudden onset and termination	Warm -up /cool-down phenomenon not in a strict sense – gain in amplitude at the start due to increasing Ca++ cycling and vice-versa at the end. (This occurs in some case)
☐ Cellular ionic leaks (Ca , Na) or sudden adrenergic drive	Premature atrial beats initiates the micro-reentrant circuit	Calcium intracellular load triggers the Na-Ca exchanger mechanism (e.g. , with digoxin toxicity)

3. What causes atrial tachycardia ? (risk factors)

Again to repeat atrial tachycardia occurs most commonly in elderly group of patients and those with other types of structural cardiac disorders, at times it may occasionally appear in children , younger generation and those with healthy hearts.

Multiple causes include :

☐ Changes in lifestyle :
- Stress , excessive caffeine or alcohol intake and lack of sleep
- Excessive use of cocaine and other stimulants

☐ Drugs , including those used to treat asthma , allergies and colds , digoxin toxicity

☐ Non-cardiac conditions
- Sleep apnea , hyperthyroidism , diabetes , lung diseases (e.g COPD) , and electrolyte imbalances

- Focal atrial tachycardia occurs in the setting of increased metabolic demand and stress , like infection , hypoxia , etc.

☐ Cardiac conditions

- A "stretched" atrium caused by hypertension , heart failure , cardiomyopathy , cardiac vulvular diseases , congenital heart defects
- Atrial scarring due to ischemic heart disease , atrial cardiomyopathy , previous heart surgery

☐ Idiopathic

These signals so created either singly or in combination may lead to the genesis of atrial tahycardia.

4. Evaluation by ECG

The study of ECG changes in this context helps in the diagnosis of atrial tachycardia. ECG features may also localize the site of origin.

Electrocardiographic features include :

- Atrial rate :100 to 250 bpm
- Ventricular conduction can be :
  - Irregular or irregularly irregular in the setting of variable AV conduction
  - Regular if 1 to 1, 2 to 1, or 4 to 1 AV conduction
- P-wave morphology
  - Unifocal , identical P-wave morphology (differs from normal sinus P-wave)
  - Altered P-axis as per the site of ectopic beat
- PR interval
  - Short PR interval due to accelerated AV conduction
  - Prolonged PR interval due to delayed AV conduction
- Normal QRS morphology (unless pre-existing bundle branch block , accessory pathway or rate –related aberrant conduction)

○ Rhythm may be paroxysmal , usually lasting for a brief period which starts and stops spontaneously :

  - May demonstrate an increase in the rate at initiation ("warm up")
  - May demonstrate a decrease in the rate at termination ("cool down")
- ST-T changes
  The ST segment depression and T-wave inversion may appear as a part of tachycardia

✓**NB** : AV block is generally a physiological response to the rapid atrial rate , except in digoxin toxicity where there is AV supression due to vagotonic effects of digoxin , resulting in a slow ventricular rate (PAT with block)

In some cases , atrial tachycardia may be asymptomatic and only detected during a routine electrocrdiogram (ECG)

5. Complications of atrial tachycardia

- ☐ Persistant atrial tachycardia may lead to cardiomyopathy with or without heart failure. This type of cardiomyopathy is often reversible if the atrial tachycardia is brought sooner under control.
- ☐ Sometimes atrial tachycardia may even lead to atrial flutter / fibrillation or even ventricular arrhythmias.

NB : Although atrial tachycardia is less common than other arrhyhmias such as atrial fibrillation , but it can significantly affect heart function and overall health if its management is being neglected.

6. Diagnosing atrial tachycardia

Diagnosis typically begins with a detailed medical history and physical examination , including some diagnostic tools as well.

Symptoms

- **A** feeling of the heart racing or fluttering
- **B**reath-shortness : caused by reduced oxygen delivery
- **C**hest discomfort : mild pain or tightness in the chest
- **D**iziness or light headachedness : due to decrease blood flow to the brain.
- **E**fficiency ↓ with diminished cardiac output to the extent of experiencing cardiac arrest or unconsciousness
- **F**atigue : the heart's inefficiency can lead to tiredness

Physical examination

- The assessment of haemodynamic status includings the detailed examination of CVS , and other systems as per need

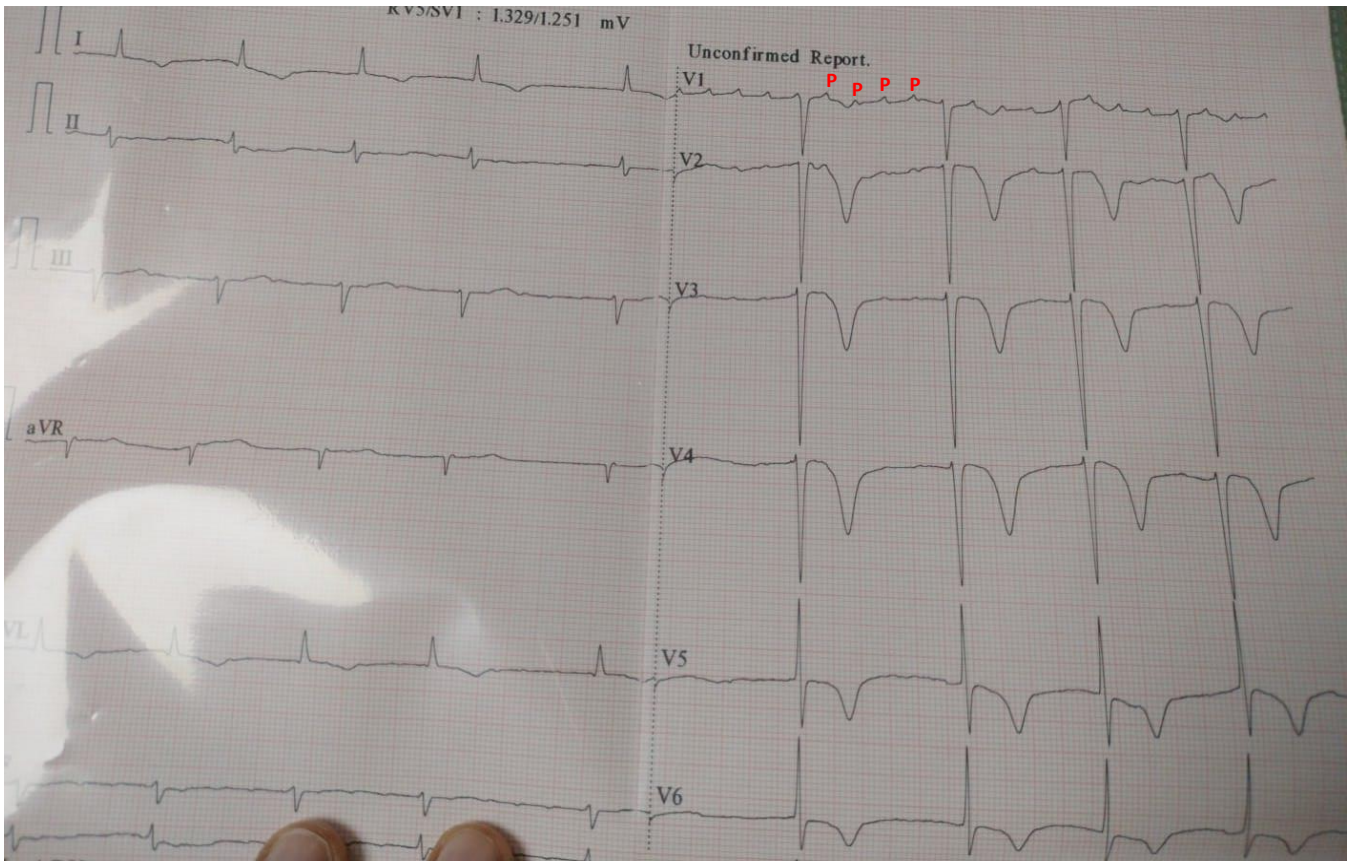
Diagnostic tools

- **Electrocardiogram (ECG)** : In some cases atrial tachycardia may be asymptomatic and only detected during a routine evaluation by ECG
- **Echocardiogram :** Provides images of the heart to identify structural abnormalities
- **Holter Monitor :** a portable device worn for 24-48 hours to capture the episodes (Event monitor for one or two months , that records the intermittent spells of tachycardia when one feels symptoms. It works by pusing a button on the monitor during the symptomatic perioid).
- **Electrophysiological studies (EPS)** : Maps the electrical signals with the heart to pickup the point-source of the arrhythmia to have catheter ablation as per need.

7. Illustration by ECGs

ECG No. 1

80 years old female admitted with urosepsis and encephalopathy with the following ECG :



Source : Dr. S.R. Ram Prasanth , Assistant professor , Dept of Medicine , Government Peripheral Hospital , Periyar Nagar , Chennai

Findings :

- Atrial tachycardia at the rate of 250 bpm , with variable AV conduction
  Identical atrial waves with intervening isoelectric lines in between , most obvious over lead V1 (atrial activities are inconspicuous in limb leads) : see above ECG
- Low voltage over limb leads
- Poor R-wave progression (see leads V3) with somewhat late transition zone at V5
- Symmetrical and somewhat deep T inversion over precordial leads ; T-wave inversion also over leads I and aVL

Discussion :

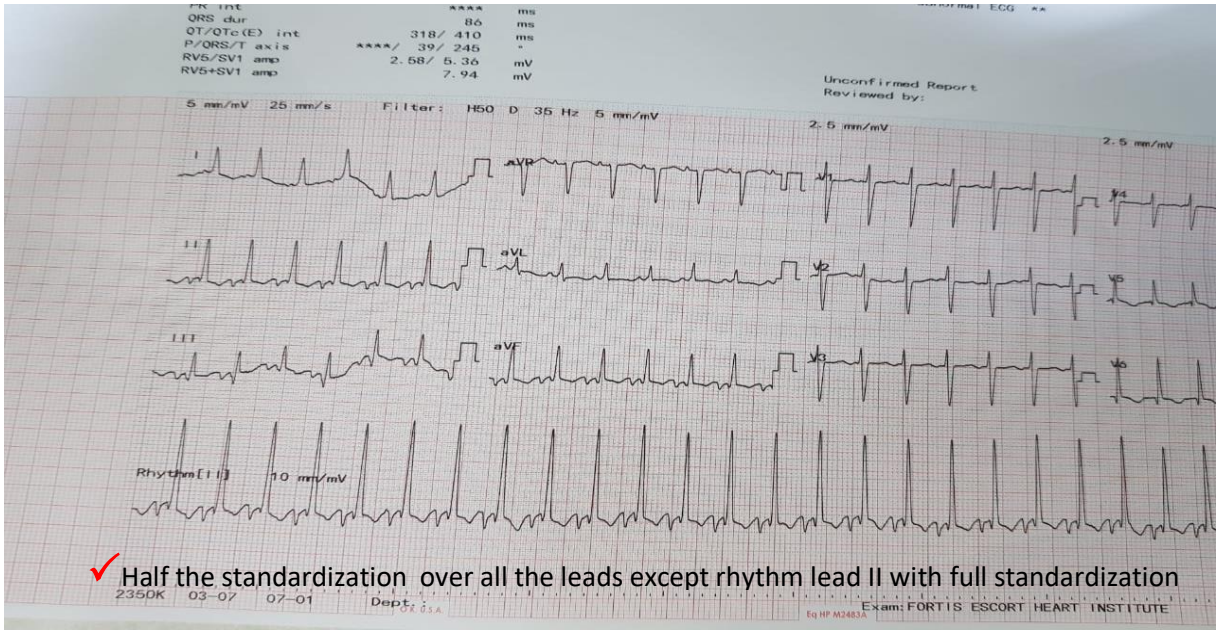
- To start with to say that atrial tachycardia occurs most commonly in elderly group of patients (here 80 years old female) and it is typically observed in the setting of increased metabolic demand and stress like infection or hypoxia as in this case
- Urosepsis with cerebral hypoxia (encephalopathy) increases the risk of cardiac dysfunction , may result in ischemia , metabolic dearrangements , or infection- related myocardial depression (septic cardiomyopathy)

- Atrial activities belonging to atrial tachycardia are best observed in lead V1. V1 is positioned to record the changes from the right atrium directly and it is also the fact that the right atrium is the most common site of focal atrial tachycardia. V1 here captures these distinct P-waves with more clarity than other leads , the intervening isoelectric lines are also distinctly observed.
- Low voltage over limb leads can be explained by sepsis induced cardiomyopathy resulting in with myocardial depression
- Poor R-wave progression with T-changes as mentioned might be explained by the co-existing ? old anterior myocardial infarction , myocardial ischemic injury or and associated 'cerebral T-waves' attributed to accompanying encephalopathy.

Overall impression :

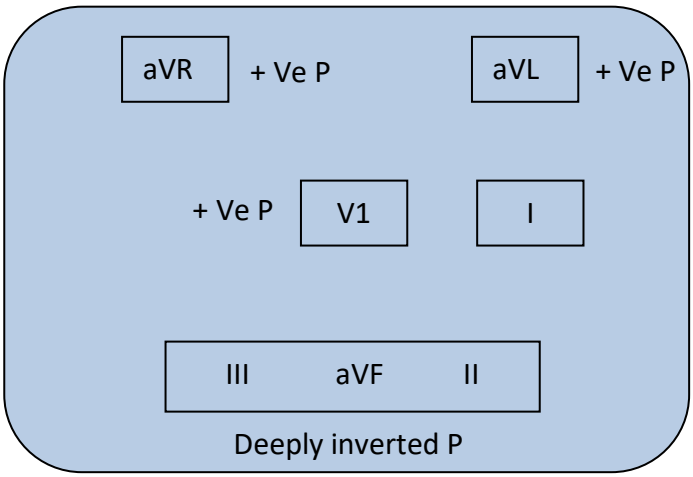
Septic cardiomyopathy with possible ischemic insult leading to atrial tachycardia.

ECG No. 2 : Incessant atrial tachycardia induced cardiomypathy with the following ECG:



Source : CME INDIA (Dr. Vijay Laxmi Ji – Bangalore – Dated June 04, 2020)

Plotting of P waves as per the posted ECG



Findings

- R in V6>R in V5 indicates possible LVH
- Plotting of P-waves from the above ECG indicates the site of atrial tachycardia to be at low right atrium (possibly from tricuspid anulus). More deeply inverted P in inferior leads indicates the sight towards low right atrium)

Discussion

" Incessant is applied to an AT that is present for at least 50 percent of the time when a patient is monitored." (automaticity is usually the mechanism of incessant atrial tachycardia).

It would be worthwhile to mention here that incessant (persistent) atrial tachycardia leads to tachycardia related cardiomyopathy , as in this case. This type of cardiomyopathy is often reversible provided the atrial tachycardia can be brought sooner under control.

8. Take Home Message

- Atrial tachycardia (AT) is a form of supraventricular tachycardia (SVT) originating from a **single** ectopic focus within the atria but outside of the sinus node – since it is having a single point of firing outside the natural pacemaker, it is also known as Focal atrial tachycardia (FAT).
- It is characterized by rapid and regular heart rate , often exceeding 100 beats/min (usually 100 to 250 bpm). But with critical atrial rate , there may occur a physiological response in the form of varible AV conduction
- There is usually a distinct isoelectic baseline in between two atrial activities (unlike atrial flutter which is having uninterrupted saw-tooth appearance)
- Rhythm may be paroxysmal , usually lasting for a brief period which starts and stops spontaneously :
  - May demonstrate an increase in the rate at initiation ("warm up")
  - May demonstrate a decrease in the rate at termination ("cool down")
- The SA node is having the highest capability of discharging impulses and therefore , this is usually responsible for pacing the heart while the other subsidiary pacemakers remain dormant. In case of Focal atrial tahcycardia the initiating impulse arises from any or a combination of the three mechanisms: abnormal automaticity, reentry or as triggered activity , making these mechanisms dominant over the SA node.
- Atrial tachycardia occurs most commonly in elderly group of patients and those with other types of structural cardiac disorders, at times it may occasionally appear in children , younger generation and those with healthy hearts .
- ECG changes – please see page 5
- Persistant atrial tachycardia may lead to cardiomyopathy with or without heart failure. This type of cardiomyopathy is often reversible if the atrial tachycardia can be brought sooner under control.
- Although atrial tachycardia is less common than other arrhyhmia such as atrial fibrillation , but it can significantly affect heart function and overall health if its management is being neglected.

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THE SI SII SIII SYNDROME ON ECG

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©DR. D.P. KHAITAN

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OUTLINE

The SI SII SIII Syndrome on ECG

A Narrative Review

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A paradigm shift at times in life becomes essential – a move more towards a comfortable and safety adaptation. This proves to be a safety essential for survival. I call this an axial shift of life , which allows a human race to survive.

The SI SII SIII syndrome is one of the examples of such a paradigm shift on ECG.

- ☐ The term 'SI SII SIII' syndrome refers to an ECG pattern where there are prominent S waves in leads I , II , III. This is not a specific marker for a single entity but rather an indicator of right heart overload or even strain.
- ☐ The enlarged or strained right ventricle produces more electrical activity which shifts the electrical axis more superiorly and rightward – this shift may be called as a new adaptation for the survival even when the heart remains firmly anchored to the underneath diaphragm by the central tendon.

The presence of SI SII SIII pattern on ECG prompts a clinician in the search of its root cause leading to right ventricular overload or strain.

1. Introduction (keynotes)

- As its name implies SI SII SIII syndrome is an electrocardiographical expression with prominent S waves in leads I II III with a positive deflection in lead aVR -------- the result of superior and rightward shift in the heart's electrical axis
- To make it simple , when the right ventricle is hypertrophied or strained , it becomes electrically dominant over the left ventricle , causing the heart's electrical axis to be shifted more rightward and upward→ this depolarization shift is away from leads I II III → prominent S waves in these leads.
  (Negative waves inscribed in those leads which are away from the electrical axis**)**

Superior and rightward shift of Electrical axis (-90^0 to -150^0)

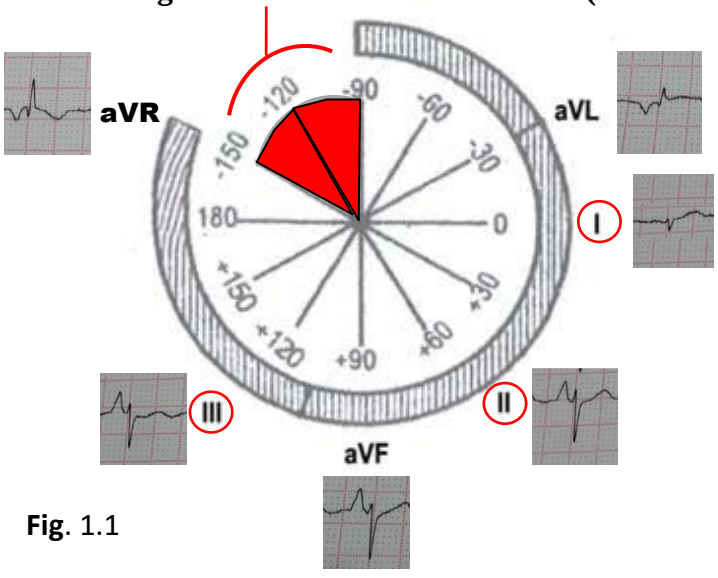


Fig. 1.1

Prominent S-waves in leads I II III with a positive deflection in lead aVR

- This pattern was initially brought to the light in 1960 by Burch and D Pasqulae in association with ventricular septal defect and shortly thereafter in adults with chronic obstructive pulmonary disease.
- The SI SII SIII syndrome is not a specific marker for a single entity as such but rather indicative of right ventricular overload or strain. It is not an uncommon electrocardiographic finding , seen usually associated with chronic pulmonary disease (eg. COPD)
- The presence of such findings on ECG prompts further investigations directed towards the underlying cause , those including echocardiography , chest radiology or at times even CT imaging to access the right heart and pulmonary vasculature.

2. Electrophysiology (Mechanism)

In a normal heart , the electrical axis is typically pointing downwards and to the left , reflecting the dominance of the left ventricle. This results in positive QRS complexes (upright R-waves) in leads I and II , which are oriented towards the left.

✓ But the enlarged or strained right ventricle sets its electrical signal more rightward and upward as a result of more electricity production attributed to the altered anatomical remodelling of the heart.

The basic reasoning behind SI SII SIII syndrome is summarized as below :

> The basic reasoning of this SI SII SIII seems to be related to somewhat delayed right ventricular depolarization due to remarkable right ventricular hypertrophy or secondary to its acute strain, shifting the electrical axis more right and upward.
> Only simple RVH or its strain might have produced only rightward shift , not so directed upward

The electrocardiographic findings have been well illustrated in the previous page (Fig. 1.1) but to understand the actual mechanism with more clarity , it become essential to delineate the electrical scenario also on the horizontal plane –.

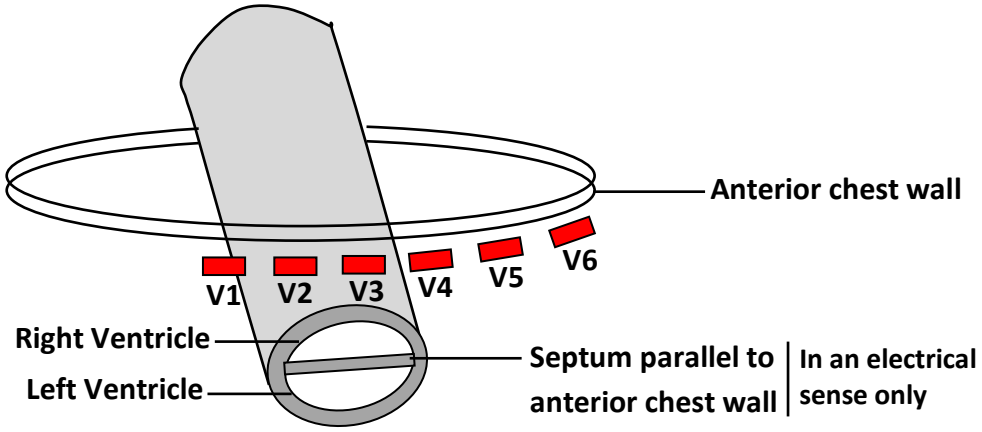
The presence of an **r'** wave in lead V1 is seen specially in the context of this SI SII SIII syndrome due to somewhat delayed activation of the hypertrophied or strained right ventricle. This makes the morphology of V1 pattern as rSr'. In other words this r' denotes the delayed activation of the right ventricle .

There may be a clockwise rotation observed with precordial leads . This entire electrocardiological episode in context with this syndrome can be explained by a single factor , being operated through both the planes – RVH or its acute strain :

Frontal plane reflects the orientation of electrical axis more towards right and upward, attributed to somewhat delayed depolarization of the hypertrophied or strained right ventricle.

Horizontal plane The initial r wave in V1 is followed by an S wave – as a mirror reflection of the left ventricle depolarization and is further followed by a next r' wave due to the delayed right ventricular activation (This r' wave is non-specific finding , not always present).

Display of SI SII SIII syndrome over the horizontal plane as r' in V1 with clockwise rotation –
— In true sense it is electrical translation of mechanical remodelling of RV:



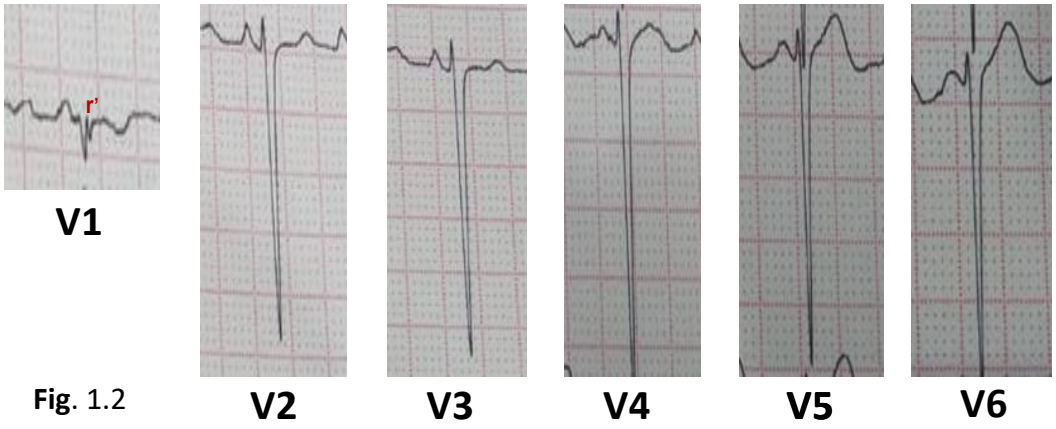


Fig. 1.2

Inscription of r' in V1 with associated clockwise rotation

3. SI SII SII Syndrome is the electrical translation of mechanical remodelling in RV

☐ **Over the frontal plane**

More electricity production by such hypertrophied or strained right ventricle

↓

Pushing the electrical axis rightward and upward

↓

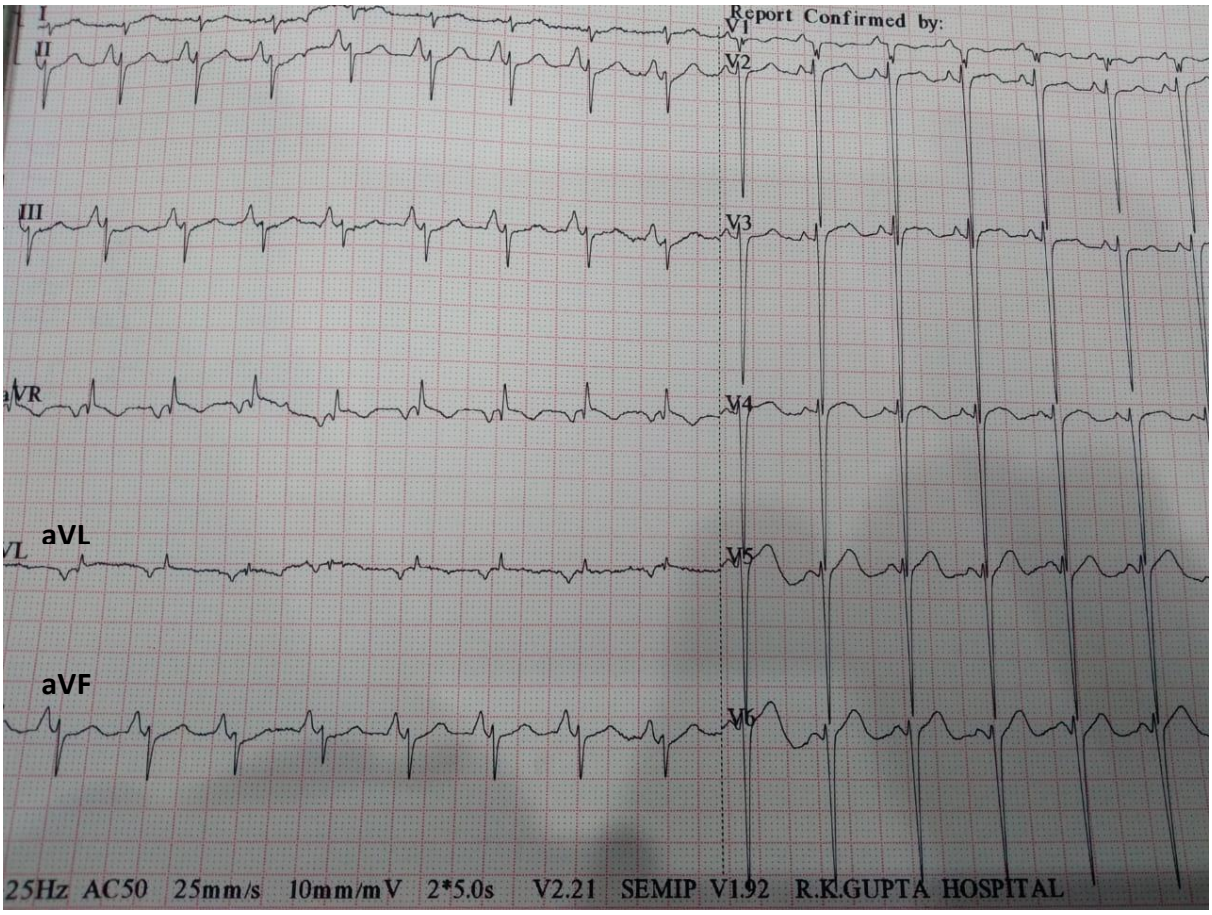
Prominent S-waves in leads I II III with a positive deflection in aVR (qR)
(this entire incidence has been illustrated as such in Fig. 1.1)

☐ **Over the horizontal plane**

With clockwise rotation around the oblique axis , the hypertrophied or strained right ventricle is assumed to adopt a more anterior postion so that the interventricular septum lies parallel to anterior chest wall. This would mean that all or most of the precordial leads – leads V1 to V5 or V6 would reflects rS complexes ——
but in true sense it is the electrical translation of mechanical remodelling of hyperophied or strained right ventricle

4. Illustration by ECG with its full explanation

This ECG belongs to 55 years male with the history of breathlessness since last 25 years



Global Heart Rhythm Forum , posted by Dr. R.K Gupta , Senior consultant Physician , Jamunanagar ,(Haryana) on 31st March 2024

Findings :

☐ **Frontal plane**

(i) SI SII SIII pattern as evident by prominent S-wave in leads I II III with a positive deflection in lead aVR (qR) – rightward and superior shift of the electrical axis

(ii) The P-pulmonale in leads II III and aVF with P-axis = $+105^0$ (Rtshift of P-axis)

☐ **Horizontal plane**

(i) Clockwise rotation as rS extending from V1 to V6

(ii) The presence of r' wave in V1

(iii) The amplitude of P wave > 1.5 mm, most prominent in V1 to V3

Discussion

- P-pulmonale (P-waves > 0.25 mV) in leads II , III , aVF
- P-axis + 105 P > 1.5 mm in V1-V3

= Right Atrial Enlargement

- Clockwise rotation with R/S ratio < 1 in lead V5 and V6
- SI SII SIII pattern , as discussed

= Right Ventricular Hypertrophy

As per Chou's criteria , this proves to be a case of COPD (Chronic obstructive pulmonary disease)

According to Chou , COPD is likely to be present if one or more of the P wave changes with one or more of the QRS changes as enumerated below , are present on ECG:

Pwave changes	1. P waves>0.25 mV in lead II,III,aVF. 2. P wave axis to the right of 80 degrees in the frontal plane. 3. Lead I sign with an isoelectric P wave,QRS amplitude <0.15 mV, and T wave amplitude <0.05 mV.
QRS changes	4. QRS amplitude in all limb leads < 0.5 mV 5. QRS axis to the right of 90 degrees in the frontal plane. 6. QRS amplitude <0.5mV in lead V5 or V6; or R wave <0.7mV in lead V5 or R wave <0.5 mV in lead V6. 7. R/S ratio <1 in lead V5 or V6. 8. $S_1S_2S_3$ pattern with R/S ratio<1 in leads I, II and III.

5. Aetiological factors leading to SI SII SIII Syndrome

The SI SII SIII Syndrome might be reflected as de novo under the following circumstances :

- As a normal variant in children due to right ventricular dominance

- As an expression of right ventricular hypertrophy such as with congenital heart disease or Acquired heart disease i.e COPD .

  It is a known fact that the SI SII SIII syndrome is not an uncommon electrocardiographic finding associated with acquired right ventricular enlargement due to chronic pulmonary disease .

- Apical myocardial infarction

  The infarction at the apex can cause abnormal conduction , leading to the electrical axis shifting more towards the right side of the heart , producing prominent S-wave in limb leads. In some cases the apical myocardial infarction can lead to increased strain on the right ventricle , most probably due to secondary defects , such as increased pressure in the pulmonary circulation (Pulmonary hypertension) with shifting of the electrical axis towards the right

- The straight back syndrome (straightening of upper dorsal spine due to loss of anterior concavity) - a congenital osseous manifestation without any heart abnormality.

6. Take Home Message

- ☐ This SI SII SIII syndrome seems to be related to somewhat delayed right ventricular depolarization due to remarkable right ventricular hypertrophy or secondary to its acute strain, shifting the electrical axis more right and upward .
  Only simple RVH or its strain might have produced only rightward shift , not so directed upward
- ☐ Prominent S waves in leads I II III with a positive deflection in lead aVR ------- hence the nomenclature SI SII SIII Syndrome
- ☐ There might be clockwise rotation on the horizontal plane. The QRS duration is normal
- ☐ The terminal QRS over the horizontal plane might be associated with a small r' deflection in lead V1. This is non-specific finding , not always present.
- ☐ This syndrome is not a specific marker for a single entity as such but rather indicative of right ventricular overload or strain. It is not an uncommon electrocardiographic finding , seen usually associated with chronic pulmonary disease (eg. COPD)
- ☐ In true sense SI SII SIII syndrome is the electrical translation of mechanical remodelling of hypertrophied or strained right ventricle
- ☐ The presence of such findings on ECG prompts further investigations directed towards the underlying cause , those including echocardiography , chest radiology or at times even CT imaging to access the right heart and pulmonary vasculature.

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