ECG Review : Some momentous articles

Series 7

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

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

These momentous ECG scripts
remind us of beauty wrapped in lyrics .....

ECG Review : Some momentous articles – Series 7
(For Academic Purpose only)

© Dr. DURGA PRASAD KHAITAN
MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

These momentous ECG scripts
remind us of beauty wrapped in lyrics ...

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

Whose wisdom and vigilance bring meaning to every heartbeat traced upon these pages

Index

HAYSTACK PRINCIPLE : SEARCHING FOR HIDDEN ‘P’ ON ECG – A MOTHER’S CRY , ‘ WHERE IS MY NANHA-MUNNA’ ?

ECG

HAYSTACK PRINCIPLE : SEARCHING FOR HIDDEN ‘P’ ON ECG - A MOTHER’S CRY, ‘WHERE IS MY NANHA-MUNNA?’

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction
Whenever one is faced with the problem of finding hidden P-waves , the attention should be directed to the lead having the least electrical deflection on ECG , as per ‘Haystack principle’.

When and why P-waves are hidden ? (Case based study)
- First case study with short RP tachycardia
- Second case study in a patient with unexplained syncope

Discussion
The phrase “finding a needle in a haystack” is a very old metaphor and has been used in electrocardiology to describe nearly precise searches for hidden P-waves.

Take Home Message

References

Haystack Principle : Searching for Hidden 'P' on ECG – A Mother's Cry, 'Where is my Nanha-Munna?'

A Narrative Review

© DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

A child was lost in the crowd. The mother ran amok, crying, 'Where is my Nanha-Munna?' And then—she found him, playing with another child.

'P' on 12-lead ECG is just like a 'Nanha-Munna' child for a clinician. It may play hide-seek game , at times making its appearance difficult to be detected. It is like a hidden star in the nighty sky, the hidden P-wave does not disappear , rather it awaits for the keen eyes to find it.

- ☐ **Whenever one is faced with the problem of finding 'hidden P' , lead showing the smallest electrical deflection should be explored for the purpose. It is like a searching a small needle in the haystack of smaller size.**
- ☐ **This searching out of hidden P-wave may yield a useful diagnostic information**

Truly to say that medicine , like science , is about finding an informative signal amidst the noise. The key to diagnosis is not just what is visible, but to reveal what is non-visible – in this way ECG is both the science and art.

1. Introduction (Keypoints)

- The P-wave denotes atrial depolarization , the first electrical activation on 12-lead ECG : it occurs just before the QRS complex , which represents ventricular depolarization. It is usually a small positive deflection on ECG. A normal P-wave with a consistent morphology and timing indicates a regular sinus rhythm.

- P-waves are most easily seen in the inferior leads (II , III and aVF) and lead V1 , as well illustrated with the following sketch but at times these P-waves are hidden at its usual sites , then it becomes a challenging task to detect them but its detection is essential as a clue to the underlying diagnosis.

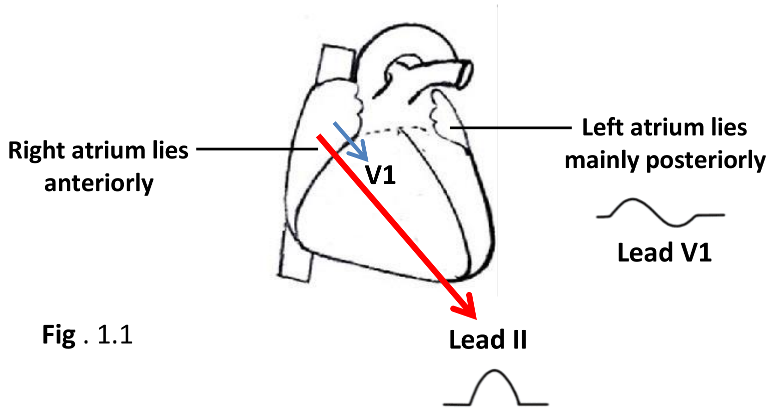


Fig . 1.1

- **How to search out these hidden P-waves ?**
  The **'Haystack Principle'** can be of great diagnostic clue when one is searching for such hidden P-waves. The basic principle is based on the fact that when one has to find a small needle in a haystack , he would obviously prefer small haystack for the purpose. Therefore , whenever one is faced with the problem of finding hidden P-waves , the attention should be directed to the lead having the least electrical deflection on the ECG. The searching out of these hidden P-waves may reveal even the alarming signals which would have been otherwise missed.

The art and science of electrocardiogram lie in witnessing the subtle message hidden therein. The secrecy of such insight lies in distinguishing meaningful content from background noises – and here , a clinician must quickly identity the message through the geographical analysis of different waveforms on ECG , as P-waves in this context.

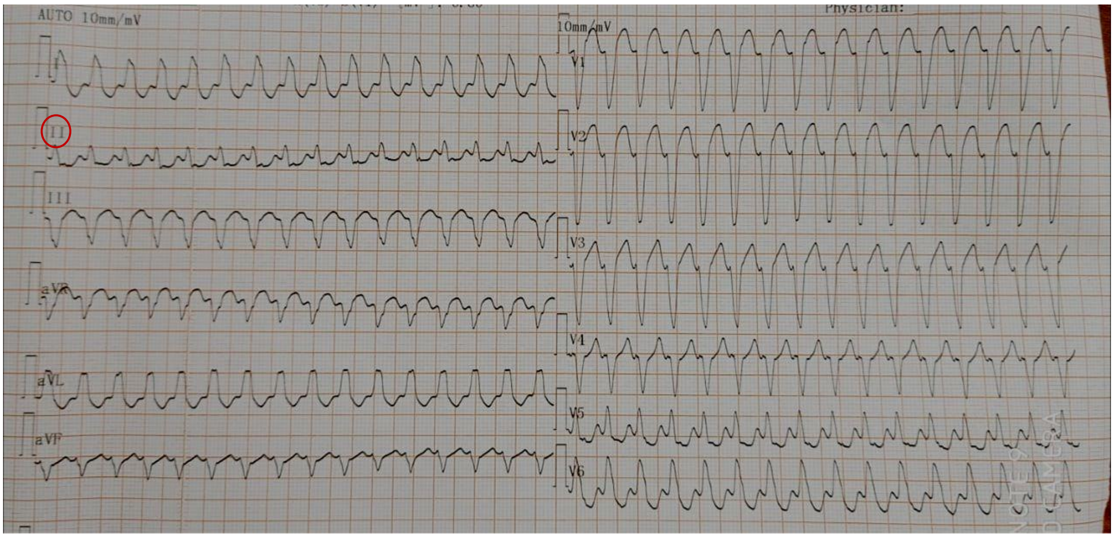
---

2. When and why P-waves are hidden ? (Case based study)

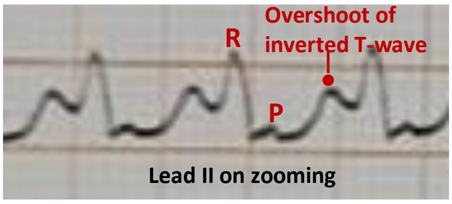
Normally P-wave is the forerunner on ECG , followed by a successive sequential display : PR interval-QRS-ST segment-T wave .

At times the P-wave loses its forerunner position on ECG and then it becomes difficult to be detected .

- **First case study :** 40 years male with the complaint of recent palpitation
  Sometimes with short RP tachycardia , the P-wave may be hidden within the preceding QRS complex and thus it is non-visible in most of the leads except in the lead II having the least electrical deflection as per Haystack principle.



Source : Global Heart Rhythm Forum on 21st Dec , 2024 by Dr. Jeffrey Alex , Senior Consulting Physician and Cardiologist



Short RP tachycardia with accompanying LBBB.
(The presence of positive P in lead II excludes the possibility of AVNRT) Most likely AT with 1:1 LBBB (aberration/prexisting one) Short RP tachycardia here is visible in lead II having the smallest electrical deflection , satisfying the 'Haystack principle'.

- **Second Case Study :** Illustrating the 'Haystack Principle' in a patient with unexplained syncope.

Source : "Reproduced from Sinha SK, Sharma AK, Razi M, Pandey U. 'Haystack Principle - Its Importance in Unearthing the Electrocardiogram in a Patient with Unexplained Syncope.' Int J Clin Cardiol. 2020;7:208. DOI: 10.23937/2378-2951/1410208." **(Open Access Article)**

History : "A 70 years old , hypertensive male presented to outdoor patient department for evaluation of dizziness and syncope. His blood pressure before initiation of treatment was 160/90 mmHg in left arm in supine position. He was receiving amlodipine 10 mg and hydrochlorrhiazzide 12.5 mg. His blood pressure was 138/78 mmHg. Postural Hypotension was ruled out. Other physical examinations were unremarkable".

As his symptoms were classical of cardiac syncope , ECG demanded a careful look

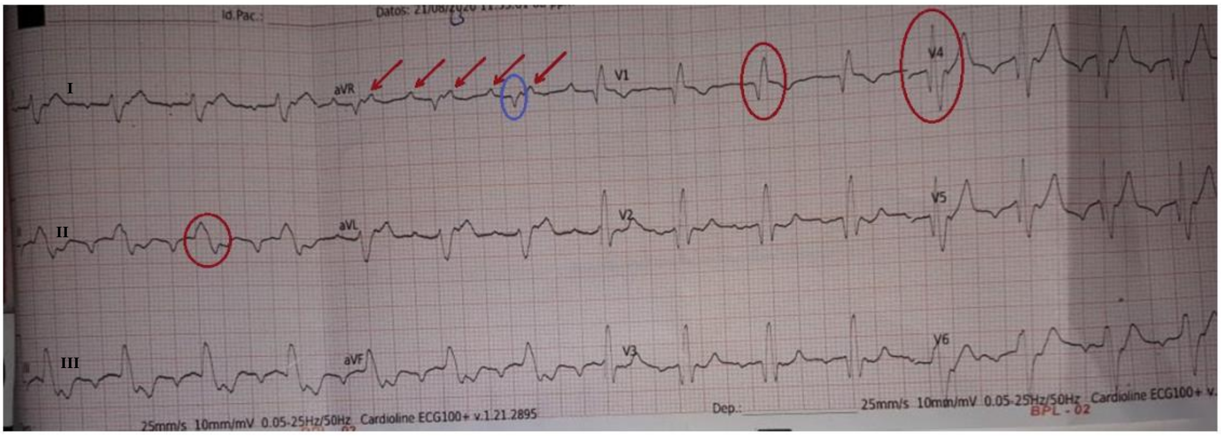


Figure 1: The rhythm strips (lead II, V1, and V4) showed RBBB pattern with bizarre QRS (red circle). Lead aVR provided a conclusive evidence of 2:1 atrioventricular (AV) conduction block which was of Mobitz type II. Every alternate P wave was nonconductive (red arrow). Blue circle represents QRS complex in lead aVR.

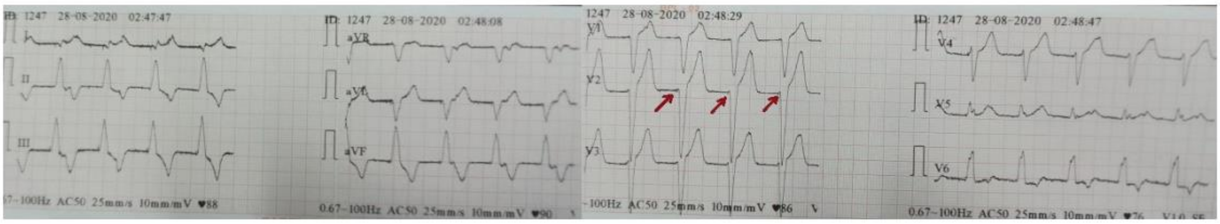


Figure 2: Pacing electrocardiogram showing left bundle branch morphology. Red arrow represents pacing spike.

ECG analysis : "ECG revealed RBBB pattern with rate of 77 per minute. The top tracing rhythm strips (lead II, V1, and V4) showed RBBB pattern but QRS was bizarre (red circle). P was inverted and giving an impression of first degree heart block. However, T wave morphology was abnormal and wide. As his symptoms were classical of cardiac syncope, ECG demanded a careful look. Lead aVR provided a conclusive evidence of 2:1 atrioventricular (AV) conduction block which was of Mobitz type II. Every alternate P wave was nonconductive (red arrow). Blocked P waves were concealed in QRS complexes in lead I and in T waves in II, III, and V2-V6. It was best discernible in lead aVR only as ventricular complexes were smallest in this lead. This was the reason which made the P wave best visible in lead aVR which followed "the haystack principle"

A permanent pacemaker was inserted and the patient was discharged in stable condition with appropriate follow up advice. .

3. Discussion

The 'Haystack principle' is not a ECG theorem but rather it is a metaphor that describes the challenging searching out of small but crucial piece of information (the needle) hidden within a vast internet of distracting data (the haystack). This principle can be of great diagnostic importance when one is searching out for hidden P-wave on ECG through the lead having the least electrical deflection. Mastering this principle enhances decision-making with improved outcome.

The phrase "finding a needle in a haystack" is a very old metaphor and has been used to describe nearly precise searches with hidden P-waves.

Dr. Henry Marriott (1917-2007) also emphasized the significance of Haystack priciple and quoted his concept in his book of Practical Electrocardiography by saying that " The haystack principle" can be of great diagnostic importance when you are searching for difficult-to-find P waves. When you have to find a needle in a haystack , you would obviously prefer a small haystack. Therefore, whenever you are faced with the problem of finding elusive items, always give the lead that shows the least disturbance of the ECG baseline (the smallest ventricular complex) a chance to help you".

Ref : Marriott's Practical Electrocardiography , 12th South Asian Edition , Page No. 510

4. Take Home Message

- P-waves are most easily seen in the inferior leads (II , III and aVF) and lead V1 , but at times these P-waves are hidden at its usual sites , then it becomes a challenging task to detect them but its detection is essential as a clue to the underlying diagnosis.
- The 'Haystack principle' is not a ECG theorem but rather it is a metaphor that describes the challenging searching of small but crucial piece of information (the needle) hidden with a vast internet of distracting data (the haystack).
- This principle can be of great diagnostic clue when one is searching out for such hidden P-waves. The basic principle is based on the fact that when one has to find a small needle in a haystack , he would obviously prefer small haystack for the purpose. Therefore , whenever one is faced with the problem of finding hidden P-waves , the attention should be directed to the lead having the least electrical deflection on the ECG. The searching out these hidden P-waves may reveal even the alarming signals which would have been otherwise missed.
- The application of this principle in ECG interpretation enables a clinician to enhance his decision-making quality with improved outcome.
- This article introduces the "Haystack Principle" in the context of interpreting ECG , illustrating how selecting specific ECG leads with smallest electrical deflection can help in identifying hidden P-waves (with two case studies this fact has been well explained on page 2 and 3)

5. References

1. Marriot's Practical Electrocardiography , 12th Edition as South Asian Edition , Page No. 510
2. Haystack Principle - Its Importance in Unearthing the Electrocardiogram in a Patient with Unexplained Syncope
   Santosh Kumar Sinha, FACC, FAESC, FSCAI , Awadesh Kumar Sharma , Mahmodullah Razi and Umeshwar Pandey
   Int J Clin Cardiol. 2020;7:208. DOI: 10.23937/2378-2951/1410208." (Open Access Article)
   https://clinmedjournals.org/articles/ijcc/international-journal-of-clinical-cardiology-ijcc-7-208.pdf
3. Wide QRS Complex Tachycardia in the Emergency Setting 2016
   https://aneskey.com/wide-qrs-complex-tachycardia-in-the-emergency-setting/

ELECTRICAL AXIS HYPOTHEIS AND THE RULE OF 90- DEGREE

،

ECG

ELECTRICAL AXIS HYPOTHESIS AND THE RULE OF 90-DEGREE

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction
The credit goes to Dr. William Einthoven who had inscribed a 'Triaxial Bipolar System' with three leads as standard leads I , II and III and accordingly the concept of manifest potential difference in the heart came first to his mind.

Electrical axis hypothesis and the rule of 90-degree
If the exploring electrode is within the 90-degree of cardiac electrical axis , it would produce positive wave with R-wave dominance and vice-versa is also true if the electrode is beyond 90-degree. If the electrode is exactly at 90-degree to the cardiac electrical axis, it would produce the equiphasic wave or no deflection.

Determination of Electrical axis as per rule of 90-degree
STEP 1 : QUARDANT DETERMINATION
STEP 2 : APPROXIMATION METHOD

Take Home Message

References

Electrical axis hypothesis and the rule of 90-degree

Research Hypothesis

© **DR. D.P. KHAITAN**
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

Every dynamic system is having its own axis – the reference point around which it takes its motion . Deviation in its expected axis is often the first pointer to the nature of its underlying forces at play. This quantitative shift tells the story of homeostatic disturbances in its territory.

The heart being the biological dynamic system behaves in the same way , it sends its message through the electrical waves in its territory , the interpretation of which paves the way of its axis determination.

- ☐ **The cardiac axis reflects the net direction of the heart's electrical depolarization , summing all individual vectors into a single dominant flow.**
- ☐ **ECG axis determination is about translating electrical impulses into meaningful clinical insight through a mathematical number.**

Clinicians determine the cardiac electrical axis with the concept – whether the heart is sending its message of electrical spread through its normal axis or it is spreading the waveforms of pathophysiological changes by shifting its axis.

1. Introduction (Keypoints)

- The heart is the supremo source of electromagnetic flow over the human body , the concept of which possibly compelled the mind of Einthoven to think in term of a hypothetical electrical equilateral triangle , which is surrounded by the right arm and left arm from above and left leg from below as an extension of its electromagnetic field. The heart with zero potential is considered to be at the centre of this triangle.
- Before understanding the rule of 90 degree in context with ECG axis determination , it would be worthwhile to discuss how the cardiac electrical axis came into existence – The credit goes to Dr. William Einthoven who had inscribed a '**Triaxial Bipolar System**' with three leads as standard leads I, II and III and accordingly the concept of manifest potential difference in the heart came first to his mind.

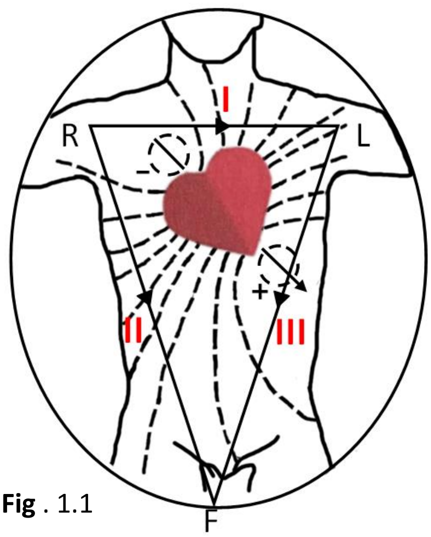
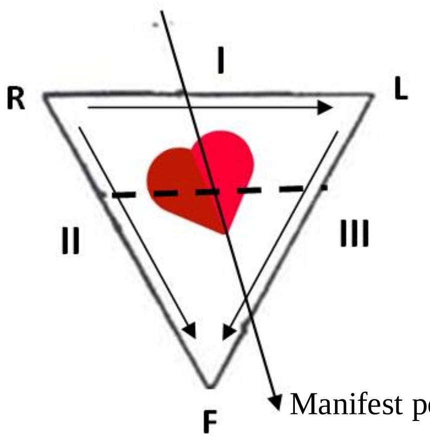


Fig . 1.1



R = Right shoulder
L = Left shoulder
F = Left foot (originally midpoint of the pubis was chosen by Einthoven as left foot representative)

Fig . 1.2

The manifest potential difference in the heart – His remarkable concept of biophysics set forth a new concept first time in the history of ECG. Einthoven inscribed the famous equilateral triangle formed by leads I, II and III at its side and derived a very important calculation what he called ‘ the manifest potential difference in the heart’ , it can be equated to the Electrical axis.

A new concept of electrical vector came into existence :

To his statement “ **The curve must represent under all circumstances and in every moment, the algebraic sum of all the potential differences which at that moment are developed in the heart**. .” His this concept might have hinted and compelled the minds of future researchers to frame the concept of electrical axis having a definite magnitude and direction.

NB :
If the exploring electrode is placed at any point on the body surface beyond 15 cm from the central position of the heart , there is hardly any noticeable effect on the intensity of the current* **.** The points of electrodes placement on the three limbs used by Einthoven were having somewhat longer distance , each more than 15 cm away from the centre of the heart. **By considering this fact the triangle is assumed to be equilateral in nature**

Ref :
* LeoSchamroth – An Introduction to ELECTROCARDIOGRAPHY - EIGHTH ADAPTED EDITION – Basic Principles – Page 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 ’).

- **The concept of hexaxial lead system**

  By combining together Einthoven triaxial lead system (I, II & III) with that of Goldberger’s lead system (aVR , aVL & aVF) it gives birth to the hexaxial lead system – the intersection of leads I , II & III is allowed to pass through the centre of so framed hexaxial lead system and leads aVR , aVL & aVF are considered to be projected from the centre to the corresponding angles. **This hexaxial lead system is thus the display of the six limb leads over the vertical plane , as illustrated below :**

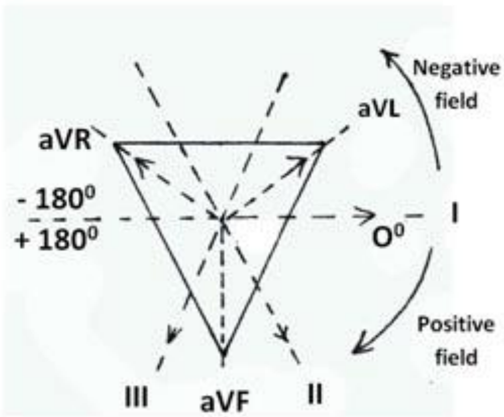


Fig . 1.3

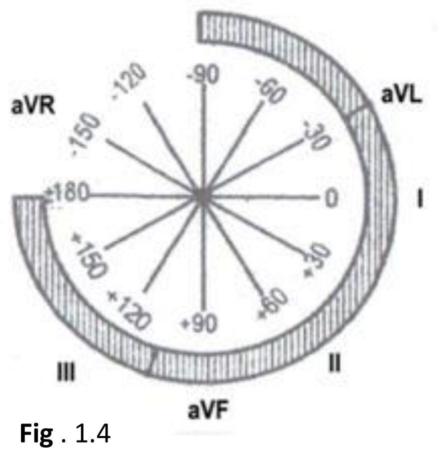


Fig . 1.4

2. Electrical axis hypothesis and the rule of 90-degree

The endless labour of **EINTHOVEN** opened the beginning of a new chapter in the history of electrocardiogram – the delivery of his lecture in 1912 addressing at the Chelsa Clinical Society in London describing the important principle – 'The manifest potential difference in the heart' in relation with his equilateral electrical triangle (Later on it was known as Einthoven's triangle). Unfortunately he died in the year 1927 , soon after being awarded with Nobel Prize in 1924 for his discovery of the Mechanism of Electrocardiogram. In 1942 EMANUEL GOLDBERGER, cardiologist of Lincoln Hospital, New York invented unipolar limbs system (aVR aVL and aVF) , which facilitated the formation of hexaxial lead system. **The author of this article feels a dire necessity of explaining this rule which can easily help in the determination of ECG axis in the light of hexaxial lead system – this can be addressed as the rule of 90-degree.** This also helps in understanding how the shape of a wave is assumed as per the rule of 90-degree.

Basic principle : Electrical axis hypothesis and the rule of 90-degree (also see the next page)

- ☐ The cardiac axis is the sum of all depolarization units of the heart and it is having the maximum flow of current along its axis , having a definite magnitude and direction .
- ☐ In any given electromagnetic field the voltage of any waveform depends upon its relative proximity to the electrical axis - the more nearer is the wave to the electrical axis , the more will be its voltage intensity being reflected by its amplitude.

By considering the above facts , the concept of **electrical axis hypothesis** came into existence – electrical axis is an electrical field vector and the voltage recorded in a particular lead is the result of angulation in between cardiac electrical axis and the lead vector itself. The amount of flow of the current through a particular lead is inversely proportional to the angulation in between cardiac vector and lead vector. The whole concept has been illustrated as below :

Lead vector is inversely propotional to angulation in between cardiac vector and lead vector itself

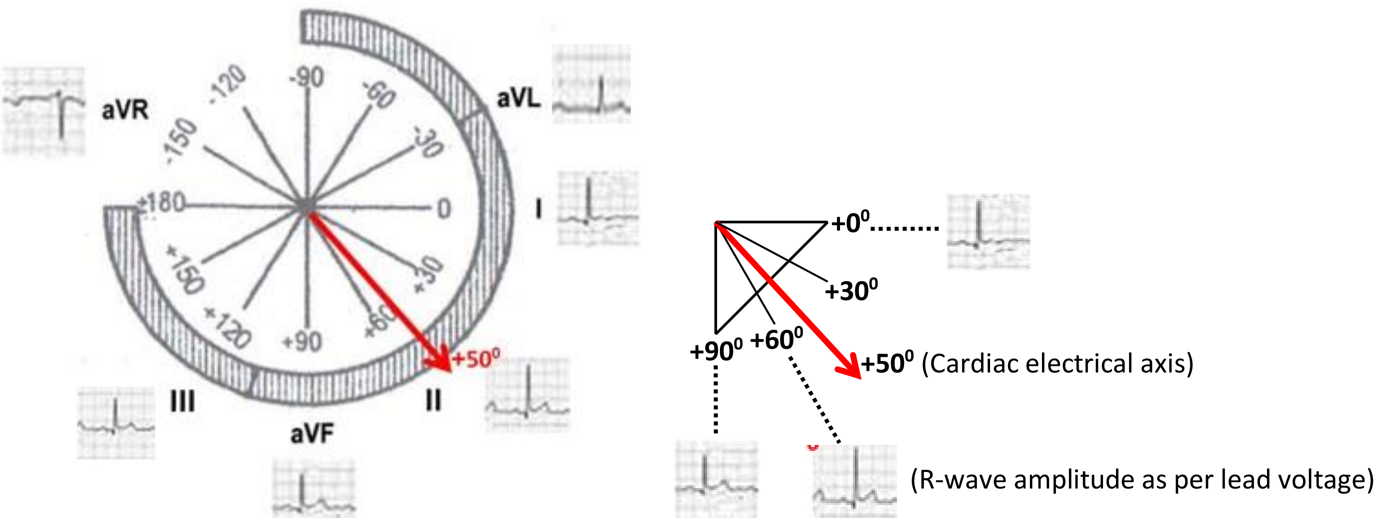


Fig . 1.5

- ☐ If the exploring electrode is within 90-degree of cardiac electrical axis , it would produce positive wave with R-wave dominance and vice-versa is also true if the electrode is beyond 90-degree. If the electrode is exactly at 90-degree to the cardiac electrical axis , it would produce the equiphasic wave or no deflection.

As per rule of 90 degree :

If an exploring electrode is within 90° of the CEA → Positive wave (R wave dominance)

If an electrode is exactly at 90° to the CEA → Equiphasic (R = S) or no deflection

If an electrode is beyond 90° from the CEA → Negative wave (S wave dominance)

CEA = Cardiac Electrical Axis

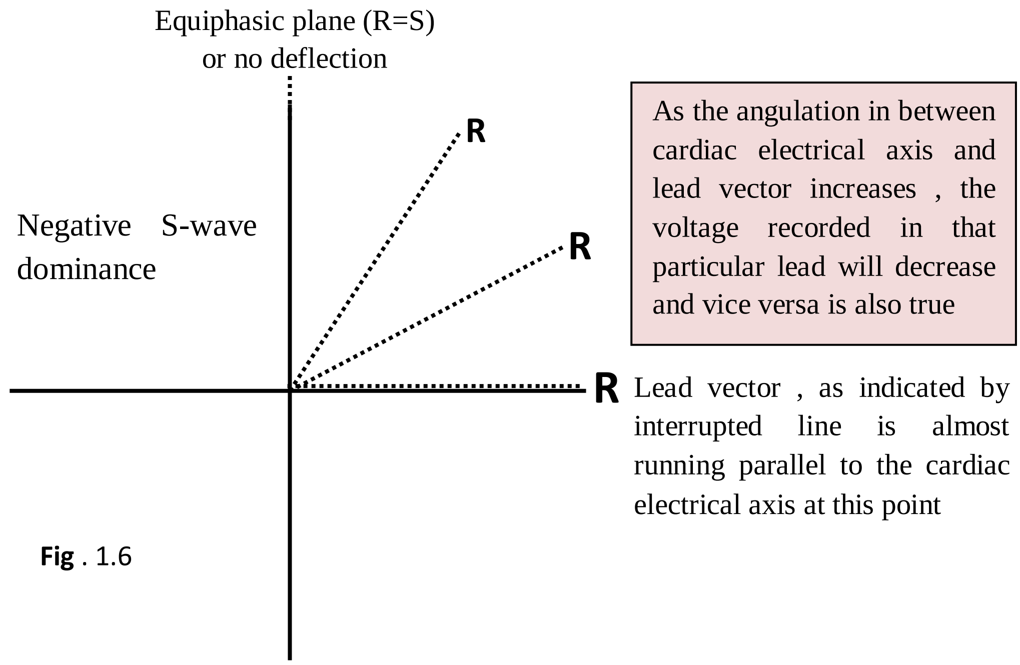


Fig . 1.6

3. Determination of electrical axis as per rule of 90-degree

All the steps described below are based on the rule of 90^0

STEP 1 : : QUADRANT DETERMINATION

Leads I and II are chosen for the purpose since they cover the normal range of QRS axis (-30^0 to $+90^0$) - QRS positivity in between these two leads indicates normal axis and deviation on either side indicates left axis and right axis deviation respectively.

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.

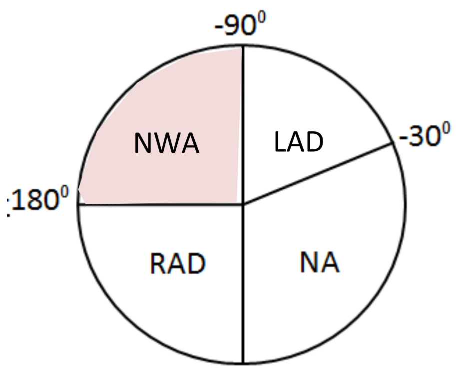


Fig . 1.7

The site of quardant determination as the first step imparts the basic clue where the QRS axis is lying.

STEP 2 : APPROXIMATION METHOD

Once the quadrant identification is completed , the next step would be adopted to arrive to the nearest multiple of 30 degree – the smallest range of angulation in between two leads of hexaxial lead system)

- ☐ Find the lead with the most positive QRS → CEA is closest to this lead axis.
- ☐ Find the equiphasic lead → The CEA is perpendicular (90°) to this lead.
- ☐ The miscellaneous way of calculation QRS axis

☐ **Find the lead with the most positive QRS → CEA is closest to this lead axis.**

- It may be found within the nearest range of 30^0 on the side , closest to the lead having the most positive QRS. Cardiac electrical axis is found to be tilted towards the lead having R-wave with more amplitude.
- The net positivity in any lead is obtained by subtracting the smaller deflection from larger deflection (qR = R - q ; Sr = S - r ; Rs = R - s) .
- Thus on reaching to the nearest multiple of 30 degree , the electrical axis would be in midway of both 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.**

☐ **Find the equiphasic lead → The CEA is perpendicular (90°) to this lead.**

Standard lead I is perpendicular to lead aVF.
Standard lead II is perpendicular to lead aVL.
Standard lead III is perpendicular to lead aVR.

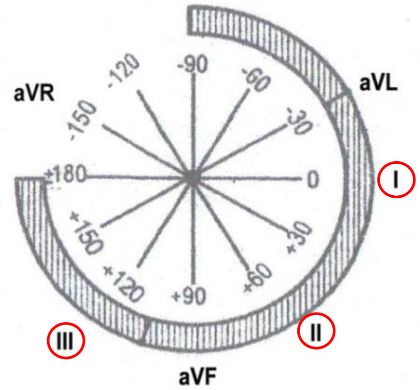


Fig . 1.8

☐ **The miscellaneous way of calculating QRS axis**

The mean QRS axis points in midway between the axes of two limb leads that show relatively tall R waves of equal amplitude.
If all the limb leads (except aVR) are showing predominantly positive wave , the axis lies in between $+30^0$ to $+60^0$.

In nutshell

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) .

4. Take Home Message

☐ The determination of the electrical axis on 12 lead ECG constitutes a very useful diagnostic pointer while interpreting electrocardiogram. And by its analysis one would also be in a position to interpret the pattern of the different waves with respect to its spatial orientation with reference to the hexaxial lead system. The rule of 90-degree imparts a logical and deductive discipline to delineate ECG axis.

If an exploring electrode is within 90° of the CEA → Positive wave (R wave dominance)

If an electrode is exactly at 90° to the CEA → Equiphasic (R = S) or no deflection

If an electrode is beyond 90° from the CEA → Negative wave (S wave dominance)

CEA = Cardiac Electrical Axis

☐ The cardiac axis is the sum of all depolarization units of the heart and it is having the maximum flow of current along its axis , having a definite magnitude and direction.

- In any given electromagnetic field the voltage of any waveform depends upon its relative proximity to the electrical axis - the more nearer is the wave to the electrical axis , the more will be its voltage intensity reflected by its amplitude.
- **Electrical axis hypothesis** is delineated which states that electrical axis is an electrical field vector and the voltage recorded in a particular lead is the result of angulation in between cardiac electrical axis and the lead vector itself. The amount of flow of the current through a particular lead is inversely proportional to the angulation in between cardiac vector and lead vector

5. References

1. A brief review : history to understand fundamentals of electrocadiography - MajdAlGhatrif, MD and Joseph Lindsay, MD (J Community Hosp Intern Med Perspect 2012) – https://pubmed.ncbi.nlm.nih.gov/23882360/
2. A Concise History of the ECG – https://en.ecgpedia.org/wiki/A_Concise_History_of_the_ECG
3. Marriott's Practical Electrocardiography (South Asian Edition - 12th Edition) , P 2 - 43
4. LeoSchamroth An introduction to Electrocardiography (Eighth adapted edition), P 20 -27
5. GOLDBERGER'S CLINICAL ELECTROCARDIOGRAPHY (First South Asia Edition), P 41-49
6. ECG Axis Interpretation , Mike Cadogan and Robert Buttner Nov 17, 2024 https://litfl.com/ecg-axis-interpretation/
7. What is Cardiac Axis ? March 6 , 2011 https://geekymedics.com/what-is-cardiac-axis/
8. The electrical axis of the heart https://ecgwaves.com/electrical-axis-of-the-heart-ecg-physiology-definition/
9. ECG Basics Determining Axis , BySteven Lome, MD https://www.healio.com/cardiology/learn-the-heart/ecg-review/ecg-interpretation-tutorial/determining-axis

QRS-T ANGLE DYNAMICS :
ITS SIGNIFICANCE IN PREDICTING THE CARDIAC HEALTH

ECG

QRS-T ANGLE DYNAMICS : ITS SIGNIFICANCE IN PREDICTING THE CARDIAC HEALTH

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction
A widened QRS-T angle is considered to be vital marker of myocardial heterogeneity and has been linked to an increased risk of ventricular arrhythmias and sudden cardiac death.

Electrophysiology of QRS-T angle dynamics
The normal angle indicates normal alignment while a large angle indicates abnormal alignment , often correlating well with an increased ventricular gradient

Determination of frontal QRS-T angle
The QRS-T angle is calculated using the vector projections of the QRS and T axes on the frontal plane of 12-lead ECG through hexaxial lead system.

QRS-T angle significance in predicting the cardiac health
In cardiology, the QRS-T angle is increasingly recognized as an important marker of ventricular heterogeneity and a potential predictor of morbidity and mortality, related to arrhythmias

Take Home Message

References

QRS-T Angle Dynamics : Its Significance in Predicting the Cardiac Health

A Narrative Review

© **DR. D.P. KHAITAN**
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

I consider the QRS-T angle to be the orchestra of life ; if rhythmic and steady , it points towards a life in harmony. If noisy , it may kill the person with its ugly triumph – the heart's song filters through its melody.

- ☐ **A widened QRS-T angle is considered to be vital marker of myocardial heterogeneity and has been linked to an increased risk of ventricular arrhythmias and sudden cardiac death.**
- ☐ **This is the angle between the axial directions of ventricular depolarization and repolarization , easily calculated on 12-lead electrocardiogram (ECG).**

It is a known fact that the clinical utility of the QRS-T angle lies in its ability to stratify risk in patients with normal and abnormal ECGs , making it a valuable predictor of the cardiac health. That's why , it is not considered just a mathematical anomaly but rather a reflection of underlying pathophysiologal changes in the myocardium.

1. Introduction (Keypoints)

- The calculation of QRS-T angle through the frontal plane of 12-lead ECG provides an insight into the interplay between ventricular depolarization and repolarization. The widening of this angle points towards myocardial heterogeneity. This is considered to be linked with an increased risk of ventricular arrhythmia and sudden cardiac death.
- **The history of this concept started when first time Wilson et al. in year 1934 accessed the value of QRS-T angle through the vectocardiogram analysis** and laid down a concept of 'ventricular gradient'. This ventricular gradient quantifies the overall imbalance between depolarization and repolarization , as shown by wilson with a three dimensional (3D) spatial view through vectocardiogram (VCG).

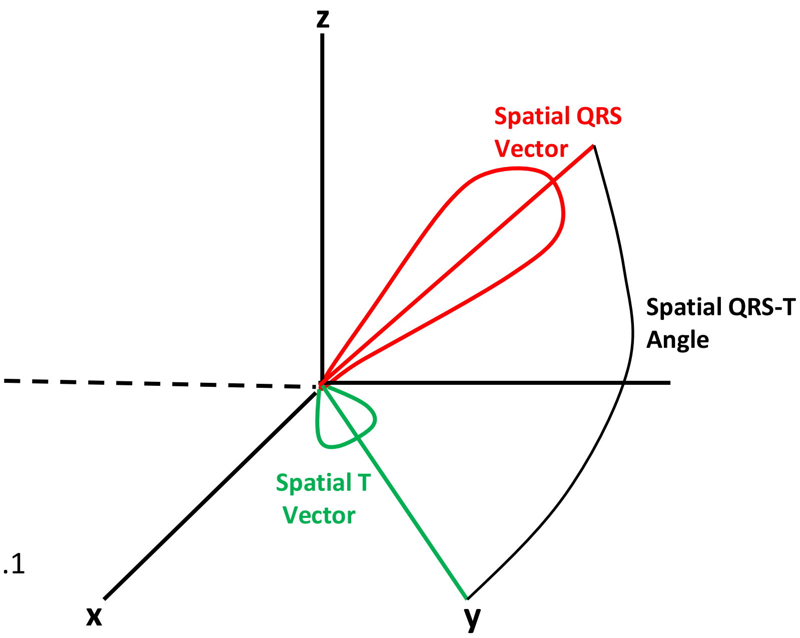


Fig. 1.1

- Thus , the credit of the concept of ‘ventricular gradient’ is accounted to Wilson et al , It is closely related to the QRS-T angle, which measures the spatial alignment or divergence between the QRS axis (direction of depolarization) and the T wave axis (direction of repolarization). A larger angle so produced often correlates with an increased ventricular gradient which may signify abnormal ventricular physiology.

Just to look at three dimensional view of vectocardiogram :

The heart itself is a three-dimensional structure and accordingly, the real-time related electrical activities are reflected onto the three-dimensional space - the orthogonal frontal , horizontal and left sagittal (depth) planes accordingly. Each cardiac cycle is having a isopotential zero plane known as **'Null plane'** - the more or less the circular plane keeping the positive and negative field within the torso in balance. This plane lies perpendicular to the vector loops created at the centre of the volume conductor.

The three inscribed loops of atrial depolarization, ventricular depolarization and ventricular repolarization are suspended in three dimensional space , as illustrated below :

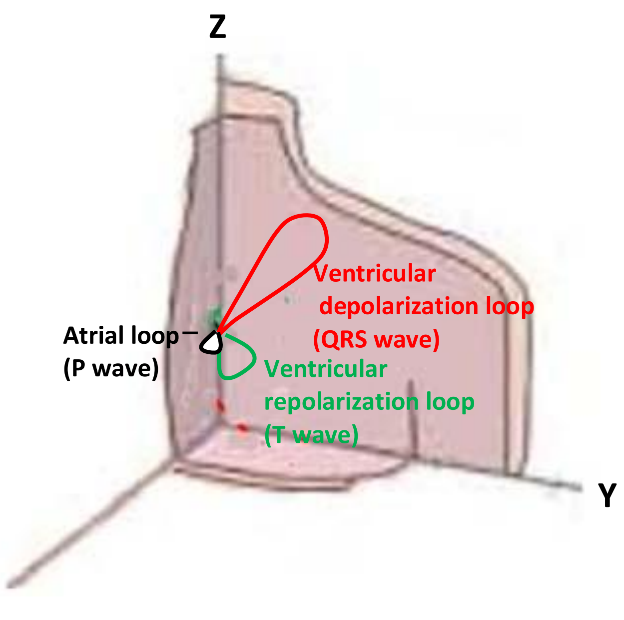


Fig. 1.2

- The spatial QRS-T angle as calculated by the vectocardiogram is a complex process , and its measurement is not familiar to most of the clinicians and it is not available routinely on the computerized electrocardiographic analysis software currently in use. **With 12-lead ECG , it is easier to calculate frontal QRS-T angle. It has been demonstrated to have a good correlation with the spatial QRS-T angle for the same purpose**. It is also important to bear in mind that 12-lead ECG remains inexpensive , non-invasive tool , easy to operate with a quick result and clinicians are well-versed with the operation of this non-invasive process.

- The QRS-T angle is proved to be associated with the increased risk of morbidity and mortality in several cardiac clinical situations. It is stronger than any of the classical cardiovascular risk factors and ECG risk indicators and provides additional value to them in predicting fatal cardiac events.

2. Electrophysiology of QRS-T angle dynamics

The keypoints involved in QRS-T angle understanding are as follows :

- QRS Axis: Represents the average direction of ventricular depolarization.
- T Wave Axis: Represents the average direction of ventricular repolarization.
- QRS-T Angle: The angular difference between these two axes on the frontal plane. The normal angle indicates normal alignment while a large angle indicates abnormal alignment , often correlating well with an increased ventricular gradient.

In other words , QRS-T angle represents the phase relationship between depolarization (QRS) and repolarization (T). It gives insights into how alignment or misalignment of these two wavefronts operate through the heart. A small QRS-T angle indicates normal alignment, meaning the processes of depolarization and repolarization are working in harmony.

✓**How does QRST angle dynamic describes the temporal disparity in the electrical activity of the ventricle** :

One has to concentrate upon the following points for the purpose :

- **The process of repolarization starts from the point where the depolarization wave QRS gets ended** . This is worthwhile to mention here that the last portion of ventricular depolarization is the posterobasal portion of the left ventricle (including the pulmonary conus and the uppermost part of the interventricular septum). That's why , the wave of repolarization starts from this posterobasal portion of the left ventricle toward the epicardial surface by propagating ahead leftward and downwards causing upward T wave in a similar positive direction as that of QRS complex.
- **Epicardial cells are normally having a shorter duration action potential than endocardial cells**. This shorter duration action potential causes epicardial cells to repolarize earlier.

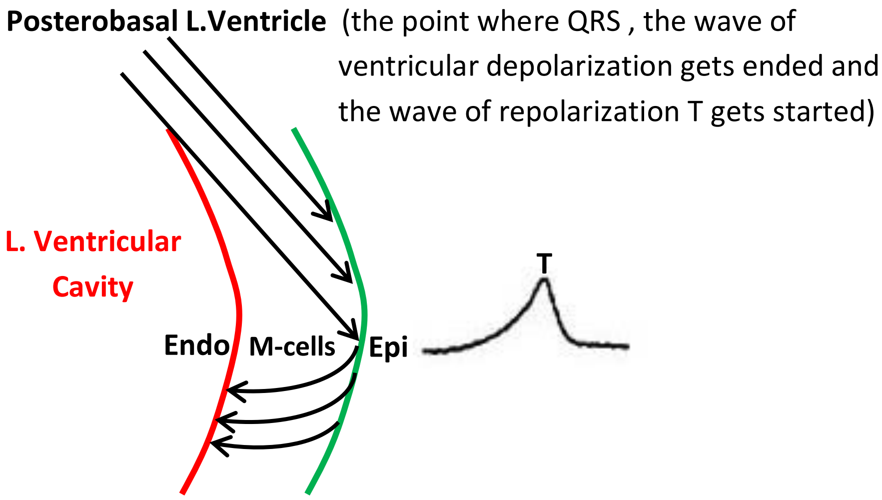
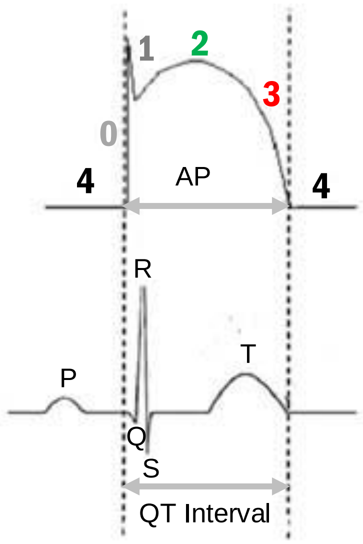
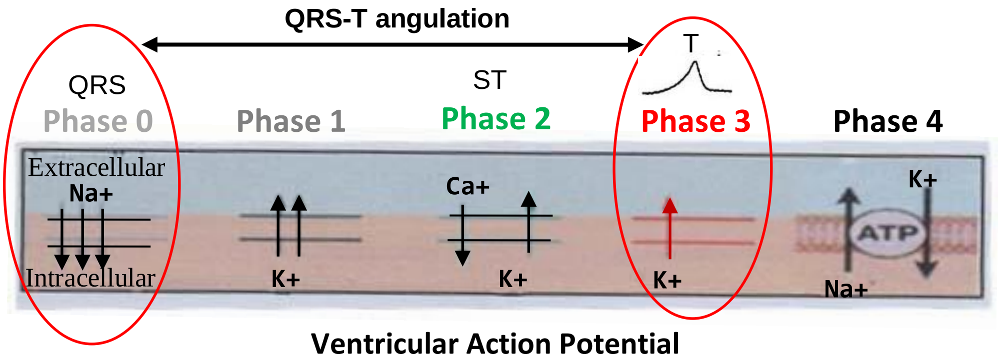


Fig. 1.3

□ The electrophysiological reason of angulation in between QRS and T waves

A. The phase 2 (systolic phase) lies in between the phase of depolarization (phase 0) - QRS and the phase of repolarization (phase 3) - T
B. Endocardial cells repolarization are delayed , since it is in contact with high intraventricular pressure of systolic phase





QRS ⟶ Isoelectric ST segment (systolic phase) ⟶ T-wave

There is a time lag in between QRS and T-wave due to
- The presence of systolic phase 2 (ST segment) in between QRS and T
- The delayed activation of the endocardial cells due to the intervening systolic phase

Fig. 1.4

NB :
"Repolarization begins when the inner membrane of the heart is exposed to high pressure. This delays the recovery of endocardial action potentials, producing a time difference between epicardial repolarization and endocardial repolarization, the ventricular gradient, which will alter the spatial QRS-T Angle. Ventricular gradients are typical of healthy myocardium".

Ref : QRS-T angle as a predictor of pulmonary arterial hypertension: A review
Li, Bo PHD[a]; Liu, Xuhan MD[a]; Wang, Baoguo MD[a]; Liu, Xiuqing MD[a]; Zhang, Weihua MD[a,*]
2023
https://journals.lww.com/md-journal/fulltext/2023/01130/qrs_t_angle_as_a_predictor_of_pulmonary_arterial.34.aspx

---

This would be worthwhile to mention here that any pathological entity involving either the subendocardial region or exposing the ventricle to high pressure would delay the recovery of endocardial cells , inducing a time-lag between epicardial repolarization and endocardial repolarization , this impact would be reflected in the form of increased ventricular gradient with myocardial dispersion. Therefore , the steepness of the descending limb of T-wave is somewhat prolonged and this prolongation attributes mainly to the prolongation of QRS-T angle. The pathological entities like myocardial ischemia , myocardial infarction and left ventricular failure are main contributing factors in this concern.

There are other factors as well , which can alter the QRS-T angle :

Structural Changes: Left ventricular hypertrophy, myocardial scarring, or dilatation.
Metabolic and Electrolyte Imbalances: Hypokalemia or hyperkalemia can affect repolarization.
Medication Effects: Drugs like antiarrhythmics or QT-prolonging agents may influence the QRS-T angle.
Conduction Abnormalities: Bundle branch blocks (e.g., left bundle branch block) often widen the QRS-T angle.

3. Determination of frontal QRS-T angle

The QRS-T angle is calculated using the vector projections of the QRS and T axes on the frontal plane of 12-lead ECG through hexaxial lead system.

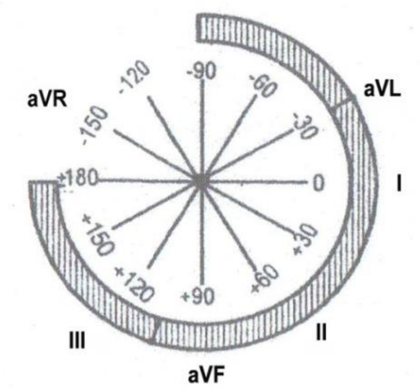
Basic principles :

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 QRS axis is determined by measuring the larger deflection of the QRS complex on the frontal plane
- ☐ The T-wave is similarly calculated
- ☐ The angle between these two axes is the QRS-T angle.

The following points should be considered while calculating the QRS and T axes

- The mean QRS axis points in midway between two limb leads that show relatively tall R waves of more or less equal amplitude. And if one deflection is having more net positivity , the axis should be considered to be tilted towards that lead by the range of $10^{0.}$



Hexaxial Lead System

Fig. 1.5

- The net positive deflection is obtained by subtracting the smaller deflections from the negative ones (qR = R - q ; Sr = S - r ; Rs = R - s)
- The other way of calculating mean QRS axis : it refers at 90^0 to the equiphasic deflection obtained with any limb lead.
- The T-wave axis can also be determined by following the same process as that of QRS axis.

Then it becomes easier to graph the mean electrical axis (QRS/T) 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)

Normal QRS-T value

Historically , upper limit of the normal QRST-angle has been considered in between 45^0 and 60^0 but now-a-days QRS-T axis is categorized as normal if $\leq 90^0$ or abnormally widened $\geq 100^0$ in context with the present article.

4. QRS-T angle significance in predicting the cardiac health

In cardiology, the QRS-T angle is increasingly recognized as an important marker of ventricular heterogeneity and a potential predictor of morbidity and mortality, related to arrhythmias. However, to use the QRS-T angle effectively as a predictive value, it is essential to recognize that it is not a standalone indicator and must be interpreted in the context of other clinical factors. This includes excluding other coexisting conditions that may influence the QRS-T angle (please see page 20 for such co-existing condition). Studies have demonstrated its predictive value for :

- **Sudden cardiac death** (SCD) :
- **All-cause mortality**: A wide QRS-T angle is associated with a nearly three-fold increased risk of all-cause mortality.
- **A marker of increased arrhythmic risk with adverse outcome** , particularly in condition like ischemic heart disease , cardiomyopathy and heart failure.
- Detection of Myocardial Ischemia and Infarction: A deviation in the QRS-T angle can signify underlying ischemic changes or myocardial infarction, even in asymptomatic patients.
- Poor prognosis : It can indicate a worsening heart failure state or a higher risk of mortality due to arrhythmic events.
- Pulmonary hypertension **:** Changes in the QRS-T angle can predict pulmonary hypertension.
- Monitoring in Chronic Conditions: Patients with hypertension, diabetes, or left ventricular hypertrophy (LVH) often exhibit an altered QRS-T angle, making it a useful marker for early intervention.
- **Post-infarction**: After a myocardial infarction (MI), the scar tissue and altered conduction pathways increase the QRS-T angle. This indicates regions of the heart that are electrically silent or conducting in an abnormal manner. A widened QRS-T angle post-MI is associated with increased mortality and arrhythmic events, particularly ventricular arrhythmias.

The interpretation of the QRS-T angle in these cases would require:

☐ Careful assessment of clinical history, including comorbidities and medications that would contribute to the electrical disturbances.

- ☐ It is critical to exclude other co-existing conditions that may contribute to the widening of QRS-T angle (please see page 20) , including systemic disorders like hypothroidism or infections.

5. Take Home Message

- ☐ The calculation of QRS-T angle through the frontal plane of 12-lead ECG provides an insight into the interplay between ventricular depolarization and repolarization. The widening of this angle points towards myocardial heterogeneity. This is considered to be linked with an increased risk of ventricular arrhythmia and sudden cardiac death.
- ☐ The normal angle indicates normal alignment while a large angle indicates abnormal alignment , often correlating well with an increased ventricular gradient. In other words , QRS-T angle represents the phase relationship between depolarization (QRS) and repolarization (T). It gives insights into how alignment or misalignment of these two wavefronts operates through the heart. A small QRS-T angle indicates normal alignment, meaning thereby . the processes of depolarization and repolarization are working in harmony.
- ☐ Historically , upper limit of the normal QRST-angle have been considered in between 45^0 and 60^0 but now-a-days QRS-T axis is categorized as normal if $\leq 90^0$ or abnormally widened $\geq 100^0$ in context with the present article.
- ☐ QRS-T angle significance in predicting the cardiac health (please see page 21 for more details)
    - Careful assessment of clinical history, including comorbidities and medications that would contribute to the electrical disturbances.
    - It is critical to exclude other co-existing conditions that may contribute to the widening of QRS-T angle (please see page 20) , including systemic disorders like hypothroidism or infections.

6. References

1. The diagnostic utility of the basal and post-exercise QRS-T angle in patients with stable coronary artery disease
   Tufan Günay MD, Alper Karakus MD
   First published: 11 August 2023
   https://onlinelibrary.wiley.com/doi/10.1111/anec.13082

2. QRS-T angle as a predictor of pulmonary arterial hypertension: A reviewLi, Bo PHDa; Liu, Xuhan MDa; Wang, Baoguo MDa; Liu, Xiuqing MDa; Zhang, Weihua MDa,* 2023

https://journals.lww.com/md-journal/fulltext/2023/01130/qrs_t_angle_as_a_predictor_of_pulmonary_arterial.34.aspx

3. Spatial QRS-T angle predicts cardiac death in a general population
Isabella Kardys, Jan A Kors, Irene M van der Meer, Albert Hofman, Deirdre A.M van der Kuip, Jacqueline C.M. Witteman
01 July 2023
https://academic.oup.com/eurheartj/article-abstract/24/14/1357/501772?redirectedFrom=fulltext

4. T wave axis deviation and QRS-T angle – Controversial indicators of incident coronary heart events 2017
Author links open overlay panelPolychronis Dilaveris MD, Christos-Konstantinos Antoniou MD, Konstantinos Gatzoulis MD, Dimitrios Tousoulis MD
https://www.sciencedirect.com/science/article/abs/pii/S0022073617300559

5. QRS-T Angle: A Review 2016
Andrew Oehler 1, Trevor Feldman 2, Charles A Henrikson 2, Larisa G Tereshchenko 2,
https://pmc.ncbi.nlm.nih.gov/articles/PMC4237708/

6. Wide QRS-T angle on the 12-lead ECG as a Predictor of Sudden Death beyond the LV Ejection Fraction April 2016
https://www.researchgate.net/publication/301550604_Wide_QRS-T_angle_on_the_12lead_ECG_as_a_Predictor_of_Sudden_Death_beyond_the_LV_Ejection_Fraction

7. QRS-T angle as a predictor of sudden cardiac death in a middle-aged general population 2012
Aapo L. Aro1,2*, Heikki V. Huikuri3, Jani T. Tikkanen3, M. Juhani Junttila3, Harri A. Rissanen4, Antti Reunanen4, and Olli Anttonen
https://academic.oup.com/europace/article-pdf/14/6/872/7277733/eur393.pdf

8. Prognostic Value and Temporal Behavior of the Planar QRS-T Angle in Patients With Nonischemic Cardiomyopathy
Behzad B. Pavri, MD, Matthew B. Hillis, MD, Haris Subačius, MA, Genevieve E. Brumberg, MD, Andi Schaechter, BSN, RN, Joseph H. Levine, MD, Alan Kadish, MD,
24 June 2008
https://www.ahajournals.org/doi/10.1161/circulationaha.107.733451

CARDIAC FOOTPRINTS :
IDENTIFYIG ARVD ON ECG

CARDIAC FOOTPRINTS : IDENTIFYING ARVD ON ECG

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction

Arrhythmogenic Right Ventricular Dysplasia (ARVD is a form of genetically induced cardiomyopathy , being characterized by progressive replacement of right ventricular myocardium with fibrofatty tissue.

Arrhythmogenic Substrate in ARVD

- ☐ A basic concept of desmosome
- ☐ Genetic mutations
- ☐ Destablization of cardiac myocytes with its pathological changes
- ☐ Changes at the molecular level (dysregulation of Ca^{++})

Electrocardiographic changes (including the concept of Fontaine leads)

ECG-related findings as a part of the diagnostic evaluation through 'Task Force criteria'

Take Home Message

References

Cardiac Footprints : Identiying ARVD on ECG

A Narrative Review

© DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

The tapestry over a cloth looks to be artistic but when its weaving tends to feather away , it tells the story of its demise with the feeling "Ah"...

The same is true with ARVD (Arrthymogenic Right Ventricular Dysplasia) where the fibres of unity of myocardium mainly in the territory of right ventricle are lost into discordance.

- ☐ **The architectural display with ARVD is the fibrofatty replacement of mainly the right ventricular (RV) myocardium**
- ☐ **The ECG writes down with its pen the electrical instability in the form of some characteristic waves – Epsilon wave , which tells the story of its fragility**

The arrhythmogenic dance of ARVD may kill the patient , emphasizing the need for precision in its diagnosis and the clinicians being dictated by clinical history focus their first attention on ECG.

1. Introduction (Keypoints)

- Arrhythmogenic Right Ventricular Dysplasia (ARVD) is a form of genetically induced cardiomyopathy , being characterized by progressive replacement of right ventricular myocardium with fibrofatty tissue.

- At the molecular level , it involves a complex interplay directed at cellular remodelling of the concerned RV , which expresses itself by dysregulation of intracellular calcium (Ca++) handling mechanism.

- Calcium ions play a critical role in the contraction of cardiac myocytes and its dysregulated signalling in ARVD contributes to its arrhythmogenic behaviour.

- The combined impact of basically three elements over right ventricular myocardium is contributing to its very nomenclature as **'Arrhythmogenic Right Ventricular Dysplasia' (ARVD) :**

  - **Arrhythmogenic** , denoting its propensity to develop ventricular arrhythmia.
  - **Right ventricular** myocardium , being its main site of expression
  - **Dysplasia** here points towards fibrofatty infiltration of the right myocardium

  Hence , 'Arrhythmogenic Right Ventricular Dysplasia' compasses all these three elements to express itself as a separate cardiomyopathic entity.

- ARVD causes a cluster of characteristic ECG changes , they dictate more or less the electrical translation of its associated histopathological changes.

ARVD with its main characteristic changes on ECG : (brief review)

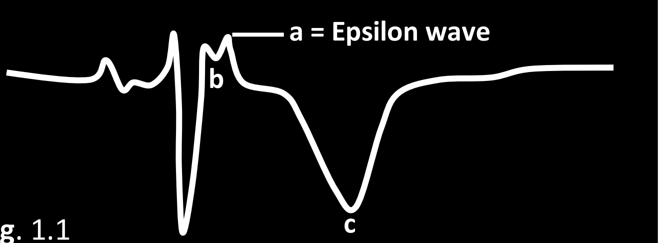


Fig. 1.1

Since architectural fibrofatty changes are mainly confined to RV , right precordial leads V1 through V3 record its associated characteristic changes on ECG :

a = Epsilon wave , a small deflection at the terminal portion of the QRS complex , looking like the shape of the Greek letter (ε) as a subtle wave.

b = Prolonged S wave upstroke

c = T-wave inversion over right precordial leads V1-V3

(The details would be discussed later on)

- ARVD causes symptoms being attributed to ventricular ectopic beats or sustained VT as a result of its arrhythmogenic behaviour. They typically present as palpitation , syncope or cardiac arrest precipitated by exercise.
  Notably to say , here the first presenting symptom may be even sudden cardiac death (there is usually a family history of sudden cardiac death in such cases)
  Over time, surviving patients develop features of right ventricular failure, which may progress to severe biventricular failure and dilated cardiomyopathy.

- The prevalence of ARVD is estimated to be 1 in 1000 to 1 in 5000 people. This disorder is usually remains undiagnosed because it can be difficult to detect in people with mild or no symptoms.

- Teenagers and young adults are more prone to ARVD compared to the old generation , though it is rare in both the groups. It can cause cardiac arrest (SCD) in young athletes.

- Risk involvement : The following patients are at high risk of sudden death if they have any one of the followings :
  - A history of syncope
  - Recurrent arrhythmias not suppressed by anti-arrhythmic drugs
  - A family history of cardiac arrest in first degree relatives

- Other terminology for ARVD :
  - Arrhythmogenic right ventricular cardiomyopathy (ARVC)
  - Right ventricular dysplasia

2. Arrhythmogenic substrate in ARVD

ARVD predisposes the sufferer to the paroxysmal episodes of ventricular arrhythmias and at times sudden cardiac death. It becomes essential to understand its arrhythmogenic substrate in such cases. The resulting arrhythmogenic substrate in patient with clinical ARVD is complex having multiple interrelated factors in its background. The following points are essential to be kept in mind for the purpose :

- ☐ **Normally the cardiac myocytes are adherent to each other by the intercellular disc filaments – known as desmosome**. These desmosomes help in interconnecting cardiac cells with each other , specially when they undergo mechanical stress. Whenever desmosome lose its normal architech , the cells of myocardium get detached from one another and are liable to die. This happens particularly when the heart muscle is placed under stress (e.g. during vigorous exercise)

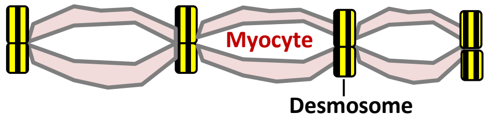


Fig. 1.2

Desmosomes attach cardiac myocytes to each other , providing strength to the myocardium as a whole and playing a pivotal role in sending electrical signaling between neighbouring cells.

Fig. 1.3

Whenever there is altered architect of desmosomes , usually induced by genetic mutations , the cardiac myocytes get detached from each other and are liable to die.

- ☐ **A number of genetic mutations adversely affect the structure and function of these desmosomes.** There have been reported mutations in at least 13 genes. Such genes are known as desmosomal genes because they provide signaling for making its components. By genetic mutations the desmosomal proteins affected include plakophilin, desmoplakin, and desmoglein. When the structure of these desmosomal protein are altered , the cardiac myocytes are prone to be damaged by mechanical stress (eg, from increased cardiac workload as from prolonged exertion).

  There are two patterns of inheritance:
  - Autosomal dominant: Where one parent has the mutation. Family members have a 50% chance of inheriting the abnormal gene.
  - Autosomal recessive: Where both parents have the mutation but no symptoms.

NB : ARVD may also be related to various non-genetic factors , e.g. viral or other inflammatory infections of the heart muscle or even other unknown causes. These factors can get operated independently or in conjunction with genetic predisposition.

☐ **Destabilization of cardiac myocytes brings a series of pathological changes :**

- Apoptosis (program cell death)
- Inflammation , enchanced fibrosis and loss of function of concerned myocytes
- Fatty replacement of myocardium
- Progressive dilatation and dysfunction of the concerned ventricle

The left ventricle is less commonly involved and the septum is relatively spared . and sometime LV involvement may preceed the RV involvement but with poor prognosis. Noteworthy to mention here that these changes usually begin in the epicardium and progress inward (the streched epicardium is the most stressed anatomical region)

☐ **Apart from the factors discussed so far there are certain changes also at the molecular level :**

- Calcium dysregulation with altered calcium handling leading to ventricular arrhythmias
  Abnormalities in calcium-handling proteins like the ryanodine receptor (**RyR2**) and sarcoplasmic reticulum Ca^{2+}-ATPase (**SERCA**) lead to intracellular calcium overload or leakage, which may lead to ventricular arrhythmias through the mechanism of 'delayed afterdepolarization' and reentry mechanism.

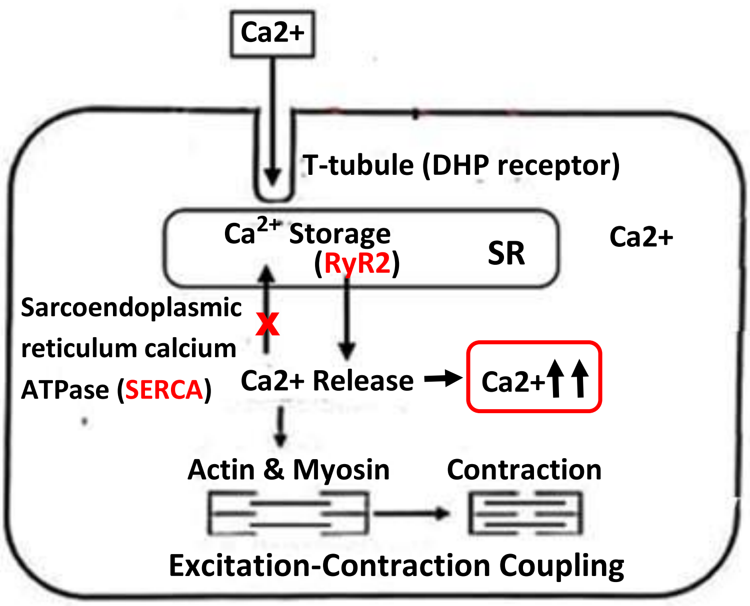


Fig. 1.4

This calcium malhandling may happen before the occurrence of gross structural changes in the myocardium , that's why , ventricular arrrhythmias may be the earlier manifestation.

- Dysregulated intracellular Ca2+ handling is particularly important during exercise, and a maladaptive response may explain the importance of exercise in the phenotypic expression and clinical outcomes in patients with ARVD.

Ref :
Arrhythmogenic Right Ventricular Cardiomyopathy
Andrew D. Krahn MD [a], Arthur A.M. Wilde MD,
PhD [b c], Hugh Calkins MD [d], Andre La Gerche MBBS, PhD [e], Julia Cadrin-Tourigny MD [f], Jason D. Roberts MD, MAS [g], Hui-Chen Han MBBS, PhD [a h]
April 2022
https://www.sciencedirect.com/science/article/pii/S2405500X21010811

---

3. Electrocardiographic changes

The typical ECG pattern of patients with arrhythmogenic right ventricular dysplasia (ARVD) resembles with that of incomplete RBBB with a negative T-wave over the right precordial leads V1 through V3 , with the additional terminal QRS deflection , known as Epsilon wave.

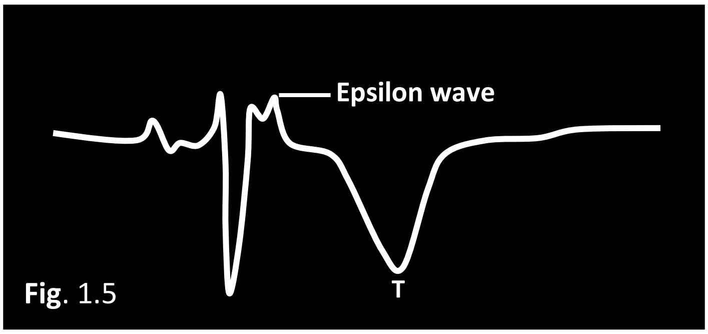


Fig. 1.5

It is not RBBB in true sense but appears so due to prolonged S-wave upstroke , as illustrated with the above sketch (a prototype wave pattern , observed over right precordial leads).

Basic of ECG changes :

- Fibrofatty changes in the right ventricular myocardium , delaying the conduction through the involved right ventricular myocardium
- The terminal late potetential (Epsilon wave) due to the late depolarization of the diseased myocardium
- Abnormal calcium handling and structural disorganization are central to the genesis of ventricular arrhythmias.

The following ECG changes have been observed with the cases of ARVD :

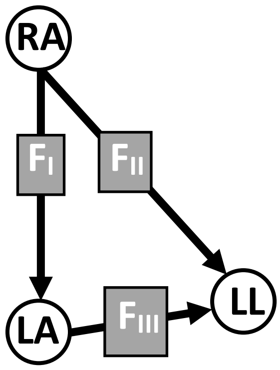
- ☐ T-wave inversion in right precordial leads V1-3 , in absence of RBBB (88% of patients) : this happens so due to the starting point of repolarization from the epicardium , in the opposite direction to that of exploring electrode → T-wave inversion.

- ☐ Epsilon wave (most specific finding) observed in 50% of cases. This Epsilon wave corresponds to a late potential due to delayed depolarization of diseased myocytes. It is probably a marker of advanced disease process.
- ☐ Localized QRS widening in V1-3 (>110 ms)
- ☐ Prolonged S-wave upstroke of $\geq$55 ms in V1-3. It is the most prevalent ECG criterion which correlates well with severity of the disease and induction of VT on ECG.
- ☐ Ventricular ectopy of LBBB morphology, with frequent PVCs > 1000 load per 24 hours
- ☐ Paroxysmal episodes of ventricular tachycardia (VT) with LBBB morphology.

NB : Some clinicians suggest recording of ECG rhythm strip lead II / V1 at double speed (50 mm / sec) and double amplitude (20 mV / mm) to have the detection of the ECG features with more clarity.

Fontaine leads : This would be worthwhile to mention here the significance of fontaine leads. They are specialized leads to increase the sensitivity of Epsilon wave detection.

Fig. 1.6



Leads are placed as shown:

- Right Arm (**RA**) over the manubrium;
- Left Arm (**LA**) over the xiphoid process;
- and Left Leg (**LL**) in the standard V4 position (5th ICS MCL).

Instead of regular leads I, II, and III, there are now three *bipolar* chest leads that are termed FI, FII, and FIII. These record potentials developed in the right ventricle, from the infundibulum to the diaphragm.

4. ECG-related findings as a part of the diagnostic evaluation through 'Task force criteria' for diagnosing ARVD

Here, only ECG-related criteria are mentioned for the purpose.

ECG-Related Major Criteria:

- Epsilon Wave in Leads V1-V3
- Ventricular Tachycardia (VT): Sustained or non-sustained VT of left bundle branch block (LBBB) morphology with a superior axis (negative QRS in leads II, III, and aVF, and positive in lead aVL). This pattern suggests origin from the inferior right ventricle.

ECG-Related Minor Criteria:

- Prolongation of the S-wave upstroke in leads V1-3
- T-Wave Inversions in lead V1-3
- Ventricular Tachycardia (VT): Sustained or non-sustained VT of LBBB morphology with an inferior axis (positive QRS in leads II, III, and aVF, and negative in lead aVL). This pattern suggests origin from the outflow tract of the right ventricle.

These ECG criteria are integral to the comprehensive assessment for ARVD, which also includes imaging studies, histological analysis, and genetic evaluation. The combination of these findings enhances the accuracy of ARVD diagnosis.

5. Take Home Message

- ☐ Arrhythmogenic Right Ventricular Dysplasia (ARVD) is a form of genetically induced cardiomyopathy , being characterized by progressive replacement of right ventricular myocardium with fibrofatty tissue.
- ☐ Calcium ions play a critical role in the contraction of cardiac myocytes and its dysregulated signalling in ARVD contributes to its arrhythmogenic behaviour.
- ☐ ARVD causes symptoms being attributed to ventricular ectopic beats or sustained VT as a result of its arrhythmogenic behaviour. They typically present as palpitation , syncope or cardiac arrest precipitated by exercise.
- ☐ Teenagers and young adults are more prone to ARVD compared to the old generation , though it is rare in both the groups. It can cause cardiac arrest (SCD) in young athletes.
- ☐ Risk involvement : The following patients are at high risk of sudden death if they have any one of the followings :
    - A history of syncope
    - Recurrent arrhythmias not suppressed by anti-arrhythmic drugs
    - A family history of cardiac arrest in first degree relatives

- Abnormalities in calcium-handling proteins like the ryanodine receptor (**RyR2**) and sarcoplasmic reticulum Ca^{2+}-ATPase (**SERCA**) lead to intracellular calcium overload or leakage, which bring about a series of contraction-excitation coupling even leading to ventricular arrhythmias.

- ECG features :

  - T wave inversion in right precordial leads V1-3, in absence of RBBB (88% of patients)
  - Epsilon wave (most specific finding, seen in 50% of patients)
  - Localised QRS widening in V1-3 (> 110ms)
  - Prolonged S wave upstroke of 55ms in V1-3
  - Ventricular ectopy of LBBB morphology, with frequent PVCs > 1000 load per 24 hours
  - Paroxysmal episodes of ventricular tachycardia (VT) with LBBB morphology

- Fontaine leads are specialized leads to increase the sensitivity of Epsilon wave detection

- ECG related findings as a part of its diagnostic evolution through the Task Force Criteria for diagnosing ARVD (please see Page 31)

6. References

1. CHOU'S ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE (Sixth Edition) , P 279-280

2. Arrhythmogenic Right Ventricular Dysplasia (ARVD)
   Robert Buttner and Ed Burns
   Oct 28, 2024
   https://litfl.com/arrhythmogenic-right-ventricular-dysplasia-arvd/

3. Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/ARVC)
   Updated: Jun 10, 2024
   https://emedicine.medscape.com/article/163856-overview?form=fpf

4. Arrhythmogenic Right Ventricular Cardiomyopathy
   (Arrhythmogenic Right Ventricular Dysplasia)
   By L. Brent Mitchell, MD, Libin Cardiovascular Institute of Alberta, University of Calgary Reviewed/Revised Jun 2024
   https://www.msdmanuals.com/professional/cardiovascular-disorders/arrhythmogenic-cardiac-disorders/arrhythmogenic-right-ventricular-cardiomyopathy

5. Arrhythmogenic Right Ventricular Dysplasia , 31 May 2024
   https://www.heart.org/en/health-topics/cardiomyopathy/what-is-cardiomyopathy-in-adults/arrhythmogenic-right-ventricular-dysplasia

6. Arrhythmogenic Right Ventricular Cardiomyopathy
Author links open overlay panelAndrew D. Krahn MD [a], Arthur A.M. Wilde MD, PhD [b] [c], Hugh Calkins MD [d], Andre La Gerche MBBS, PhD [e], Julia Cadrin-Tourigny MD [f], Jason D. Roberts MD, MAS [g], Hui-Chen Han MBBS, PhD [a] [h]
April 2022
https://www.sciencedirect.com/science/article/pii/S2405500X21010811
7. Arrhythmogenic right ventricular cardiomyopathy
https://medlineplus.gov/genetics/condition/arrhythmogenic-right-ventricular-cardiomyopathy/
8. Arrhythmogenic Right Ventricular Dysplasia
https://my.clevelandclinic.org/health/diseases/16752-arrhythmogenic-right-ventricular-dysplasia-arvd

LVH IN DCMP : A PUZZLE ON ECG

LVH IN DCMP – A PUZZLE ON ECG

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction

In dilated cardiomyopathy (DCMP), the left ventricle (LV) enlarges and its contractile function declines. Despite this dilatation, the myocardium often exhibits hypertrophy. This form of hypertrophy differs from that observed with pressure overload conditions such as hypertension or aortic stenosis.

Difference in LVH between DCMP and pressure overload conditions

- In DCMP , the heart exhibits eccentric hypertrophy
- The pressure overload conditions such as hypertension or aortic stenosis exhibit concentric hypertrophy

Pathology of LVH in DCMP

- ☐ Ventricular overload leading to LV dilatation , initially compensated by ventricular stretching as per **Frank-Starling law**
- ☐ Neurohormonal Activation (RAAS & Sympathetic Overdrive)
  - Myocardial hypertrophy as an adaptive mechanism
  - Fibrosis , which contributes to electrical remodelling

Concerned ECG changes with its basic reasoning

Illustration by ECGs

- ☐ **ECG Case 1 DCMP**
- ☐ **ECG Case 2 Degenerative Aortic Valvular Disease plus Diabetic cardiomyopathy (DCMP)**

Take Home Message

References

LVH in DCMP – A Puzzle on ECG

A Narrative Review

© **DR. D.P. KHAITAN**
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

Everyone loves solving puzzles from an early age. Even scientists find joy in unraveling complex puzzles , using their intellect to search out its solutions. It looks to be a very fascinating challenge that captivates the mind and sparks curiosity in everyone.

Dilated cardiomyopathy (DCMP) presents a diagnostic paradox ; the dilated heart exhibits hypertrophy. This discrepancy posses a compelling puzzle in the minds of those clinicians who are very much interested in cardiology and electrocardiography.

- ☐ **In dilated cardiomyopathy (DCMP), the left ventricle (LV) enlarges and its contractile function declines. Despite this dilatation, the myocardium often exhibits hypertrophy**
- ☐ **This form of hypertrophy differs from that observed with pressure overload conditions such as hypertension or aortic stenosis.**

Understanding the concept of left ventricular hypertrophy (LVH) in dilated cardiomyopathy is of paramount importance , as its management differs from that of classical LVH associated with pressure overload conditions.

1. Introduction (Keypoints)

- The basic histopathological changes in DCMP show myofibrillatory loss , accompanied with interstitial fibrosis and cellular infiltrates. Here the left ventricle (LV) is subjected to chronic overload due to systolic dysfunction (weakened contractibility) and increased end-diastolic volume (impaired ventricular ejection).

- Contrary to this dilatation , the myocardium often exhibits eccentric hypertrophy , different from concentric hypertrophy as seen with pressure overload conditions such as hypertension or aortic stenosis

- This eccentric hypertrophy in DCMP presents initially as compensatory phase and this is followed by the decompensatory phase as the heart failure ensues.

- There is a gradually declining left ventricular function due to global myocardial dysfunctioning (ejection fraction <40%).
  Patients usually present with symptoms of biventricular failure , e.g. fatigue , dyspnoea , orthopnoea , ankle oedema , etc.

- The documentation of precise incidence and prevalence rates of LVH in DCMP is lacking in the available literature. This lack of specific data may be due to the complex interaction between myocardial dilatation and hypertrophy in DCMP, further research is necessary for the purpose..

- Causes :

  Genetic

  Upto 25% are familial (primarily autosomal dominant , some types are X-linked)

  Ischaemic

  Dilated cardiomyopathy may occur here following massive anterior STEMI due to extensive myocardial necrosis and loss of contractility

  Other causes :

  - Late autoimmune reaction to viral myocarditis (coxsackie B)
  - Chronic excessive alcohol intake including even cocaine.
  - Some anti-neoplastic agents (doxorubicin , trastuzumab , imatinib)
  - Autoimmune diseases like sarcoidosis and SLE
  - Metabolic disorders such as diabetes mellitus and thyroid dysfunction
  - Pregnancy (peripartum cardiomyopathy)
  - Nutritional deficiencies : lack of essential nutrients particularly vitamin B1
  - Idiopathic cases : **majority** cases occur without identifiable cause

  NB : Long standing cases of Type 2 Diabetes mellitus (DM2) can lead to dilated cardiomyopathy : contributory factors might be chronic hyperglycaemia and increased fatty acid oxidation , microangiopathy , autonomic neuropathy impairing the myocardial contractility , oxidative stress with chronic low-grade inflammation.

- Patients with DCMP and LVH may respond differently to heart failure therapies. Regression of LVH with optimal therapy (ACE inhibitors, beta-blockers, and MRAs) correlates with better prognosis.

  Persistent LVH despite medical therapy may indicate poor response to reverse remodeling, necessitating CRT or ICD therapy in high-risk cases.

2. Difference in LVH between DCMP and pressure overload conditions

In DCMP , the heart exhibits eccentric hypertrophy –

The hypertrophied mass is laid down around the dilated ventricular cavity without its obliteration→ the overall myocardial mass is increased.

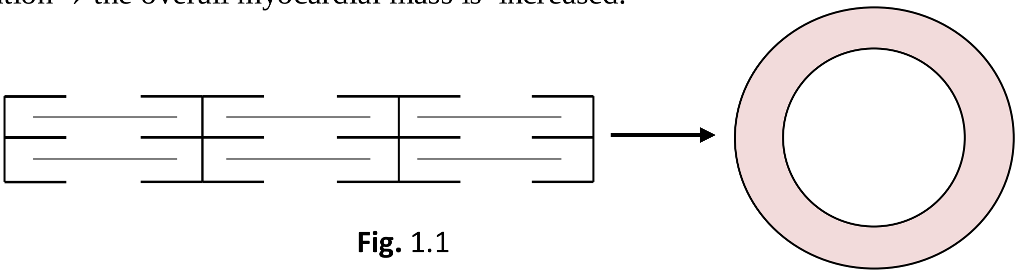


Fig. 1.1

Sarcomeres – the fundamental contractile units of muscle fibres , are added in series , i.e. , with end-to-end arrangement along the length of existing ones leading to enlongated muscle fibres with overall increase in ventricular mass.

This is known as eccentric hypertrophy.

The pressure overload conditions such as hypertension or aortic stenosis exhibit concentric hypertrophy.

Here the hypertrophied mass extending towards the centre of LV cavity , causing its significant obliteration.

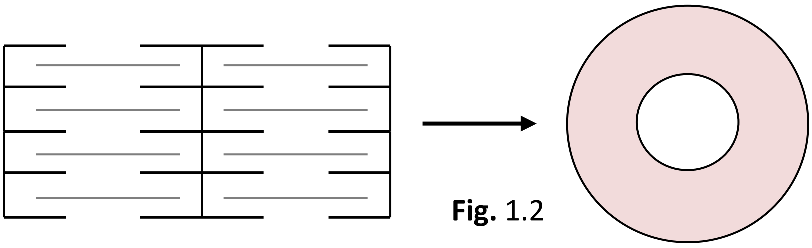


Fig. 1.2

Sarcomeres are added here in parallel , meaning thereby, new sarcomers are superposed upone each other leading to increased myocardial thickness with increased ventricular mass $\rightarrow$ thus obliterating LV cavity without its significant dilatation.

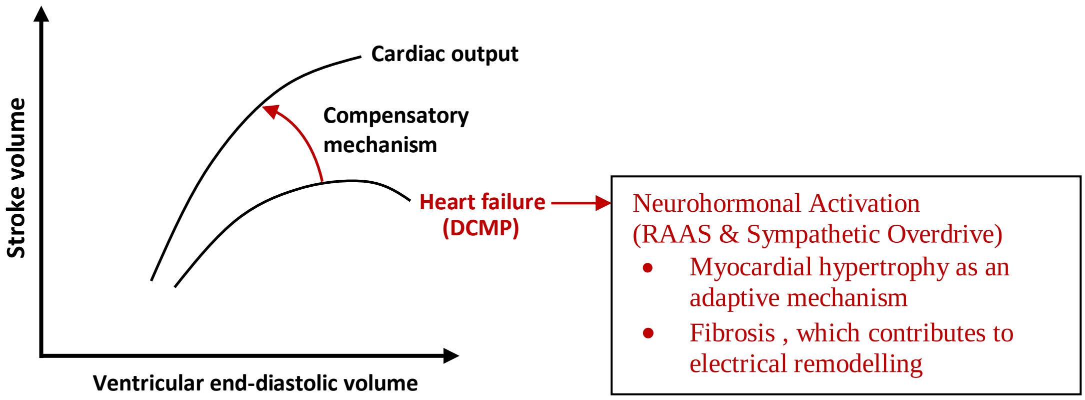
This is known as concentric hypertrophy.

Functional differences

DCMP	Pressure overload
Primarily associated with systolic dysfunction , with or without accompanying diastolic dysfunction.	Initially preserved systolic function , with diastolic dysfunction emerging over time.

3. Pathophysiology of LVH in DCMP

- ☐ Ventricular overload leading to LV dilatation , initially compensated by ventricular stretching as per **Frank-Starling law** (perceived as pseudo LVH on ECG)
- ☐ Neurohormonal Activation (RAAS & Sympathetic Overdrive)
  - Myocardial hypertrophy as an adaptive mechanism
  - Fibrosis , which contributes to electrical remodelling
- ☐ Progressive eccentric LVH with terminal decline in left ventricular functioning (Heart failure)
- ☐ **Secondary causes** : If any prior pressure overload , such as aortic stenosis or hypertension is superadded to the preexisting DCMP , then hypertrophy may supervene over the existing LV dilatation.



Frank-Starling law

Fig. 1.3

4. Concerned ECG changes with its basic reasoning

Basic reasoning :

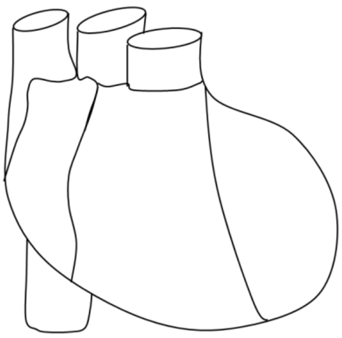


Fig. 1.4

- LVH with LV dilatation (eccentric hypertrophy) with associated Fibrosis → shifting of electrical vector laterally and posteriorly :
  Poor R-wave progression (PRWP)
  Late QRS transition
  Clockwise rotation
- In the presence of chamber dilatation , LVH pattern may be less pronounced than in pressure overload conditions.
- Interstitial fibrosis reflected in limb leads as relatively low voltage (limb leads are being away from the heart compared to those of chest leads).

Goldberger's triad is an electrocardiographic (ECG) pattern identified by Dr. Ary Louis Goldberger in 1982, associated with dilated cardiomyopathy (DCMP).

ECG triad

1. Relatively low voltage in limb leads (✱ QRS voltage < 0.8 mV in each of the limb leads)
2. Poor R-wave progression across precordial leads: suboptimal increase in R-wave amplitude from leads V1 to V6 (e.g. R-wave ≤3 mm in lead V3)
3. Relatively prominent QRS voltage in the chest leads (LVH)

The quantum of these changes would depend upon the balance in between heart failure and compensatory mechanism operating therein.

This triad is considered highly specific for DCMP but is relatively insensitive, meaning its presence strongly suggests DCMP, yet its absence doesn't rule out this condition.
However , due to chamber dilatation , LVH pattern may be less pronounced than in pressure overload conditions.
Persistent LVH in DCMP suggests ongoing maladaptive remodelling.

LVH with associated fibrosis → slowed conduction across the myocardium

- Poor R-wave progression (PRWP) : This would be worthwhile to mention here that **as the LV enlarges , the heart's electrical vector shifts more laterally and posteriorly** , resulting in poor anterior forces leading to PRWP despite no infarction.
- Late QRS transition (normally V3-V4 but in DCMP V5-V6)
- Clockwise rotation due to LV dilatation and septal fibrosis , the depolarization wave moves more posteriorly , causing an apparent clockwise rotation

✱ **Ref :** LeoSchamroth An Introduction to Electrocardiography (Eighth Adapted Edition) P , 135

Conduction defects

- LBBB : This pattern usually indicates diffuse myocardial disease with fibrosis. The contributing factors to its causation are enumerated as below :

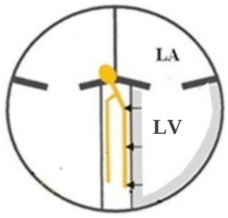


Fig. 1.5

- The mechanical stretch affecting the HIS-Purkinje system particularly the left bundle branch with slowing of the conduction therein.
- Creeping fibrosis disrupts conduction pathway especially in the left bundle branch , further promoting LBBB in DCMP patients.
- Delayed septal activation due to assocaited fibrosis may lead to ventricular dyssynchrony , further adding element to LBBB

The development of new LBBB during follow-up is a strong independent prognostic predictor of all cause mortality

- Intra Ventricular Conduction Delay (IVCD) without LBBB
- Other conduction defects like left anterior fascicular block , at times even first degree AV block may occur.

Arrhythmias

A high incidence of supraventricular and ventricular arrhythmias has been reported in most of the cases in DCMP. Ventricular arrhythmias have been observed terminally. Supraventricular tachycardia occurs occasionally**. Atrial fibrillation tends to develop late and it is a sign of structural disease progression** (it can lead to sudden decompensation with heart failure).

It has been documented that QRS duration $\geq$ 120 ms is a significant predictor of ventricular arrhythmias.

Peguero-Lo Presti and Sokolow-Lyon indices are commonly in use for diagnosing LVH

Due to shifting of electrical vector laterally and posteriorly with eccentric hypertrophy , Peguero-Lo Presti criteria seems to be more logical in diagnosing eccentric LVH

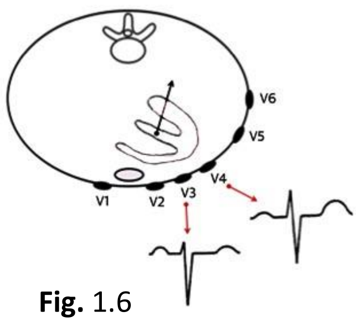


Fig. 1.6

Supplementary assessment, including imaging modalities like echocardiography or cardiac MRI, is highly recommended for precise evaluation.

Peguero-Lo Presti Criteria

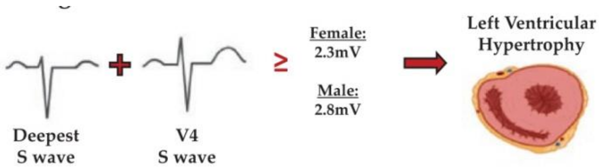


Fig. 1.7

Sensitivity: 47% to 55%, improving detection rates.
Specificity: Approx 72%, a higher rate of false positive , compared to Sokolow-Lyon.

Sokolow-Lyon criteria

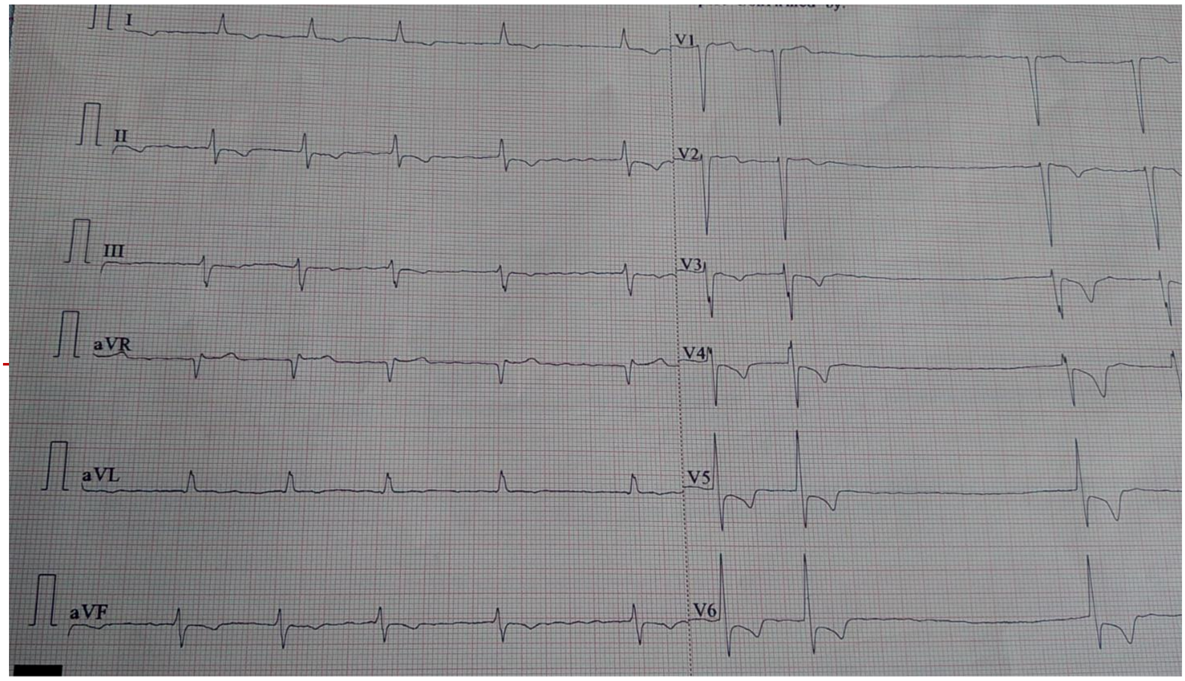
- S-wave depth in V1 + tallest R wave height in V5 or V6 > 35 mm
- R_{aVL} > 11 mm (with left axis deviation R in aVL should be >13 mm)

Sokolow-Lyon's index is the most used index, despite having the lowest sensitivity (20%) of all indexes. The specificity is high (>85%).

5. Illustration by ECGs

ECG Case 1 DCMP

50 Years old male with h/o Dysnoea



Source : Global Heart Rhythm Forum on 05.03.2025 **by Dr. R.K. Gupta** ,
Senior Consultant Physician , Yamunanagar , Haryana

ECG Findings	Comments
1. Atrial fibrillation (fine) with occasional long pause , lasting for 2.24 sec (see chest leads).	Atrial fibrillation tends to develop late and is a sign of structural disease progression. Longer pauses (over 4 seconds) or pauses with significant symptoms simulating that of SA block warrant medical attention.
2. Relatively low voltage in limb leads (QRS voltage < 8 mm in each of the limb leads)	Interstitial fibrosis reflected in limb leads as relatively low voltage (please see page 4)
3. LVH by Peguero-Lo Presti Criteria (deepest S-wave in V2 19 mm + S wave in V4 10.5 mm = 29.5 mm with accompanying global non-specific ST/T changes)	
4. Triad • Poor R-wave progression (PRWP) : (R-wave<3 mm in lead V3 • Late QRS transition at V5 • Clockwise rotation	LVH with associated fibrosis → slowed conduction across the myocardium. As the LV enlarges , the heart's electrical vector shifts more laterally and posteriorly , resulting in poor anterior forces leading to such changes
5. fQRS (QRS fragmentation) at V3-V4 (obvious on zooming the tracings)	QRS fragmentation indicates fibrosis ± conduction defects

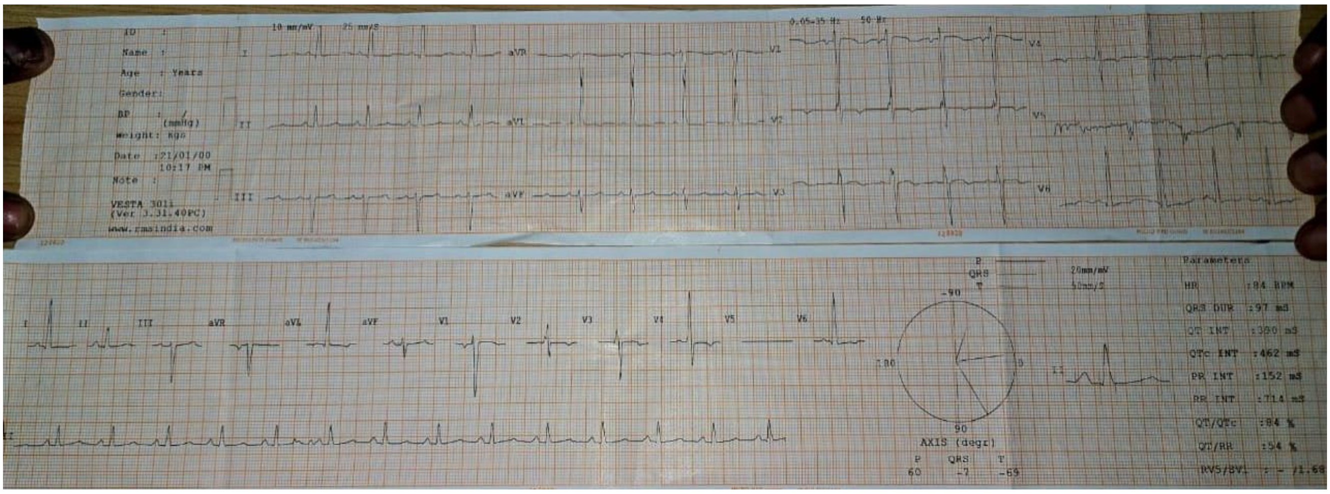
☐ **Cardiac Echo** : Severe left ventricular dysfunction with EF 25%

☐ **CAG** Normal

FINAL IMPRESSION : DCMP (the patient was put on Dytor plus 10 , Arney 50 bd , Dapa 10 , Lanoxin ½ , Ecosprin/AV)

ECG Case 2 **Degenerative Aortic Valvular Disease plus Diabetic cardiomyopathy (DCMP)**

46 Years old female presented with SOB and swelling of the body , Normotensive , No past history of diabetes but random blood sugar 250 mg % with HbA1c 8.2



Source : CME INDIA on 05.03.2025 by **Dr. N.K. Singh** , Director , Diabetes and Heart Research Centre, Dhanbad , Editor , www.cmeindia.in

Findings on ECG :

- R in aVL > 11 mm with secondary ST/T changes in lead I and aVL , associated with somewhat axis tilting towards the left (frontal QRS axis -20 degree , with reciprocal mild ST elevation in lead V1-V2)
  All these indicates LVH with systolic strain.
- Poor R-wave progression : R amplitude is more in V2 than in V3 associated with Counterclockwise rotation
- fQRS (fragmentation) at V2-V3

Comments : The followings reports done with this case are also taken into consideration:

- ☐ Random blood sugar 250 mg % with HbA1c 8.2
- ☐ HRCT scan of thorax : Marked cardiomegaly with dilated main pulmonary artery , evidence of pulmoanry edema
- ☐ Cardiac Echo findings : Degenerative Aortic Valve Disease (Severe Aortic Stenosis with Moderate Aortic Regurgitration) , Mild MR
  Dilated LV with ejection fraction 25%
- ☐ NT-pro BNP 3903 pg/mL

❒ Degenerative severe aortic stenosis (AS) with moderate aortic regurgitation (AR) and functional mild mitral regurgitation (MR), aggravated by long-standing hyperglycemia.

- ○ AS is most likely calcific and degenerative, when associated with diabetes
- ○ AR is likely due to aortic root changes, possibly worsened by vascular stiffening from diabetes.
- ○ Mild MR appears functional, secondary to LV dilation and remodeling rather than primary valve disease.

- LVH pattern only with lead aVL, counterclockwise rotation, and severely reduced EF (25%) suggest LV stretching rather than true LVH.
  - The lack of widespread LVH criteria suggests ventricular remodeling due to volume overload & dysfunction, rather than classical concentric hypertrophy.
  - Counterclockwise rotation : This suggest the heart is rotating opposite to normal , often due to leftward displacement or dilatation of the right ventricle.
- Diabetic Cardiomyopathy (DCMP) with heart failure with reduced EF (25%) interacting with degenerative valvular AS/AR is likely responsible for LVH being limited to aVL.
  - Diabetic cardiomyopathy contributes to LV dysfunction & fibrosis, further influencing the remodeling pattern seen on ECG.
  - The combination of severe AS and heart failure (with pulmonary oedema) explains why LVH is not more widespread on ECG, as the ventricle is more stretched than truly hypertrophied.

NB : There is no finding on ECG suggestive of pulmonary hypertension as revealed by HRCT of thorax but this is equally true that in the presence of diffuse DCMP , the ECG evidence of pulmonary hypertension would be lacking.

Final impression :

1. Degenerative Aortic Stenosis with Aortic Regurgitation (with functional MR) , aggravated by coexisting DM2.
2. Diabetic cardiomyopathy (DCMP)

6. Take Home Message

- In dilated cardiomyopathy (DCMP), the left ventricle (LV) enlarges and its contractile function declines. Despite this dilatation, the myocardium often exhibits hypertrophy. This form of hypertrophy differs from that observed with pressure overload conditions such as hypertension or aortic stenosis.

- LVH in DCMP is an important marker of disease severity and remodeling. While it results primarily from chronic volume overload and neurohormonal activation, additional factors like fibrosis, conduction abnormalities, and genetic predisposition contribute to its presence.

- Pathophysiology of LVH in DCMP
  - Ventricular overload leading to LV dilatation , initially compensated by ventricular stretching as per **Frank-Starling law** (perceived as pseudo LVH on ECG)
  - Neurohormonal Activation (RAAS & Sympathetic Overdrive)
    - Myocardial hypertrophy as an adaptive mechanism
    - Fibrosis , which contributes to electrical remodelling

- In DCMP , the heart exhibits eccentric hypertrophy .
  The pressure overload conditions such as hypertension or aortic stenosis exhibit concentric hypertrophy

- ECG findings in DCMP is having lower volatage criteria with poor R wave progression (PRWP) , late QRS transition at V5-V6 and clockwise rotation. The presence of LVH in DCMP correlates with worse prognosis and increased arrhythmic risk.
- Key Takeaways
  - Concentric LVH → High voltage, LV strain pattern, normal QRS duration
  - Eccentric LVH → Normal/low voltage, PRWP, QRS widening, conduction delays
- ECG Features in Diabetic DCMP + Aortic Stenosis
  - If DCMP is dominant → ECG resembles eccentric LVH (PRWP, low voltage, LBBB)
  - If AS is dominant → ECG resembles concentric LVH (high voltage, LV strain, normal QRS duration)
- Supplementary assessment, including imaging modalities like echocardiography or cardiac MRI, is highly recommended for precise evaluation.
- Patients with DCMP and LVH may respond differently to heart failure therapies. Regression of LVH with optimal therapy (ACE inhibitors, beta-blockers, and MRAs) correlates with better prognosis.
  Persistent LVH despite medical therapy may indicate poor response to reverse remodeling, necessitating CRT or ICD therapy in high-risk cases.

7. References

1. Goldberger's Clinical Electrocardiography (First South Asia Edition)
   Dilated Cardiomyopathy - P 249 , 118
2. LeoSchmroth An Introduction to Electrocardiography (Eighth Adapted Edition)
   Importance of Electrocardiogram in heart failure – P 134-137
3. CHOU'S ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE (Sixth Edition)
   Dilated Cardiomyopathy , P 274-276
4. Davidson principle and practice of medicine (22nd Edition)
   Dilated Cardiomyopathy , P 636-637
5. Harrison's Manual of Medicine (19th Edition)
   Caridomyopathies and myocarditis , P 639-640
6. Dilated Cardiomyopathy (DCM)
   Ed Burns and Robert Buttner
   Oct 8, 2024
   https://litfl.com/dilated-cardiomyopathy-dcm-ecg-library/

7. Left Ventricular Hypertrophy
   Abraham B. Bornstein; Suman S. Rao; Komal Marwaha. 2023
   https://www.ncbi.nlm.nih.gov/books/NBK557534/

8. Performance of the Peguero-Lo Presti Index in Diagnosis of Left Ventricular Hypertrophy at CIMAK Hospital Center in North Kivu, Democratic Republic of the Congo , Feb 2023
   Ferdinand Ng'ekieb Mukoso1,2,3*, Aliocha Natuhoyila Nkodila4,5, Thierry-Samuel Mbuyi Muhemed2, Trésor Mosomo Kashinde2, Sévérin Juamungu Ananas2, Salomo Ndoole Myatsi2, Ariane Namwiza Gakuru2, Olivier Gasana Muratwa3, Zéphirin Tudienzelz Kamuanga6, Yves Lubenga7, Bernard Phanzu Kianu7, Benjamin Longo-Mbenza5,7, Zacharie Kibendelwa Tsongo2
   https://www.scirp.org/journal/paperinformation?paperid=123354

9. Do We Need New Electrocardiographic Criteria for Left Ventricular Hypertrophy ? The Case of the Peguero-Lo Presti Criterion. A Narrative Review
   Andrea Faggiano, 1,2, Elisa Gherbesi, 2 Marijana Tadic, 3 Stefano Carugo, 1,2 Guido Grassi, 4 and Cesare Cuspidi , 19 Dec , 2023
   https://colab.ws/articles/10.1093%2Fajh%2Fhpad117

10. Peguero-Lo Presti criteria for the diagnosis of left ventricular hypertrophy: A systematic review and meta-analysis , 2021
    Zongying Yu 1, Jie Song 1, Li Cheng 1, Shasha Li 2, Qun Lu 1, Yafeng Zhang 3, Xiaoci Lin 4, Dadong Liu 5,*
    http://pmc.ncbi.nlm.nih.gov/articles/PMC7846009/

11. Assessment of Peguero Lo-Presti Criteria for Electrocardiographic Diagnosis of LVH in Indian Subjects April 2018
    Corresponding Author: Akshay S Chincholi, Department of Medicine, Jawaharlal Nehru Medical College, Belagavi-590010, Karnataka, India ,
    https://www.fortunejournals.com/articles/passessment-of-peguero-lopresti-criteria-for-electrocardiographic-diagnosis-of-lvh-in-indian-subjectsp.html

12. Electrocardiographic Criteria for the Diagnosis of Left Ventricular HypertrophyFree
    Julio G. Peguero Saberio Lo Presti Jorge Perez Omar Issa Juan C. Brenes and Alfonso Tolentino , 2017 Apr,
    https://www.jacc.org/doi/10.1016/j.jacc.2017.01.037

13. Pathophysiologic significance of left ventricular hypertrophy in dilated cardiomyopathy 1996
    K Iida 1, M el Sersi, K Fujieda, S Kawano, F Tabei, Y Iwasaki, T Masumi, I Yamaguchi, Y Sugishita
    https://pubmed.ncbi.nlm.nih.gov/8874989/

14. Peguero–Lo Presti criteria modified by body surface area for the electrocardiographic diagnosis of left ventricular hypertrophy in Thai patients
    Wongsakorn Luangphiphat 1, Atthaphon Phaisitkriengkrai 1, Ratikorn Methavigul 1, Komsing Methavigul 1

https://pmc.ncbi.nlm.nih.gov/articles/PMC10388768/#:~:text=Peguero%E2%80%93Lo%20Presti%20criteria%20were%20modified%20by%20dividing%20the%20criteria,considered%20a%20diagnosis%20for%20LVH.

15. Dilated Cardiomyopathy (DCM)
https://www.heart.org/en/health-topics/cardiomyopathy/what-is-cardiomyopathy-in-adults/dilated-cardiomyopathy-dcm

16. Dilated cardiomyopathy: MedlinePlus Medical Encyclopedia
https://medlineplus.gov/ency/article/000168.htm

17. Dilated Cardiomyopathy – Wikipedia
https://en.wikipedia.org/wiki/Dilated_cardiomyopathy

18. Dilated Cardiomyopathy - StatPearls - NCBI Bookshelf
Hadi Mahmaljy; Varun S. Yelamanchili; Mayank Singhal.
https://www.ncbi.nlm.nih.gov/books/NBK441911/

19. Dilated cardiomyopathy
Heinz-Peter Schultheiss, DeLisa Fairweather, Alida L. P. Caforio, Felicitas Escher, Ray E. shberger, Steven E. Lipshultz, Peter P. Liu, Akira Matsumori, Andrea Mazzanti, John McMurray & Silvia G. Priori
https://www.nature.com/articles/s41572-019-0084-1

DIABETIC CARDIOMYOPATHY AND ECG CHANGES : AN EMERGING CONCEPT

DIABETIC CARDIOMYOPATHY AND ECG CHANGES : AN EMERGING CONCEPT

©DR. D.P. KHAITAN
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

OUTLINE

Introduction

As Diabetic cardiomyopathy has become an increasingly recognized clinical entity among clinicians ; its better understanding is a 'must' for early diagnosis and the implementation of treatment strategies accordingly

Pathological-cum-clinical classification

Stage 1 : Early (Preclinical)

Stage 2 : Intermediate (Compensated Phase) Fibrosis increases →Stiff LV, Worsening diastolic dysfunction

Stage 3 : Advanced (Dilated , Failing Heart)

The pathological footsteps as correlative with ECG changes

Concentric hypertrophy → creeping fibrosis → LV dilatation with residual hypertrophy

Associated microvascular involvement

✓How to assess these findings on ECG ?

Illustration by ECG (An interesting case)

Take Home Message

References

Diabetic Cardiomyopathy and ECG Changes : an emerging concept

A Narrative Review

© **DR. D.P. KHAITAN**
MD (MEDICINE) FCGP(IND) FIAMS(MEDICINE) FICP FICCMD FIACM

When the heart is hurt by its associated accompanion , it is broken down with a grieved cut – it will heal , but there will always be a scar. This tormentation does not fly away on the wings of time but rather deepens more. The shattering of a heart when being broken may remain tearful till the end.

The associated accompanion diabetes mellitus does not sweeten the heart with glucose-dependent energy but rather shifts the metabolic pathway towards a bitter turn.

- **Insulin resistance , a hallmark of Type 2 diabetes leads to inadequate glucose uptake by cardiomyocytes , resulting in energy deprivation and metabolic turmoil leading to a cascade of anatomical and functional remodelling of the heart.**
- **Diabetes mellitus in early stage expresses itself as HFpEF , while advanced cases expresses itself as HFrEF.**

Clinicians know that ECG may prove itself the storyteller of the diabetic heart ; it deciphers the concerned patterns even before the heart succumbs to failure. The heart expresses its agony through the flickering signals on ECG.

1. Introduction (Keypoints)

- As Diabetic cardiomyopathy has become an increasingly recognized clinical entity among clinicians ; its better understanding is a 'must' for early diagnosis and the implementation of treatment strategies accordingly.
- This is a unique but distinct form of cardiomyopathy that occurs in patients with diabetes mellitus (DM), independent of other heart diseases like hypertension , any valvular or coronary artery disease (CAD). It is characterized by a combination of both diastolic and systolic dysfunction.
- It was first detected and described in 1972 by Rubler et al. when postmortem pathological reports revealed its nature in 4 diabetic patients who presented as heart failure without evidence of any concomitant pathology like coronary artery or valvular disease or even hypertension.
- Truly to say , diabetic cardiomyopathy looks to be an entity with shifting of energy substrate utilization – from a more balanced use of glucose to more reliance on fatty acid oxidation but this metabolic pathway is not so efficient so as to compensate the energy deficit in a diabetic patient. There are multiple factors as well involved in the pathogenesis of diabetic cardiomyopathy.

- It is manifested mainly as two subgroups :
  - Diastolic dysfunction (HFpEF-like phase) – Common in early diabetic cardiomyopathy ; ventricular hypertrophy preserves the myocardial function.
  - Systolic dysfunction (HFrEF-like phase) – ventricular dilatation develops as remodeling progresses.

  NB : Mixed DCMP phenotype (dilated with hypertrophy) – seen in advanced stages, with a combination of fibrosis, chamber dilation, and residual hypertrophy.

- Sex Differences in Diabetic Cardiomyopathy
  - Males with diabetes are more prone to develop systolic dysfunction (HFrEF)
  - Females with diabetes more commonly develop diastolic dysfunction (HFpEF) with preserved ejection fraction but in association with higher heart failure related mortality.
  - Estrogen's Protective Role: Females tend to have better mitochondrial function and endothelial response to diabetes compared to males.

- The ECG is very easily available tool which may bring the story of the diabetic heart on the surface – deciphers its pattern and whispers to the ears of clinicians – the different footsteps the heart meets with. Cardiac echo adds to its evaluation.

2. Pathological-cum-clinical classification

The pathophysiological spectrum in diabetic cardiomyopathy is a chain of events in continuum , which may be witnessed as below with its accompanying clinical scenario :

Stage 1 : Early (Preclinical)

Concentric hypertrophy → LV walls thicken due to hyperinsulinemia & AGEs (advanced glycation end-products).

Diastolic dysfunction appears first (HFpEF-like phase with EF $\geq$50%)

No major symptoms, but fatigue, mild dyspnea may be present.

Stage 2 : Intermediate (Compensated Phase)Fibrosis increases → Stiff LV, worsening diastolic dysfunction.

Microvascular dysfunction → Reduced myocardial perfusion. Subtle systolic dysfunction begins (mid-range EF 41-49%)

Arrhythmias (atrial fibrillation, QT prolongation) become common.

Stage 3 : Advanced (Dilated, Failing Heart)

LV dilates, leading to mixed remodeling (DCMP-like features).

Systolic function declines (HFrEF-like phase with EF<40%)

Heart failure symptoms (edema, pulmonary congestion, fatigue, exercise intolerance).

High risk of sudden cardiac death (SCD) due to ventricular arrhythmias.

NB :

- ☐ **Microvascular Disease**
  Capillary rarefaction (reduced density of small vessels) → Silent ischemia & impaired coronary flow reserve.
  Endothelial dysfunction → Reduced nitric oxide (NO) → Worse vasodilation & perfusion mismatch.

- ☐ **Autonomic Dysfunction & Arrhythmias**
  Diabetic neuropathy affects the heart → Reduced heart rate variability (HRV).
  Increased sympathetic tone → QT prolongation → Risk of sudden cardiac death (SCD).

3. The pathological footsteps as correlative with ECG changes

Here ECG changes can be well correlated with the pathological findings of diabetic cardiomyopathy , as enumerated below :

- Concentric hypertrophy → creeping fibrosis → LV dilatation with residual hypertrophy
- Other associated pathological changes
  - Microvascular involvement as reduction in density of small vessels with its concomitant endothelial dysfunction
  - Autonomic dysfunction with increased sympathetic tone

RV systolic and diastolic dysfunction

Type 1 DM and Type 2 DM have been found to be associated with RV dysfunction . This would be worthwhile to mention here that RV diastolic impairment seems to precede RV systolic dysfunction.

"Impairment of RV systolic function in patients with T2DM includes lower RV long-axis fractional shortening, reduced systolic excursion of tricuspid annulus, reduced pulmonary valve peak and mean velocities and impaired systolic strain. RV strain has also been correlated with levels of HBA1c. RV 3D echocardiography data in DM patients are limited. A study comparing 3D- and 2-D echocardiography showed that RV and right atrial strain were decreased in patients with pre-diabetes and DM as compared to controls. The early and deteriorating impact of diabetes on RV systolic and diastolic function develops independently from LV diastolic dysfunction, pulmonary hypertension and CAD."

Ref :
Diabetic cardiomyopathy: pathophysiology, imaging assessment and therapeutical strategies , December 2024
Author links open overlay panelVincenzo Rizza [a], Lara Tondi [b c], Angelo Maria Patti [d], Damiano Cecchi [a], Massimo Lombardi [b], Francesco Perone [e], Marco Ambrosetti [f], Manfredi Rizzo [d], Domenico Cianflone [a g], Francesco Maranta [a g]
https://www.sciencedirect.com/science/article/pii/S277248752400103X

How to reveal ECG changes correlative with pathological footsteps

Concentric hypertrophy : (Increased LV wall thickness without significant dilatation)

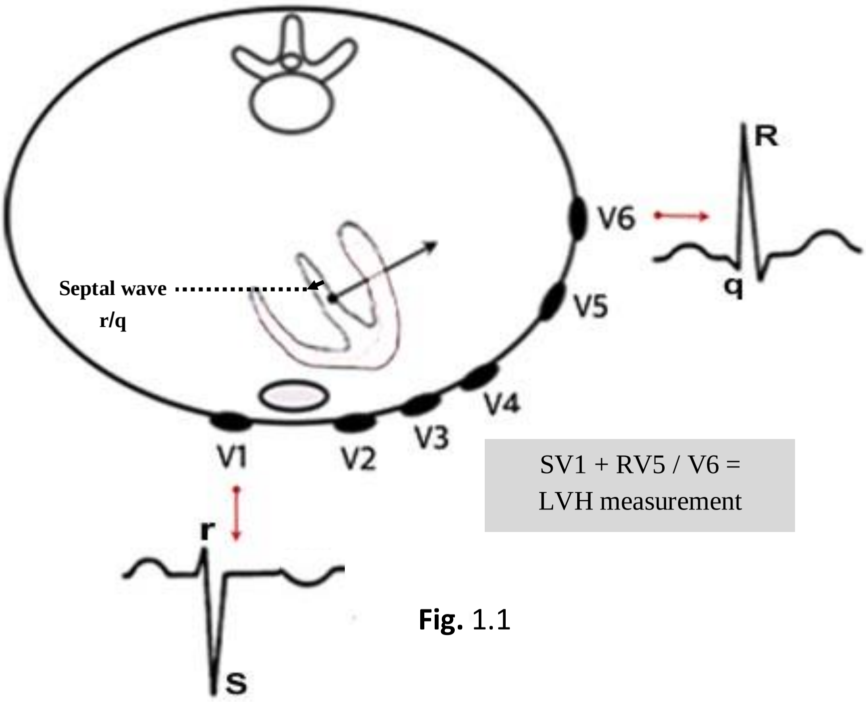


Fig. 1.1

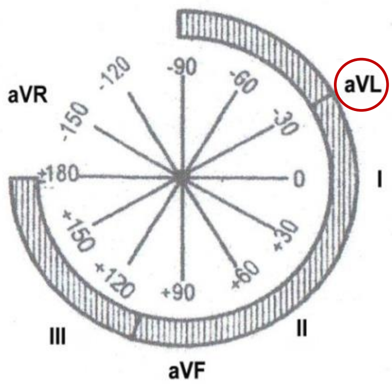


Fig. 1.2

LV dilatation with residual hypertrophy

- **Normally** , since septal wave is directed towards V1 **,** it inscribes 'r' as the first positive deflection in V1 and since septal wave is away from V6 , it inscribes q wave as a first negative deflection in V5/V6. And negative S in V1 and positive R in V6 both indicate the depolarization occuring through the L.Ventricle but at different thoracic levels.

- **Concentric hypertrophy :**
  The common criteria for left concentric hypertrophy in common use is
  Sokolow-Lyon criteria
  S-wave depth in V1 + tallest R wave height in V5 or V6 > 35 mm
  Sokolow-Lyon's index is the most used index, despite having the lowest sensitivity (20%) of all indexes. The specificity is high (>85%).

  Other criteria like cornell-voltage criteria Romhilt-Estes Point scoring system may also be used (see the 'Text' for details)

  R $_{aVL}$ > 11 mm (with left axis deviation R in aVL should be >13 mm)

  Supporting Non-voltage criteria :
  Strain over lateral leads (I , aVL , V5, V6) : ST depression with T-inversion , caused by hypertrophy and related endocardial ischemia during repolarization phase.

- **Masked Hypertrophy :**
  Long -standing diabetic cardiomyopathy may present as low voltage in limb leads , inspite of hypertrophy (LVH paradox)

- **Assisted by imaging techniques**
  Echocardiography : confirms LV wall thickness , mass index , and systolic / diastolic dysfunction.
  Cardiac MRI (more sensitive than echocardiography in this context)

NB : Mild intraventricular conduction delay (IVCD) or left bundle branch block (LBBB) may be there due to subendocardial fibrosis and conduction slowing in diabetic cardiomyopathy.

Electrical axis assessment (Hexaxial lead system)

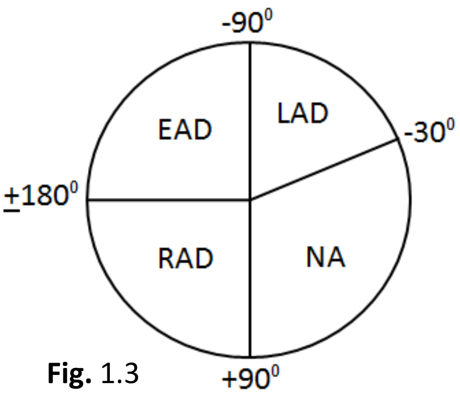


Fig. 1.3

□	Normal Axis	=	-30^0 to $+90^0$
□	Left Axis Deviation (LAD)	=	-30^0 to -90^0
□	Right Axis Deviation (RAD)	=	$+90^0$ to $+180^0$
□	North-West Deviation (NWD) This is also known as Extreme Axis Deviation	=	-90^0 to -180^0

∗Rule of 90^0

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)

- **QRS Vector with Concentric hypertrophy**
  Normal axis or Left axis deviation (LAD) due to thickened LV walls (the electrical vector may be shifted leftward between -30^0 to -90^0).

- **Possible QRS vector in masked hypertrophy** (LV dilatation with residual hypertrophy)
  - Normal to left axis deviation (LAD -30^0 to -90^0) – most common
  - **Indeterminate axis** : It is not equivalent to north-west axis. This terminology is used if QRS complexes are equiphasic in almost all the limb leads or if it cannot be plotted by the rule of 90^0. ∗

    Mechanism :

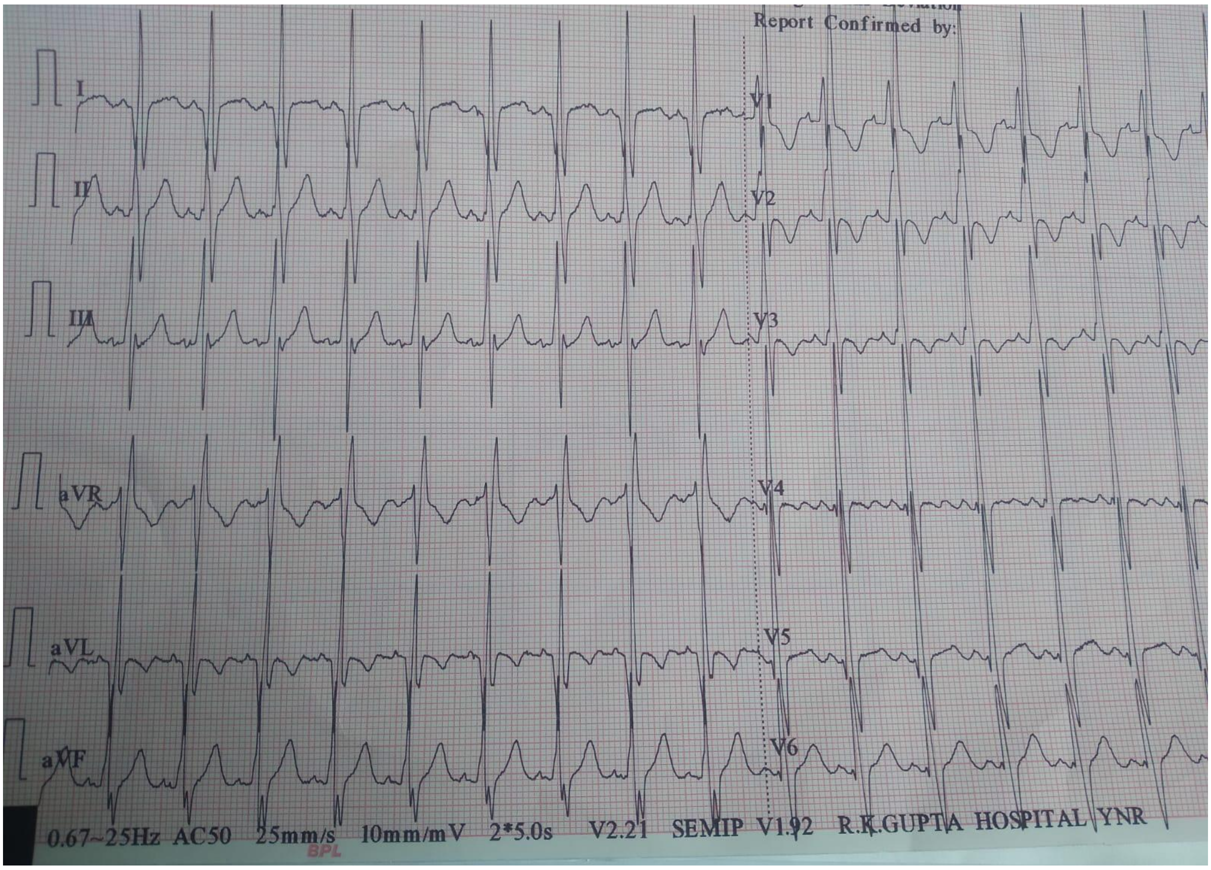
    - Extensive myocardial fibrosis with disorganized depolarization pattern.
    - ✓ Loss of anterior septum (common in diabetic cardiomyopathy) can result in anterior infarction patterns, further altering the axis.
    - Biventricular enlargement can create conflicting electrical forces, leading to an indeterminate axis.
  - Right Axis Deviation (RAD) (+90° to +180°) – Less Common but Possible

    Mechanism :

    - Severe LV dilatation → secondary pulmonary hypertension → Right ventricular hypertrophy (RVH) → RAD
    - Independent RVH (see page 49)

4. Illustration by ECG

History : 60 yrs ✓diabetic female presented with acute breathlessness , managed as acute LVF and B/L bronchoneumonia (Angiography could not be done because of concomitant pulmonary infections).



Source : Global Heart Rhythm Forum , on 16.03.2025 by Dr. R.K Gupta , Senior Consultant Physician , Yamunanagar , Haryana

Her echocardiography and X-ray Chest are posted below :

ECHOCRDIOGRAPHY :

- Left Ventricle Dilated, Concentric Left Ventricular Hypertrophy
- Regional Wall Motion Abnormally
- Anterior Wall, Anterior Septum Hypokinetic
- Severe Left Ventricular Systolic Dysfunction (LVEF 35%)
- Moderate Mitral Regurgitation , Mild TR , No PAH

Fig. 1.4

ECG findings	Comments
1. Indeterminate QRS axis (QRS complexes are equiphasic in so many limb leads and the axis cannot be determined by the rule of 90^0)	This may be due to the structural damage (fibrosis ± ischemic changes) to the interventricular septum involvement leading to marked conduction defect therein. (anterior septum hypokinesia on cardiac echo also points towards the same)

Contd.

ECG findings	**Comments**
2. qR in leads I and aVL (Initial q is having much depth with R>11 mm)	Left ventricular hypertrophy with strain / ? old high lateral MI (deep q waves in lateral leads might be due to fibrotic tissue unable to depolarize)
3. RBBB-like pattern in leads V1-V3	Abnormal conduction through the fibrosed interventricular septum
4. R in V5 > 26 mm Total QRS voltage in all the 12-leads >175 mm	Left Ventricular hypertrophy
5. Sinus rhythm with ventricular rate > 100 bpm	Sinus tachycardia

Comments :

Based on the combined findings of ECG and Echocardiography

- The presence of indeterminate axis with RBBB like pattern in leads V1-V3 is most likely due to fibrosis creeping over the anterior interventricular septum (also evident on cardiac echo by the presence of anterior septum hypokinesia). All these indicate abnormal conduction through the fibrosed / ischemic interventricular septum.
- ECG and cardiac echo both demonstrate left ventricular hypertrophy
- The pattern of qR may indicate old high lateral MI , the amplitude of R>11 mm in lead aVL also indicates the left ventricular hypertrophy in association.
- On cardiac echo , the presence of moderate mitral regurgitation is possible with left ventricular dilatation.

In nutshell , ECG plus cardiac echo findings are suggestive of **advanced stage of diabetic cardiomyopathy.**

- Left ventricular dilatation , fibrosis plus concomitant concentric LVH
- Severe left ventricular systolic dysfunction (LVEF 35% , <40%)

(The patient presented with acute breathlessness which might be due to the combined impact of declined systolic function plus associated bilateral broncopneumonic changes)

Final impression

Diabetic cardiomyopathy with severe Left Ventricular Systolic Function (LVEF 35%) associated with bilateral bronchopulmonary infection

5. Take Home Message

- Diabetic cardiomyopathy is a distinct entity that occurs in patients with diabetes mellitus (DM), independent of other heart diseases like hypertension , any valvular or coronary artery disease (CAD). It is characterized by a combination of both diastolic and systolic dysfunction.

- This looks to be an entity with shifting of energy substrate utilization – from a more balanced use of glucose to more reliance on fatty acid oxidation but this metabolic pathway is not so efficient so as to compensate the energy deficit in a diabetic patient. There are multiple factors as well involved in the pathogenesis of diabetic cardiomyopathy.

- Diabetic-induced cardiomyopathy is an underdiagnosed but clinically significant cardiovascular complication. It begins as subclinical diastolic dysfunction which progresses to overt heart failure if not tackled early.
- Recent research suggests that diabetic cardiomyopathy is not a single entity but may have distinct subtypes based on structural abnormalities , assessed also with LVEF index on cardiac echo.
  - Diastolic dysfunction (HFpEF-like phase) – Common in early diabetic cardiomyopathy ; ventricular hypertrophy preserves the myocardial function.
  - Systolic dysfunction (HFrEF-like phase) – ventricular dilatation develops as remodeling progresses.
  - Mixed DCMP phenotype (dilated with hypertrophy) – seen in advanced stages, with a combination of fibrosis, chamber dilation, and residual hypertrophy.
  - Other associated findings
    ⇒ Microvascular involvement as reduction in density of small vessels with its concomitant endothelial dysfunction
    ⇒ Autonomic dysfunction with increased sympathetic tone

In nutshell , Diabetic cardiomyopathy is a specific cardiac manifestation of patients with diabetes characterized by left ventricular hypertrophy and diastolic dysfunction in the early phase and later on overt heart failure with reduced systolic function in the advanced stages.
The pathogenesis of this condition is multifactorial , being recognised as a specific entity in the presence of insulin resistance and hyperglycemia.

6. References

1. CLINICAL MEDICINE UPDATE (A Publication of the Indian Association of Clinical Medicine) , 2025
   Heart Failure with Preserved Ejection Fraction : Diagnostic Pitfalls and Management
   Subhash Chandra , SN Yadav , P 89-102
2. McMaster Cardiology 2023 (South Asian Edition)
   Heart Failure , P 178-216
3. Diabetic cardiomyopathy: pathophysiology, imaging assessment and therapeutical strategies , December 2024
   Author links open overlay panelVincenzo Rizza [a], Lara Tondi [b c], Angelo Maria Patti [d], Damiano Cecchi [a], Massimo Lombardi [b], Francesco Perone [e], Marco A mbrosetti [f], Manfredi Rizzo [d], Domenico Cianflone [a g], Francesco Maranta [a g]
   https://www.sciencedirect.com/science/article/pii/S277248752400103X
4. Diabetic Cardiomyopathy , 2023
   https://www.sciencedirect.com/topics/medicine-and-dentistry/diabetic-cardiomyopathy

5. Diabetic cardiomyopathy: The role of microRNAs and long non-coding RNAs
   Mirjana T. Macvanin1*Zoran Gluvic2Jelena Radovanovic1Magbubah Essack3Xin Gao3Esma R. Isenovic , 7 March 2023
   https://www.frontiersin.org/journals/endocrinology/articles/10.3389/fendo.2023.1124613/full

6. Mechanisms of diabetic cardiomyopathy and potential therapeutic strategies: preclinical and clinical evidence , 20 Feb 2020
   Yi Tan, Zhiguo Zhang, Chao Zheng, Kupper A. Wintergerst, Bradley B. Keller & Lu C
   https://www.nature.com/articles/s41569-020-0339-2

7. **Diabetic Cardiomyopathy: Definition, Diagnosis, and Therapeutic Implications , 2019**
   Stefania Paolillo 1, Fabio Marsico 2, Maria Prastaro 3, Francesco Renga 3, Luca Esposito 3, Fabiana De Martino 3, Pierfrancesco Di Napoli 3, Immacolata Esposito 3, Antonio Ambrosio 3, Monica Ianniruberto 3, Rosa Mennella 3, Roberta Paolillo 3, Paola Gargiulo 4
   https://pubmed.ncbi.nlm.nih.gov/31079692/

8. Diabetic Cardiomyopathy: An Update of Mechanisms Contributing to This Clinical Entity , 16 Feb , 2018
   Guanghong Jia, **Michael A. Hill**, and **James R. Sowers**
   https://www.ahajournals.org/doi/10.1161/circresaha.117.311586

9. Diabetic Cardiomyopathy Wikipedia
   https://en.wikipedia.org/wiki/Diabetic_cardiomyopathy

10. Diabetic cardiomyopathy, causes and effects
    Sihem Boudina [1], Evan Dale Abel , 2010
    https://pmc.ncbi.nlm.nih.gov/articles/PMC2914514/

11. Diabetic Cardiomyopathy—From Basics through Diagnosis to Treatment
    by Ewa Radzioch Bartłomiej Dąbek Marta Balcerczyk-Lis eronika Frąk Piotr Fularski
    https://www.mdpi.com/2227-9059/12/4/765

12. Referred to ChatGPT whenever needed

ISOLATED LVH IN aVL WITH CLOCKWISE ROTATION : A SUBTLE ECG CLUE TO HOCM

ISOLATED LVH IN aVL WITH CLOCKWISE ROTATION : A SUBTLE ECG CLUE TO HOCM

©DR. D.P. KHAITAN

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

OUTLINE

Introduction

This article explores the phenomenon of isolated LVH in lead aVL with clockwise rotation on ECG as a possible early electrocardiographic marker of HOCM.

Why isolated LVH in aVL with clockwise rotation in HOCM ?

A. Isolated LVH in aVL (positioned at basal and high lateral LV wall) directing the QRS axis leftward and superiorly
B. Clockwise rotation : A clue to Posteriorly Directed Hypertrophy

A consideration to pertinent ECG changes as a subtle pointer to HOCM

The combination of isolated LVH in aVL with systolic overload and clockwise rotation suggests that the hypertrophy is affecting the basal septum and high lateral LV wall in a way that shifts electrical forces posteriorly.

Illustration by ECGs

Two interesting cases showing the pattern of 'isolated LVH in aVL with clockwise rotation' in favour of HOCM (confirmed on echocardiography)

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

Take Home Message

References

Isolated LVH in aVL with Clockwise Rotation : A Subtle ECG Clue to HOCM

A Narrative Review

© DR. D.P. KHAITAN
MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

A scientific fact usually comes into limelight when one does not go where the path may lead , goes instead where there is no path but only a trail is visible. It is very easy to establish a fact by holding the key with its established norms , but recognizing an atypical presentation in medicine requires thinking beyond the conventional standards.

By keeping these facts in mind , one should not immediately exclude a particular diagnosis on ECG even when it has only a subtle clue to its diagnosis. HOCM (hypertrophic obstructive cardiomyopathy) doesn't always announce itself loudly on ECG ; sometimes , it whispers only through a single lead.

- ☐ **A subtle clue , like isolated LVH in aVL , might hold the key to diagnose HOCM (Lead aVL views the high lateral left ventricle—an area often preferentially hypertrophied in HOCM, especially in its asymmetric septal variant)**
- ☐ **Its diagnosis is strengthened by the presence of accompanying clockwise rotation. The hypertrophied basal septum with high lateral LV wall in HOCM may shift the ventricular orientation towards the clockwise direction.**

This is the scientific way through which ECG tracings continue to reveal new triumphs of diagnostic informations , even with conditions like HOCM , which may be further evaluated through cardiac imaging.

1. Introduction (Keypoints)

- Hypertrophic cardiomyopathy usually encompasses an inappropriate hypertrophy of the myocardium occuring in the absence of any obvious cause like aortic stenosis or systemic hypertension and predominantly involving the interventricular septum in an asymmetrical manner with some accompanying clues on ECG , including dagger-like Q waves , high-voltage QRS complexes and deep T-wave inversions in the concerned leads , as per anatomical involvement of the myocardial territories.

- **Certain cases of HOCM meet with diagnostic challenges in the absence of conventional patterns as just described.**

- One such condition which is usually overlooked is an isolated LVH in lead aVL , particularly when associated with clockwise rotation in addition.

- Recognizing such an atypical pattern with hypertrophic cardiomyopathy is an interesting topic to be reviewed through ECG.

- This study may explore the significance of high R-wave voltage in lead aVL as an indicator of LVH , associated with HOCM , specially if linked with clockwise rotation as an associate.
- Currently , there seems a paucity of established literatures specifically describing “isolated LVH in lead aVL with the associted clockwise rotation” as a distinct phenotype of HOCM. However , several studies and case reports have discussed other atypical ECG presentations with HOCM.
- Hypertrophic cardiomyopathy (HOCM) is a complex myocardial disorder often diagnosed based on echocardiographic findings of asymmetric left ventricular hypertrophy (LVH). However, early ECG recognition can be crucial, particularly in cases where classical ECG criteria for HOCM are absent. This article explores the phenomenon of isolated LVH in lead aVL with clockwise rotation on ECG as a possible early electrocardiographic marker of HOCM, needs to be confirmed by echocardiography.

2. Why isolated LVH in aVL with clockwise rotation in HOCM ?

This atypical pattern “isolated LVH in aVL with clockwise rotation” is a finding that, although subtle, may suggest underlying segmental hypertrophy and warrants further investigation with echocardiography.

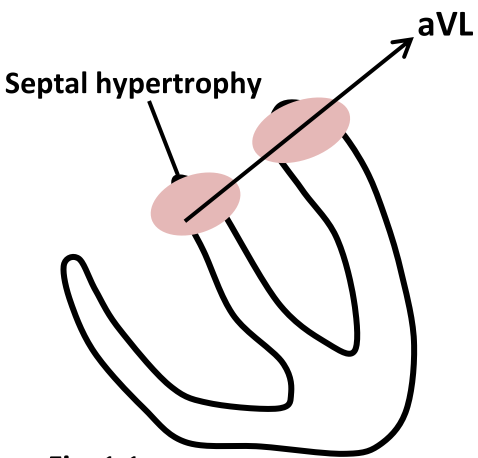


Fig. 1.1

A. Isolated LVH in aVL: A Unique ECG Signature

This is likely due to localized hypertrophy in the basal and upper lateral LV wall, directing the QRS axis leftward and superiorly

B. Clockwise Rotation: A Clue to Posteriorly Directed Hypertrophy

Clockwise rotation (delayed R-wave progression in precordial leads, with late transition in V5–V6) suggests posteriorly directed forces due to basal LV hypertrophy (more obvious if the hypertrophy is more localized to the basal and posterior LV segment rather than the septum)

3. A consideration to pertinent ECG changes as a subtle pointer to HOCM

LVH in aVL with clockwise rotation

Isolated LVH in lead aVL

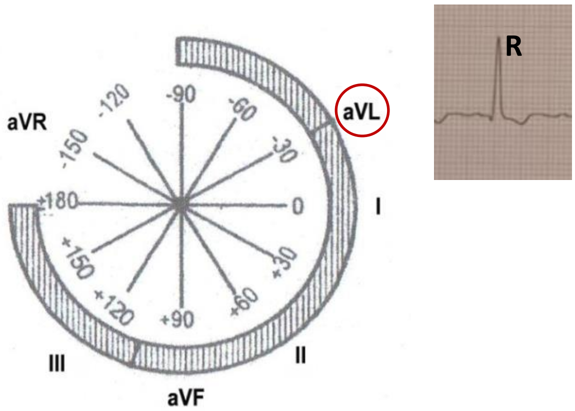


Fig. 1.2

- **Why aVL?**
  Lead aVL views the high lateral left ventricle—an area often preferentially hypertrophied in HOCM, especially in its asymmetric septal variant.
- R **aVL** > 11 mm (with left axis deviation R in aVL should be >13 mm) may be an isolated but early marker of HOCM.
  This R may be attributed to increased afterload from left ventricular outflow tract (LVOT) obstruction.
- Strain over leads I and aVL :
  ST depression with T-inversion , caused by hypertrophy and related endocardial ischemia during repolarization phase.

Clockwise rotation

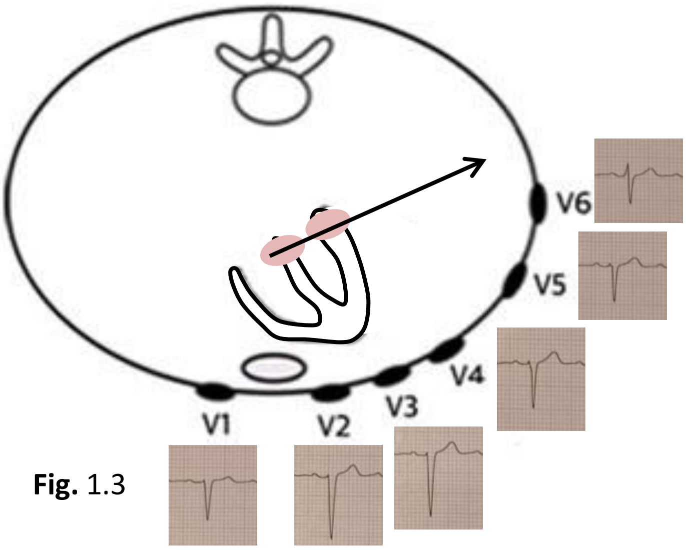


Fig. 1.3

- **Clockwise rotation**
  Normally, the R-wave progresses from V1 to V6, with the transition (R = S) occurring around V3 or V4. Here , in clockwise rotation, this transition shifts later (V5 or V6), indicating a relative posterior displacement of the involved segments.
- This may be attributed to :
  - ✓ Septal / high lateral LV wall hypertrophy pushing the heart posteriorly
  - ✓ Electrophysiological delay due to myocardial disarray (disorganised myocardial fibers)
  - ✓ Left ventricular outflow tract (LVOT) obstruction and its effect on ventricular geometry

---

NB : The combination of isolated LVH in aVL with systolic overload, and clockwise rotation suggests that the hypertrophy is affecting the basal septum and high lateral LV wall in a way that shifts electrical forces posteriorly.

Be precautious of red signal

- ❒ When isolated LVH in aVL is seen with clockwise rotation on ECG , it is an alarming sign and HOCM should be strongly suspected
- ❒ This should prompt further echocardiographic evaluation, especially if there are symptoms like exertional dyspnea, chest pain , syncope, palpitation or a family history of sudden cardiac death.
- ❒ One should also auscultate the chest for the presence of mid-systolic murmur over the left parasternal area (ejection systolic murmur may be confused with valvular aortic stenosis)

Important clarification for some important findings on ECG in the present clinical scenario

- ➢ The absence of Q wave : The usual deep septal Q waves of HOCM may be absent, as hypertrophy in these cases does not predominantly involve the anteroseptal wall.
- ➢ Pseudo-Delta Waves : In some cases, initial slurring of the QRS complex may be observed, mimicking a pseudo-delta wave. This may result from fibrosis-related conduction delay in hypertrophied myocardial segments (prone to ventricular arrhythmias→SCD).
- ➢ T-Wave inversion may not be deep in this scenario due to the following consideration:

  In typical HOCM with severe asymmetric septal hypertrophy, we expect deep T-wave inversions in the concerned leads due to repolarization abnormalities caused by massive anteroseptal hypertrophy.However, when hypertrophy is distributed across both anterior and posterior segments at the basal/high lateral LV wall, the repolarization forces may partially cancel each other on the frontal plane.This neutralization effect can blunt the expected T-wave inversions, making them less prominent than in classical cases of HOCM.

The traditional reliance on global LVH patterns and deep T-wave inversions in precordial leads may cause certain cases of HOCM to go unnoticed. Recognizing the significance of isolated LVH in aVL with systolic overload , especially when accompanied by clockwise rotation , can help in unmasking HOCM at its earlier stages.

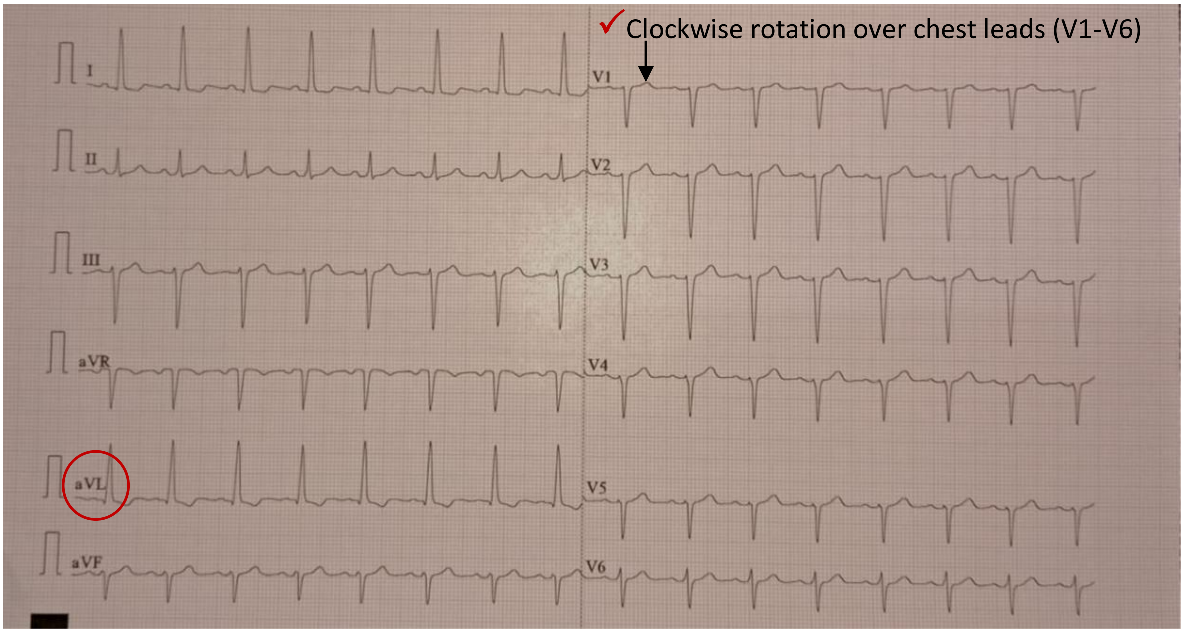
Assisted by imaging techniques

- **Echocardiography :** Echo is diagnostic of HOCM
  Asymmetrical septal hypertrophy (ASH) , systolic anterior motion (SAM) of the MV apparatus which may abut the IVS , mid-systolic AV closure and fluttering ,etc. And the continuous Doppler may show a peak velocity across the left ventricular outflow tract.
- **Cardiac MRI with late gadolinium enhancement (LGE)** to assess and quantify fibrosis.

4. Illustration by ECGs

Case No. 1

50 years aged female presented with 2 months h/o distension abdomen , Fever 2 months , Dyspnoea on exertion - NYHA IV (USG showed ascites , 4L of fluid drained from abdominal cavity – exudate in nature , ATT started) CVS Exam : systolic murmur over LPSA



Source : Global Heart Rhythm Forum on 20-03-2025 by **Dr. R.K Gupta** , Senior Consultant Physician , Yamunanagar , Haryana

❒ **Please review again page 59 for proper understanding the case**

ECG findings	Comments
• **R in aVL>11 mm** (QRS axis +20 degree) , associated with ST depression / T-inversion in leads aVL and I)	Isolated LVH in lead aVL with systolic overload
• **Clockwise rotation over chest leads**	This indicates shifting of electrical forces posteriorly by the hypertrophied basal septum and high lateral LV wall
• Mild slurring at the beginning of the ascending limb of QRS complexes , obvious over leads II and V6	Possibly fibrosis –related conduction delay in hypertrophied myocardial segments (this subtle QRS slurring may be mistaken for pre-excitation - WPW). It is a risk factor for ventricular arrhythmias and sudden cardiac death (SCD) in HOCM.
• Accompanying sinus tachycardia	----

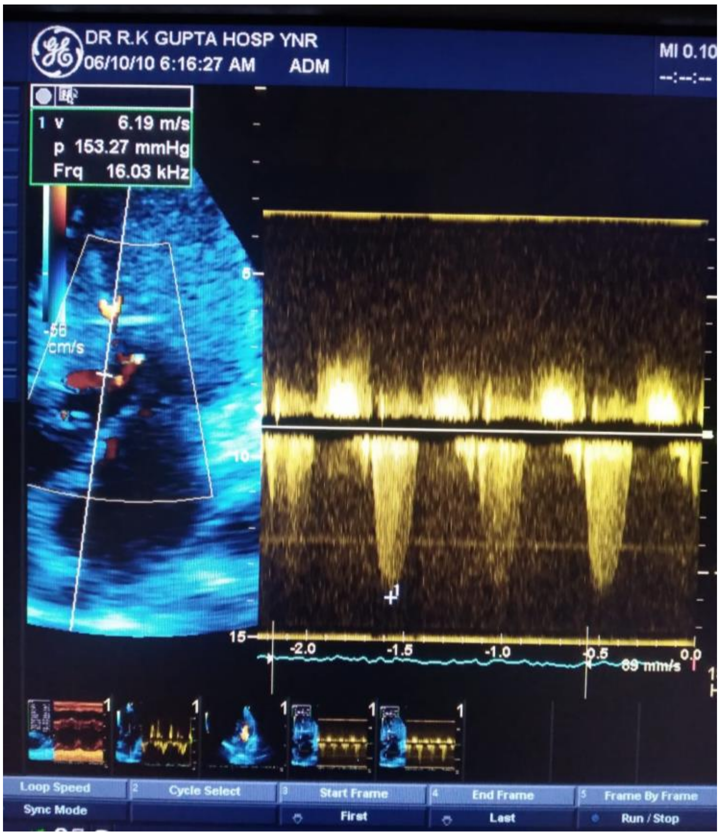


Fig. 1.4

❒ **Cardiac echo : Salient features**

- Hypertrophic cardiomyopathy
- *Peak LVOT gradient = 66 mmHg Significant LVOT obstruction +, SAM+
- Continuous Doppler showing a peak velocity across the left ventricular outflow tract
- Mild MR , mild AR ; No AS/TR

*'This dynamic obstruction is more pronouned at the later stages of systole. As the LV becomes empty, LV cavity size is comparatively smaller and the anterior mitral leaflet moves anteriorly to abut against the septum. It may be present at rest or become more pronounced with valsalva maneuvre or exercise.

NB : In some individuals with HOCM, the most dangerous period is at the end of vigorous exercise , e.g. during a race competition. At this time, ventricular volume diminishes as cardiac output and heart rate decrease , catecholamine drive is having a downgoing pattern, and there may be changes in circulating electrolyte concentrations , such as K+. These features all combine to increase the risk of syncope and sudden cardiac death by increasing the likelihood of increasing left ventricular outflow gradient and ventricular arrhythmias (**Ref :** ECHO MADE EASY , Second Edition by Sam Kaddoura**)**

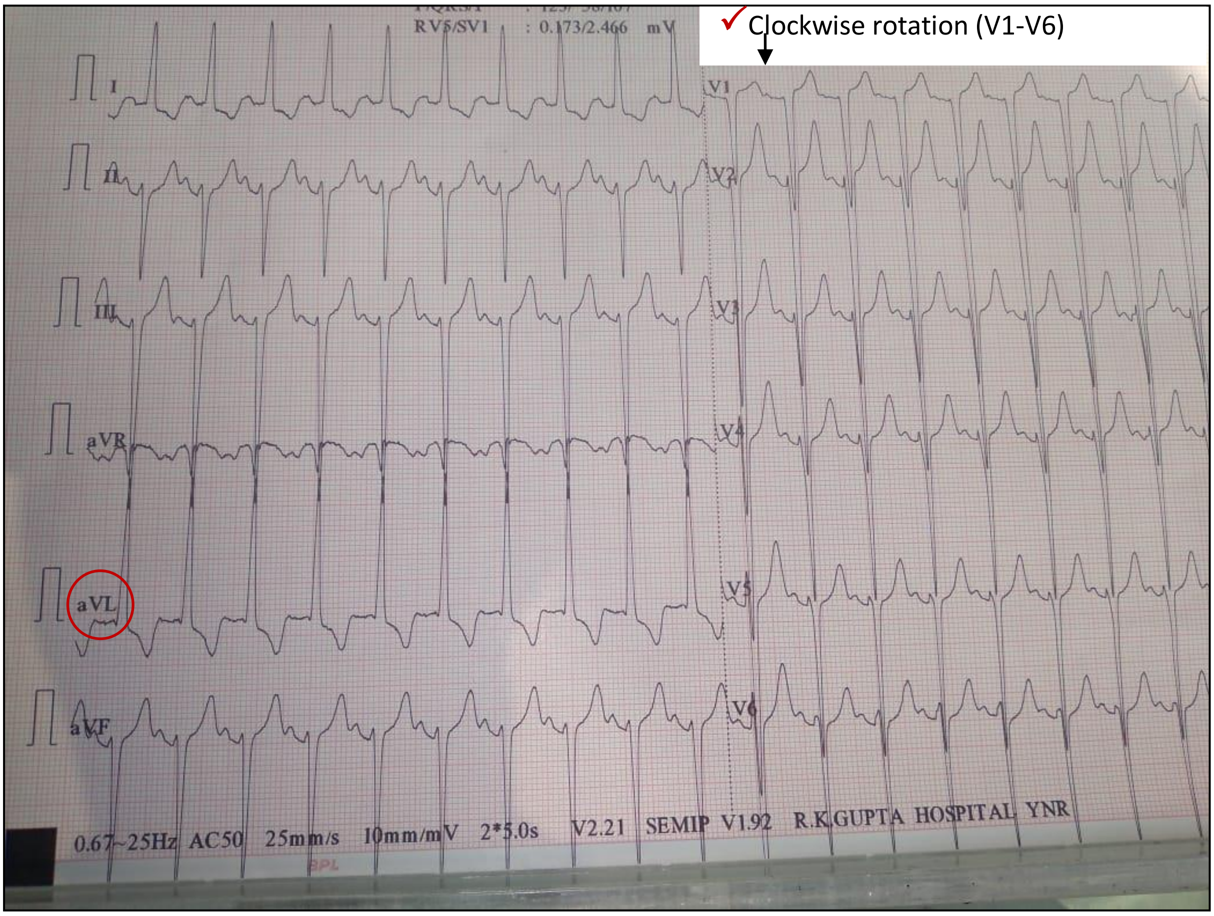
❒ **On auscultation ejection systolic murmur heard over LPSA**

Comments :

- The combination of isolated LVH in aVL with systolic overload associated with clockwise rotation over the chest leads suggests hypertrophic cardiomyopathy.
- Cardiac echo findings , as discussed above are diagnostic of HOCM.
  - SAM is hallmark of HOCM – related LVOT obstruction.
  - Patient with peak LVOT gradients ≥ 50 mmHg are at higher risk for HOCM related symptoms.
- The presence of mid-systolic murmur over LPSA in association is also the pointer to the same diagnosis.

Case No. 2

59 years female , diabetic 20 years , hypertensive 7 years , came for medical fitness for surgery
BP 160/80 mmHg , hemodynamically stable no h/o chest pain or dyspnoea, systolic murmur over LPSA also heard.



Source : Global Heart Rhythm Forum on 24-01-2025 by **Dr. R.K Gupta** , Senior Consultant Physician , Yamunanagar , Haryana

❒ Please see the followings :

ECG findings	**Comments**
• **Marked R in aVL>13 mm** (left axis deviation , QRS axis -50^0) , associated with ST depression / T-inversion in leads aVL and I)	With left axis deviation R in aVL > 13 mm is the pointer of isolated LVH in lead aVL Associated systolic overload.
• **Clockwise rotation over chest leads with prominent upright T-waves**	This indicates shifting of electrical forces posteriorly by the hypertrophied basal septum and high lateral LV wall
• Accompanying sinus tachycardia	----

❐ Cardiac echo : Salient features

Hypertrophic Cardiomyopathy with mid - cavity gradient of 85 mm Hg

A "dagger-shaped" signal with CW

NB :

- **Mid-Cavity Obstruction** (MCO) : Unlike classical hypertrophic obstructive cardiomyopathy (HOCM) , here obstruction stands within the body of the LV.
- A gradient of 85 mmHg indicates severe obstruction
- **Dagger-Shaped CW Doppler's signal** suggest a progressive increase in velocity during systole , creating a late peaking , with dagger-like appearance ; this is considered a key diagnostic feature for obstructive HOCM

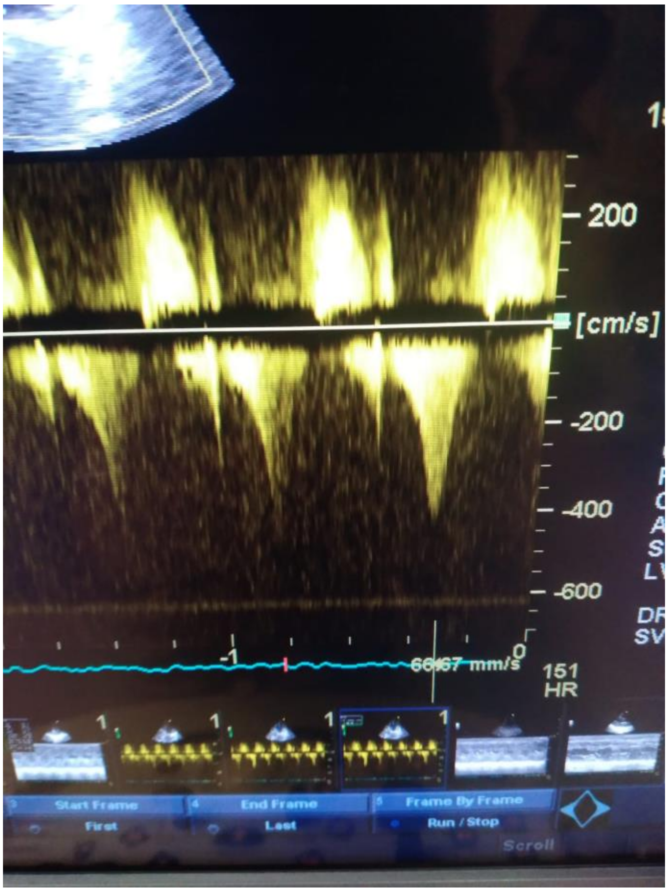


Fig. 1.5

❐ On auscultation with ejection systolic murmur heard over LPSA

Comments : All the followings are in favour of HOCM

- Isolated LVH in aVL in the presence of left axis deviation with systolic overload , linked with clockwise rotation over the chest leads
- Cardiac echo : Hypertrophic cardiomyopathy with cavity gradient of 85 mmHg and dagger-shaped signal on Continuous Wave Doppler.
- The presence of ejection-systolic murmur in association over LPSA is also the pointer to the same diagnosis.

5. Take Home Message

- The conventional reliance on global LVH patterns and deep T-wave inversions in precordial leads may cause certain cases of HOCM to go unnoticed. Recognizing the significance of isolated LVH in aVL, especially when accompanied by clockwise rotation and systolic overload can unmask HOCM at its earlier stages
- While LVH in aVL in association with clockwise rotation alone is not diagnostic of HOCM, in the right clinical context—especially with systolic overload—it may serve as an early or subtle ECG marker. Therefore , there is a dire need for vigilance in ECG interpretation , as early detection of HOCM can prevent complication like sudden cardiac death.
- Lead aVL, placed at –30 degree on the frontal plane, provides an anatomical view of the high lateral and basal septal regions of the left ventricle. In HOCM with asymmetric septal hypertrophy (ASH), these regions are often preferentially thickened, directing electrical forces towards aVL. As a result, the R-wave amplitude in aVL may become disproportionately prominent, even in the absence of significant precordial LVH findings.
- Clockwise rotation, detected by delayed R-wave progression in precordial leads (shifted transition to V5 or V6), is another subtle but crucial clue. The hypertrophied basal septum and high lateral LV wall in HOCM can shift the electrical axis posteriorly, delaying septal activation and altering QRS transition.
- In this context isolated LVH in aVL with clockwise rotation should prompt further imaging with echocardiography or cardiac MRI to confirm hypertrophy distribution and associated fibrosis if any (subtle QRS slurring on ECG may indicate underlying myocardial fibrosis).
- More case studies are required to validate the hypothesis – "Isolated LVH in aVL with clockwise rotation is a subtle ECG clue to the diagnosis of HOCM".

6. References

1. LeoSchamroth An Introduction to Electrocardiography (Eighth Adapted Edition) , Hypertrophic Cardiomyopathy , Revised by K.K Narayanan Namboodiri , P 208-211
2. CHOU'S ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE (Sixth Edition) Hypertrophic Cardiomyopathy , P 276-279
3. Marriot's Practical Electrocardiography (12th Edition) Cardiomyopathies , P 261-263

4. Textbook of Clinical Electrocardiography for Postgraduates , residents and Practising Physician (3rd Edition) by Dr. S.N. Chugh ,
Hypertrophic Cardiomyopathy , P 308-313

5. ECHO MADE EASY (Second Edition) by Sam Kaddoura
Hypertrophic cardiomyopathy , P 80-82

6. Hypertrophic cardiomyopathy - Diagnosis and treatment , 23 Feb 2024
https://www.mayoclinic.org/diseases-conditions/hypertrophic-cardiomyopathy/diagnosis-treatment/drc-20350204

7. Hypertrophic Cardiomyopathy (HCM)
Ed Burns and Robert Buttner
Oct 8, 2024
https://litfl.com/hypertrophic-cardiomyopathy-hcm-ecg-library/

8. Diagnostic and prognostic electrocardiographic features in patients with hypertrophic cardiomyopathy
Andrea Bernardini [1,2,✉,2], Lia Crotti [3,4], Iacopo Olivotto [5,6], Franco Cecchi
by A Bernardini · 2023
https://pmc.ncbi.nlm.nih.gov/articles/PMC10132576/

9. Apical hypertrophic cardiomyopathy (AHC)
Robert Buttner and Mike Cadogan
Jun 8, 2022
https://litfl.com/apical-hypertrophic-cardiomyopathy-ahc/

10. Hypertrophic cardiomyopathy electrocardiogram
9 December 2022
Editors-In-Chief: C. Michael Gibson, M.S., M.D. [1], Martin S. Maron, M.D., and Barry J. Maron, M.D. Soroush Seifirad, M.D
https://www.wikidoc.org/index.php/Hypertrophic_cardiomyopathy_electrocardiogram

11. Hypertrophic Obstructive Cardiomyopathy (HOCM) ECG Review
BySteven Lome, MD
http://healio.com/cardiology/learn-the-heart/ecg-review/ecg-topic-reviews-and-criteria/hypertrophic-obstructive-cardiomyopathy-review

12. Referred to ChatGPT whenever needed