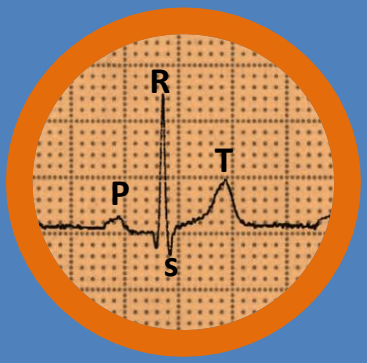

ECG Review :

Chamber Enlargement and Hypertrophy

(eBook for Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

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

O Heart
Thou Chamber enlargement and hypertrophy
We clinicians search on ECG ...

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O Heart
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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 everything of 'Chamber Enlargement and Hypertrophy on ECG' is not covered within this book. Here I am putting only some important aspects related to the articles discussed herein. It is only a step toward the vast ocean of knowledge. I may be excused for any error or omission.

With thanks and regards


DEDICATED
TO ALL THE
FELLOW COLLEAGUES

Index

ASSESSMENT OF THE ATRIAL ENLARGEMENT ON THE ELECTROCARDIOGRAM

ECG

ASSESSMENT OF THE ATRIAL ENLARGEMENT ON THE ELECTROCARDIOGRAM

©DR. D.P. KHAITAN

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OUTLINE

Introduction (Keypoint consideration)

Normally there are two components of P-wave , an initial component is inscribed by the depolarization of the right atrium and the terminal component is inscribed by the depolarization of the left atrium. The P-wave is the combined deflection of right and left atria both.

Right Atrial Enlargement	**Left Atrial Enlargement**
In right atrial enlargement the P-wave becomes taller in both limb lead II and right precordial lead V1.	Biphasic P-wave with a notch in between its two peaks in limb lead II. In chest lead V1 , the terminal negative component of the biphasic P-wave is delayed in its duration and depth both.

- Causes
- Electrophysiological basis
- ECG criteria
- Comments

Biatrial enlargement

In Biatrial enlargement the ECG shows signs of both right and left atrial enlargement.

Concluding remark

References

Assessment of Atrial Enlargement on the Electrocardiogram

A Narrative Review

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The human mind grasps the image of an object by its shape and size. Most possibly this observation had given birth to the science of measurement , which came to the existence by repeated perception of the concerned object in space and time. These changes rendered the basic principle of measurement – the vehicle of important informations that can be labelled as the science of morphometry.

This science of morphometry is of very much significance in the assessment of atrial enlargement on the electrocardiogram. The researchers have expanded their mind to bring about the vocabulary of calculating the atrial enlargement on ECG.
The passage of atrial impulses through both the atria is reflected as the P-wave

- ☐ **Normally there are two components of P-wave , an initial component is inscribed by the depolarization of the right atrium and the terminal component is inscribed by the depolarization of the left atrium. The P-wave is the combined deflection of right and left atria both.**
- ☐ **The P-axis lies normally at 0 to +75^0 but mostly in between +45 to +70^0. Its axis < +45^0 usually reflects left atrial enlargement (LAE) , if > +70^0 , it reflects right atrial enlargement (RAE).**

The clinicians move forward to access the atrial enlargement based on these two basic principles.

1. Introduction (keypoints consideration)

- Though the right and left atria are anatomically two distinct chambers but electrophysiologically they behaves as a single unit – the impulse from the SA node first depolarizes the right atrium and then simultaneously the left atrium through a special connective link known as **Bachmann's Bundle**. Thus , there is no electrical barrier in between these two atria.

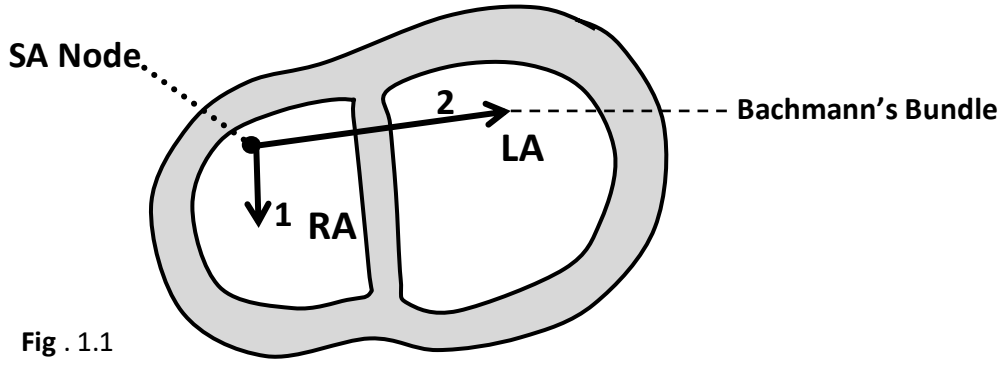


Fig . 1.1

- P-axis consideration on the frontal plane

  Normally the P-axis on the frontal plane varies from 0 to $+75^0$ but mostly lies between +45 to $+70^0$. Its axis less than $+45^0$ reflects left atrial enlargement (**LAE**) , if more than $+70^0$, it reflects right atrial enlargement (**RAE**) , as shown by the following sketch of Hexaxial lead system.

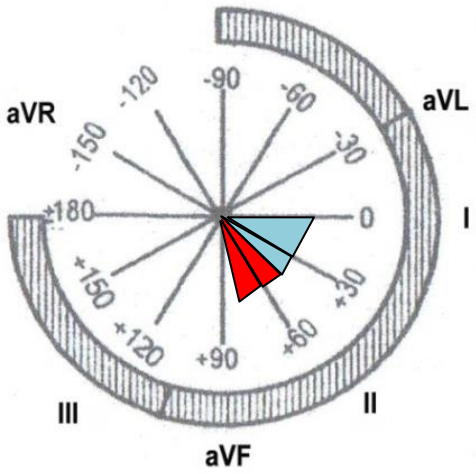


Fig . 1.2

Comments

Two atria conjoined together behave as a single electrical unit ; wherein SA node in the right atrium is the point-source of electrical flow → simultaneous flow of electrical current towards both the atria in two different directions → the resultant of this bidirectional atrial force is the P-axis.

- The left atrium is thicker than that of right atrium and this is to be noted that the right atrium lies anterior to the left atrium, which takes longer time to be depolarized due to its relative thickness.

  As already mentioned , the right atrium is depolarized first , inscribing the P-wave on the frontal plane with its dominant vector directed inferiorly with its positive deflection in inferior leads II, III and aVF.

  And over the horizontal plane the right atrium gets depolarized towards V1 , tending to cause an initial positive deflection , and the left atrium depolarizes later with its posterior extension , tending to cause a terminal negative P deflection.

  (**The current flow towards the exploring lead produces positive deflection and if away it produces the negative deflection**).

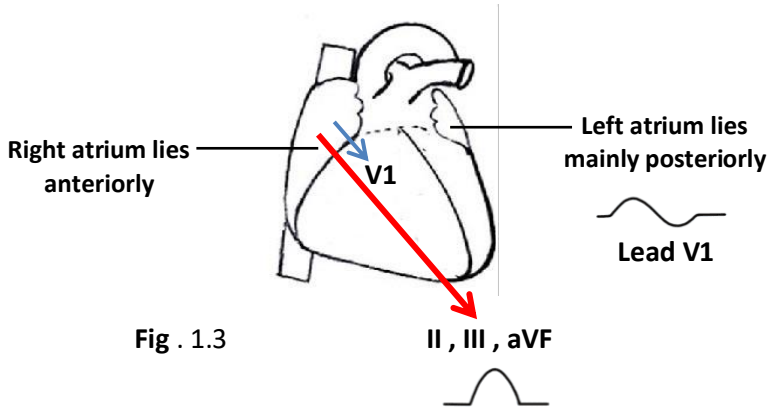


Fig . 1.3

The P-wave is the combined deflection of right atrial and left atrial depolarization.

- The normal morphology of P-wave is mentioned as below :

P-wave morphology in frontal lead II
The normal P-wave is usually best visioned in frontal lead II because P-wave axis is directed towards the inferior leads , mostly lead II. The normal P-wave in lead II is having a smooth round contour (The right and left atrial counterparts usually summate together to form a monophasic P-wave with positive deflection). The amplitude of P-wave is < 2.5 mm (0.25 mV) with its duration not greater than 110 ms.

P-wave morphology in lead V1
This is usually best visioned in lead V1 since both the initial and terminal components of P-wave are visible on this lead – the morphology here is biphasic , having an initial positivity and terminal negativity. Here the initial component of P-wave (right atrial component) is normally less than 1.5 mm (0.15 mV) in amplitude. The terminal component is having negative deflection (left atrial component) is less than 0.04 sec duration with depth < 1mm.

- Here the causative pathology may be due to dilatation / hypertrophy or increased atrial pressure in isolation or combination. Though atrial abnormality is considered to be a better terminology **but the term 'atrial enlargement' is used here in this context to eliminate the confusion in between the causative factors**.

2. Right Atrial Enlargement

- **Causes of Right atrial enlargement :**
  In association with increased pressure in the pulmonary artery , which gets transmitted from the right ventricle to the right atrium.
  The causes are listed as below :
  - Chronic lung disease , commonly COPD (cor pulmonale)
  - Primary pulmonary hypertension
  - Congenital heart disease (Tetralogy of Fallot with left to right shunt)

  In isolation :
  - Congenital heart disease (Ebstein anomaly, Tricuspid atresia)
  - Tricuspid stenosis

- **Electrophysiological basis of Right atrial enlargement :**
  The right atrial force plays its dominant role in dictating the right atrial enlargement (RAE) over both the vertical and horizontal planes – inscribing tall P-wave in both limb lead II and right precordial lead V1. Because this enlargement involves only the early part of the P-wave , any increase in the duration of right atrial depolarization does not result in the prolongation of the total duration of the P-wave.
  Predominance of right atrial forces causes P-wave axis to be more than 70^0 on the frontal plane.
  On the above electrophysiological basis the right atrial enlargement is assessed :

- **ECG Criteria of Right Atrial Enlargement**

  With right atrial enlargement , the amplitude of the initial component of P-wave increases , i.e. the overall amplitude of P-wave in lead II and the amplitude of inital component of P in lead V1. The width remains unchanged because the terminal portion of the P-wave is left atrial in origin.

  Right atrial enlargement also makes the right atrium to dominate over the left atrium from the electrical point of view , the P-axis is deviated clockwise towards more than $+70^0$.

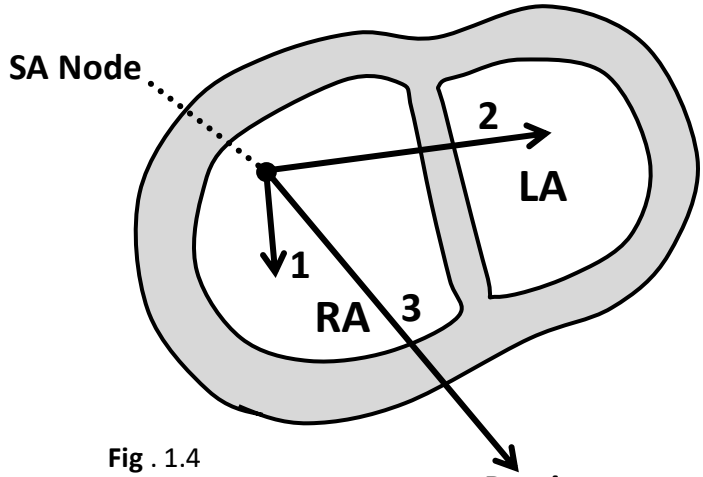


Fig . 1.4

The ECG changes suggestive of right atrial enlargement are mentioned as below :

- > 2.5 mm in the inferior leads (II, III and AVF) , known as **P-pulmonale**.
- > 1.5 mm in V1 and $\pm$V2

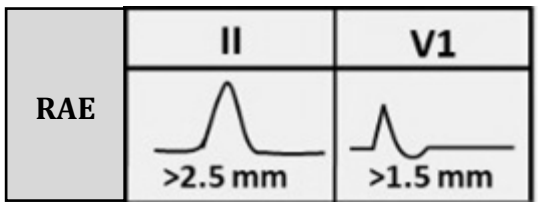


Fig . 1.5

- P-wave axis in the frontal plane is more than $+70^0$.

The ECG changes suggestive of right atrial enlargement correlate poorly with its clinical and anatomic findings

- Better criteria can be laid down from the QRS complex since QRS changes may occur due to high incidence of RVH in association with RAE.
  - QR, Qr, qR, or qRs morphology in lead V1 (in absence of coronary heart disease)

    This is to be noted that Q/q is the septal wave which is directed towards the left ventricle , away from the right ventricle.

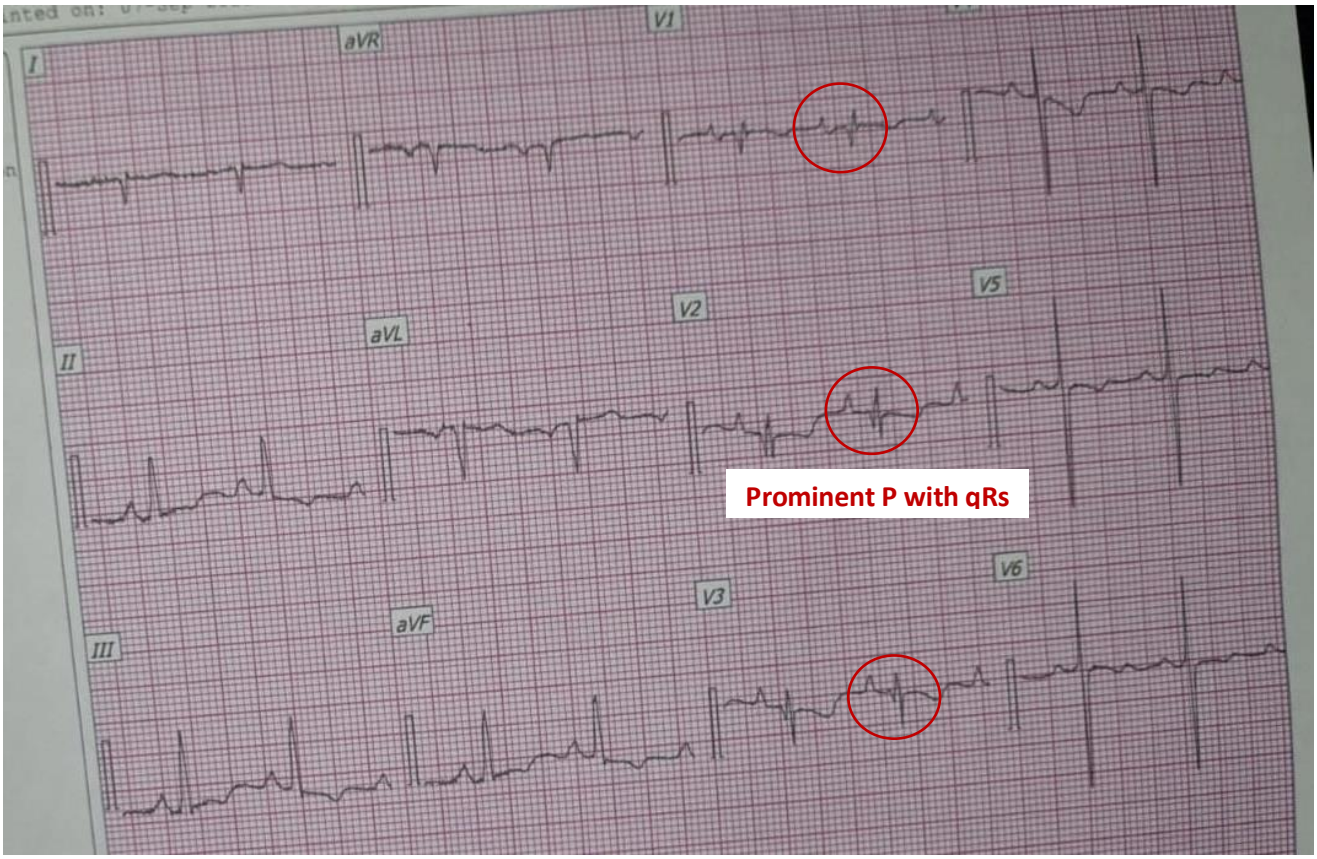
Ref : ECG Learning Centre (Created by Eccles Health Science Library)
https://ecg.utah.edu/lesson/7

NB : With marked RAE , the P-wave may be inverted in lead V1 , making the pseudo impression of LAE.

ECG to show the presence of Qr/qRs morphology in lead V1-3 supporting the criteria of RVH in favour of associated RAE

(Better QRS criteria to diagnose RAE in the presence of RVH , as mentioned in page 4)

45 years female with respiratory distress off and on having intermittent spells of cough and SOB over 6 years (diagnosed to be a possible case of Cor Pulmonale)



Source : CME INDIA on 07.09.2023 by Dr. N.K. Singh , Senior Consultant Physician and Diabetologist , Dhanbad ; Editor: www.cmeindia.in

ECG Findings :

- The presence of Qr/qRs in V1-3 (prominent septal wave over right ventricular leads) in association with clockwise rotation and T-inversion from V1-5.
- Other findings are right axis deviation with ST/T changes over the inferior leads , more marked in lead III ; the amplitude of P-wave in V2-3 is >1.5mm ; P-pulmonale in lead aVF (questionable in lead II and III) with P-axis at $+80^0$.

'As a rule , an abnormally tall P wave in the right precordial leads is a more specific finding for right atrial enlargement than the diagnostic criteria based on the limb leads.'

Ref : CHOU'S ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE , P 33
Sixth Edition

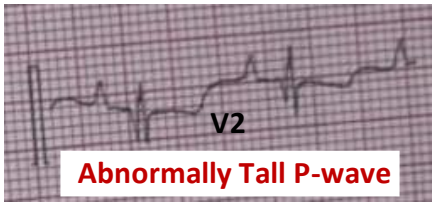


Fig . 1.6

Comments :

- ☐ The presence of the rightward direction of P-wave axis strengthens the diagnosis of right atrial enlargement. This rightward axis shift is considered to be the most discriminating P-wave change when evaluating the severity of COPD.
- ☐ The P-pulmonale pattern is a mostly recognized hallmark of RAE , but it may also be caused by the low diaphragm position and increased sympathetic stimulation in COPD. The low diaphragm causes the P-axis to be deviated vertically and increased signal due to sympathetic stimulation causes the increased amplitude of P-wave. Under such circumstances these factors may vary , and accordingly the pattern of P-pulmonale is also variable in the same patient depending on the heart rate severity and associated bronchospasm.
- ☐ A tall, peaked T-wave may be visioned in healthy individuals with thin body build.
- ☐ Pseudo P-pulmonale may also be observed sometimes in cases of hypokalemia due to the summation of preceding larger U-wave with that of P-wave.
- ☐ RA enlargement has been consistently associated with the poor outcome in patient with pulmonary hypertension. This is considered to reflect right ventricular failure. RAE is also considered to be an independent predictor for severity of tricuspid regurgitation with pulmonary hypertension

(Ref : Heart Atrial Enlargement
Judy R. Mangion MD, ... Steven A. Goldstein MD, in ASE's Comprehensive Echocardiography (Second Edition), 2016
https://www.sciencedirect.com/topics/medicine-and-dentistry/heart-atrium-enlargement)

3. Left Atrial Enlargement

- **Causes of left atrial enlargement :**

  In isolation:
  - Classically seen with **mitral stenosis**

  In association with left ventricular hypertrophy:
  - Systemic hypertension
  - Aortic stenosis
  - Mitral incompetence
  - Diastolic dysfunction in the left ventricle / Left ventricular failure.
  - Congenital heart disease – ventricular septal defect , Patent ductus arteriosus with reverse shunting.
  - Hypertrophic cardiomyopathy

- **Electrophysiological basis of left atrial enlargement**

  Left atrial enlargement causes its depolarization to be delayed due to its increased relative stretching or pressure therein in association with ± dilatation / hypertrophy. Therefore , the normal monophasic P-wave in frontal plane gets converted into a positive biphasic P having a notch in between.

  Over the chest lead V1 there is also a biphasic P but with terminal negativity. This terminal negativity is due to delayed depolarization of the larger left atrium , its impulse passing away from lead V1. An increase in the left atrial force exaggerates the normal terminal component of the P-wave.

  The electrocardiographic pattern of left atrial enlargement is called **P-mitrale** because it is most commonly seen with mitral valve disease - a common cause of left atrial enlargement.

- **ECG criteria of left atrial enlargement**

  The evaluation of left atrial enlargement is based on the electrophysiological principle , as discussed in the page no. 6.

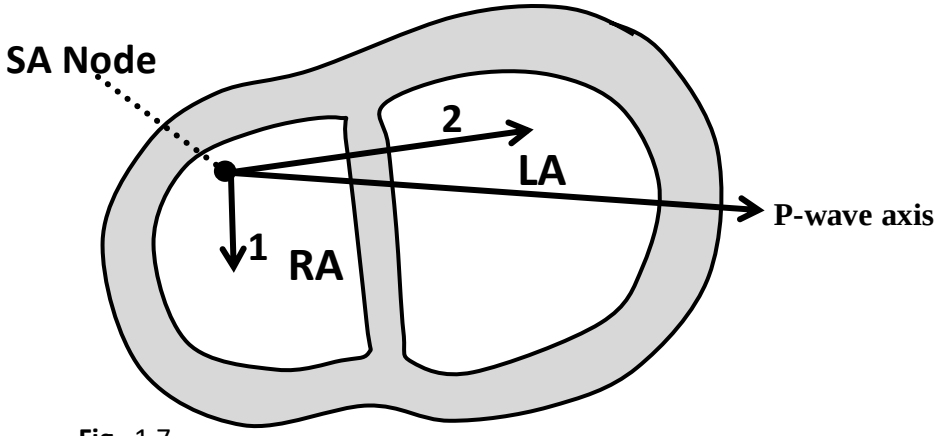


Fig . 1.7

The ECG changes suggestive of left atrial enlargement are discussed as below :

- Notched P wave in limb lead II with the inter-peak gap ≥ 0.04s , **known as P-mitrale**. Total P-wave duration >110 ms.
- Terminal P negativity in lead V1 (i.e., 'P-terminal force') duration ≥ 0.04s, depth ≥ 1 mm.

(Sensitivity = 50% ; Specificity = 90%)

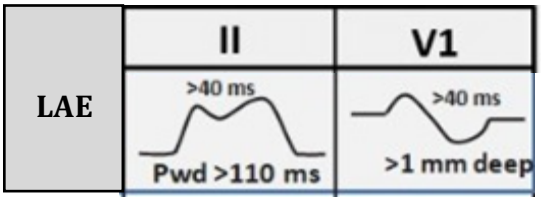


Fig . 1.8

The classical **Morris Index** (mVsec) is the product of the depth of the terminal negative component (in millivolts or millimeter) and its duration (in second). This index cannot be considered as sensitive parameter to detect left atrial enlargement. Therefore , this index must be evaluated with clinical findings and complementary test such as echocardiogram.

- In left atrial enlargement the degree of orientation of P-axis is usually $\leq +45^0$ and accordingly P-wave prominence would be reflected in its nearby lead – either lead II or in lead I or even lead aVL.

Comments :

☐ Atrial fibrillation : Its association with LAE is having increased mortality and it has been claimed to be a cause and complication of left atrial enlargement both.

☐ Stroke : In a study with older people , an increase in left atrial size was found to be an independent risk factor for ischemic stroke. The risk of stroke is more if the person is having simultaneous atrial fibrillation.

☐ Congestive heart failure : The geriatric population has been found to have more incidence of congestive heart failure in association with LAE.

- ☐ The electrocardiographic criteria for detecting LAE is also used to diagnose left ventricular hypertrophy – LVH (This is of more significance with right bundle branch block where the left ventricular hypertrophy criteria become dubious). This is also to be noted that LAE is an early finding in hypertensive heart disease.
- ☐ Sometimes interatrial block pattern may be seen on ECG as a biphasic wave having a notch in between but here the interval in between two peaks is less than 0.04 sec. This pattern is mostly a normal variant.
- ☐ In some patients with ischemic heart disease , a wide P-wave with a left atrium of normal dimension can be observed possibly due to ischemic delay in atrial conduction.

4. Biatrial enlargement

- **In Biatrial enlargement the ECG shows signs of both right and left atrial enlargement**.

The diagnostic clues on ECG are mentioned below :

- In lead II biphasic P-wave is having increased amplitude of both the positive components with increment in the width.
- In V1 the initial component of P is taller and peaked than normal and the terminal component is also deeper and wider.

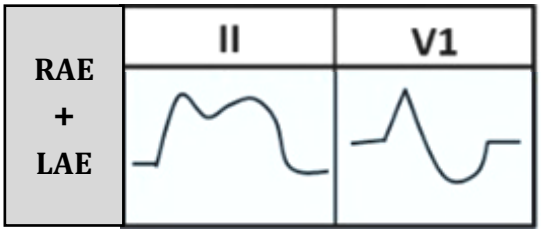


Fig . 1.9

- **Causes of combined right and left atrial enlargement**
  - Mitral stenosis associated with marked pulmonary hypertension or ± with tricuspid regurgitation / stenosis (when the initial component of P-wave is taller than its terminal component , it is sometimes nomenclatured as a 'P Tricuspidale' because of its frequent association with tricuspid valve disease).
  - Atrial septal defect
  - Lutembacher's syndrome : Atrial septal defect associated with acquired mitral stenosis.

5. Concluding remark

- ➢ Two atria (right and left) conjoined together behave as a single electrical unit ; wherein SA node in the right atrium is the point source of electrical flow , normally resulting in two components : the initial component is inscribed by the depolarization of the right atrium and terminal component is inscribed by the depolarization of the left atrium. Thus , the P-wave is the combined deflection of right and left atria both.
- ➢ The P-axis lies normally at 0 to $+75^0$ but mostly in between +45 to $+70^0$. Its axis < $+45^0$ usually reflects left atrial enlargement (LAE) , if > $+70^0$, it reflects right atrial enlargement (RAE).
- ➢ In right atrial enlargement the P-wave becomes taller in both limb lead II and right precordial lead V1. Because this enlargement involves only the early part of the P-wave , any increase in the duration of right atrial depolarization does not result in the prolongation of the total duration of the P-wave.

- Left atrial enlargement causes its depolarization to be delayed , thereby resulting in a positive biphasic P-wave having a notch in between as seen in limb lead II. Over the chest lead V1 , the terminal negative component of the biphasic P-wave is also delayed in its duration and depth both.
- In Biatrial enlargement the ECG shows signs of both right and left atrial enlargement.

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RIGHT VENTRICULAR HYPERTROPHY (RVH) : AN ANALYTIC APPROACH TO ITS ECG DIAGNOSIS

ECG

RIGHT VENTRICULAR HYPERTROPHY (RVH) : AN ANALYTIC APPROACH TO ITS ECG DIAGNOSIS

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OUTLINE

Right Ventricular Hypertrophy (RVH) : An analytic approach to its ECG diagnosis

A Narrative Review

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O heart ! thou are the wonderful bodybuilder causing the ventricular mass to increase its size whenever there is a pressure overload or volume overload. This puts a further strength to the ventricle to push out the blood from its interior to the outside. This is a sort of biological adaptation by which the ventricle gets adjusted to this new set up of working.

- ☐ **Right ventricle is a thin-walled muscular chamber , producing electrical forces which get masked by those generated by the thicker left ventricle. The right ventricle must have a pronounced muscular mass to overcome the masking effect of the left ventricle forces so as to be expressed on 12 lead ECG.**
- ☐ **The main clue to diagnose RVH on ECG is to observe the reversal of precordial pattern , obviously due to the fact that here the right ventricle is having the dominance over the left one.**

The task of diagnosing RVH becomes easier on finding the evidences of right ventricular predominance over the left one. However , the clinicians may face difficulties due to less sensitivite ECG criteria for RVH. Thus , it become essential to review this situation of RVH in depth.

1. Introduction : Keypoints

- ○ Right ventricular hypertrophy (RVH) is rather a physiological-cum-pathological entity being reflected by increase in muscle mass of the right ventricle , usually in response to pressure overload , most commonly as a result of severe lung diseases. COPD is the classical example of such situation.

- ○ A considerable amount of increase in right ventricular muscle mass is needed to reverse the balance towards the right ventricle , dictated also by the reversal of cardiac vector. Since such an association occurs in late stages of right ventricular involvement , the ability of ECG to detect mild or moderate RVH is usually low.

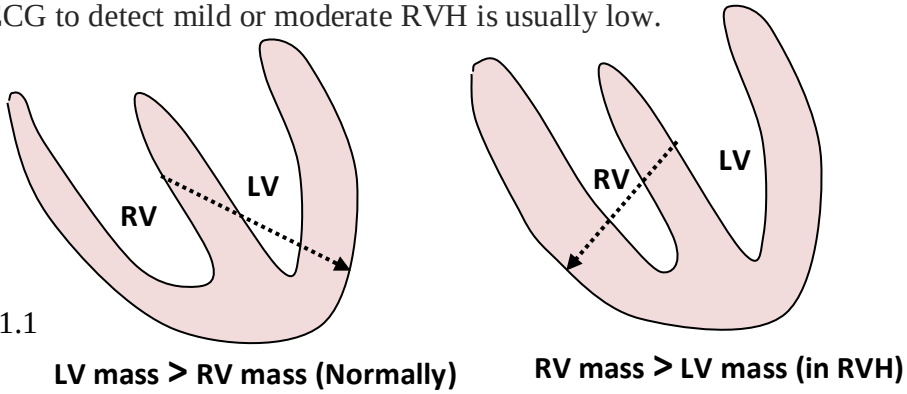


Fig.1.1

RVH : 'Reversal of ventricular muscle mass with its vector '

- The electrocardiographic criteria for diagnosing RVH are well known , which could provide a non-invasive and well-tolerated method of screening such cases , but these criteria are not having a clear cut sensitivity and predictive value in the earlier stages of RVH.
  This takes longer time to show the evidence of RVH on ECG , the increase in right ventricular mass must be sufficient enough to have predominance over the left ventricular mass.

2. Physiological-cum-pathological events in the evolution of right ventricular hypertrophy

- Right ventricle being a thin-walled cavity (free wall thickness 0.3-0.5 cm) imparts a larger distensible volume space , compared to the left ventricle. Thus , this gets initially distended to accommodate pressure overload and volume overload , as per situation.
- Right ventricular hypertrophy sets in gradually to further strengthen its wall. This enables it to pump blood further to the lungs for more oxygen to be delivered.
- This hypertrophy pattern is a physiological and pathological process both in itself. It can be pathological by damaging the ventricular wall when there is excessive laying out of hypertrophy. Much hypertrophic ventricular muscle can supervene in due course of time and the cavity of the right ventricle might be obliterated to the extent that lesser amount of blood would be accommodated inside. Even right ventricle myocardial ischemia may set in occasionally by the compressive impact of hypertrophic muscle on the concerned coronary circulation.
- **Right atrium may get enlarged (RAE) due to the pumping of its contents against resistance created by the hypertrophied right ventricle.**
- **Normally the right ventricle is located anteriorly and to the right of the left ventricle. The hypertrophic right ventricle may be extended somewhat laterally what is known as 'clockwise rotation' on ECG.**

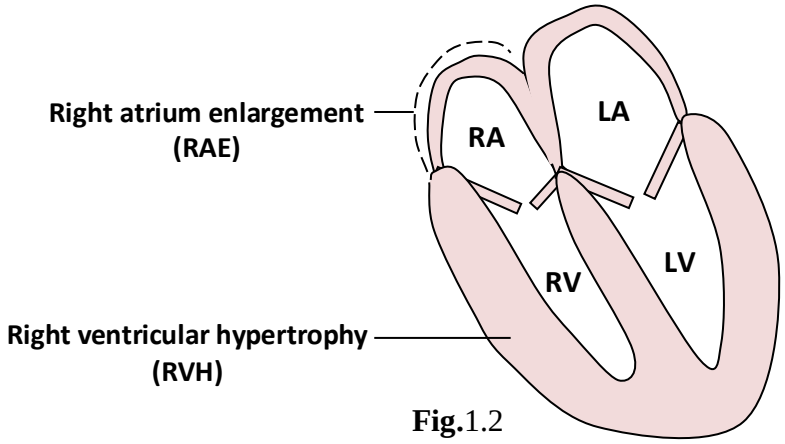


Fig.1.2

Summary of events
Right ventricle hypertrophy (RVH) – Right atrium enlargement (RAE) – extension of the hypertrophied right ventricle somewhat laterally 'clockwise rotation'.

3. Electrocardiographic leads / electrical axis in accessing RVH

This would be more rewarding to interpret ECG if one is well versed – which group of leads are employed to access the different parts of the heart , say the right ventricular chamber in this context. This would be also advantageous to record the concerned electrical axis as well.

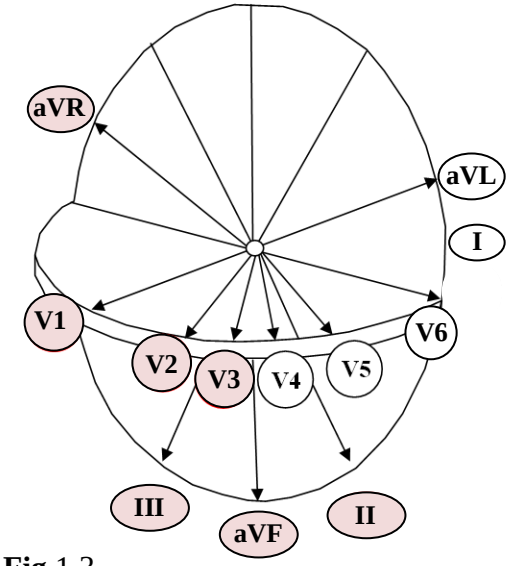


Fig.1.3 **'12-lead system'**

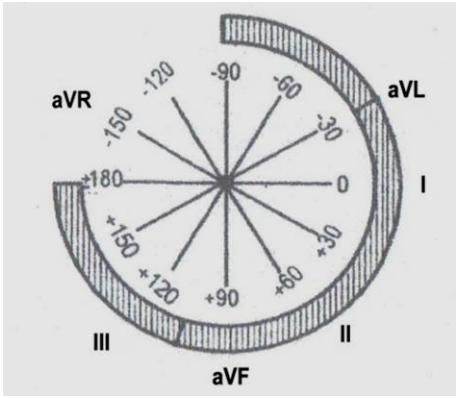


Fig.1.4 **⁕ Hexaxial Lead System 'To determine the electrical axis'**

The leads to be looked at with right ventricular hypertrophy (RVH) :

Right ventricular leads	Left ventricular leads
• Precordial leads : V1 ,V2 (at times V3) to visualise the dominance of right ventricle as R wave. aVR (views outflow tract of the right ventricle and basal part of the septum) • Inferior leads II , III , aVF and lead V1 Right atrial enlargement. Secondary ST-T changes	• Deep S wave in leads (V5-V6 , I , aVL) – **mirror effect of RVH.**

⁕Hexaxial lead system for axis determination

Main principle (Rule of 90^0)

Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive current , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection.

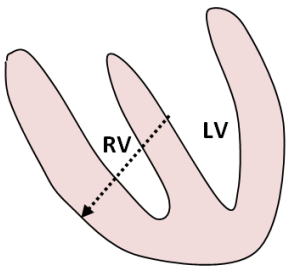


Fig.1.5

The electrical axis is virtually always shifted to the right –
Right axis deviation is almost mandatory with RVH

4. ECG changes in right ventricular hypertrophy

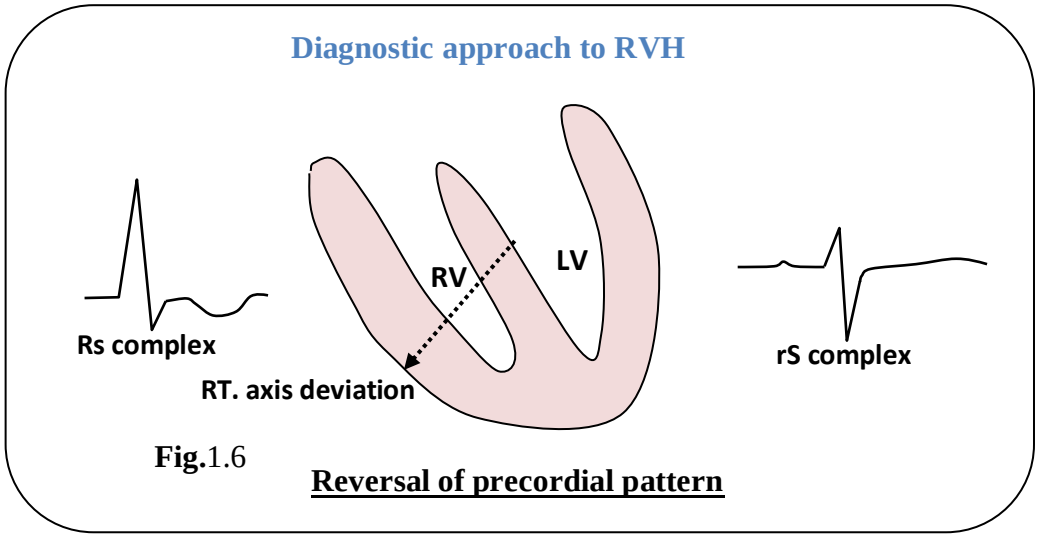


Fig.1.6

Reversal of precordial pattern

ECG features for RVH on ECG

☐ **Diagnostic criteria (Reversal of precordial pattern)**

- Dominant R wave in V1 (> 7mm tall or R/S ratio > 1) with peak time is typically prolonged (35-55 ms)
- Dominant S wave in V5 or V6 (> 7mm deep or R/S ratio < 1).
- Right axis deviation of +110° or more in addition (Virtually the electrical axis is always shifted to the right – almost mandatory)

☐ **Supporting criteria**

- Right atrial enlargement - P-pulmonale : right atrial dilatation / overload.
- Secondary changes : ST depression / T-wave inversion in the right precordial (V1-V2 ,at times V3) and inferior (II , III , aVF) leads due to repolarization abnormalities of the right ventricular myocardium / subendocardial ischmeia.
- Deep S wave in the lateral leads (I , aVL , V5-V6) – mirror effect of RVH
- Dominant S wave is lead I , II and III (S1 S2 S3 syndrome associated with axis deviation superiorly and to the right towards the 'north-west' region)

☐ **Other abnormalities such as**

- Right bundle branch block (complete or incomplete). It signifies dilatation or overload of the right ventricle.

NB : There is a lack of accepted criteria to diagnose RVH in the presence of RBBB. The presence of tall R wave in V1 in association with right axis deviation of 110^0 or more with supporting criteria would be considered as suggestive of RVH.

Further to Say :

☐ **If the ECG criteria is not conclusive of RVH , extended right-sided precordial leads (V3R or V4R) may be explored to obtain R/S ratio greater than 1 as a more reliable indicator of RVH.**

☐ Other criteria based on 12-lead ECG (not commonly used and not well correlated with the echo cardiographic findings)

- Sokolow-Lyon criterion
  R voltage of V1 plus S voltage of V5 or V6 >10.5 mm
- (R 1 + S III) – (S I + R III) <15mm

5. Other issues and diagnostic pearls on ECG : An analytic approach

Clinicians should be alert that they may meet with certain limitations while interpreting right ventricular hypertrophy – **different pathological subsets may be encountered with somewhat different ECG findings**. This fact should also be taken into consideration.

- Typical right ventricular hypertrophic pattern (Rs , R or qR) with right axis deviation is **encountered** with severe RV pressure overload even from causes other than COPD , such as pulmonary stenosis or pulmonary hypertension.
- Pulmonary hypertension with right axis deviation due to COPD is having somewhat different ECG pattern that reflects mainly the low diaphragm resulting from the increased lung volume.

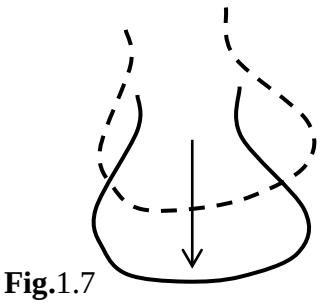
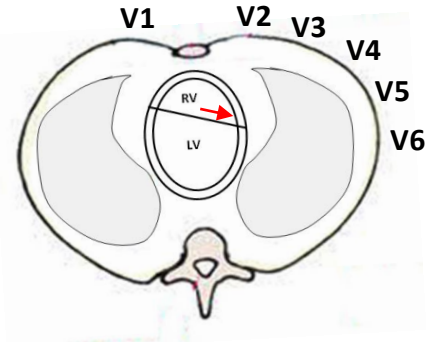


Fig.1.7



Clockwise rotation with overlying emphysematus lungs

I. Low voltage QRS complex (low diaphragm with inflated lungs)
II. Very slow R wave progression with delayed precordial transition zone (due to anteriorly displaced right ventricle with clockwise rotation with overlying emphysematous lungs)

Sometimes predominant R in V1 is not seen in COPD cases in spite of the presence of RVH on cardiac echo. Under this prevailing circumstances **the criteria suggested by Chou is very much of significance , as outlined below**.

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

P wave changes

1. P waves > 0.25 mV in lead II, III, aVF.
2. P wave axis to the right of 80 degrees in the frontal plane.
3. Lead I sign with an isoelectric P wave , QRS amplitude <0.15 mV, and T wave amplitude <0.05 mV.

QRS changes

4. QRS amplitude in all limb leads <0.5 mV
5. QRS axis to the right of 90 degrees in the frontal plane.
6. QRS amplitude <0.5 mV in lead V5 or V6; or R wave <0.7 mV in lead V5 or R wave <0.5 mV in lead V6.
7. R/S ratio < 1 in lead V5 or V6.
8. $S_1S_2S_3$ pattern with R/S ratio <1 in leads I, II and III.

- But this would be worthwhile to mention here that right ventricular involvement due to classical volume overload is having somewhat different picture than RVH , as in Atrial septal defect (secundum). Here the right ventricular overload with somewhat dilatation may be associated with incomplete right bundle branch block pattern in the right precordial leads , most possibly attributed to delayed activation of the hypertrophied right ventricular outflow tract. There is also simultaneous presence of **'Crochetage sign'**. This is defined as M-shaped pattern involving initial 80 ms of the QRS complex in inferior leads.

  Ref : Crochetage Sign JAPI , 2016
  Author : Pratibha Himral, Susheel Kudial, Kailash Nath Sharma, Jitender Kumar
  https://japi.org/y2e4c4/crochetage-sign
  The sensitivity and specificity of crochetage sign is 92-100% in inferior leads for ASD secundum – almost diagnostic.

Incomplete RBBB + crochetage sign = **ASD secundum**.

- Congenital heart disease should be strongly suspected in the presence of right ventricular hypertrophy associated with biatrial enlargement + left ventricular hypertrophy with diastolic overload.

- RVH with P-mitrale in lead II / P terminale in V1 should raise the suspicion of mitral stenosis.

- **Right ventricular preponderance in new born infants :** Newborn infants have right ventricular preponderance with prominent R waves in the right precordial leads and deep S waves in the left lateral precordial leads

Briefly to say (in context with RVH)

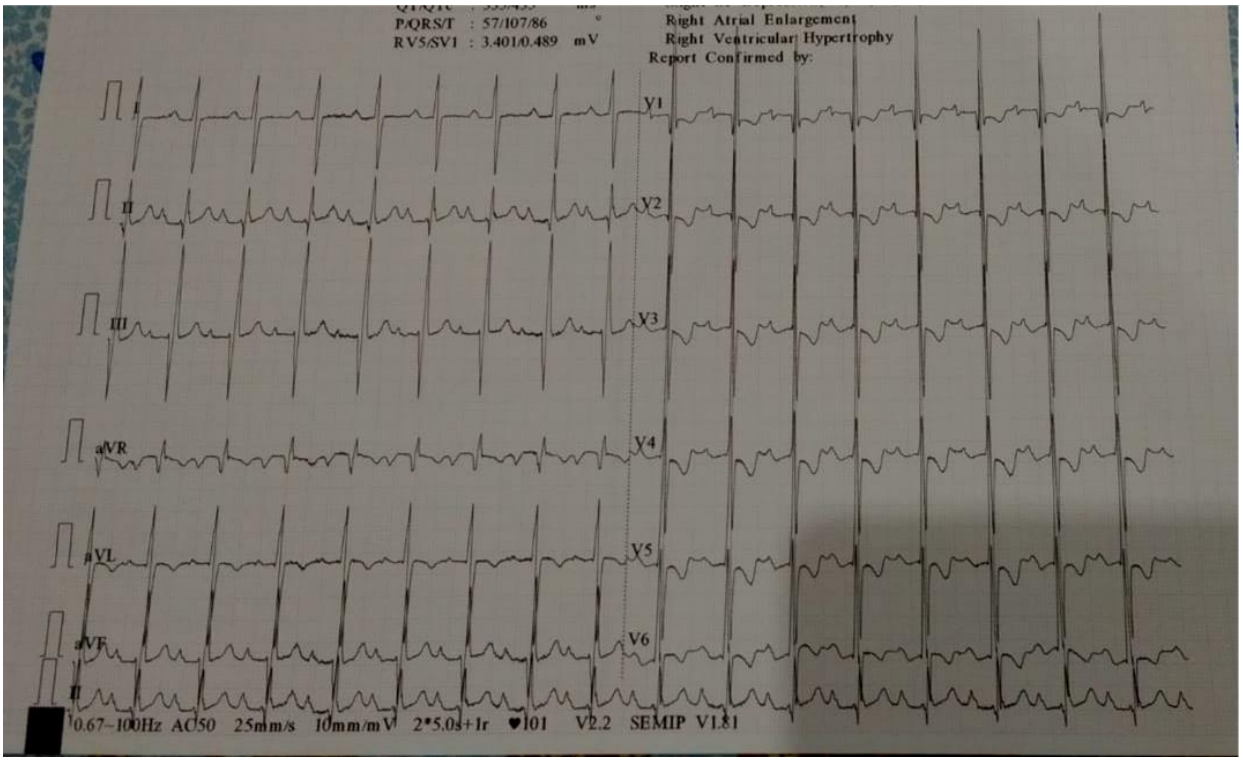
'The greatest accuracy is in congenital heart disease, with intermediate accuracy in acquired heart disease and primary pulmonary hypertension in adults. The lowest accuracy occurs in chronic lung disease'.

Ref : Right Ventricular Hypertrophy on the Electrocardiogram – MY EKG
https://en.my-ekg.com/hypertrophy-dilation/right-ventricular-hypertrophy.html

It becomes essential to record clinical history somewhat in details. Affected patient due to pulmonary hypertension might have symptoms such as excertional chest pain and syncope , peripheral edema and symptoms attributed to passive hepatic congestion (right upper quadrant pain). Likewise symptom in COPD cases would depend on its severity.

6. Illustration by ECGs

ECG No. 1



Source : Dr. Bhanu Pratap Singh , MD , CCEBDM , Senior consultant Physician , Siwan (Bihar)

17 years female (known case of pulmonary hypertension)

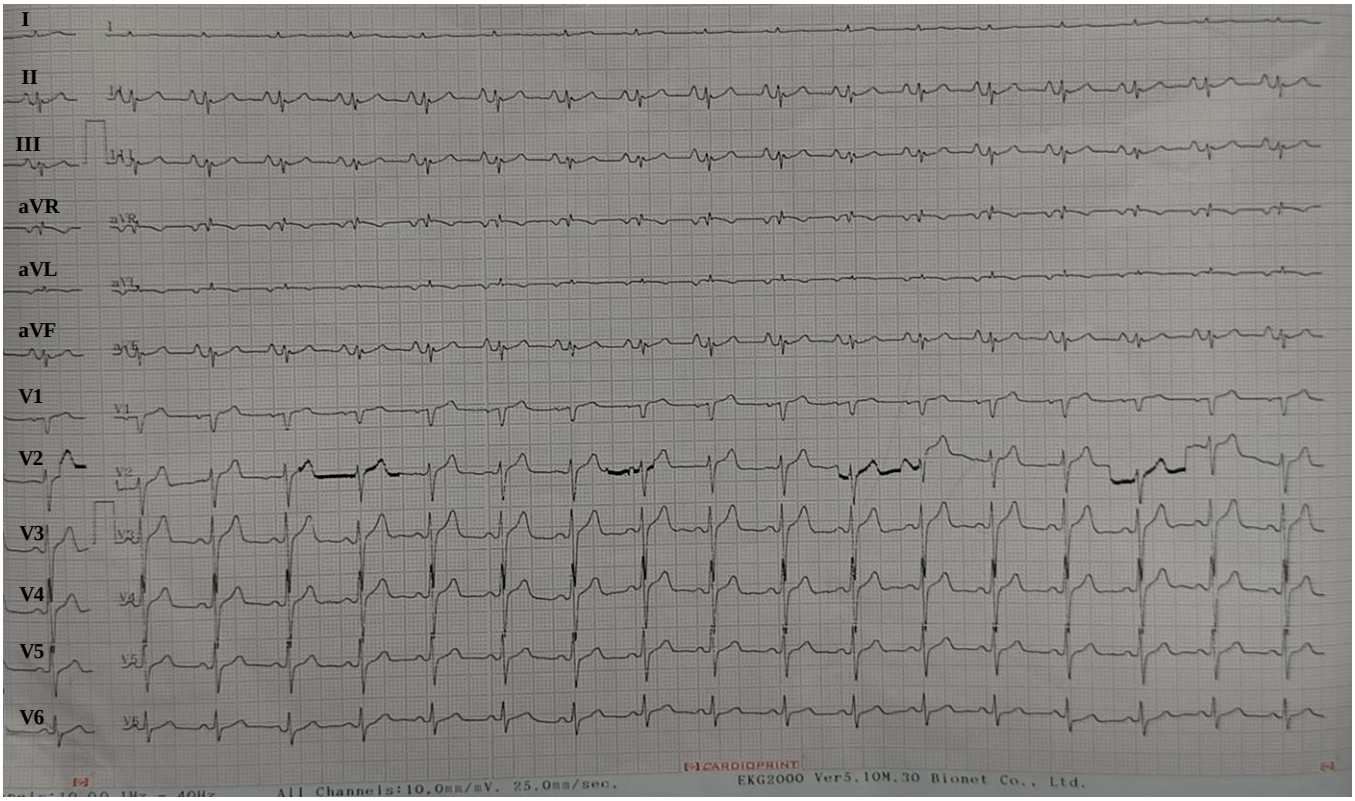
Findings on ECG

- P pulmonale obviously seen in lead II
- qR pattern in V1 (suggestive of right ventricular hypertrophy)
- Secondary ST-T wave changes (strain patten) extending from V1-V6 (with clockwise rotation)

Discussion

Pulmonary hypertension may have its association with multiple clinical conditions as encountered in cardiovascular and respiratory diseases. 12- lead ECG may demonstrate the presence of right ventricular hypertrophy with diffuse ST and T strain pattern. This is usually associated with right ventricular hypertrophy.

ECG No. 2



Source : Dr. M. Gowri Sankar, MD , Senior Assistant Professor in Medicine , Government Medical College & ESI Hospital , Coimbatore

60 years male having smoking habit and working in a cotton mill.
(Known case of COPD since last 15 years)

Findings on ECG

- Over limb leads
  - Low voltage QRS in all limb leads
  - P pulmonale in inferior leads II , III , aVF
  - Lead I sign consistent with modified Schamroth criteria : very low amplitude P , QRS , T wave complexes in lead I
  - P wave axis rightward at about + 80^0
- Over chest leads
  - R wave < 5 mm in lead V6 , R/S ratio < 1 in V5

Discussion

The presence of P pulmonale in leads II , III and aVF ; with low voltage QRS in all limb leads ; P wave axis is rightward shifted on the frontal plane ; presence of lead I sign (as per modified Schamroth criteria). R wave <5 mm in lead V6 , R/S ratio <1 in V5 – all these criteria are in favour of COPD (consistent with Chou's criteria). **Chou's criteria is more beneficial in diagnosing COPD whenever there is a lack of predominant R wave in V1.**

7. Concluding remark

Normally electrical forces generated by thin-walled right ventricle is cancelled by the electrical force of the thicker left ventrcile. The right ventricle should have a pronounced muscular mass to overcome the masking effect of the left ventricular forces so as to be noticed on ECG as right ventricular hypertrophy. Right atrium may also get enlarged (RAE) due to the pumping of its contents against resistance created by the hypertrophic right ventricle. There are certain limitations while interpreting right ventricular hypertrophy – different pathological subsets may be encountered with somewhat different ECG patterns.
Chou's criteria is very useful in the assessment of COPD when definite diagnostic crietria of dominant R wave in V1 is lacking.

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LEFT VENTRICULAR HYPERTROPHY : ITS SPECTRUM OF DIAGNOSIS ON ECG

ECG

LEFT VENTRICULAR HYPERTROPHY : ITS SPECTRUM OF DIAGNOSIS ON ECG

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OUTLINE

Introduction
Left ventricular hypertrophy (LVH) is a physiological-cum-pathological entity in response to an increase in pressure/ volume overload in the left ventricle resulting in an increased left ventricular mass.

A concept of systolic and diastolic overload leading to left ventricular hypertrophy
- ☐ LVH with systolic overload (concentric hypertrophy)
- ☐ LVH with diastolic overload (Eccentric hypertrophy)

Electrophysiology of LVH
- ☐ Left ventricular free wall thickness
- ☐ Delay in impulse conduction
- ☐ New setup of repolarization event

Diagnostic spectrum of LVH on ECG
Various diagnostic criteria have been laid down in accordance with the observed increased amplitude of QRS complex in different left sided leads – either singly or in combination.

Concluding remark

References

Left ventricular hypertrophy : Its spectrum of diagnosis on ECG

A Narrative Review

© **DR. D.P. KHAITAN**
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The heart is gifted with a wonderful dynamics of conduction system so as to have a disciplined path of the supraventricular impulse to flow down simultaneously to both the ventricles – **a symphony of synchronized orchestra.**

The ventricular system of the 'Heart' is possessing two different masses conjoined together, wherein supraventricular electrical force passes through the common point of HIS-BUNDLE and descends down through two separate bundle branches to reach to both the ventricles simultaneously to have its synchronized contraction , **the resultant force is tilted towards the thicker left ventricle.**

- ☐ **The measurement of left ventricular mass on ECG is dependent upon the dominant electrical force passing through this – in true sense it implies the principle of indirect morphometry by accessing the magnitude of its resultant QRS complexes.**
- ☐ **This indirect measurement of left ventricular mass in LVH is considered to be less sensitive compared to its specificity and also true to say the sametime that left ventricular hypertrophy can be diagnosed on ECG with good specificity.**

The datas-pool on LVH morphometry put by different workers is still remains a valuable initial ECG screening test adopted by the clinicians for the purpose.

7. Introduction (keypoints)

- Left ventricular hypertrophy (LVH) is a physiological-cum-pathological entity in response to an increase in pressure / volume overload in the left ventricle resulting in an increased left ventricular mass.
- In context with the term ventricular hypertrophy , this would be a wiser step to mention in the beginning that the adult cardiac myocytes are embryologically final differentiated cells that do not divide in response to any stress. Thus , the ventricular myocytes adapts itself to a pressure / volume overload by the increment in its size , just to strengthen the cardiac pumping system. **This hypertrophy is a sort of compensatory remodeling of the concerned ventricular system.**
- The electrocardiogram is the least expensive and more readily available tool , often used as a screening test to evaluate the individual who would undergo further testing. This is worthwhile to mention here that left ventricular hypertrophy can be diagnosed on ECG with good specificity.

- The hypertrophied left ventricles yields a larger area of myocardium for electrical activation to pass through ; the resultant increased amplitude of the QRS complex with its different indices creates the basis of recording electrical changes on ECG.

- **Why to study Left ventricular hypertrophy (LVH)**
  - LVH is an independent risk factor for cardiovascular mortality , associated with a 60% increased risk of sudden death. There are so many associated long term consequences in this context :
    - Chronic cases of hypertension might be associated with coronary artery disease + heart failure.
    - LVH sets in an earlier progression of CKD possibly because of associated high hypertension prevalence , including the occurrence of frequent nocturnal hypertension. Virtually all patients with end-stage renal disease (ESRD) have LVH.
    - LVH is mechanically a potential substrate of ventricular arrhythmias due to the development of subsequent fibrosis , altered action potential duration and increased incidence of early afterdepolarization dynamic.
    - In addition there are so many risk factors of LVH prevalent in the society - such as older people generation , obese individuals , diabetic patients , positive family history for LVH , etc.
  - Recent studies have revealed that LVH regression is feasible to be achieved with improved clinical outcome by the use of certain drugs , particularly angiotensin receptor blockers (ARBs) and angiotensin-converting enzyme inhibitors (ACE-Is).
  - This is also pertinent to known that " LVH is more prevalent in women on ECG compared with men, occurring in 17% of women with SCD and 10.6% of men ($P < .001$). Additionally, LVH combined with repolarization abnormalities was also more common in women than men (8.2% vs. 4.9%) . A promising line of investigation is whether ECG LVH may convey sex-specific information with respect to risk of SCD that may reflect electrical rather than anatomic remodeling."

    Ref : Sex-specific risk assessment of sudden cardiac death

    Anne M. Kroman , Kristen K. Patton , in Sex and Cardiac Electrophysiology, 2020

    https://www.sciencedirect.com/topics/medicine-and-dentistry/left-ventricular-hypertrophy

8. A concept of systolic and diastolic overload leading to left ventricular hypertrophy

This becomes important and essential to discuss the concept of pressure overload (systolic LVH) and volume overload (diastolic LVH) because these two conditions are associated with somewhat different repolarisation abnormality on ECG.

The systolic LVH is mainly associated with afterload mechanism, while diastolic LVH is associated with preload mechanism. Both these processes are having somewhat different remodeling changes over the left ventricle.

With no doubt to say that LVH is a compensatory mechanism which ultimately leads to an abnormal increase in the mass of the myocardium. The most common factors causing an elevated afterload are seen in hypertension and also seen in aortic stenosis. The second group is due to an increased filling of the left ventricle , induced by diastolic preload , commonly seen with regurgitant valvular lesions such as aortic regurgitation or mitral regurgitation and also with dilated cardiomyopathy.
On the basis of discussion as above LVH is classified into two subtypes :

i) With systolic overload and ii) With diastolic overload

The differences in between these two subsets can be visioned as noted below :

☐ **LVH with systolic overload** (the heart is contracting against an elevated afterload)

Pressure afterload (as in hypertension , aortic stenosis) → gradual evolution of **concentric hypertrophy** → a compressive impact over the myocardium, mainly subendocardial region → a marked secondary repolarization abnormality , as reflected by ST depression and asymmetrical T-wave inversion on ECG.

☐ **LVH with diastolic overload** (response to volumetric overload)

Volume preload (as in regurgitant valvular lesions , dilated cardiomyopapthy) → increased tension over myocardium → **eccentric hypertrophy** → negligible compressive effect over subendocardial region → repolarization abnormality , as reflected by prominent q wave and taller T-wave on ECG.

- Concentric LVH is an abnormal response to long standing increased resistance to the ventricular outflow , commonly resulting from pressure overload as occurs in cases of chronic hypertension or aortic stenosis.
- Ecentric LVH is induced by an increased preload filling of the left ventricle, termed as diastolic overload, the underlying causes are seen in patients with regurgitant valve lesions such as aortic or mitral regurgitation , also in the case of dilated cardiomyopathy.

Both these conditions are associted with a significant increase in the ventricular wall stiffness with increase in diastolic ventricular pressures , which can also be subsequently transmitted back into the left atrium as well as in the pulmonary vasculature

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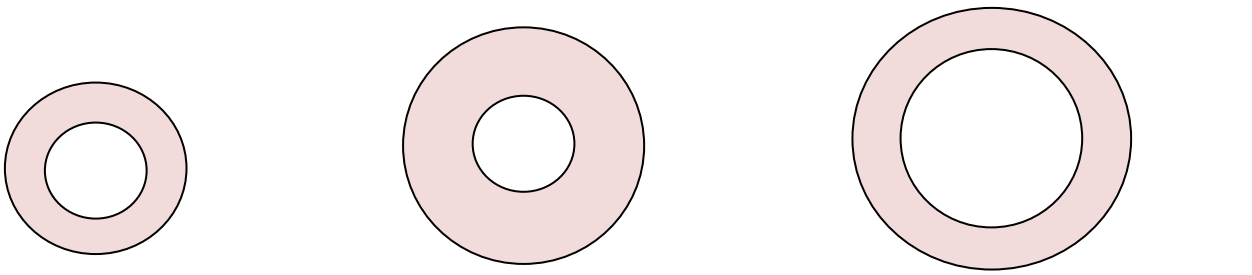
A concept of concentric and ecentric hypertrophy



Normal

Concentric hypertrophy
(The hypertrophied mass extending towards the centre of LV cavity , causing its marked obliteration)

Ecentric hypertrophy
(Dilated LV cavity , around which the hypertrophied mass is laid down without the obliteration of its cavity)

Fig. 1.1

9. Electrophysiology of LVH

The electrophysiology of LVH can be discussed under the following subheads :

- ☐ Left ventricular free wall thickness
- ☐ Delay in impulse conduction
- ☐ New setup of repolarization event

☐ **Left ventricular free wall thickness**

The axis direction of increased voltage in left ventricle is not having well established reasoning but it is said to be related to the altered geometric projection of its electrical forces , rather than increased ventricular muscle mass by itself.

The left ventricle shows arrangement of its myofibres in a spiral way – one group is with rapidly descending spiral loops and the second is with its less rapidly ascending spiral whorls , as illustrated with this sketch :

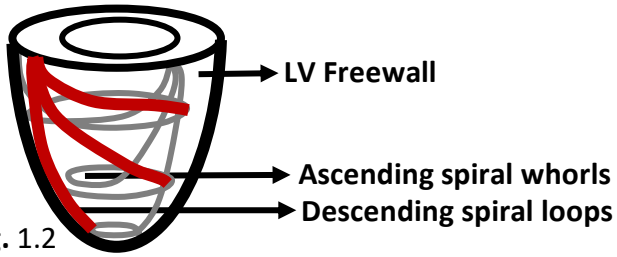


Fig. 1.2

The impact of LVH is on the geometric projection of this spiral arrangement of the concerned myofibrils. The resultant electrical force in the left ventricle is reflected as increased amplitude of R-wave over the left ventricular leads and the corresponding deep S-wave in leads represented by non-hypertrophied right ventricle.

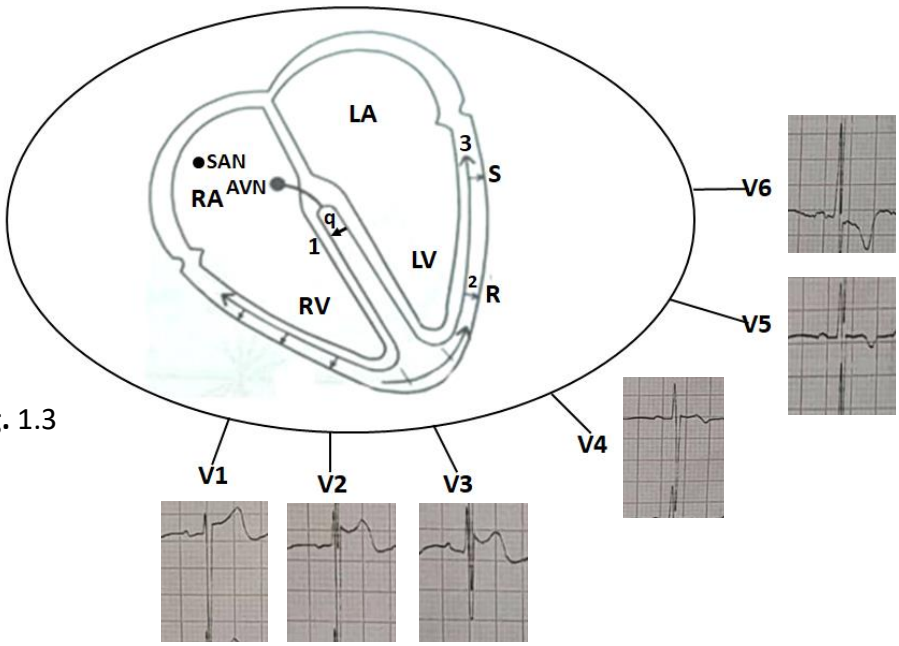


Fig. 1.3

<u>Over the Horizontal plane (chest leads) :</u> In LVH the predominant electrical force is directed towards left ventricular leads V5-6 with waves morphology as per sequential activation of the ventricular system from right to left (V1 to V6) , as illustrated with the above ECG tracings.

Over the frontal plane (limb leads) :

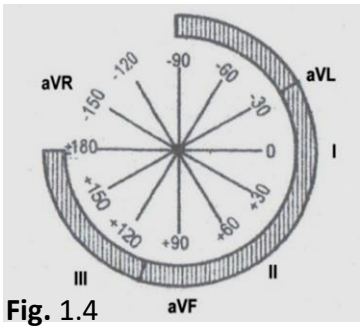


Fig. 1.4

The electrical axis may be shifted more towards the left – even as left axis deviation which lies in between -30^0 to -90^0 (normal axis lies in between -30^0 to $+90^0$). The extent of axis deviation would depend upon the severity of LVH. In mild cases the axis may be within normal limits , more nearer to -30^0 i.e. the couterclockwise movement of the electrical axis depends upon the severity of LVH and the increased amplitude of the resultant QRS would be running parallel with the recorded electrical axis , as per rule of 90^0 , as stated below :

Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive current , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection

The maximum positive deflection is in the lead , lying nearest to the electrical axis.

☐ **Delay in impulse conduction**

The QRS originating from hypertrophied ventricle is widened due to delayed conduction through the thickened muscular wall but does not exceed 0.12 sec.

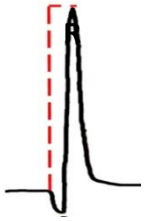


Fig. 1.5

- This delay in impulse conduction can also be demonstrated by **an increase in the left ventricular activation time (VAT) in left oriented leads, duration of more than 0.05 sec (50 ms).**
  This is a more sensitive indicator with upto 90% sensitivity to indicate the degree of left ventricular stiffness with diastolic dysfunction.

☐ **New setup of repolarization event**

The pattern of repolarization abnromalities (ST and T-wave changes) is dependent upon whether it is systolic overload or diastolic overload in the left ventricle.

- **LVH with systolic overload**

  The left ventricle is strained due to systolic overload , there is more compression over the subendocardium and also there is relative ischemia of the same zone – creating a subendocardial zone of relative strain injury resulting in ST depression and asymmetrical T-wave inversion

  Since the exploring electrode is placed over the leads facing epicardium away from subendocardium , there is downward ST depression and asymmetrical T-wave inversion.

  In this context ST depression with inverted T- wave inversion indicates left ventricular hypertrophy with systolic overload.

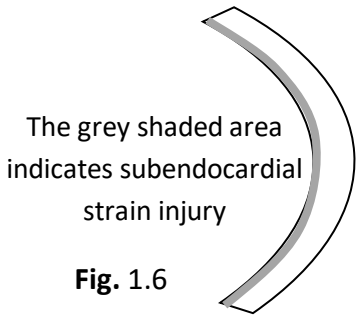
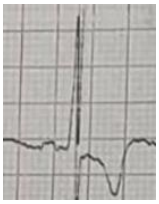

The grey shaded area indicates subendocardial strain injury

Fig. 1.6


ST depression and asymmetrical T-wave inversion (somewhat delay in impulse conduction through the hypertrophied LV also causes the shift of epicardial repolarization towards endocardial zone – this also attributes to these secondary ST and T changes)

- **LVH with diastolic overload**
  LVH wtih diastolic volume overload also causes forceful contraction of the left ventricle but without systolic overload resistance , that's why , there is no zone of subendocardial strain induced subendocardial injury. Here this event of diastolic overload is associated with somewhat more prominent septal wave depolarization (prominent q wave) and with prominent force of T-wave repolarization (prominent upward T-wave).
  In this context prominent upward T-wave with prominent initial q of QRS complex indicates left ventricular hypertrophy with diastolic overload.

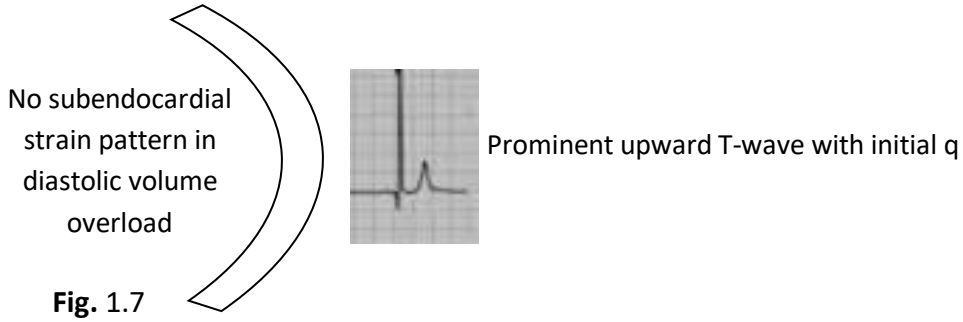


Fig. 1.7

10. Diagnostic spectrum of LVH on ECG

- **Electrophysiolgical basis**
  - The resultant increased voltage of the QRS complex with its different indices creates the basis of diagnostic spectrum of LVH on ECG.
  - This results in increased R wave amplitude in the left oriented leads (I, aVL and V5-6) and increased S wave depth in the right-sided leads (V1-2).
  - The corresponding secondary ST-T changes are recorded in left-sided leads , and also in inferior leads II , III and aVF , as per direction of electrical axis on the frontal plane.

- **Various diagnostic criteria**
  - Various diagnostic criteria have been laid down in accordance with the observed increased amplitude of QRS complex in different left sided leads - either singly or in combination.
  - The evidence of increased amplitude of QRS complexes consequent to LV hypertrophy is not seen in all the left sided leads , most possibly due to the projection of altered geometric projection of the spiral fibres with respect to concerned leads.
  - Therefore , the sensitivity of detecting LVH is low while the specificity is somewhat high. Further to say , ECG changes are said to be insensitive means of detecting LVH but it is also true to say that left ventricular hypertrophy can be diagnosed on ECG with good specificity.
  - The predictive value of the voltage criteria is cumulative i.e. the more voltage criteria makes the greater likelihood of LVH.

- There are various voltage criteria for diagnosing LVH on ECG, as summarized below
- Voltage criteria must be accompanied by non-voltage criteria such as increased ventricular activation time (VAT) in leads V5 or V6 ; accompanying ST-T changes in the concerned leads.

The most commonly used ECG criteria for left ventricular hypertrophy (LVH) – are summarized below :

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%).

Cornell-voltage criteria

- Male: $S_{(V3)} + R_{(aVL)}$ > 28mm
- Women: $S_{(V3)} + R_{(aVL)}$ > 20 mm

Sensitivity 42%, specificity 95%

In the European Society of Hypertension / European Society of Cardiology , the Sokolow-Lyon voltage criterion was recommended as a part of all routine assessment of patient with hypertension.
The Sokolow-Lyon voltage criteria was successfully used together with the Cornell voltage duration product in detecting patient with LVH in the Lorsatan Intervention for Endpoint Reduction in Hypertension (LIFE) study.

Cornell product criteria

- (R_{aVL}+ S_{V3}) • QRS duration > 2440 mVms
  This criteria also takes QRS duration in consideration – a better approach.

✓*Presumably the best index. Sensitivity 51%, specificity 95%.*

The next important LVH criterion is Romhilt-Estes Point scoring system. This scoring system includes QRS voltage criteria + voltage independent signs (QRS duration , VAT in V5 or V6 , left atrial enlargement , left axis deviation and left ventricular strain) and so this criterion considers all the possible ECG parameters together for the assessment of LVH.

Romhilt–Estes point score	(1) Any limb lead R or S ⩾20 mm or $S_{V1/2}$⩾30 mm or $R_{V5/6}$⩾30 mm **(3 points)** (2) Left ventricular strain pattern with digitalis (1 point) / without digitalis **(3 point)** (3) Left atrial enlargement **(3 points)** (4) Left axis deviation **(2 points)** (5) QRS duration ⩾ 90 ms **(1 point)** (6) Intrinsicoid QRS deflection of ⩾50 ms in V5/6 **(1 point)**	⩾ 5 points–definite LVH 4 points–probable LVH 3 points–unlikely LVH

Other miscellaneous voltage criteria attributed to

limb leads

- R-wave in lead I + S-wave in lead III > 25 mm
- R-wave in aVF > 20 mm
- S-wave in aVR > 14 mm

Precordial leads

- R-wave in V4 , V5 or V6 >26 mm
- Largest R-wave plus largest S-wave in precordial leads > 45 mm
- S in V2 + R in V6 > 43 mm
- Normally R-wave is larger in V5 compared to V6. With reverse pattern there is likelihood of LVH.

Total QRS voltage in combination

- if > 175 mm , it indicates LVH

Supporting Non-voltage criteria

An ECG diagnosis of LVH must be accompanied by non-voltage criteria.

- Increased R-wave peak time (VAT) > 50 ms in lead V5 or V6
- ST and T changes as discussed previously with systolic overload / diastolic overload

NB : Some important consideration while considering LVH

- The combination of two different ECG criteria for left ventricular hypertrophy can improve risk stratification compared with either criterion alone.
- The persistence or development of ECG left ventricular hypertrophy by both Cornell product and Sokolow-Lyon voltage was associated with >3-fold increased risks of myocardial infarction, stroke, cardiovascular mortality : the composite end point of these three outcomes, and all-cause mortality after adjusting for other known or suspected predictors of risk.
- **The predictive value of the voltage criteria is cumulative i.e the more voltage criteria make the greater likelihood of LVH.**
- The secondary changes are greatest in the lead with the tallest QRS complex
- Lean individuals tend to have higher voltage as pseudo LVH due to a shorter distance between the heart and the electrode.
- The distance between the heart and the electrodes is greater in obese persons , as well as those with COPD due to hyperinflation of the lungs – the voltage criteria of LVH is less sensitive in these individuals.
- Athletes will often show large QRS amplitude due to their ventricular remodelling as the evidence of LVH but they do not have pathological hypertrophy.
- It would be worthwhile to mention here that LVH in due course of time (with long standing cases) might show the evidence of MI / coronary insufficiency and the reverse is also true , the old cases of MI may also show compensatory LVH.
  If LVH with systolic overload is associated with coronary insufficiency , the T-wave would show symmetrical and deep inversion in the concerned leads.

11. Concluding remarks

- ☐ Left ventricular hypertrophy (LVH) is a physiological-cum-pathological entity in response to an increase in pressure / volume overload in the left ventricle resulting in an increased left ventricular mass.
- ☐ LVH is an independent risk factor for cardiovascular mortality , associated with a 60% increased risk of sudden death. There are so many associated long term consequences in this context :
- ☐ Concentric LVH is an abnormal response to long standing increased resistance to the ventricular outflow , commonly resulting from pressure overload as occurs in cases of chronic hypertension or aortic stenosis.
- ☐ Ecentric LVH is induced by an increased preload filling of the left ventricle, termed as diastolic overload, the underlying causes are seen in patients with regurgitant valve lesions such as aortic or mitral regurgitation , also in the case of dilated cardiomyopathy.
- ☐ The axis direction of increased voltage in left ventricle is not having well established reasoning but it is said to be related to the altered geometric projection of its electrical forces , rather than increased ventricular muscle mass by itself.
- ☐ Various diagnostic criteria have been laid down in accordance with the observed increased amplitude of QRS complex in different left sided leads - either singly or in combination.
- ☐ The predictive value of the voltage criteria is cumulative i.e the more voltage criteria make the greater likelihood of LVH.

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BIVENTRICULAR HYPERTROPHY (BVH) ON ECG

ECG

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OUTLINE

Biventricular hypertrophy (BVH) on ECG

A Narrative Review

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A tug of War by rope pulling in between two opponent groups on either side is a very famous sports game. Each group tries with its maximum effort to pull the opponent towards its side by cancelling the efforts of each other. This is obvious that both the groups apply their maximum efforts but the effort of the winner group is mainly appreciated by the surrounding , annulling the effort of looser one. Only the careful eyes can see the implied physical forces by both the groups.

The same story is true with biventricular hypertrophy wherein both the ventricles are having their dominating electrical forces – a ground created to have a tug of war in between.

- ☐ **On the horizontal plane both the hypertrophied ventricles try to imprint their dominating images on ECG.**
  (RVH over V1-2 , V3/V4 transition zone may have more or less equal but larger biphasic R/S waves and LVH over V5-6)
- ☐ **But on the frontal plane there is added element of axis deviation - usually toward the right in favour of RVH.**

NB : ± Associated evidence of atrial enlargement (lt/rt)

This becomes a challenging task to the clinicians how to witness this intangible problem with biventricular hypertrophy on 12-lead ECG.

1. Introduction

- Biventricular hypertrophy simply means that both the left and right ventricles are simultaneously hypertrophied. The 12 lead ECG has a low sensitivity for such a diagnosis of biventricular hypertrophy , as the counteracting left and right ventricular forces tend to cancel each other.
- In clinical practice biventricular hypertrophy may be seen with :
  - Dilated cardiomyopathy
  - Congential heart disease – Eisenmenger syndrome
  - Valvular lesions (as in rheumatic heart disease)
  - Hypertensive congestive heart
  - High cardiac output status ± failure
- This is worthwhile to mention in this context that in biventricular hypertrophy the electrical forces governing the left ventricular hypertrophy dominate over right ventricular hypertrophy ; thereby characteristic pattern of LVH is obviously witnessed on ECG. But there needs a careful observation to see the concomitant RVH in addition by utilizing the laid down criteria with biventricular hypertrophy.

- In 1937 Katz and Watchel laid down the foundation of biventicular hypertrophy by examining the ECGs of 43 children with congenital heart disease and proposed a very important and pertinent diagnostic criteria – popularly known by their combined name as **'Kartz and Watchel phenomenon'** on ECG.

2. Natural progression of ECG changes in biventricular hypertrophy

Normally on ECG tracings there is left ventricular predominance over the right ventricle. Even with biventricular hypertrophy this also stands true – the forces of left ventricular hypertrophy usually dominates over right ventricular forces ; thereby characteristic pattern of LVH is usually well recorded in left precordial leads. Sometimes right ventricular hypertrophy may also dominate and becomes equal to LVH , then ECG changes of each ventricular hypertrophy could be obvious in leads representing them , with transition zone remaining more or less at normal position (V3/V4) – equiphasic R/S waves with increased amplitude indicating a balanced state of right and left ventricular forces.

Natural history of progression

LVH ⟶ a balanced state when RVH equals to LVH with transition zone remaining as such at normal position (V3/V4) – ECG changes of each ventricular hypertrophy are recorded in the concerned leads. The transition zone may be shifted on either side as per their relative predominance.

3. Electrocardiographic criteria of diagnosing Biventricular hypertrophy (BVH)

Many criteria have been laid down for the electrocardiographic (ECG) diagnosis of biventricular hypertrophy (BVH). This would be worthwhile to mention here the observation made by Jain et al. , who studied electrocardiographic patterns in patients who also showed the evidence of biventricular hypertrophy (BVH) on cardiac echo , as well. They included 69 such patients in their study group –

All the concerned ECGs of such patients were analysed by using the established criteria in the diagnosis of LVH and RVH. Their study group had 25% of such cases consistent with BVH , 30% cases with LVH and 20% cases with RVH.

The sensitivity of ECG criteria was 24.6% with specificity 86.4% in their study. They pointed out the difficulty of ECG in diagnosing RVH but they concluded that the ECG had a low sensitivity but satisfactory specificity.

'An S wave in V5/V6 > 7 mm was the most frequent finding in 17 patients with BVH on the electrocardiogram' (Later this was also included as a diagnostic criteria in BVH).

Ref :

Electrocardiographic patterns of patients with echocardiographically determined biventricular hypertrophy

A Jain[1], H Chandna, E N Silber, W A Clark, P Denes

J Electrocardiol. 1999 Jul;32(3):269-73.

https://pubmed.ncbi.nlm.nih.gov/10465570/

In order to understand the diagnostic criteria with biventricular hypertrophy on ECG it would be better to correlate this with the anatomical arrangement of hypertrophied ventricles in relation to the cardiac plane , as sited below :

Basic ECG changes as per anatomical arrangement of hypertrophied ventricle

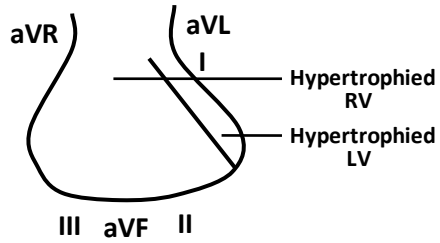


Fig. 1.1

On the Vertical plane

- Normally right ventricle lies anteriorly and medially to the left ventricle , both the hypertrophied right and left ventricles occupy the same position respectively , but with RVH the right ventricle may be somewhat shifted to the left.
- **On ECG** , Right axis deviation (due to associated RVH) ± evidence of atrial enlargement

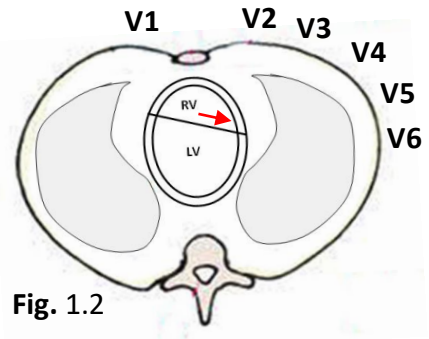


Fig. 1.2

(the red arrow depicted above points toward RVH with clockwise rotation)

On the horizontal plane

- The hypertrophied right ventricle occupies the area more or less corresponding to the leads V1-V2 but with somewhat more extension towards the left , and the left hypertrophied ventricle occupies the area corresponding to leads V5-V6 with the transition zone in between.
- **On ECG** : Both the hypertrophied ventricles record tall R-wave in their respective precordial leads. Since there is a clockwise rotation due to associated RVH , it causes deep S-wave in leads V5 or V6. Transitional zone with larger equiphasic R/S may start at V3/V4 or at times it may be shifted on either side as per relative predominance of either hypertrophied ventricle.
  ± evidence of atrial enlargement

Based on the above discussion the diagnostic ECG criteria for biventricular hypertrophy (BVH) is laid down as follows :

- ☐ **Features of LVH with one of the followings**
  - Right axis deviation (greater than 90^0)
  - Clockwise rotation of the heart associated with RVH causes the leftward shift of the right ventricle. This happen so when the right ventricular hypertrophy is associated with right ventricular dilatation as well.
  - A relatively tall R-wave in lead V1 , especially with an R/S ratio > 1 which is frequently observed in congenital heart disease (such as in VSD and PDA) with pulmonary hypertension - the Eisenmenger syndrome. This gets manifested on ECG as :
    - Tall R-waves in both right and left precordial leads
    - **With** large equiphasic QRS complexes in the mid precordial leads – 'Kartz and Watchel phenomenon' (the total sum of R and S may approach ≥ 60 mm in children or ≥ 50 mm in others)

☐ **Left atrial abnormality (LAE) with one of the followings**

- R/S in V5 or V6 is ≤1
- S wave in V5 or V6 ≥7 mm

This points towards clockwise rotation associated with RVH

- Right axis deviation – when marked , this causes greater R-wave than the corresponding Q wave in lead aVR.

NB : One should try to pickup the diagnostic signals coming from both the hypertrophied ventricles – and the mind should be alert to grasp the simultaneous presence of clockwise rotation associated with RVH and to analyze all the electrocardiographic findings accordingly. 'Kartz and Watchel phenomenon' should also be accessed if present therein.

4. Illustration by ECG

29 years old female with h/o large PDA since age 2 ; with a history of SOB since early childhood with cyanosis for last 10 years (established case by cardiac echo)

Source : Dr. Neel Snehal R. , DM Cardiology resident, GMC & SSH , Nagpur

(ECG tracings have been arranged as per horizontal and frontal planes for better understanding)

ECG findings on horizontal plane

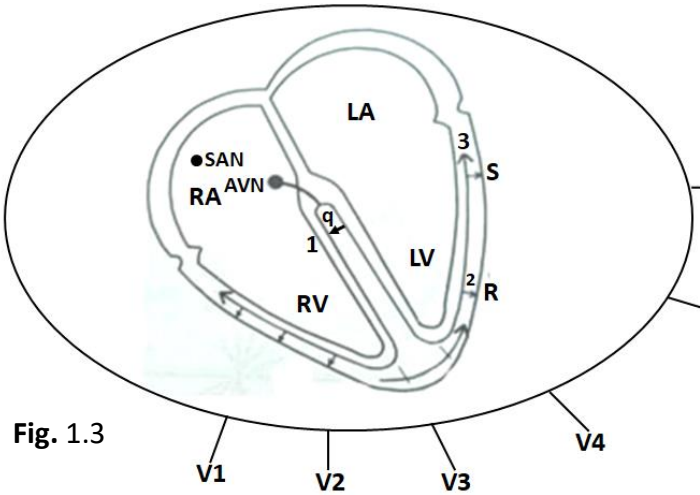


Fig. 1.3

Tall R with deeper S wave > 7 mm in V6 with the background of clockwise rotation associated with prominent upright T-wave (Diastolic overload)

Tall R with secondary ST -T changes (Systolic overload) plus prominent P-terminale (LAE)

Transition zone with prominent R/S (larger R-wave and deeper S-wave)

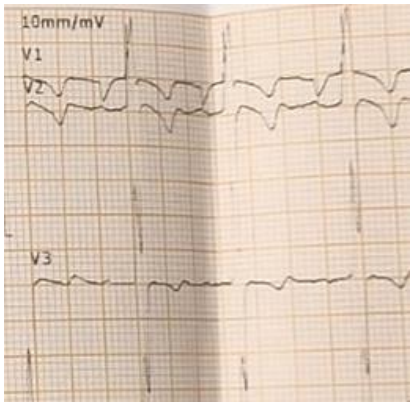


Fig. 1.4

- **Tall R-wave in both right and left precordial leads with deep S-wave (> 7 mm) in V6 with the background of clockwise rotation**
- **Kartz and Watchel phenomenon (V3)**
- **Associated LAE as in V1**

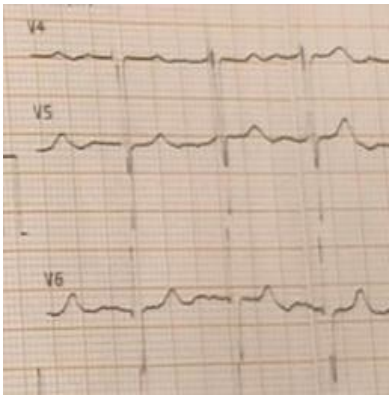
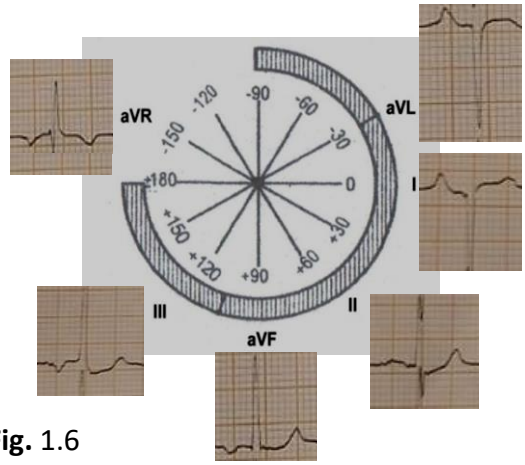


Fig. 1.5

ECG findings on frontal plane (The tracings are of the same patient but a sincere effort is applied to make them somewhat darker with a view to have more clarity)

R is more in amplitude than corresponding Q in lead aVR (this happens so due to marked right axis deviation - here at +140^0)



Negative QRS complexes in both leads aVL and I point towards **right axis deviation**

Fig. 1.6

Comments :

The combination of biventricular hypertrophy (BVH) with left atrial enlargement (LAE) in this 29 years aged female patient with early childhood cyanosis for last 10 years points towards PDA (Eisenmenger syndrome).

(Such is the proven case on cardiac echo)

The ductus arteriosus is a vascular channel which connects the pulmonary trunk and the aorta distal to the origin of left subclavian artery. Normally it gets closed immediately after birth. Persistent patency of the ductus after birth is relatively common, occuring more frequently in female.

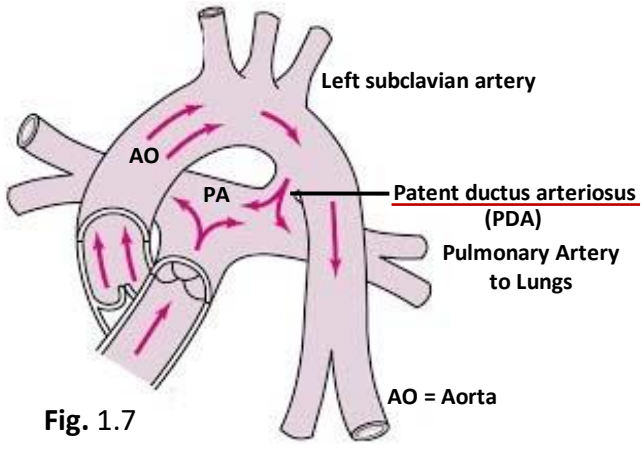


Fig. 1.7

Illustration to show the basic hemodynamic flow in PDA

HEMODYNAMIC FLOW

Without Reversal of Shunt Non-cyanotic state

Aortic flow through PDA and incoming flow from right ventricle → both pass through pulmonary artery to the lungs →left atrium and then to the left ventricle → Volume overload leading to LAE and LVH with diastolic overload

With Reversal of Shunt Cyanotic state

Elevation of pulmonary vascular pressure , exceeding that of aorta → right ventricular hypertrophy (RVH) → reversal of shunt (R→ L) , a condition known as Eisenmenger syndrome

5. Concluding remark

☐ Biventricular hypertrophy simply means that both the left and right ventricles are simultaneously hypertrophied. The 12 lead ECG has a low sensitivity for such a diagnosis of biventricular hypertrophy , as the counteracting left and right ventricular forces tend to cancel each other.

- ☐ Observation made by Jain et. al in their study showed that the sensitivity of ECG criteria was 24.6% with specificity 86.4%. They pointed out the difficulty of ECG in diagnosing RVH but they concluded that the ECG had a low sensitivity but satisfactory specificity.
- ☐ There may be signs of both Left Ventricular Hypertrophy (**LVH**) and Right Ventricular Hypertrophy **(RVH)** on the same ECG – i.e. positive diagnostic criteria for LVH with some additional features suggestive of RVH.
    - Right axis deviation (>90°) – This never occurs in LVH as such
    - Deep S-wave in V5 or V6 (>7 mm) – pointing towards clockwise rotated RVH
    - Large RS complexes in multiple leads
    - Evidence of atrial enlargement.
- ☐ One should not hesitate to have cardiac echo of the concerned patient as a confirmatory evidence of biventricular hypertrophy.

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