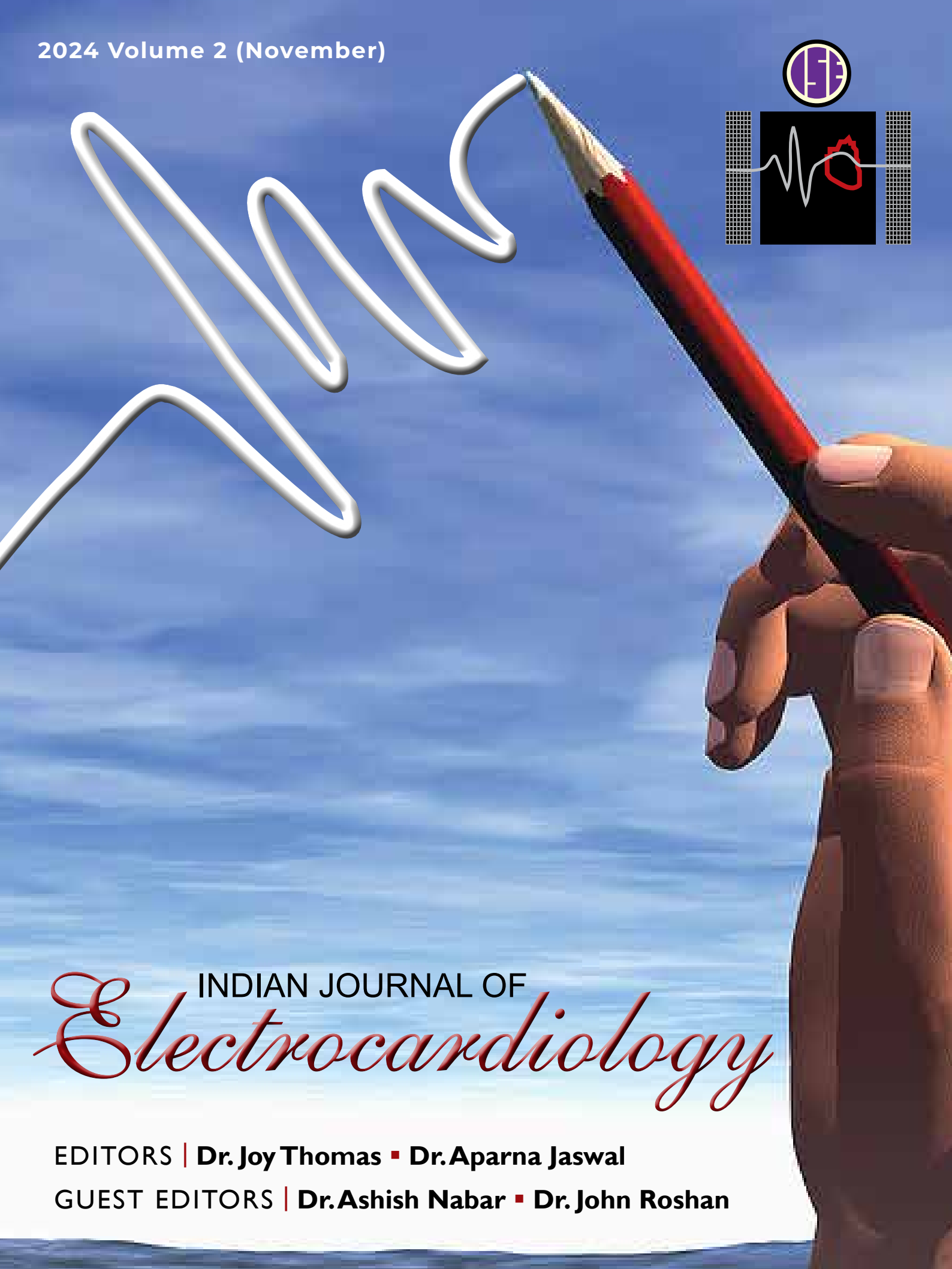
2024 Volume 2 (November)



INDIAN JOURNAL OF Electrocardiology

EDITORS | **Dr. Joy Thomas** ▪ **Dr. Aparna Jaswal**

GUEST EDITORS | **Dr. Ashish Nabar** ▪ **Dr. John Roshan**

UPCOMING EVENTS

ISECON 2025

21-23 February 2025 • Kolkata

Mid-Term ISECON 2025

August 2025 • Srinagar

ISECON 2026

February 2026 • Lucknow

C O N T E N T S

SECRETARIAT
Ketan K. Mehta
HON. GENERAL-SECRETARY

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Editorial

The ECG- A never ending tryst

Putting together yet another issue of the Indian Electrocardiology Journal, a honour granted by our advisor Prof. Dr S B Gupta, President Prof. Dr Aditya Kapoor and Honorary General Secretary Prof Dr Ketan K Mehta to be released at the Mid Term Meeting in November 2024 had hit some air pockets due to the personal pre-occupations of the editors. However with the undying enthusiasm of Prof. Dr Ashish Nabar the task was completed with the assistance of Guest editors- Prof. Dr John Roshan and Prof. Ashish Nabar.

The ECG continues to guide us through the decades after more than a century of its arrival on the medical scene in diagnosing, triaging, managing and follow-up of patients. The Bundle branch blocks that one confronts us in day to day practice can confound us at times. The importance of Right Bundle branch branch, the commoner bundle bramch block has been dealt with in a comprehensive manner, dwelling on its association with various syndromes that exhibits its morphology and its association with congenital and other structural heart diseases.

The axis of the ECG, a basic attribute of the vector characteristics of the waves that make up the ECG should enable us to understand the significance of the ECG we are assessing. A normal axis usually denotes a more benign condition.

Abnormal depolarising beats like ventricular tachycardia and Parasystole have mechanisms that are quite revealing and give us the specific method of managing them. Bidirectional ventricular tachycardia could be caused by the differential effects of drugs on the conduction system on the right and left side of the heart or by the effects of calcium or the disordered ion channels present in genetically deviant individuals. Parasystole is caused by the presence of an entrance block around the parasystolic ectopic which can occur in the atrium or the ventricle.

Cardiac Resynchronisation Therapy (CRT)has been around for more than two decades and has opened up a novel non drug management of patients with Heart Failure who have wide QRS with a Left Bundle Branch Block morphology and severe Left ventricular dysfunction by echocardiography. However the success of the procedure depends on the selection of the optimal pacing site and this has been discussed in this issue.

Muscular dystrophy does not spare the muscles of the heart and the electrocardiographic changes precede the echocardiographic and the MRI changes in these patients adding a tool in the diagnosis and prognostication of these patients.

We are grateful to the authors for having given us these articles at short notice and hope that the topics covered in this issue will be yet another step in the understanding and application of the ECG in daily clinical practice.

Dr. Joy M Thomas and Dr. Aparna Jaswal
Editors

Dr. Ashish Nabar and. Dr John Roshan
Guest Editors

From the Desk of Advisor

Dear Members,

It is indeed a great pleasure that Indian Journal of Electrocardiology, the Official Journal of Indian Society of Electrocardiology has become a regular feature. It has happened because of the untiring efforts of our esteemed editors, Dr Joy Thomas and Dr Aparna Jaswal, who are in constant touch with the contributors and looking into the contents so that the journal is worth reading by the post-graduates, physicians, cardiologists and even by the electrophysiologists.

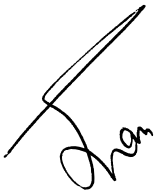
Current issue of Indian Journal of Electrocardiology has very useful articles like Axis in ECG, Bidirectional VT, Role of ECG in CRT, parasystole, Significance of RBBB and ECG in Duchenne Muscular Dystrophy for practicing physicians, who will enjoy reading them and be benefitted,

I would like to thank Dr Aditya Kapoor, President ISE, Dr Ashish Nabar, Treasurer ISE and the back bone to bring out this issue and Dr Ketan Mehta, Secretary ISE for his support.

My heartfelt thanks to the Journal Editors, Dr Joy Thomas and Dr Aparna Jaswal for their hard work to bring the IJE November 2024 issue in time.

I am sure the readers will be benefitted by going through the articles.

Long Live ISE.



Dr. S.B. Gupta
Advisor
Indian Society of Electrocardiology

From the President's Desk

Dear Friends,

It gives me immense pleasure to welcome you to this latest edition of the Journal of the Indian Society of Electrocardiology. As President of this prestigious society, I am honored to introduce a platform that fosters the exchange of knowledge, advances in research, and the latest in the field of electrocardiology. Electrocardiology has long been at the forefront of cardiovascular diagnosis and treatment, and the importance of continued research and clinical insights in this domain cannot be overstated. Our journal serves as a vital medium for sharing important clinical findings, and thought-provoking discussions that shape the future of our specialty.

I extend my heartfelt gratitude to all the contributors, reviewers, and the editorial board led by Dr Joy Thomas and Dr Aparna Jaswal who have worked tirelessly to ensure that this journal reflects the highest standards of academic excellence. The dedication and commitment of our colleagues who have contributed their articles in this edition, are what make this publication an invaluable resource for professionals in the field.

This is the opportune moment also to thank DR SB Gupta, Advisor, ISE, Dr. Ketan Mehta, Honorary Secretary, ISE and Dr. Ashish Nabar, Treasurer ISE. Dr Gupta's passion for advancing electrocardiology has made this journal a trusted source of knowledge and a proud reflection of our society's mission. Thank you for your leadership, dedication, and for going above and beyond to make this publication possible.

The organizational skills of Dr Mehta have been instrumental in bringing together contributors, reviewers, and experts, ensuring that the journal reflects the highest standards of quality and relevance in the field of electrocardiology.

Dr. Nabar's meticulous management of resources and support behind the scenes have always been invaluable in enabling the seamless production and distribution of the journal.

I am confident that the work presented in this journal will enhance patient care and further advance our collective knowledge. On behalf of the Indian Society of Electrocardiology, I request you to explore the contents of this journal and encourage you to engage with the ideas and perspectives it offers.

Thank you for your continued support, and we look forward to our shared journey toward improving cardiovascular health through the advancement of electrocardiology.

Dr. Aditya Kapoor
President
Indian Society of Electrocardiology

Right Bundle Branch Block-Unveiling its Significance

Ajeeth Arul Kumar[1], Praveen Anto[2]

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Introduction

Right bundle-branch block (RBBB) characterised by ECG findings (Table 1) suggestive of a significant delay in the electrical conduction through the right bundle branch and distal Purkinje fibres. This results in delayed right ventricular depolarisation occurring primarily via the left bundle branch and fascicular system. RBBB is generally known to a be benign conduction abnormality however, prompt diagnosis and appropriate evaluation is needed to rule out any underlying cardiac disease which can worsen the prognosis.[1] This article focuses on an approach to the management of patient with incidentally detected RBBB.

Table 1: Features of Complete RBBB as defined by the AHA/ HRS in 2009[2]

Complete RBBB	1. **Widened QRS** duration ≥120ms 2. In leads V1 and V2 (right precordial leads): **Rsr', rSr or rsR'** 3. In leads 1 and V6 (lateral leads): **S wave of greater duration than R wave or >40ms** 4. In leads V5, V6, normal R peak time but >50ms in V1 Of the above criteria, the first 3 should be present to make the diagnosis. When a pure dominant R wave with or without a notch is present in V1, criterion 4 should be satisfied.
Incomplete RBBB	1. No widened QRS 2. Other criteria are the same as for complete RBBB.

Incidence

Right bundle branch block (RBBB) is often discovered incidentally on an ECG, with a reported prevalence ranging from 0.2% to 1.3%. The prevalence of RBBB increases with age, being observed in 18% of individuals by the age of 80, compared to just 1% at the age of 50. This pattern suggests that RBBB is a gradually progressive degenerative condition of the cardiac conduction system. Additionally, RBBB is more common in men, as shown by the Copenhagen City Heart study, which found a prevalence of 1.4% in men compared to 0.5% in women. Incomplete RBBB is at least three times more common than complete RBBB, though its association with advancing age is less pronounced.[1]

ECG Criteria for Diagnosis

What defines RBBB? (Figure 1)

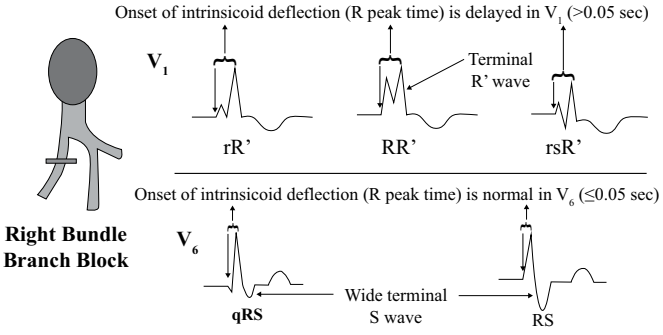


Figure 1: ECG features to identify RBBB

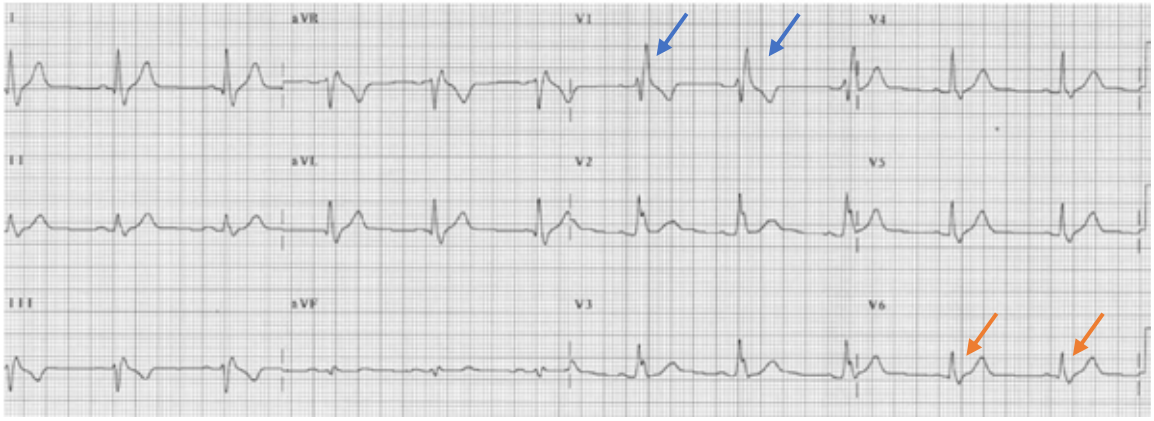


Figure 2: The QRS complexes are wide measuring 0.12 seconds. rsR configuration is present in V1(blue arrows) and qRs configuration is present in V6. Wide S waves are present in leads I and V6(orange arrows). The axis of the QRS complex in the frontal plane is normal.

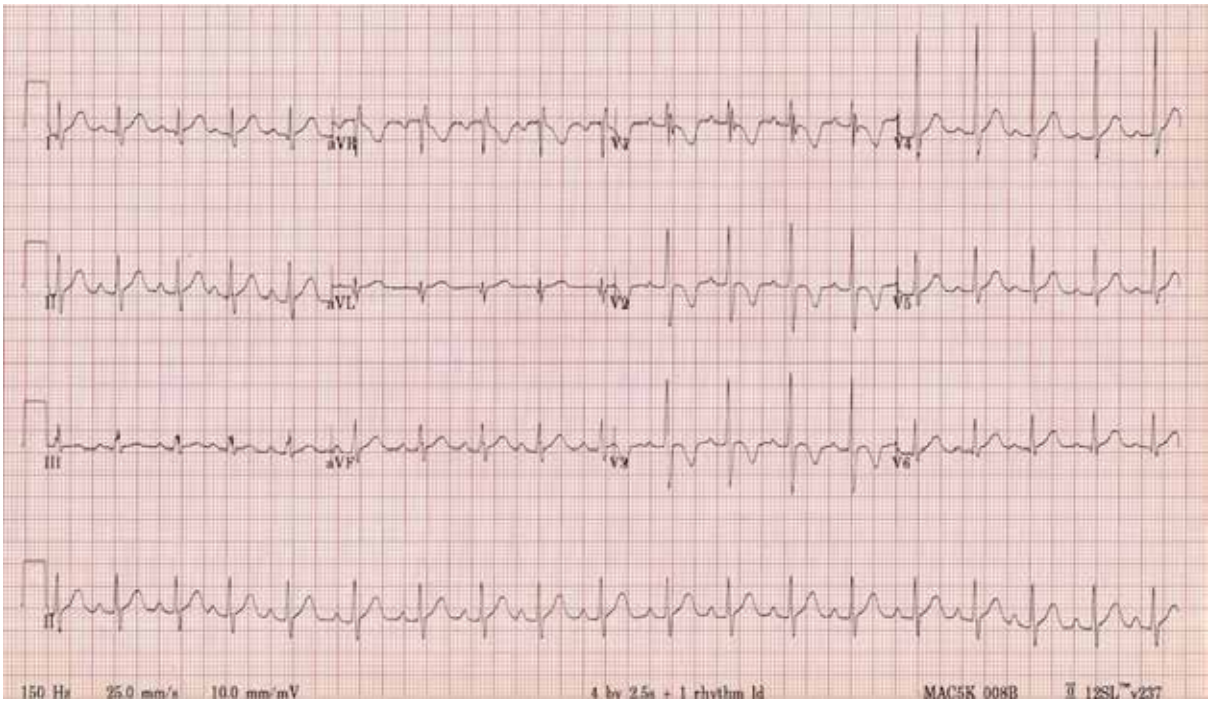


Figure 3: Incomplete RBBB. Normal QRS duration with RSR' pattern from V1-V3

What is not RBBB?

RBBB follows a distinct pattern on ECG however it should be distinguished from certain significant patterns that resemble it.[1,3,4]

Condition	ECG pattern
Ventricular arrhythmias:*	
Ventricular tachycardia	Widened QRS with HR> 100 bpm
Accelerated idioventricular rhythm	Widened QRS with HR < 100 bpm
Biventricular Pacing	Widened QRS preceded by pacing spikes
Brugada syndrome[γ]	Pseudo-RBBB characterised with a coved ST segment elevation, with inverted T waves in leads V1 and V2.[3]

* A rate dependant RBBB can be mistaken for a ventricular tachycardia or accelerated idioventricular rhythm. When a QRS complex appears in an RBBB pattern intermittently it is known as rate dependant RBBB, seen occasionally during increased heart rate. The QRS becomes narrow again once the HR slows down.

[γ]Patients with Brugada syndrome can develop a new onset RBBB pattern on ECG or can have an RBBB that is masking characteristic signs of Brugada syndrome on ECG, which can be evident either spontaneously, pharmacologically or through ventricular pacing (Chiale et al) (using right apical ventricular pacing with appropriately timed A-V intervals)[3]

Bifascicular block occurs when an RBBB occurs combined with other conduction disturbances such as left anterior fascicular block (LAFB) and left posterior fascicular block, left septal fascicular block (LSFB) or 1st degree AV block. RBBB with LAFB is more commonly seen in clinical practice. A bifascicular block combined with a 1st degree AV block is considered to be an equivalent of a trifasicular block.[1]

Etiology:[1]

Category	Causes
Most common association	Systemic hypertension
Structural heart disease	Ischemic heart disease, cardiomyopathy, myocarditis, valvular heart disease
Congenital heart disease	Tetralogy of Fallot, Ebstein's Anomaly
Metabolic causes	Hyperkalemia
RV pressure / volume overload	Pulmonary embolism, Cor pulmonale
Iatrogenic cause	Right heart catheterization, Ethanol ablation for septal reduction in hypertrophic cardiomyopathy
Age-related degeneration	Lev's disease, Lenegre's disease
Genetic disorders	Brugada's syndrome

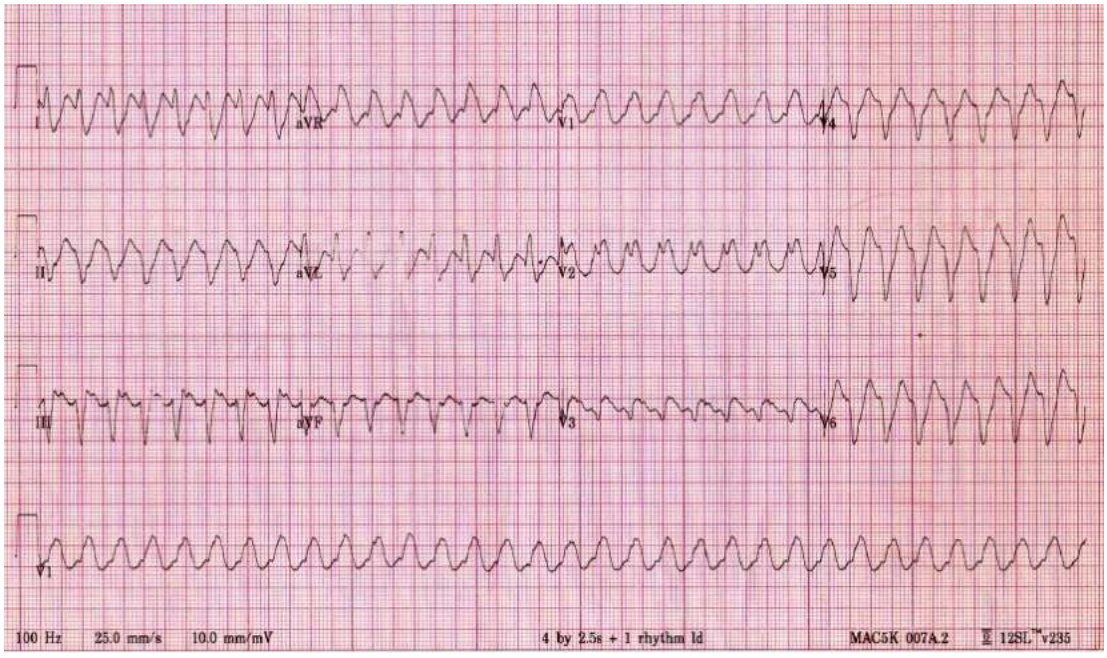


Figure 4: Ventricular tachycardia, with widened QRS and heart rate of >150 bpm

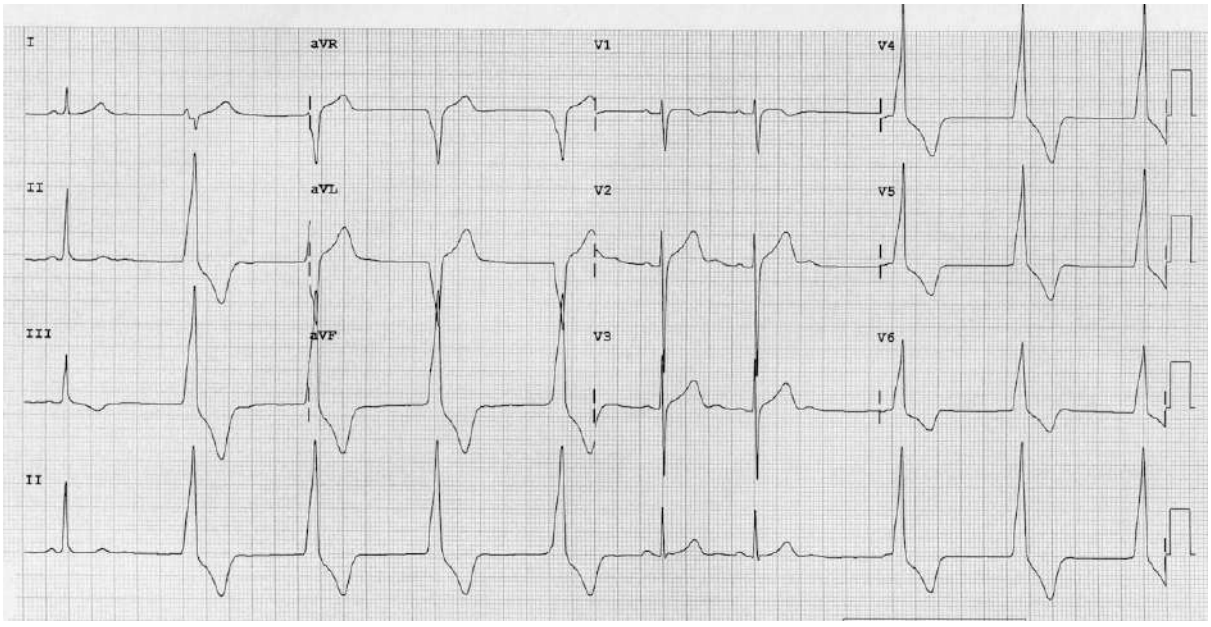


Figure 5: Accelerated Idioventricular rhythm. ECG shows heart rate of 60bpm, widened QRS and capture beats

Further Evaluation

RBBB although considered benign and presents asymptomatically, it requires further evaluation to identify the type and severity of any concurrent underlying heart disease and presence of other conduction disturbances.

Delayed onset of RV depolarisation with prolongation of total RV activation results in late opening of the pulmonic valve and delayed ejection of the RV, which can impact RV function. With worsening RV electromechanical coupling, conduction across the RV myocardium is expected to become delayed and can be detected as R' prolongation on the surface ECG. Studies show that prolonged R' duration in lead V1 is an indicator of RV dysfunction in patients with RBBB as correlated by functional echocardiographic parameters and cardiac magnetic resonance imaging. Echocardiographic findings from a study comparing patients with RBBB without RVF and those with RVF demonstrated that patients with RBBB and right ventricular failure (RVF) showed significantly poorer RV function compared to those without RVF. Specifically **Tricuspid Annular Plane Systolic Excursion** (**TAPSE**: 19.2 ± 1.7 mm in patients with RBBB without RVF vs. 13.6 ± 2.8 mm in patients with RBBB and RVF ($p < 0.001$),

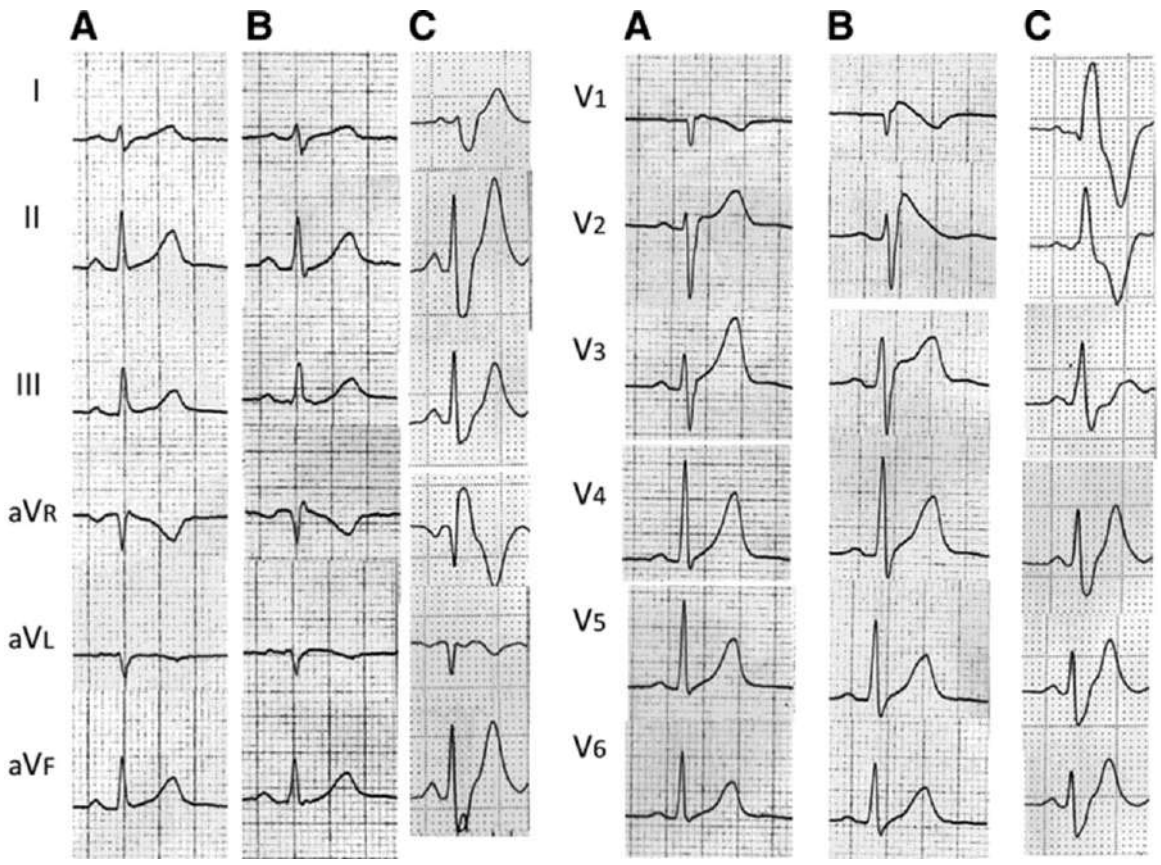
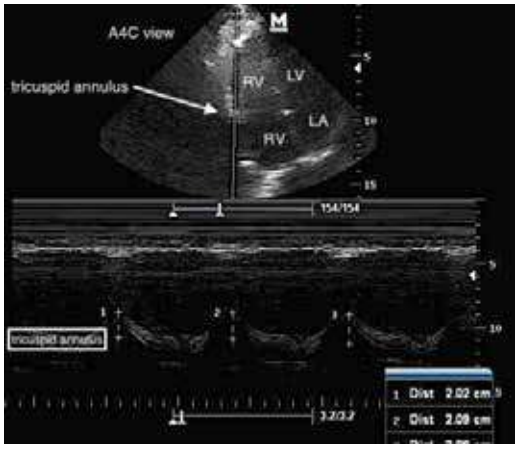
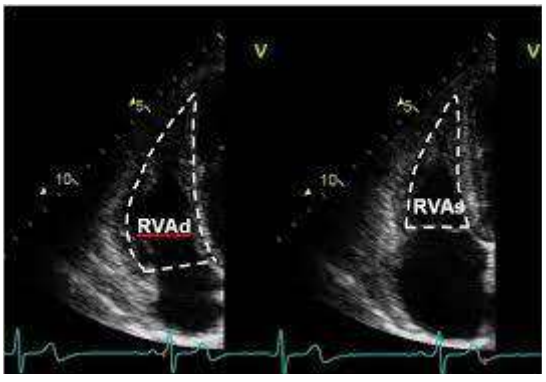


Figure 6: Patient showed variable ECG in the right leads (A and B). During the 2-year follow-up, complete right bundle-branch block developed without any precipitating cause (C). The ECG showed a tall R′ in V1, and the QRS duration was wide with a slurred, wide



Tricuspid Annular Plane Systolic Excursion is the excursion of the lateral tricuspid annulus towards the right ventricular apex in systole



Fractional area change is the difference in area from diastole to systole

Right ventricular **fractional area change** (**FAC**: 41.4 ± 4.5% in patients without RVF vs. 27.7 ± 5.6% in patients with RVF ($p < 0.001$) and **RV Myocardial Performance Index,** (the ratio of RV isovolumetric time to RV ejection time): 0.35 ± 0.17 in patients with RVF vs. 0.43 ± 0.19 in patients without RVF ($p < 0.001$) all demonstrated worse values in patients with RVF, highlighting reduced RV systolic function and impaired myocardial performance. RV dimension was larger in patients with RVF, suggesting RV dilation. (**RV Dimension**: 3.20 ± 0.64 mm in patients without RVF vs. 3.53 ± 0.69 mm in patients with RVF ($p < 0.001$)) Overall, these findings emphasize the need for comprehensive evaluation of RV

function in patients with RBBB (Figure 7) to identify those at risk for RVF and to tailor management strategies accordingly.[6]

Systolic RV contraction is delayed due to the delayed RV free wall depolarization, resulting in prolongation of the QRS duration as demonstrated in a study based on CMR, where RV free wall contraction occurred 90.7 ms ± 42.6 later than at the septum. Ignoring RV physiology in RBBB patients leads to a statistically significant underscoring of RV performance in up to two-thirds of patients. To avoid this, a RBBB or LBBB should always be taken into account when performing CMR assessment of cardiac function. This is mandatory, both for patients with congenital heart diseases as well as for patients with acquired heart diseases, especially RBBB, if right ventricular function is used as a prognostic factor.[7]

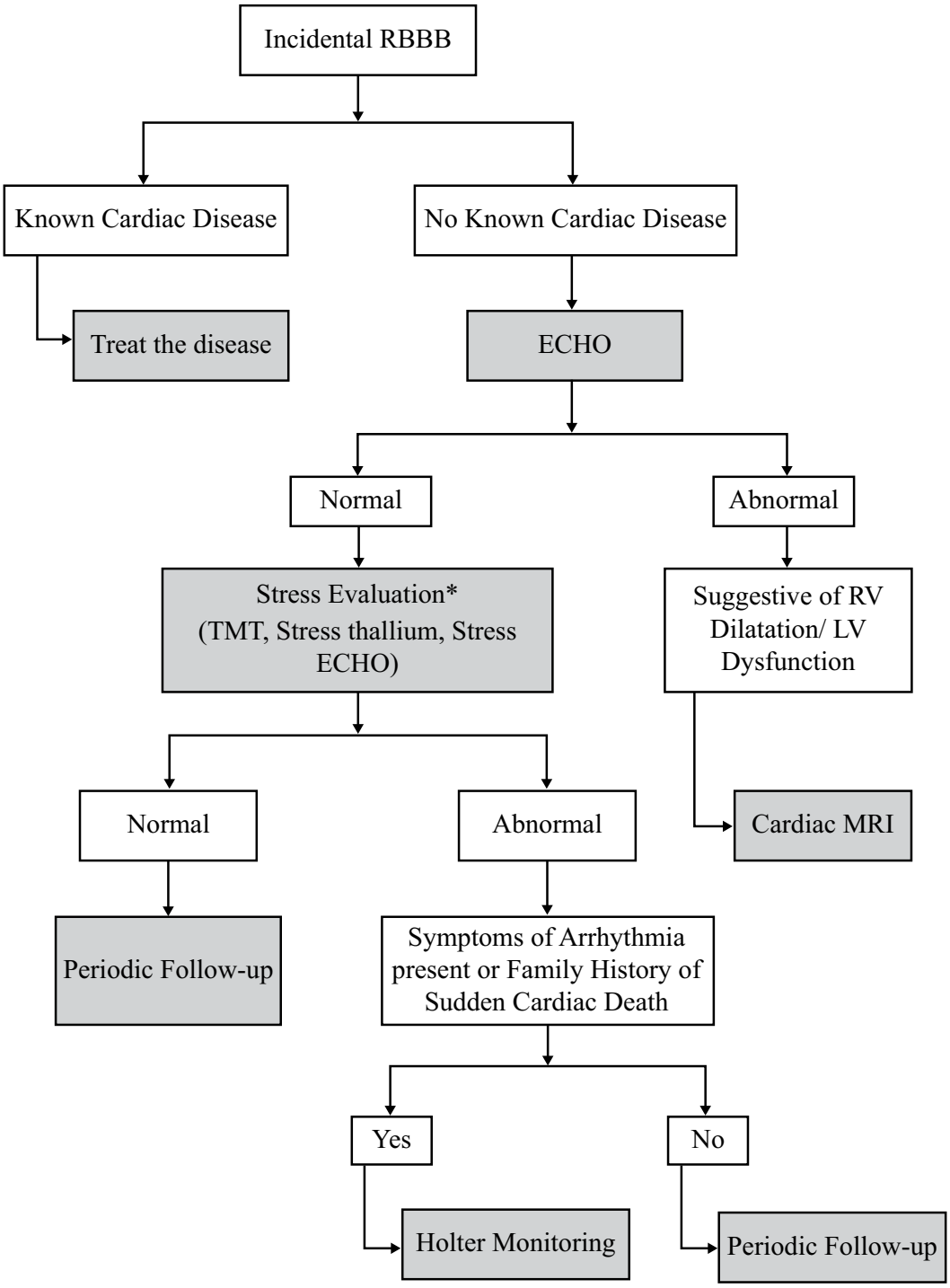


Figure 7: Approach for the Evaluation and Management of a Patient with RBBB. *Although the incidence of CAD is low in patients with asymptomatic RBBB, stress evaluation can still be performed to out CAD as it is of high incidence in our population.

Exercise-induced right bundle branch block (RBBB) or left bundle branch block (LBBB) is usually considered nonspecific unless it is associated with evidence of ischemia. It should be included in the evaluation of asymptomatic RBBB to rule out coronary artery disease, despite its low prevalence in patients with RBBB.

Treatment:[1]

Chronic RBBB should be followed up closely due to its association with poor cardiovascular outcomes, however it does not require treatment. For New onset RBBB it is paramount to identify the underlying cause of RBBB and it should be treated. For patients with CAD, concordant T waves on ECG can be suggestive of ischemia or MI (as opposed to discordant T waves in patients without MI with RBBB) and should be planned for an invasive coronary intervention. Pacemaker should be used in the treatments of patients with syncope, accompanied by other conduction disturbances such as second-degree AV block. For patients with RBBB with specific ECG patterns, such as LAFB and long PR interval CRT can also be used as in heart failure, which showed less favourable outcomes in patients with RBBB.

Prognosis:[5]

Long term outcomes are generally favourable for patients without any apparent heart disease. However in patients with CVD such as CAD and heart failure, it is a predictor of all-cause mortality, as observed in multiple large cohort studies and systematic reviews. In patients with acute coronary syndrome presentations, RBBB is an independent predictor of in-hospital and early (≈6 month) mortality. It has also been shown to be an independent predictor of decreased right ventricular ejection fraction (EF), which in itself is a predictor of adverse outcomes in patients with ischemic cardiomyopathy. Data from the Copenhagen City Heart Study, for example, which included 18, 441 participants without prior myocardial infarction or HF, found that RBBB was a strong predictor of increased all-cause mortality and cardiac death.

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The Axis in ECG - What it Points to?

Deep Chand Raja[1], Sharon Jebamala[2], Selva Maheshwari M[2]
[1]Cardiologist and Clinical lead Cardiac Electrophysiologist, [2]Physician Assistant, Kauvery Hospital, Chennai

Introduction

The ECG axis represents the direction of the heart's electrical activity in the frontal plane, providing a crucial diagnostic tool in electrocardiography (ECG). It summarizes the overall orientation of electrical impulses during the depolarization and repolarization of the ventricles.[1] Determining the QRS axis is the most crucial for diagnosing cardiac conditions, as it reflects the direction of ventricular depolarization. It is measured using the limb leads (I, II, III, aVR, aVL, aVF) on the ECG. The axis is typically expressed in degrees and helps in assessing the heart's electrical health.[2-5] Additionally, measuring the axes of the P wave and T wave can provide further insights into atrial and ventricular repolarization. In this article, we will be discussing about the various methods for measurement of QRS axis and causes of abnormal QRS, P, T wave axes.

Measuring QRS Axis

Measuring the QRS axis involves determining the direction of the electrical impulses during ventricular depolarization as represented on an ECG. Accurate measurement is essential for diagnosing various cardiac conditions. Here is a detailed guide for measuring the QRS axis:

1. Quadrant Method

This is a basic and commonly used method for determining the QRS axis. Examine the QRS complex in Lead I and Lead aVF.[4,6] Calculate if the QRS complexes are positive or negative in these leads:

LEAD 1	LEAD aVF	AXIS
POSITIVE	POSITIVE	NORMAL AXIS (-30°- +90°)
POSITIVE	NEGATIVE	POSSIBLE LAD (0° TO -90°)
NEGATIVE	POSITIVE	POSSIBLE RAD (+90° TO +180°)
NEGATIVE	NEGATIVE	EXTREME AXIS (-90° TO +180°)

2. Hexaxial reference system (Figure 1)

It is a more precise method and involves plotting the QRS axis on a diagram:

- Calculate the QRS amplitude in the limb leads (I, II, III, aVR, aVL, aVF).
- Hexaxial reference system diagram is used to chart the net amplitude of the QRS complex in Leads I and aVF.
- Determine the angle of the QRS axis relative to reference, which correspond to 0°, 60°, 120°, and 180°.
- The axis will be perpendicular to this on the positive side. It represents the direction in which the maximum positive voltage is observed.[4,7,8]

3. Axis Calculation Using the Maximal QRS Amplitude Method

This method involves calculating the QRS axis based on the amplitude of the QRS complexes:

- Identify the limb leads with the biphasic QRS complex (Equiphasic R/S)
- Use this limb lead as the reference line
- Identify the limb lead with the highest positive amplitude which is perpendicular to the reference line. Mean QRS axis is around this lead.[8,9]

Causes of Left Axis Deviation (Figure2, Figure 3)

Left axis deviation occurs when the QRS axis on an ECG is shifted to the left, typically indicating an angle of less than -30°. Several conditions and factors can cause left axis deviation, reflecting various underlying cardiac or systemic issues.[4,10]

1. **Left Ventricular Hypertrophy (LVH):** Thickening of the walls of the left ventricle.

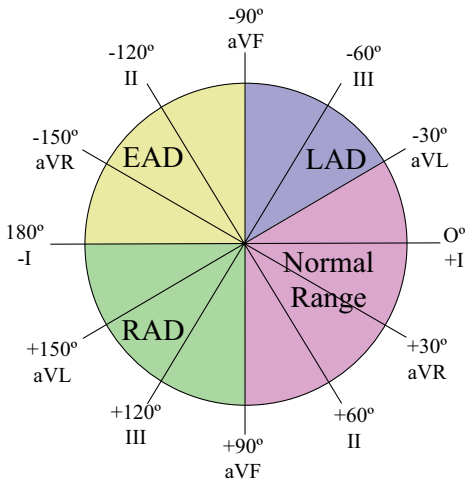

Figure 1: Hexaxial reference system

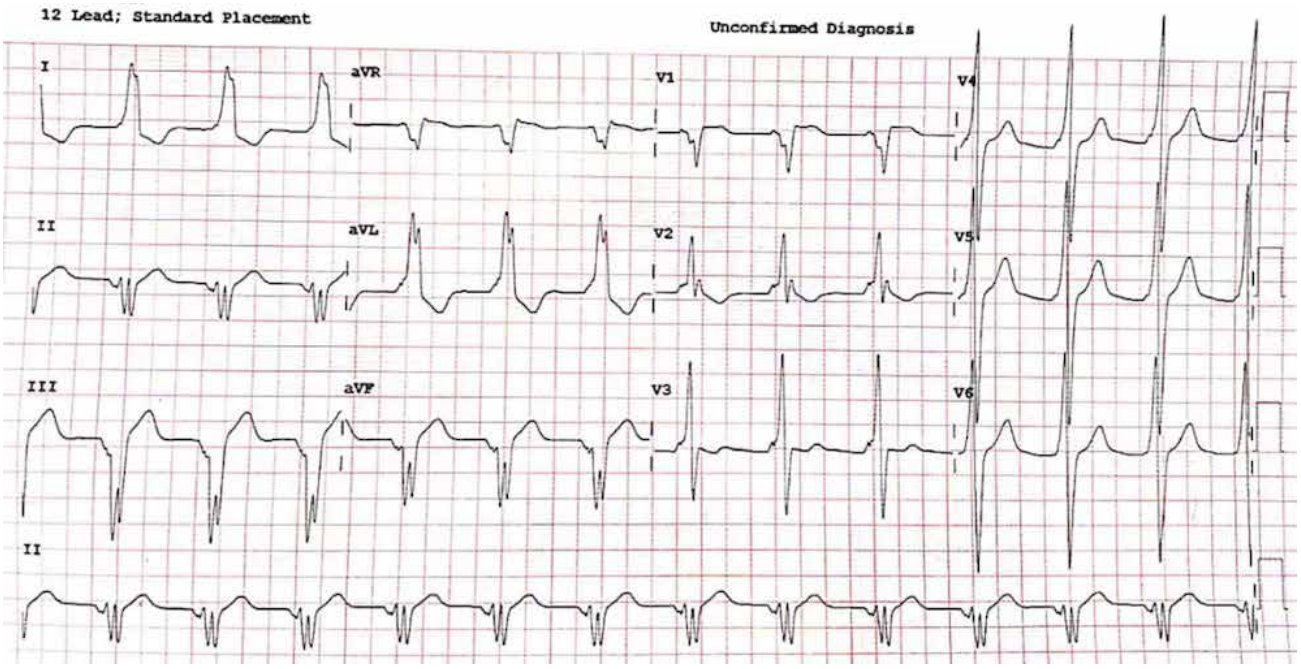

Figure 2: Left axis deviation in a patient with WPW Syndrome with right posteroseptal accessory pathway (QRS axis -30°)

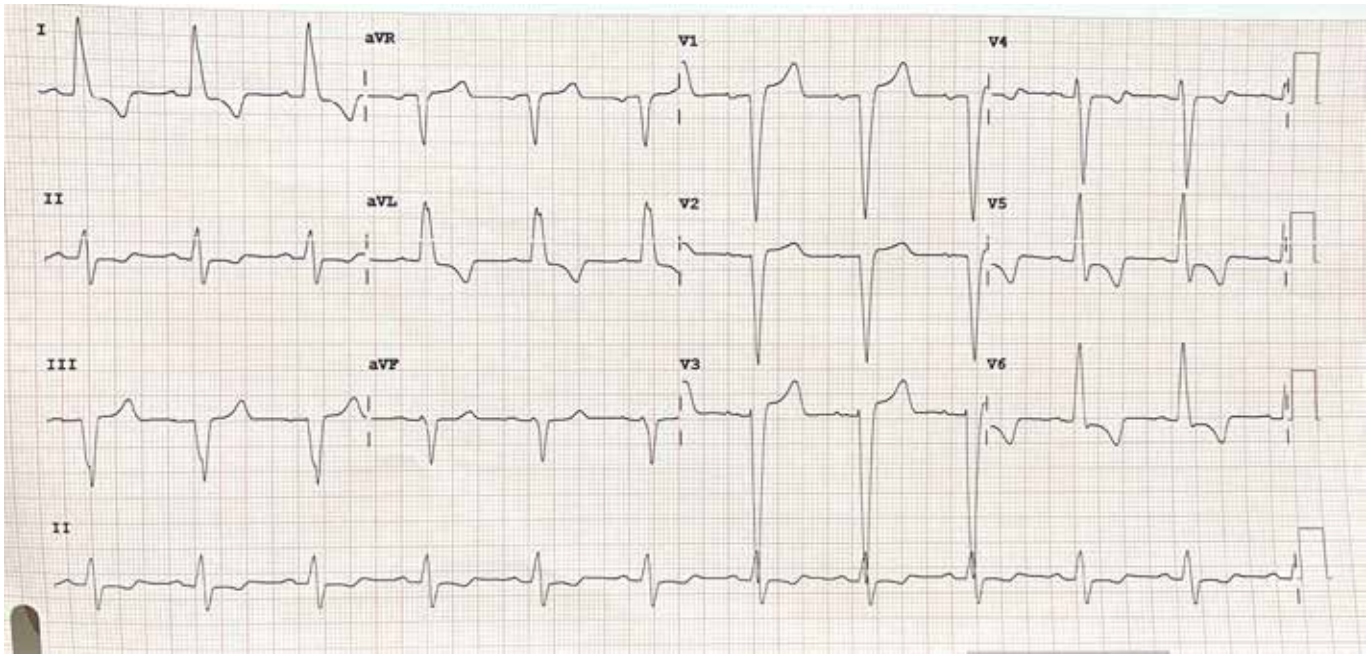

Figure 3: Left axis deviation, Left ventricular Hypertrophy with strain, Complete LBBB, in a patient of chronic kidney disease (QRS axis -30°)

2. **Left Bundle Branch Block (LBBB):** A blockage in the electrical conduction system of the left bundle branch, affecting the depolarization of the left ventricle.
3. **Left Anterior Fascicular Block (LAFB):** A block affecting the left anterior fascicle of the left bundle branch, leading to altered depolarization patterns.
4. **Hypertrophic Cardiomyopathy (HCM):** changes in the electrical axis due to altered ventricular structure.
5. **Aortic Stenosis:** Narrowing of the aortic valve, causing increased pressure load on the left ventricle, Leads to left ventricular hypertrophy and subsequent Left axis deviation.
6. **Emphysema**
7. **WPW syndrome:** right lateral or right posteroseptal accessary pathway

8. **Congenital heart diseases:** Tricuspid atresia, atrio-ventricular septal defect.
9. **Inferior Myocardial Infarction**
10. **Ventricular tachycardia**

Causes of Right Axis Deviation (Figure 4)

Right ais deviation occurs when the QRS axis on an ECG is shifted to the right, typically indicating an axis greater than +90°.

1. **Right Ventricular Hypertrophy**: Increased muscle mass in the right ventricle, often due to conditions like chronic lung disease or congenital heart disease.
2. **Pulmonary Embolism**: A blockage in the pulmonary arteries can cause increased pressure and strain on the right side of the heart, leading to RAD.
3. **Right Bundle Branch Block (RBBB)**: This conduction abnormality affects the electrical impulse as it travels through the right bundle branch, potentially leading to RAD.
4. **Cor Pulmonale**: Right-sided heart failure due to lung disease, causing strain on the right ventricle.
5. **Acute Right Ventricular Myocardial Infarction**: A heart attack affecting the right side of the heart can cause RAD.
6. **Acute right heart strain/pressure overload** - also known as McGinn-White Sign or S1Q3T3 that occurs in pulmonary embolus
7. **WPW**- left lateral or left posteroseptal accessary pathway
8. **Dextrocardia**
9. **Left posterior hemiblock**
10. **Anterolateral MI**
11. **Hyperkalemia**: Elevated potassium levels can affect the heart's electrical conduction, sometimes resulting in RAD.

Causes of Northwest Axis Deviation (Figure 5, Figure 6)

North-West axis deviation is also known as extreme axis deviation, and it defined as it frontal QRS axis between +180° and +270°. The following are potential causes of North-West axis deviation:

1. **Ventricular rhythm:** Ventricular ectopics, VT, accelerated ventricular rhythm
2. **Pulmonary Embolism:** A blockage in the pulmonary arteries can cause significant strain on the right side of the heart, resulting in a northwest axis.
3. **Right ventricular ectopy:** Ventricular ectopy originating from the anterior fascicle can result in North-West axis deviation.
4. **New-onset extreme axis deviation in acute myocardial infarction:** During acute myocardial infarction, North-West axis deviation may be related to extensive myocardial ischemia and/or necrosis, causing an "electrical escaping" with an extreme dislocation of the QRS axis.

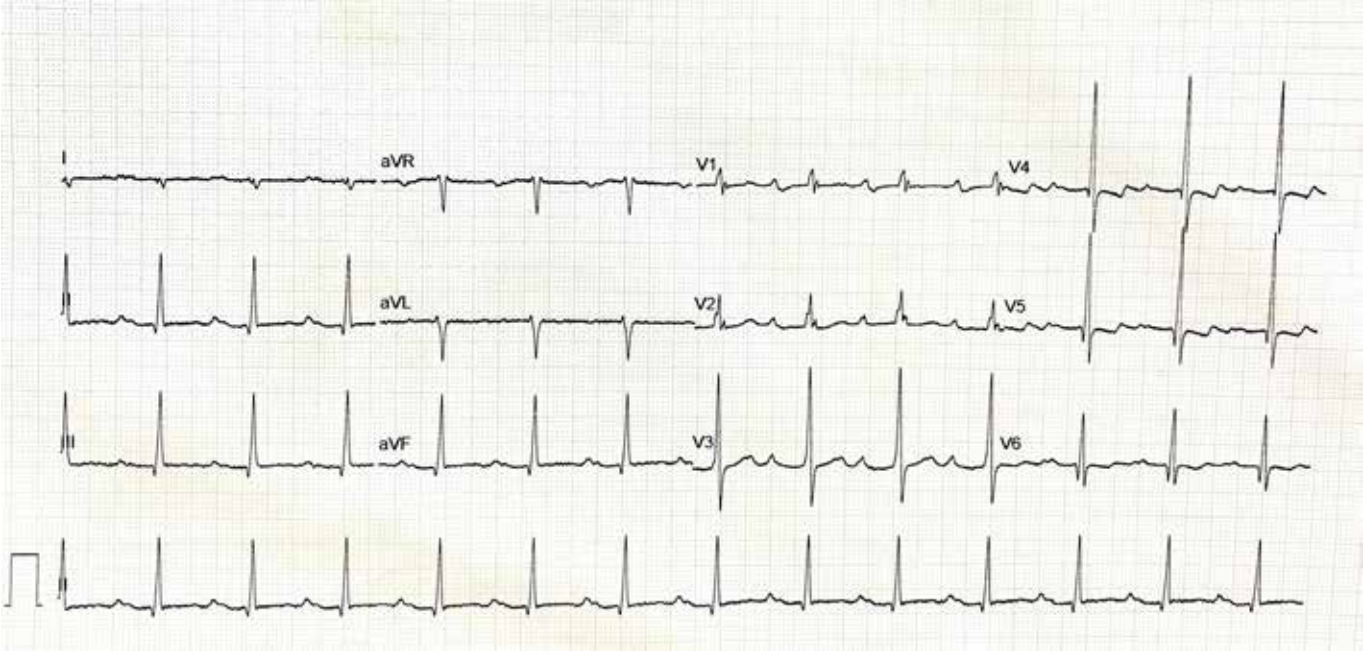

Figure 4: Right axis deviation, first degree AV block with incomplete RBBB, right ventricular hypertrophy, Left ventricular hypertrophy, Rheumatic heart disease with P-mitrale, P wave axis – 90 (QRS angle +60°)

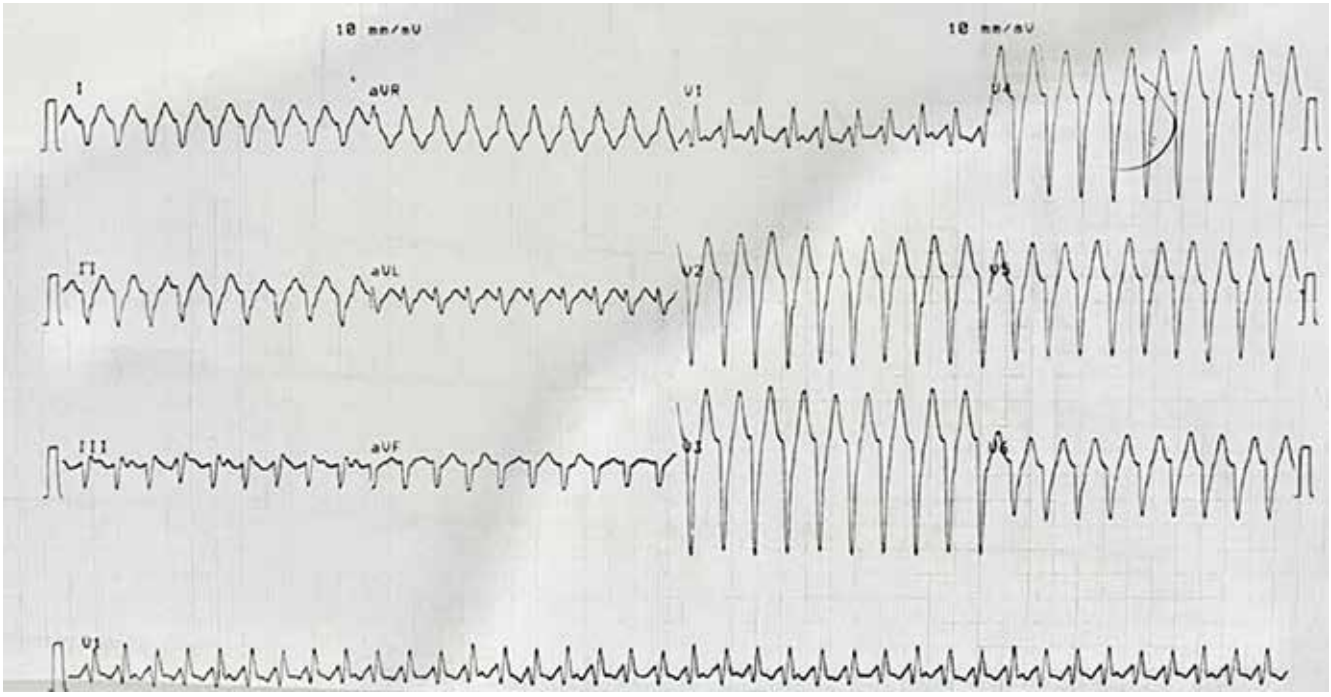

Figure 5: North-West axis deviation in a patient having Ventricular tachycardia with RBBB morphology at a rate of 300 bpm (QRS angle -120°)

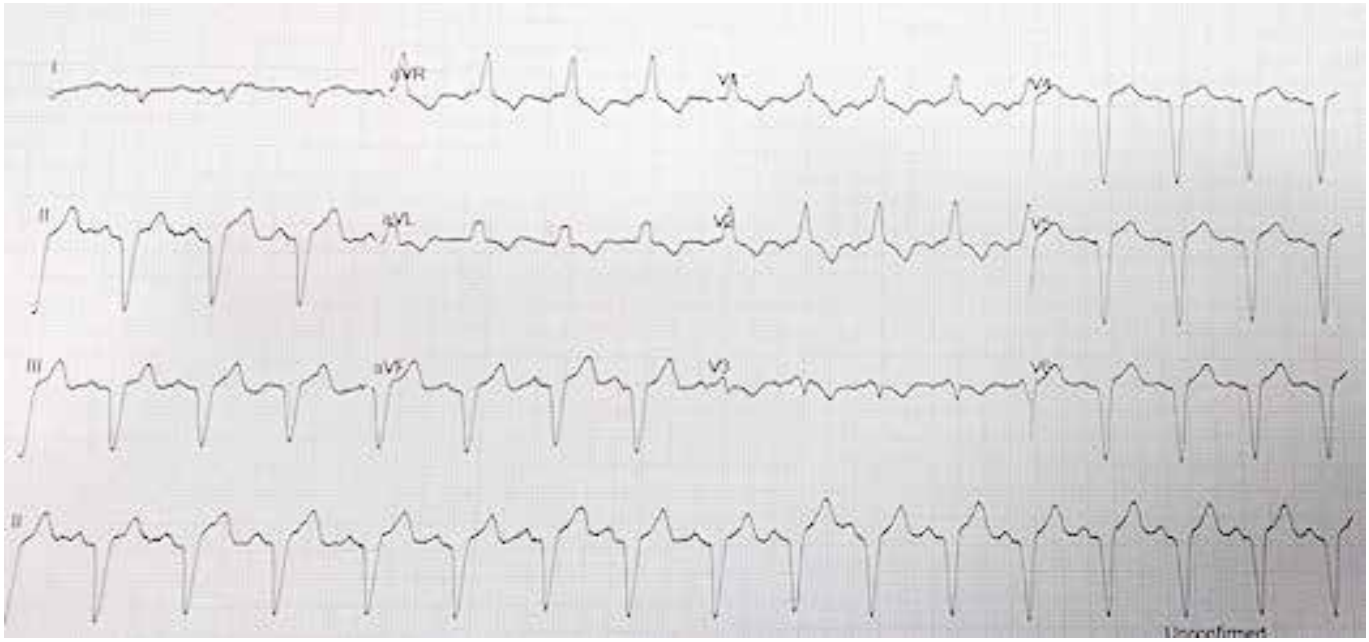

Figure 6: North-West axis deviation in a patient implanted with Dual chamber pacemaker, ECG showing Atrial sensed and ventricular paced rhythm. (QRS angle +250°)

5. **Congenital heart disease**
6. **Paced rhythm**
7. **Hyperkalemia:** Elevated potassium levels can affect cardiac conduction and result in an extreme right axis deviation.

P Wave Axis and its Importance (Figure 4)

The dominant vector of atrial depolarization is reflected as P wave axis. Normal P wave axis is 0° to +75°. This measurement provides information about the atrial electrical conduction, anatomy, and the orientation of the atrium. Abnormalities in the P wave axis have been associated with an increased risk of atrial fibrillation and stroke.[4,11]

Associated conditions:

1. In patients with WPW syndrome: right accessory pathway (-80° to -90°)
2. In patients with WPW syndrome: left AP (-90° to -150°) North-West region
3. In patients with Chronic obstructive pulmonary disease and Emphysema (directed to +90°)
4. In patients with Ebstein's anomaly (left ward deviation -30°)
5. A rightward deviation of the P wave axis is observed in mirror-image dextrocardia and when arm electrodes are reversed.

T Wave Axis and its Importance

The T wave axis on an ECG represents the direction of ventricular repolarization, by which the ventricles return to their resting state after contraction. An abnormal T wave axis orientation and an increased QRS-T angle is an indicators of repolarization abnormalities and used to evaluate ventricular repolarization. In myocardial ischemia caused by coronary artery disease, the T wave axis shifts away from the affected area of the left ventricle.[11,12]

Normal T wave axis= +15° to +60°

Conclusion

The ECG axis, encompassing the QRS, P wave, and T wave axes, is essential for evaluating the heart's electrical activity. The QRS axis, which indicates the direction of ventricular depolarization, is crucial for diagnosing cardiac conditions. Deviations such as Left axis deviation, Right axis deviation, and North-West Axis Deviation can signal various issues including ventricular hypertrophy, myocardial infarction, or pulmonary embolism.[4] Accurate measurement using methods like the quadrant method and hexaxial reference system is vital for pinpointing these deviations. The P wave axis, reflecting atrial depolarization, helps identify atrial abnormalities.[11] T wave axis represents ventricular repolarization, and its deviation from the normal range (+15° to +60°) can indicate myocardial ischemia or electrolyte imbalances.[12] Analyzing this axis provides a comprehensive view of the heart's electrical health, guiding diagnosis and treatment. Each axis offers unique insights into different phases of cardiac function, making them crucial tools in cardiology for accurate assessment and effective management of heart conditions.

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Parasystole

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Introduction

Parasystole , first described by Kaufmann and Rothberger in the 1920s, is a type of cardiac arrhythmia characterized by the coexistence of two independent pacemakers within the heart, one of which is usually the sinoatrial (SA) node, and the other an ectopic pacemaker that operates autonomously. The key feature of parasystole is that the ectopic pacemaker is "protected" from the influence of the normal sinus rhythm, leading to the generation of ectopic beats that are not reset by the sinus node. This results in a distinctive and complex arrhythmic pattern that can be challenging to diagnose and interpret.[1]

Epidemiology and Demographics of Parasystole

Incidence: Parasystole occurs in about 0.13% of all electrocardiograms in a general hospital setting. It is observed twice as often in males compared to females, with a male-to-female ratio of 2:1. While parasystole can affect individuals of any age, it is more commonly seen in patients over 60 years old, particularly the elderly.[2]

Risk factors: Around 60% of cases involve atherosclerotic heart disease and hypertensive cardiovascular disease, with half of these patients also experiencing congestive heart failure.[3] While most cases are associated with acquired or congenital heart diseases, 14% of patients show no evidence of underlying heart disease.

Pathophysiology

The ectopic pacemaker in parasystole operates independently due to a unique feature known as an entrance block, which protects the ectopic focus from being reset by the sinus impulses. The entrance block can be partial or complete:[4]

- **Complete Entrance Block (Classic variant):** The ectopic focus is entirely protected from the surrounding myocardial tissue, meaning that the impulses from the normal conduction system do not affect the timing of the ectopic pacemaker.
- **Partial Entrance Block (Modulated Variant):** Some sinus impulses may influence the ectopic focus, but not to the extent that it resets or completely alters the ectopic rhythm. Here the parasystole rate is not constant due to the electrotonic interference from the sinus impluses.

Types of Parasystole

Parasystole can occur in different parts of the heart, leading to various types:

1. **Atrial Parasystole:** The ectopic pacemaker is located in the atria. This can coexist with normal sinus rhythm or other atrial arrhythmias like atrial fibrillation.[3]
2. **Ventricular Parasystole:** The ectopic focus is within the ventricles, often leading to ventricular ectopic beats or more complex ventricular arrhythmias.
3. **Junctional Parasystole:** The ectopic pacemaker is located in the atrioventricular (AV) junction.

Clinical Manifestations

Parasystole is often benign and may not require treatment, especially if the patient is asymptomatic.

In some cases, parasystole can lead to symptoms like palpitations, dizziness, or syncope, particularly if the ectopic focus leads to significant ventricular arrhythmias or contributes to a decline in cardiac output.

However, it can be associated with underlying cardiac conditions such as ischemic heart disease, myocarditis, or structural heart disease, where the presence of parasystole might indicate more significant pathology.

Diagnosis

ECG (Electrocardiogram)

The diagnosis of parasystole is primarily made through electrocardiography (ECG), where several distinct features may be observed:

- **Variable Coupling Intervals:** Unlike other arrhythmias, where premature beats have a fixed coupling interval with the preceding beat, parasystolic beats may show varying coupling intervals due to the independent nature of the ectopic pacemaker.
- **Fusion And Capture Beats:** These occur when an ectopic beat coincides with a normal sinus beat, leading to a QRS complex that has features of both normal and ectopic origins.

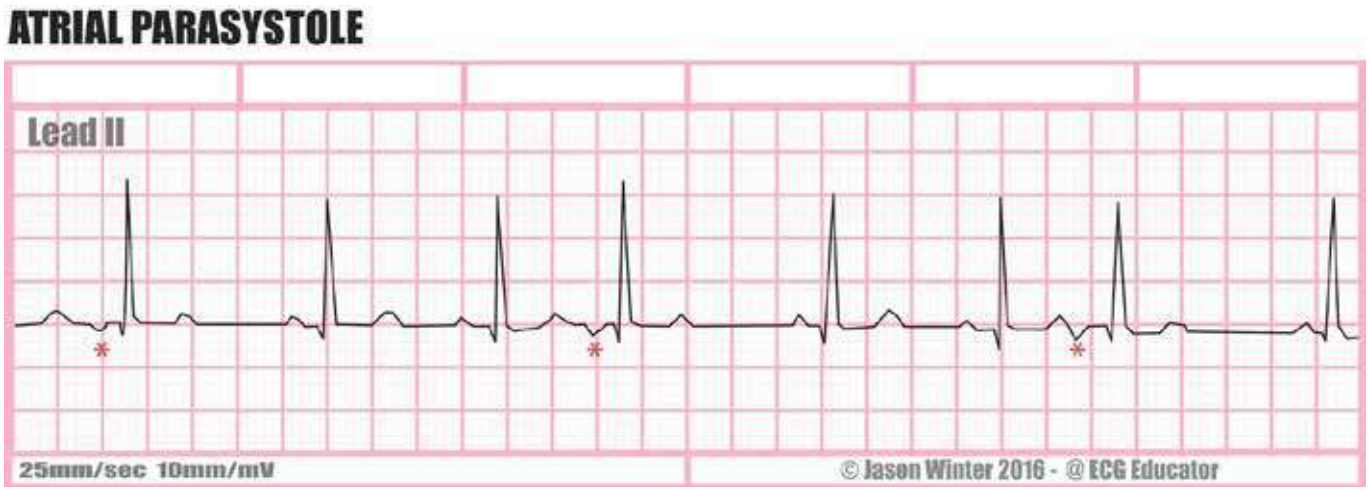

Figure 1: Narrow complex QRS with variable coupling intervals, constant firing interval suggestive of atrial parasystole.

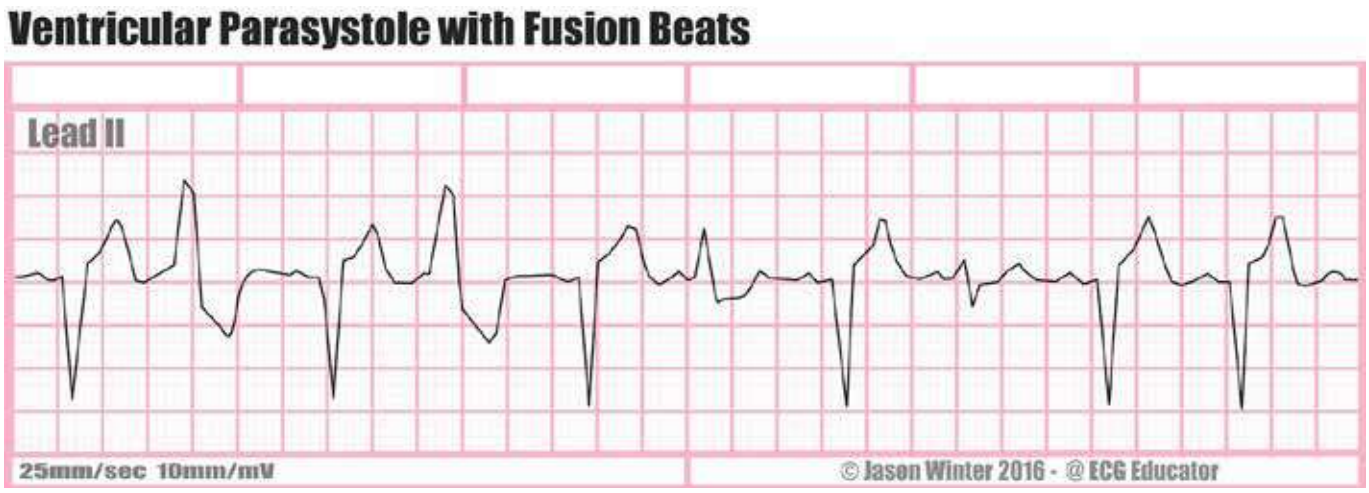

Figure 2: Wide complex QRS with fusion beat, fixed ectopic rhythm, varying coupling intervals suggestive of Ventricular parasystole.

- **Intermittent Aberration:** If the parasystolic focus intermittently captures the ventricles or atria, it may lead to aberrant conduction, such as bundle branch block patterns.
- **Fixed Ectopic Rhythm:** The ectopic pacemaker typically fires at a regular interval, which is evident on the ECG as a series of ectopic beats that occur at a consistent rate.

Differential Diagnosis

Parasystole must be distinguished from other arrhythmias, such as:

- **Premature Atrial or Ventricular Complexes (PACs/PVCs):** These usually have fixed coupling intervals.
- **Atrial or Ventricular Bigeminy:** A pattern of alternating normal and ectopic beats, but with a fixed coupling interval.
- **Multifocal Atrial Tachycardia (MAT):** Characterized by multiple P-wave morphologies, but not due to independent pacemakers.

Treatment:

Management of parasystole is typically conservative, focusing on:

- **Observation:** In asymptomatic patients, no specific treatment may be necessary.
- **Addressing Underlying Conditions:** If parasystole is associated with ischemic heart disease or other structural abnormalities, treating the underlying condition is the primary goal.
- **Symptomatic Management:** In cases where parasystole leads to significant symptoms, beta-blockers or calcium channel blockers may be used to reduce ectopic activity, although this is rare.

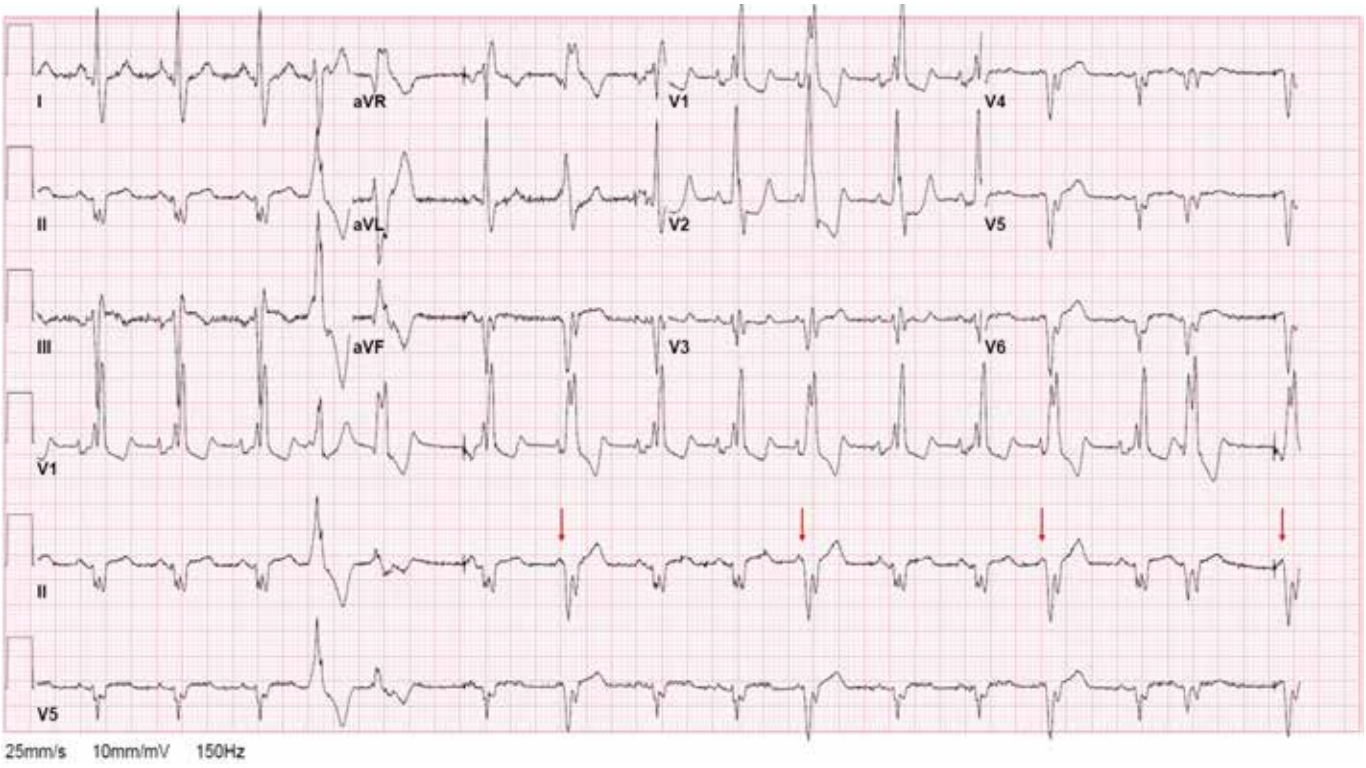

Figure 3: 12 lead ECG shows a bifascicular block, consisting of a right bundle branch block and a left anterior fascicular block. Additionally, it shows ventricular parasystole originating from a left posterior fascicular focus, marked by evenly spaced ventricular beats (indicated by red arrows) that are variably coupled.

- **Catheter ablation:** Maybe be rarely indicated in patients with severe symptomatic ventricular parasystole.[5]

In summary, parasystole is a unique arrhythmia characterized by the presence of an independent ectopic pacemaker operating alongside the normal sinus rhythm. The diagnosis relies heavily on ECG findings, and treatment is typically conservative unless the arrhythmia is symptomatic or associated with more severe underlying cardiac pathology.

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Bidirectional Ventricular Tachycardia: Mechanisms, Causes, and Management

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Introduction

Bidirectional ventricular tachycardia (BDVT) is a rare and distinctive arrhythmia characterized by the alternating morphology of QRS complexes on the electrocardiogram (ECG) at a regular ventricular rate [Figure 1]. This arrhythmia presents unique diagnostic and management challenges due to its diverse underlying mechanisms and associated conditions. This comprehensive review explores the mechanisms, causes, diagnostic approaches, and management strategies for BDVT, synthesizing current knowledge and recent advancements in the field.

Mechanisms of Bidirectional Ventricular Tachycardia

Understanding the mechanisms underlying BDVT is crucial for accurate diagnosis and effective treatment. The key mechanisms include:

1. Digitalis Toxicity

   Digitalis toxicity is one of the best established causes of BDVT. Digitalis, derived from the foxglove plant, is used to treat various cardiac conditions, including atrial fibrillation and heart failure. The drug increases vagal tone and affects cardiac conduction by inhibiting the Na+/K+ ATPase pump, which leads to an increase in intracellular calcium and a subsequent alteration in cardiac excitability.[1]

   The alternation in BDVT due to digitalis toxicity is thought to result from its varying effects on different regions of the heart. Digitalis can produce alternating patterns of depolarization by differentially affecting the conduction system's function in the right and left ventricles. This is often seen as a result of the drug's effect on the AV node and Purkinje fibers, leading to alternation in the QRS morphology.[2]

2. Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

   CPVT is a genetic arrhythmia syndrome characterized by bidirectional or polymorphic ventricular tachycardia that occurs during exercise or emotional stress(Figure 2). This condition is linked to abnormalities in intracellular calcium handling due to mutations in genes such as RYR2 (ryanodine receptor) or CASQ2 (calsequestrin)

   In CPVT, the mechanism involves abnormal calcium handling within cardiomyocytes, which can cause

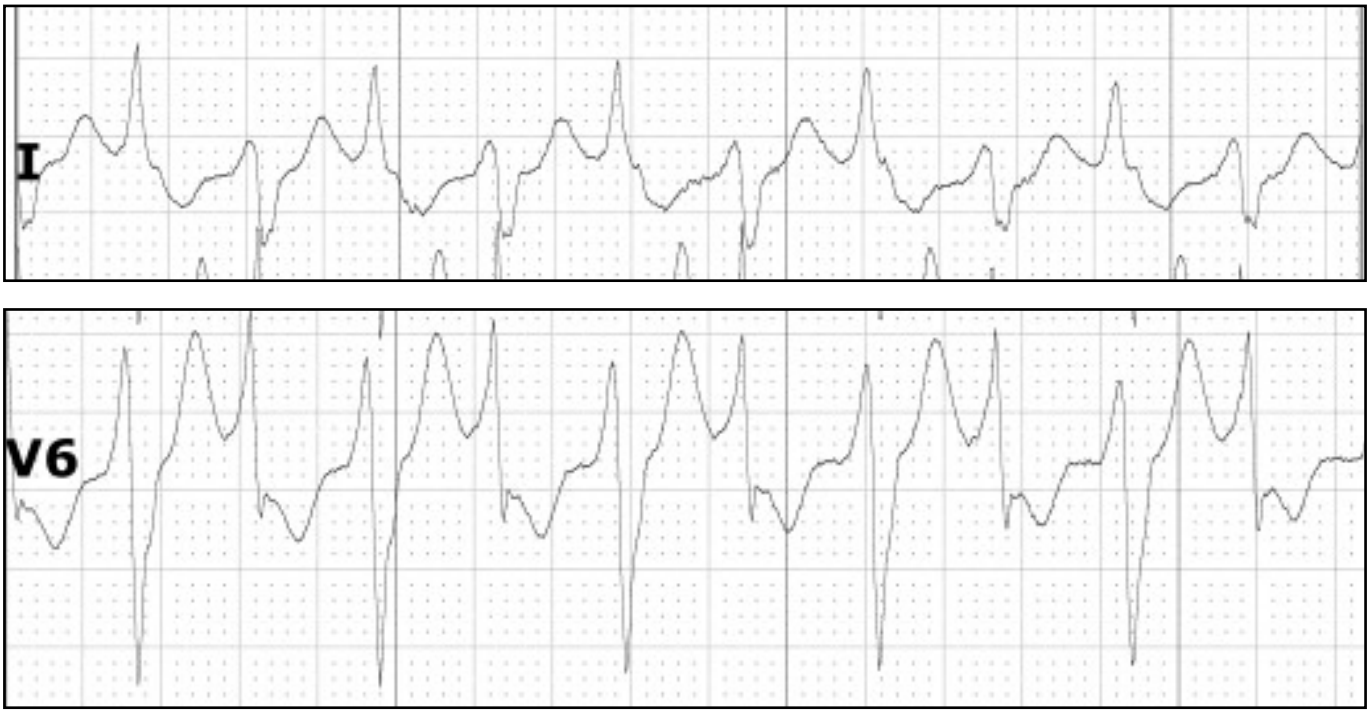

Figure 1: CPVT-1 (RYR2) patient, Holter recording showing bidirectional VT while playing

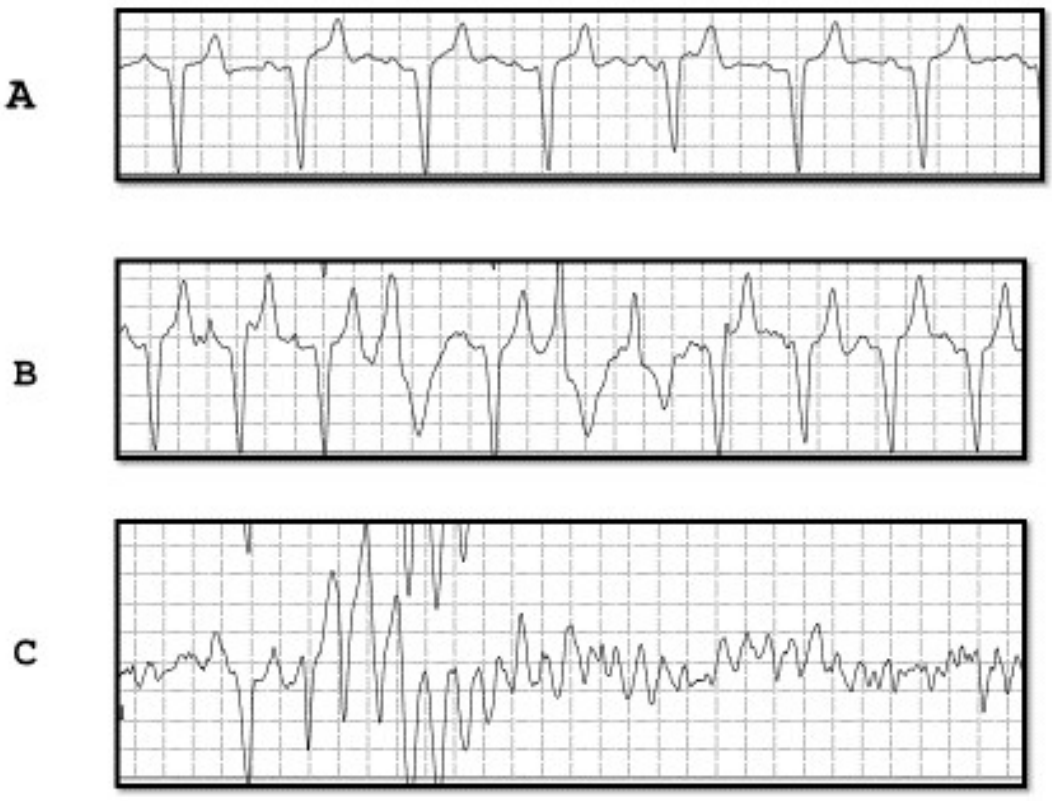

Figure 2: In CPVT ,Progressive stages of the patient's graded exercise stress test. Sinus rhythm in stage 1 (A). An increase in ventricular ectopy(Bidirectional) noted with increased exercise in stage 2 (B). With continued exertion and increasing heart rate in stage 3 (C),it degenerated into polymorphic VT/VF

delayed afterdepolarizations and spontaneous calcium release. This results in variability in the activation sequence of the ventricles. The alternation in the QRS morphology seen in BDVT is due to the variability in the timing and direction of ventricular activation caused by these abnormal calcium-induced afterdepolarizations.[3]

3. Andersen–Tawil syndrome (ATS) and other Long QT syndromes [LQTS]:

   ATS is characterized by three main factors: periodic muscle paralysis, repolarization changes in the ECG, and structural body changes [Figure 3]. Episodes of flaccid muscle weakness are a common muscle manifestation. Typical repolarization changes include a tendency toward ventricular arrhythmias and a prolongation of the QU/QUc intervals and normal or minimally prolonged QT/QTc intervals. Ventricular arrhythmias are characterized by bidirectional ventricular tachycardia, premature ventricular contractions, and infrequently, polymorphic ventricular tachycardia of the torsade de pointes type (Figure 4).

   In approximately 50–60% of cases, mutations in the KCNJ2 gene, which codes for the α subunit of the K+ channel protein Kir2.1, result in ATS type 1, an autosomal dominant disorder. A rare mutation in the KCNJ5-GIRK4 gene, which codes for the G protein-sensitive-activated inwardly rectifying K+ channel Kir3.4 (15%) and transmits the acetylcholine-induced potassium current, is further connected to ATS type 2. De novo/sporadic cases account for about 30% of cases, indicating that the disorder is also caused by additional, as-yet-unidentified genes [4]

   In LQTS, prolonged cardiac repolarization due to defective ion channels can lead to early afterdepolarizations, which may contribute to arrhythmias including BDVT [5]

4. Herbal Aconite Poisoning:

   Aconite appears to trigger automaticity by direct, persistent activation of inward Na+ channels during the plateau (phase II) of the action potential, prolonging repolarization and inducing afterdepolarizations with triggered automaticity.[6]

5. Structural Heart Disease

   Structural heart diseases such as dilated cardiomyopathy, hypertrophic cardiomyopathy, and myocarditis can lead

Figure 3: LQT7 Anderson Tawil syndrome showing dysmorphic features: Broad forehead, hypertelorism, telecanthus, low set ears, broad nasal bridge, micrognathia, dental enamel abnormalities, crowding and malocclusion, high arched palate and bilateral short fifth fingers

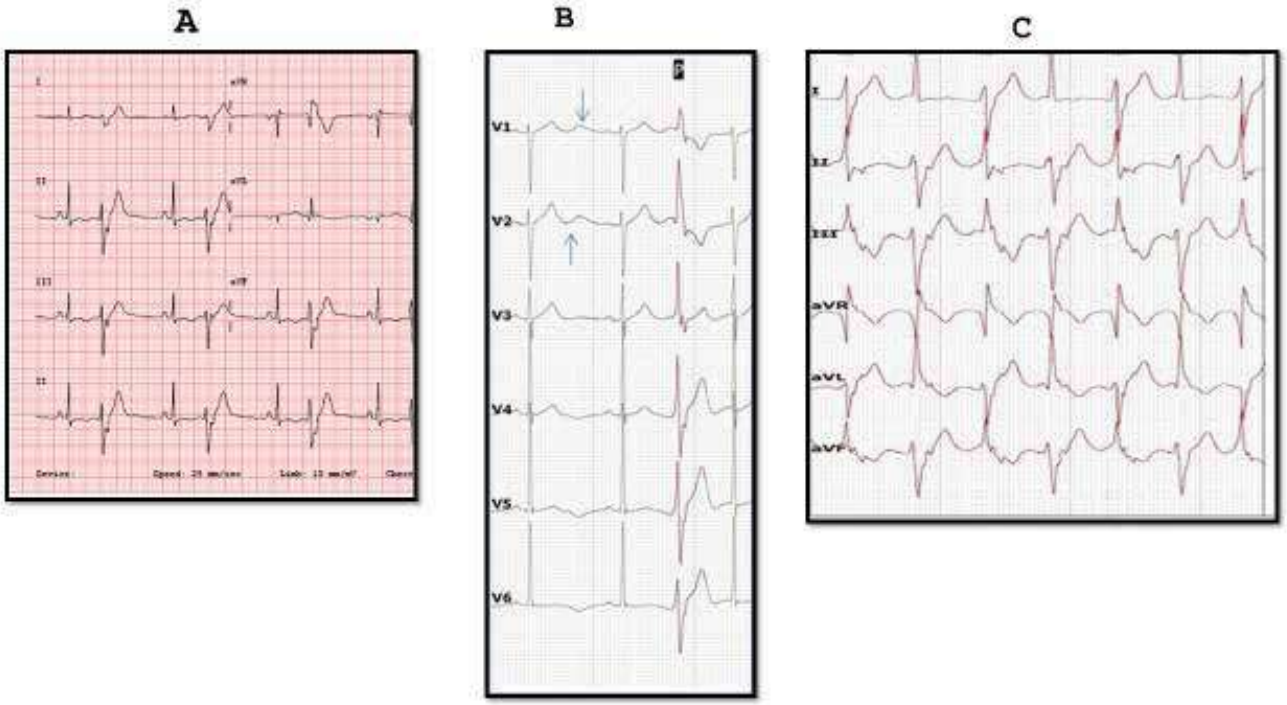

Figure 4: Anderson Tawil Syndrome A. Ventricular bigeminy of RBBB(right bundle-branch block) morphology , B Wide T-U junction, enlarged U waves noted in V1-V2 leads, C. Significant ventricular ectopy burden of 39.9% with runs of bi-directional ventricular tachycardia

to BDVT. These conditions alter the normal anatomical and electrical properties of the heart, creating a substrate conducive for arrhythmias.

In dilated cardiomyopathy, the presence of fibrotic tissue and altered ventricular geometry can disrupt normal electrical conduction. This disruption may result in the formation of reentrant circuits or altered conduction pathways that produce the alternating QRS complexes seen in BDVT. In myocarditis, inflammation and fibrosis

can similarly disrupt normal conduction and predispose to BDVT.[7]

Diagnosis

The diagnosis of BDVT requires a careful evaluation of the ECG and consideration of the clinical context. The key diagnostic features include:

1. Electrocardiogram (ECG) Findings

   As a rare clinical entity, bidirectional VT can be caused by a number of conditions, including myocarditis, fatty replacement in the RV, ATS, familial hypokalemic periodic paralysis, aconite poisoning, digoxin toxicity, and acute ischemia.

   With a normal echocardiogram and no history of aconite or digitalis intake, the majority of etiologies are ruled out, making channelopathies the most likely cause of BDVT in the index case.

2. Clinical Evaluation

   A thorough clinical evaluation is essential to identify underlying conditions or predisposing factors. This evaluation includes a detailed patient history, physical examination, and assessment of symptoms such as palpitations, syncope, or heart failure. A history of digitalis use, heart failure, or recent infections should be documented. Additional tests, such as echocardiography, cardiac MRI, or genetic testing, may be performed based on the suspected underlying cause.

Management

The management of BDVT focuses on addressing the underlying cause and alleviating symptoms. Treatment strategies include:

1. Treatment of Digitalis Toxicity

   For patients with BDVT secondary to digitalis toxicity, discontinuation of the drug is the primary treatment. In cases of severe toxicity, antidotes such as digoxin-specific antibody fragments may be administered to neutralize digitalis. Monitoring and managing electrolyte imbalances, such as hypokalemia, is also crucial in the treatment of digitalis toxicity. While digoxin-specific antibodies are typically unavailable, phenytoin is a useful and widely accessible medication. Phenytoin is a Class 1B antiarrhythmic that inhibits digitalis binding to the sodium-potassium-ATPase pump and antagonizes digitalis induced delayed after depolarization. The effective dose is 5–15 mg/kg/day infusion with a targeted serum level of 10–18 mcg/mL[8] Phenytoin however has multiple drug interactions, besides possible side effects that include vertigo, nystagmus, and lethargy.

2. Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) and ATS interventions to modulate autonomic tone may be beneficial. These interventions may include lifestyle modifications, such as reducing stress and avoiding triggers, or pharmacological treatments to modulate vagal or sympathetic activity. Beta-blockers are very effective for CPVT by preventing exercise-induced arrhythmias. Flecainide has been consistently shown to reduce ventricular ectopy and arrhythmias in patients with CPVT. Flecainide, a class IC anti-arrhythmic agent, has Na channel blocking properties. Due to its highly malignant nature and high risk of sudden cardiac death, CPVT may necessitate bilateral cervical sympathectomy for cardiac sympathetic denervation and placement of an implantable cardioverter defibrillator (ICD).

   In ATS flecainide acts by increasing the inward current generated by Kir2.1 channels (IKir2.1) in ventricular myocytes and by suppression of irregular calcium ion releases through modulation of Na/Ca exchanger.[9]

   For channelopathies genetic counseling and family screening are important for identifying at-risk relatives and providing appropriate management recommendations.

3. Herbal Aconite Poisoning:

   Aconite induces automaticity by persistently activating inward Na+ channels during the action potential's plateau (phase II), prolonging repolarization and causing afterdepolarizations with triggered automaticity, a process effectively counteracted by the Na-channel blocker flecainide and β-blockers.

4. Management of Heart Failure

   In patients with heart failure-related BDVT, optimizing heart failure management is essential. This includes the use of medications such as angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, and diuretics to improve cardiac function and reduce arrhythmic risk. Device therapy, such as implantable cardioverter-defibrillators (ICDs), may be indicated for secondary prevention of sudden cardiac death in patients with severe heart failure.

5. Treatment of Myocarditis

   Management of myocarditis-related BDVT involves treating the underlying inflammation. Corticosteroids and other immunosuppressive agents may be used to reduce inflammation and prevent further myocardial damage. In cases of severe myocarditis, additional treatments such as intravenous immunoglobulin (IVIG) may be considered.

Conclusion

Bidirectional ventricular tachycardia is a rare arrhythmia with diverse mechanisms and causes. Understanding the underlying mechanism is crucial for accurate diagnosis and effective management. Advances in genetic testing and a deeper understanding of autonomic regulation will continue to enhance our approach to BDVT, improving patient outcomes and guiding therapeutic strategies.

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Role of Electrocardiography in Cardiac Resynchronization Therapy Candidature and its Follow Up

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Introduction

Delayed electrical activation of the left ventricle(LV) is considered the underlying substrate of LV dysfunction in patients with systolic dysfunction and a conduction delay, mainly due to left bundle branch block(LBBB).[1] Cardiac resynchronisation therapy(CRT) with biventricular pacing (BIVP) aims to correct the underlying electrical substrate by paced pre-excitation of late depolarized and contracting LV regions, thereby restoring synchronous ventricular electrical activation and contraction. Thus, it improves LV systolic function, quality of life (QoL), and reduces heart failure (HF) symptoms, hospitalizations, and mortality.[2,3] Till date, one of the most important criteria for selection of patients for CRT is Electrocardiography (ECG) based. Wider the QRS duration, better would be the response to CRT in terms of both HF and mortality.[4-9] There is no role of CRT in patients with HF with underlying normal narrow QRS and sinus rhythm(SR). This review focuses on the ECG criteria for patient selection for CRT and its follow-up.

ECG Criteria for CRT

An electrical substrate, in the form of sufficient amount of LV activation delay, needs to be present for CRT to be efficient.[10-14] In this regard the following ECG patterns are considered best to predict LV activation delay and hence a better response to CRT.[15]

1. Left bundle branch block
2. Intraventricular conduction delay (IVCD) with QRS duration ≥150ms.
3. Right bundle branch block (RBBB) with masquerading LBBB.

We will discuss in detail about the definitions of each.

Left bundle branch block: It is considered the hallmark conduction disturbance that is associated with delayed LV activation. The sequence of LV endocardial activation in these patients is heterogeneous. The activation wavefront originating from the right ventricle (RV) is shown to cause LV endocardial breakthrough in different septal regions.[16-19] There is reversal of septal activation from right to left and after breaking out from LV septal endocardium activation wavefront propagates to inferior wall and then to lateral wall. A characteristic finding in true LBBB patients seems to be a long (>40 ms) transseptal conduction time.[20] Although there are discrepancies across different guidelines and criteria for the standard definition of LBBB, overall LBBB can be defined by the following criteria (Figure 1a):[15,21-26]

1. QRS duration of ≥120 ms.
2. Notches or slurring in the middle third of QRS in at least two of the following leads: V1, V2, V5, V6, I, and aVL - with a prolongation at the delayed peak in R in V5 / V6 to longer than 60 ms.
3. Generally, the ST segment is slightly opposed to the QRS polarity, and particularly when it is at least 140 ms, is rapidly followed by an asymmetrical T wave of opposed polarity.
4. Horizontal plane: QS or rS in V1 with small ‘r’ with ST

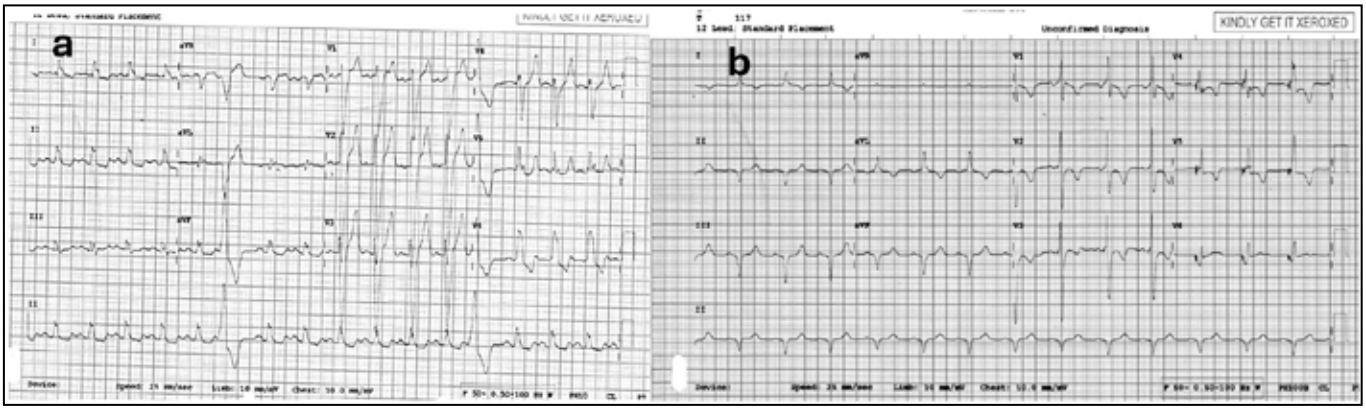

Figure 1: Cardiac resynchronization therapy (CRT) in a patient with dilated cardiomyopathy with typical left bundle branch block. Figure a and b shows the pre and post CRT implantation 12 lead electrocardiogram of the patient. See text for details.

slightly elevated and positive asymmetrical T wave and unique R wave in V6 with negative asymmetric T wave. When the QRS is less than 140 ms, the T wave in V6 may be positive.

5. Frontal plane: Exclusive R wave in I and aVL often with a negative asymmetrical T wave, slight ST depression, and usually QS in aVR with positive T wave.

6. The QRS axis can be variable.

Based on the evidence described above, the definition of a true LBBB seems to be the best starting point with which to select patients for CRT.

Non-specific intraventricular conduction delay (IVCD): Patients with IVCD generally exhibit more complex and heterogeneous ventricular activation patterns than do patients with a typical BBB on the ECG. ECG shows QRSd >110ms but not meeting morphological criteria of typical BBB.[15] These are often not primarily related to conduction system disease but are predominantly caused by an underlying myocardial disease e.g. ischemic, inheritable cardiomyopathies like hypertrophic cardiomyopathy, sarcoidosis etc (Figure 2a).[27,28]

Moreover, LV activation times in IVCD patients are generally shorter than those for patients with LBBB and the location of the region of the latest electrical activation is highly variable. Absence of sufficient LV electrical delay together with more extensive underlying myocardial disease likely results in the lower response rate observed in patients with IVCD.[27,29,30] In an analysis of the MADIT-CRT (Multicenter Automatic Defibrillator Implantation Trial With Cardiac Resynchronization Therapy) trial, Zareba et al.[21] observed that IVCD patients with LBBB features may obtain some benefit after CRT.

RBBB with masquerading LBBB: In contrast to LBBB, RBBB is typically associated with delayed RV activation, but not delayed LV activation. However, in some RBBB patients, the QRS morphology differs significantly from the characteristic RBBB pattern. These patients show a specific ECG pattern defined as RBBB masking LBBB,[31,32] which is characterized by precordial lead findings consistent with RBBB and limb lead findings consistent with LBBB. RBBB morphology in the chest leads, but with the absence of a wide negative terminal deflection in the lateral limb leads and characterized by a broad, slurred, sometimes notched R wave on leads I and aVL, together with a leftward axis deviation (Figure 3a).

Extensive measurements of both RV and LV endocardial electrical activation in HF patients with RBBB using 3 dimensional electroanatomical mapping showed that patients with RBBB masking LBBB have LV activation delay like that found in LBBB.[33] This characteristic helps to identify patients with RBBB who not only have a delayed RV activation, but also a delayed activation of the LV free wall. These patients may also show a good response to CRT.[34,35] Patients with isolated RBBB do not benefit from CRT unless as mentioned above there is associated masquerading LBBB or a prolonged PR interval.[15]

An important recent notion is the possible role played by a prolonged PR in HF patients with non-LBBB. In MADIT-CRT,[21] the subgroup of non-LBBB patients who had a prolonged PR did benefit from CRT-D, with a 73% reduction in the risk of HF or death and an 81% reduction in the risk of all-cause mortality compared with ICD-only therapy. Patients with LBBB pattern demonstrates more benefit than those with RBBB or non-specific IVCD patterns. Patients with the combination of a normal PR interval and non-LBBB morphology may even have a higher mortality risk after CRT.[36-38] Currently, only symptomatic patients with HF and LVEF ≤ 35%, in SR with LBBB and a QRS duration of at least 150 ms in their native ECG have a class IA indication for CRT. Recommendations are weaker (IIA) for patients with symptomatic HF patients with LVEF ≤ 35%, an LBBB pattern and a QRS duration of 130-149 ms and for symptomatic HF patients with LVEF≤35% with a non-LBBB pattern and a QRS duration of ≥150 ms. Recommendations are weakest and questionable (IIB) for patients with symptomatic HF patients with LVEF ≤35%, non-LBBB ECG pattern and a QRS duration of 130–149 ms.[39,15]

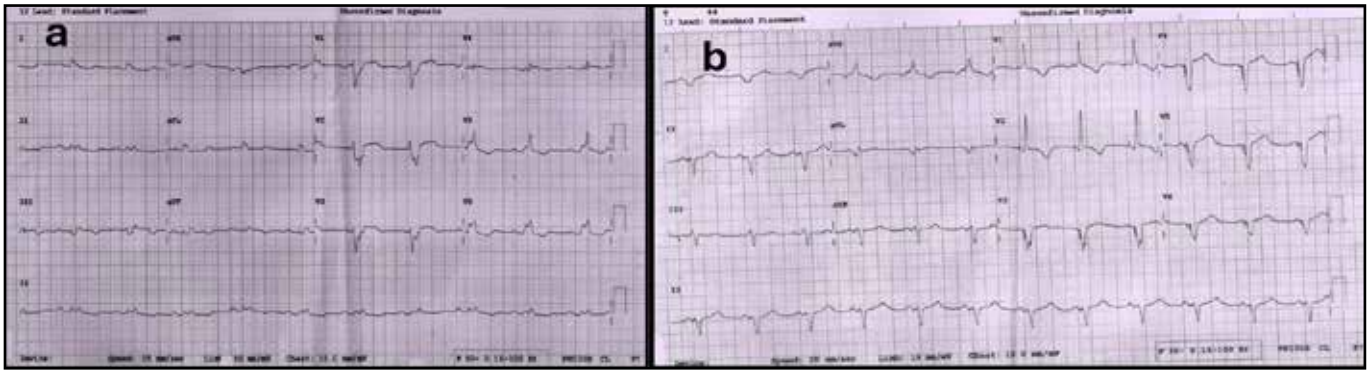

Figure 2: A case of ischemic cardiomyopathy with refractory heart failure with intraventricular conduction delay (IVCD). Twelve lead electrocardiogram before and after cardiac resynchronization therapy. See text for details.

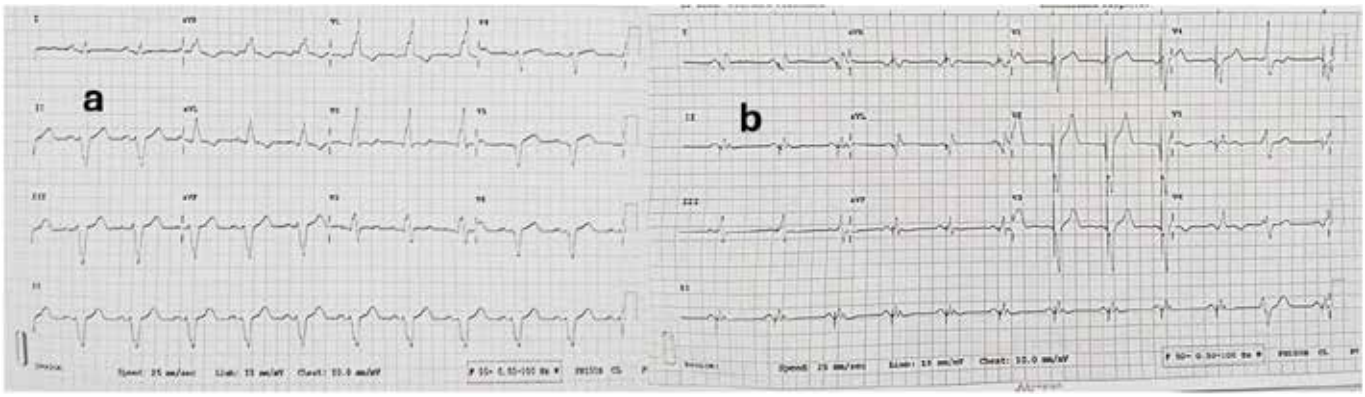

Figure 3: Cardiac resynchronization therapy (CRT) in a patient with masquerading bundle branch block. Twelve lead electrocardiogram (ECG) showing pre (a) and post (b) CRT implantation ECG in a patient with ischemic cardiomyopathy with drug refractory heart failure. See text for details

Follow Up

The CRT device allows programming of the atrioventricular (AV) and interventricular delay (VV) to optimize the positive effects of BIVP. Various studies have shown that LV pacing alone can be as effective as BIVP.[40-42] Especially in patients with normal AV conduction, when LV only pacing is adequately timed with intrinsic activation, response in cardiac function improvement can be even superior to that with BIVP.[41,42] Surface ECG on follow up is key to assess weather proper resynchronisation of LV has been achieved or not.

Axis: The QRS axis in the frontal plane during BIV pacing is most often directed towards the right superior quadrant resulting in a dominant R wave in lead aVR. Sometimes, with a more posterior LV lead position, the QRS axis is directed towards a left superior quadrant.[43]

QRS in precordial leads: To assess whether there is contribution of LV pacing, QRS morphology must be evaluated in the precordial leads. The QRS complex during BIVP most often has a dominant R wave in lead V1-2, suggestive of contribution from LV pacing. However, a dominant R wave in lead V1 is not diagnostic of BIVP as RV only pacing occasionally produces the same pattern. Therefore, adequate assessment of BIVP should include evaluation of RV only and LV only pacing. A negative QRS complex in lead V1 should warrant further evaluation, although it does not necessarily indicate inadequate CRT, as this may occur in incorrect lead V1 placement, marked LV pacing latency or slow activation from the LV pacing site, LV lead dislodgement or inappropriate LV lead placement (middle or anterior cardiac vein).[(43)] Expression of biventricular fusion on the ECG by new or increasing R wave in lead V1 and V2 is significantly related to the probability of reverse remodelling after adjusting for the degree of myocardial scar (Figure 1b,2b,3b).[44]

Lateral leads: With contribution from LV pacing the overall activation reverses to dominant negativity in leads I and aVL (Figure 2b). It has been shown to correlate with acute hemodynamic improvement after AV- and VV- optimisation.[45]

QRS width: With good BIVP, the QRS becomes narrow, and the LV activation time is shortened**.** When the QRS duration is not decreasing and no contribution from LV pacing is seen, further analysis of the ECG during intrinsic rhythm, RV only, and LV only pacing can reveal inadequate CRT programming and LV lead positioning.[46]

Conclusion

Evaluation of 12 lead surface ECG is the gold standard for both selecting the patients for CRT and their follow-up, in patients with severe LV systolic dysfunction with HF.

Conflict of Interest: None to declare

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ECG Predictors of the Optimal Pacing Site

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Introduction

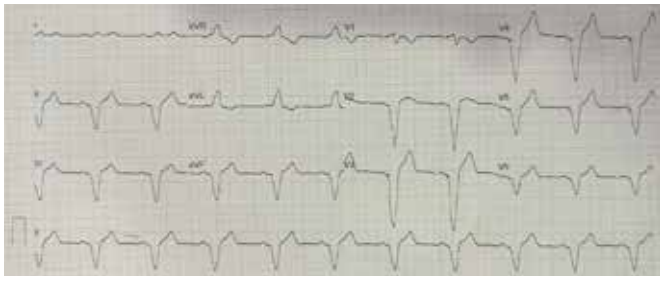
The advent of multisite pacing for the treatment of congestive heart failure (CHF) has added a new dimension to the electrocardiographic evaluation of pacemaker function.[1] The "low-tech" paced 12-lead ECG was relegated to a minor role and often neglected.[2] The reading of a 12 lead ECG is an art. Identifying the vector of the ventricular activation, the QRS, determines the site of earliest activation. It is described best by studying the concepts provided by Willem Einthoven and his theory of the Einthoven Triangle to describe the ECG vectors more than a century ago.[3] Einthoven recognized the great potential importance of the ECG as a diagnostic and investigative tool and his achievements made him the founder of modern electrocardiography. The Einthoven triangle is shown in **Figure 1** for reference of the readers. Based on these principles, the anatomical location of the pacemaker lead can be ascertained in a patient with an implanted pacing device. This follows the principle of Vector law, which states that the net vector will be negative if the activation wavefront is going away from the sensing electrode and vice versa. Int eh ECG, for right atrial pacing, there is a paced P wave confirming atrial depolarisation while with ventricular pacing, the QRS has a bundle branch block configuration. Nowadays, biventricular (BV) pacing has generated a well deserved renaissance of the 12-lead paced ECG that has become an indispensable tool in the evaluation of cardiac resynchronization. However, It is important to understand the characteristics of traditionally paced rhythms before embarking on the journey of deciphering the biventricular and conduction system paced ones.

The common sites of ventricular lead placement have been described below along with their characteristic paced QRS patterns and the pros and cons of the individual sites. This review focuses on the 12 Lead ECG aspects of each pacing site. The technical specification of procedural nuances are beyond the scope of this review

The Potential sites for pacing :

1. RV Apical pacing:

   The RV Apical pacing is a traditional pacing site taught to the Cardiologists during their training. It generally involves placing the RV lead in the apex or the apical septal location in the right ventricle.



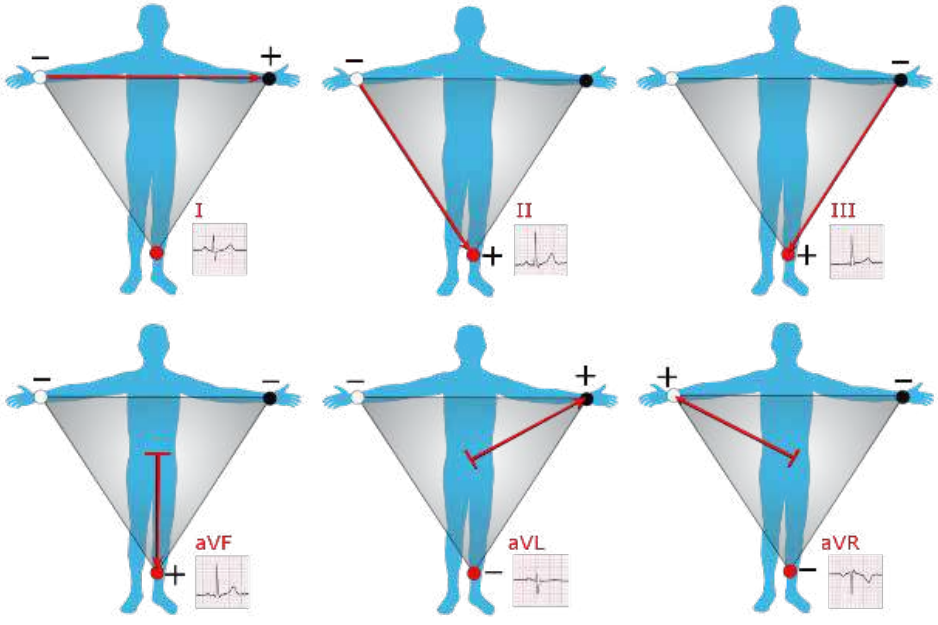


Figure 1: The Einthoven's Triangle:
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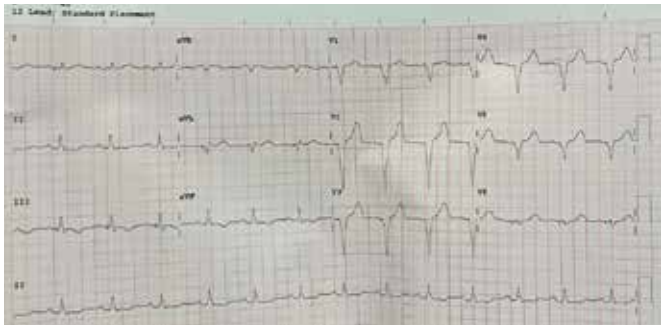
- The paced ECG of this anatomical site is very characteristic and as follows:
  - QRS width: Wide paced QRS (>150 ms)
  - QRS Axis- Left and Superior (0 to -90)
  - LBBB morphology with negative QRS vector in Inferior leads (II,III,aVF)
- Pros:
  - Easy to perform and a simple learning curve.
  - Least chance of macro-dislodgement
- Cons:
  - Paced wavefront is not physiological.
  - Greater risk of perforation
  - Clinical data suggests that there is a higher risk of pacing induced cardiomyopathy in these individuals.

2. Mid Septal Pacing:

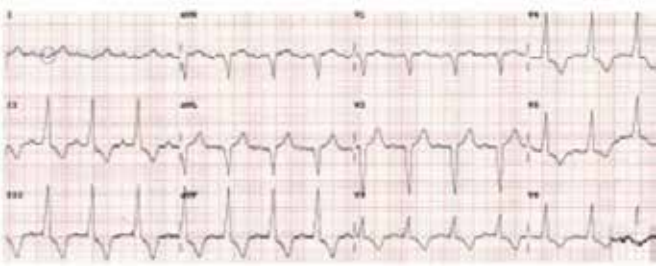
   This was the most commonly placed lead position in pacemaker implantations before the advent of the so called conduction system pacing. The pacing lead is placed arbritrarily between the apical septum and the outflow septum and consists of a large area of septum as a potential site. The lead placement is based on fluroscopic landmarks alone,in contrast to conduction system pacing where the surface ECG is also taken into account.[4]



- The paced ECG of this anatomical site are varied and as follows:
  - QRS width: variable from 120-150
  - QRS Axis- Left and Superior (0 to -90)
  - IVCD type of morphology with Rs in left sided leads (Lead II, avL) and a predominant positive QRS deflection in inferior leads.
  - The Transition of QRS occurs around V3-V4.
- Pros:
  - Relatively narrower paced QRS with evidence to suggest lower risk pacing induced cardiomyopathy.
  - Lower risk of perforation
- Cons:
  - Some training and experience required while implanting at this site.
  - Risk of lead dislodgement is technically higher than apically placed leads.

3. Outflow tract (High Septal Pacing):

   Outflow tract is a rare anatomical site of pacing. It is usually chosen as the target site when there is an impediment to place the pacing lead at other sites owing to extensive fibrosis of the septum in cases of Ischemic Heart Disease or Structural heart disease like an operated Ventricular Septal Defect. On rare occasions, the lead may be placed electively at this position in view of very poor pacing and sensing lead parameters at other sites.[4]



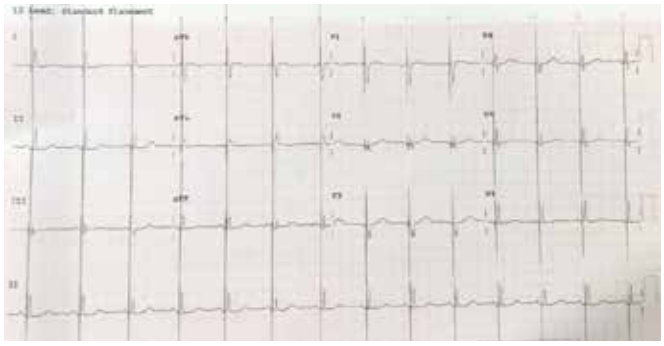
Adapted from : Das A, Kahali D. Ventricular septal pacing: Optimum method to position the lead. Indian Heart J. 2018 Sep-Oct;70(5):713-720.

- The paced ECG of this anatomical site is as follows:
  - QRS width: variable from 120-150
  - QRS Axis- Right and Inferior (+90 - 120)
  - LBBB type morphology with a Positive QRS vector in Inferior leads and a negative QRS vector in Leads aVL and aVR.
  - rS in V1 suggesting high septal origin of the paced QRS.
- Pros:
  - Relatively narrower paced QRS with evidence to suggest lower risk pacing induced cardiomyopathy.
  - Lower risk of perforation

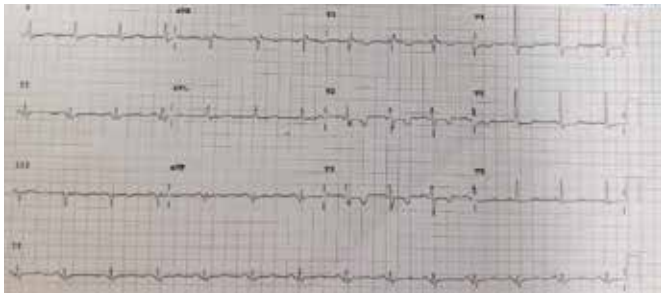
- Cons:
  - Significant Risk of lead dislodgement

4. Conduction System Pacing; the His Bundle/ Left Bundle Branch Area Pacing:

   The Conduction system acing pacing is considered to be most physiological method of pacing the right ventricle. It involves placing the right ventricle lead under support of a pre shaped sheath along the His Bundle area of His Bundle pacing or along the interventricular septum for Left Bundel area pacing and screwing the lead deep into these structures to get a very characteristic narrow paced QRS pattern of ECG. The 12 lead surface ECG is vital not while locating the best site of screwing but also for ensuring that we have adequately captured the desired conduction system target.[5]



Left Bundle Area Pacing in Unipolar configuration



Left Bundle Area Pacing in Bipolar configuration

- The paced ECG of this anatomical site is as follows:
  - QRS width: 100- 120 ms (may vary with pacemaker output settings)
  - QRS Axis- left (0 - +60)
  - Characteristic QRS vectors: positive QRS in Lead II and avL; Negative QRs axis in III and aVF.
  - A characteristic terminal R with a RSr' (RBBB) pattern in lead V1.
  - The so called R wave peak time (V6) is less than 85 msec, suggesting early LV activation. This is calculated from the pacing spike to the peak of R wave in V6 or any other left sided leads.
  - The V6-V1 interval – The time difference between the R waves in V6 & V1 leads of more than 45 ms suggests a good selective Left Bundle area capture.
- Pros:
  - Narrow and physiologically paced QRS with evidence to suggest lower risk pacing induced cardiomyopathy.
  - Lower risk of perforation
- Cons:

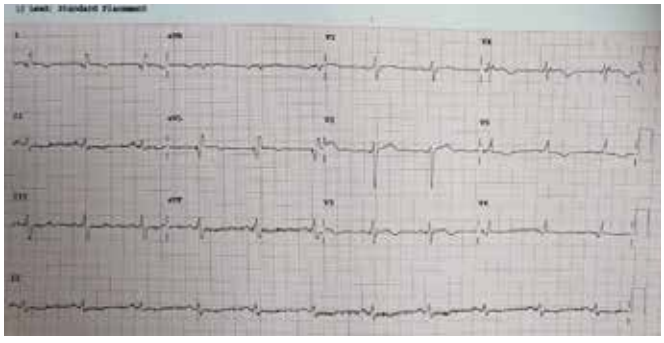
  Requires special hardware to place the lead.

  A continuous 12 lead ECG monitoring is required.

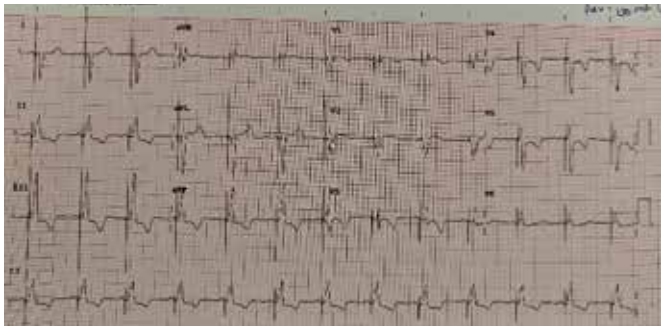
  Long term data regarding the lead stability and success outcomes is still evolving.

5. Biventricular Pacing :

   This is a traditional pacing modality for patients with LBBB and Heart failure or any Left Ventricular Systolic Dysfunction with AV block with a pacing indication. It involves placing a Right Atrial lead, Right Ventricular lead and the Coronary Sinus / Left Ventricular lead in the Right Atrial Appendage, IV septum and along the posterolateral or lateral target tributary veins in the Coronary sinus respectively. The principle is to pace the right and left ventricles in a synchronised fashion to avoid any worsening of heart failure due to non physiological right ventricular activation.[6]



Cardiac Resynchronisation Therapy in Bipolar configuration



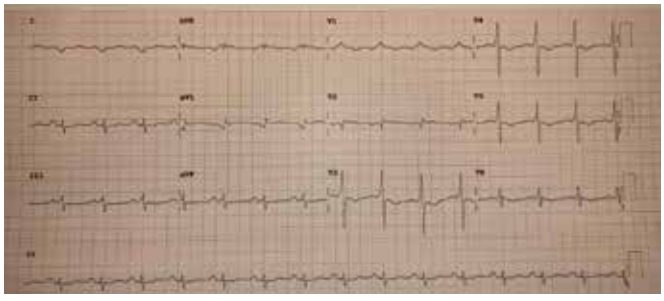
Cardiac Resynchronisation Therapy in Unipolar configuration

- The paced ECG of this anatomical site is as follows:
  - QRS width: variable from 110-140 (depending upon the level of LV scarring & Dilation)
  - QRS Axis- Right to Norwest (+120 to +270)
  - RBBB type morphology with 2 pacing spikes noted before the QRS suggesting Biventricular activation.
  - RBBB in V1 with a predominant negative QRS vector in leads aVL and Lead I suggesting a LV pre-excitation.
- Pros:
  - Narrow paced QRS with evidence confirming its credibility Heart failure optimisation and mortality reduction.
- Cons:
  - Challenging venous anatomy can prolong the procedure time as well as affect the procedural success.

6. Left Ventricular Only Pacing (Left Ventricular Predominant pacing) :

   It is a special variant of Cardiac Resynchronisation therapy in patients with an intact AV nodal conduction. The Right Atrium and Left Ventricle are paced in synchronic and the left ventricular activation is fused with the native RV activation. This results in a narrow and sharp paced QRS. This is a relatively more efficient way to pacing the left ventricle in heart failure patients.

   - The paced ECG of this anatomical site is as follows:[7]



  - QRS width: variable from 120-140
  - QRS Axis: Right to Norwest (+90 - 120)
  - RBBB morphology with a sharp QRS upstroke
  - predominant negative QRS vector in leads aVL and Lead I suggesting a LV pre-excitation.
- Pros:
  - Relatively narrower paced QRS with evidence to suggest lower risk pacing induced cardiomyopathy.
- Cons:
  - Challenging venous anatomy can prolong the procedure time as well as affect the procedural success.
  - Has concerns of pacing safety in patients with an AV nodal block due to epicardial placement of the lead.

Conclusion

A thorough knowledge of the basics of reading a 12 lead ECG can help to not only locate the anatomical pacing site in a patient with a pacemaker, but also help to detect any potential pacing lead faults, especially in patients with a biventricular pacemaker. Hence, it is vital to have a conscious and thorough understanding of paced ECGs in the practice of Cardiology.

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Electrocardiographic Abnormalities in Duchenne Muscular Dystrophy Patients and its Affecting Factors

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Introduction

Duchenne muscular dystrophy (DMD), the most common type of muscular dystrophy is a X-linked genetic disorder, caused by mutation of the gene encoding dystrophin on chromosome Xp21. This gene is the largest known human gene, containing 79 exons. It has an incidence of ≈1 in every 5000 live male births (≈20 000 new cases worldwide each year). The loss of dystrophin protein results in a progressive, symmetric myasthenia and amyotrophy. Symptoms start with abnormal gait at 3-4 years of age, which progresses to loss of walking ability at 10-12 years and most of them do not survive beyond 18-20 years of age. With advancement in supportive care and multidisciplinary management DMD patient's survival can be extended up to 30 years. Previously respiratory failure was the most common cause of death in DMD patients but with improvement in survival, cardiomyopathy is becoming the primary cause of death.

DMD -Cardiomyopathy

DMD causes dilated cardiomyopathy which presents with heart failure or arrhythmia. Patient becomes symptomatic during second decade of life. Nigro et al showed that the cardiomyopathy is progressive and can be detected preclinically at 6 years of age.[1] Clinical cardiomyopathy is seldom seen before 10 years of age while after 18 years of age almost all patients have signs and symptoms of cardiomyopathy.[1]

A randomized, clinical trial reported that institution of ACE inhibitors before the second decade delayed the onset of LV dysfunction and reduced mortality.[2]

Detecting the cardiomyopathy in preclinical stage and initiation of early and aggressive treatment can delay the progression of disease and will result in improved survival.

ECG in DMD

Population and animal studies have suggested that ECG changes may be the forerunner of cardiac involvement in DMD patients. ECG abnormalities can appear before myocardial fibrosis. ECG may be more sensitive than echocardiography and even CMR imaging in detecting early manifestations of DMD cardiomyopathy.[3]

Pathophysiology of ECG changes

Dystrophin is a cytoskeletal protein located on the plasma membrane of skeletal muscle cells and cardiomyocytes. It acts as a cell scaffold, maintains the integrity of muscle fibres, and protects against contraction-induced damage. Lack of dystrophin causes the rupture of muscle membranes during contraction, leading to rise in intracellular calcium levels, which in followed by a cascade reaction in cells, resulting in inflammation, necrosis, and fibrosis.

Subcellular changes in patients with DMD often involve the sub-epicardium of the posterior basal part of the left ventricle, which could explain the fact that the most common abnormal ECG changes in children with DMD are deep Q waves at the anterolateral wall leads.[4]

Animal experiments have confirmed that abnormal ECG changes in animals with muscular dystrophy occur earlier than myocardial fibrosis[5] (i.e., ECG changes appeared without cardiomyocyte necrosis or fibrosis), suggesting that there may be other mechanisms underlying ECG changes in children with DMD.

Tang et al. speculated that deletion of dystrophin in the membrane of Purkinje fibrocytes is the pathophysiological basis of early-stage ECG abnormalities in children with DMD.[6]

Abnormal ECG findings

Higher resting heart rate

This higher resting heart rate is a form of abnormal heart rate variability caused by impaired cardio-autonomic regulation and is associated with myocardial fibrosis. These boys do not have an age-related decline in heart rate but rather an increase in heart rate that precedes the onset of cardiomyopathy. Those DMD boys who are in the upper quartile for heart rate are more likely to progress to cardiomyopathy within 5 years.[7]

Deep Q waves

Deep Q waves predominantly seen in anterolateral leads(I,Avl,V5,V6) is the most common ECG abnormality in DMD patients.[3,8] It is sometimes seen in inferior leads also. A characteristic pattern of myocardial damage involving inferolateral wall resulting in changes in electromotive forces is responsible for these ECG changes in DMD patients.

Prominent R:S ratio in lead V1

Prominent R in V1 is the second most common finding in DMD patients (Figure.1A). Similar findings are referred to as right ventricular hypertrophy in some studies.[3,8,9] Damage to

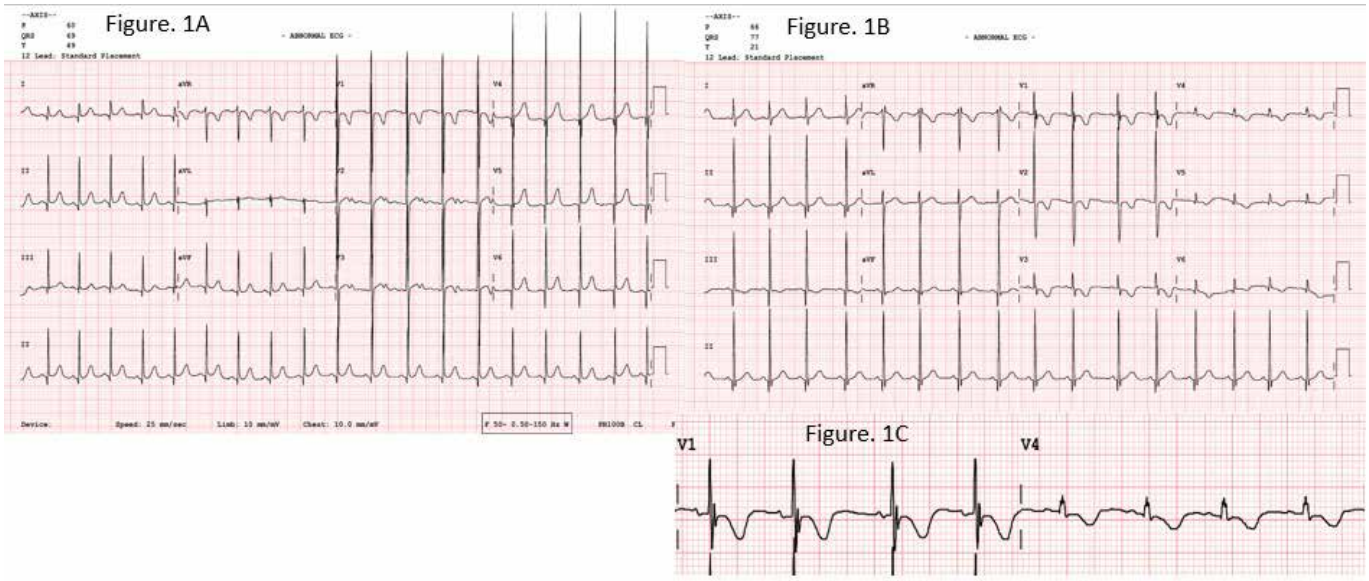

Figure 1A: ECG of A 6 old DMD patient showing prominent R in (V1 >20 mm) with deep Q V5 V6 (>5 mm) as well as Q waves seen in anterior and inferior leads. **Figure 1B and 1C:** (zoom view): ECG of 9 year old DMD patient with fragmented QRS in V1 as well notching is seen in V2-V4.

inferolateral wall leading to alteration in QRS vector may be a reasonable explanation for this finding.

Short PR interval

Kazuhiko Sega speculated that the absence of dystrophin in the conduction system would lead to left axis deviation and a shortened PR interval.[10] According to Peroloff[11] this is likely due to accelerated AV conduction and rarely due to presence of an atriofascicular pathway. As atrial arrhythmias are rare in DMD patients this finding is unlikely to be clinically significant.

Prolonged QTc interval

Prolonged QTc is a rare finding in DMD patients. QTc interval increases with age and the difference as compared to normal controls becomes statistically significant above 10 years age.[12]. In some larger studies on DMD patients the prevalence of prolonged QTc was reported between 0-4%.[3,9,13]

Fragmented QRS (fQRS) and Bundle branch blocks

The fQRS (defined by the presence of an additional R wave (R') or notching in the nadir of the R-wave or the S-wave, or the presence of more than one R' without a typical bundle branch block) which is marker of regional myocardial damage, is common in DMD patients (figure 1B,1C).[12] The leads showing fQRS increase along with age. The presence and progress of fQRS may be a useful marker of cardiac involvement for detection of cardiomyopathy at an early stage.[12]

Prevalence of bundle branch blocks in DMD patients increases with age. It is almost absent below 10 years of age and becomes more common in twenties. RBBB is 2-3 times more common than LBBB.[10,14]

Factors influencing ECG abnormalities in children with DMD

Age

Deep Q wave and number of individuals with low amplitude of RV5+SV1 increases significantly with age, while other types of ECG abnormalities exhibited no significant difference.[8] Conduction blocks, atrial and ventricular arrhythmias increased with age especially those with impaired cardiac function.

Genotype

Understanding the relationship between the ECG abnormalities and genotype can help us to predict the future course of development of cardiomyopathy as well as to diagnose and treat it in preclinical stage.

Distribution of gene exon deletion in DMD patients was more common in exons 3–21 and 45–52, and the frequency of ECG abnormalities in the exons 1–2 and 44–45 deletion groups demonstrated statistically significant differences from groups with deletion(s) at other loci.[14]

Cardiac Function

As previously described, Impaired cardiac function is a risk factor for various types of arrhythmias in DMD patients. Incidence of ECG abnormalities were significantly higher in DMD patients with dilated cardiomyopathy as compared to those with normal LV function.[13]

Although Dominant R wave in V1 is presumed to be due to fibrosis of postero-basal part of heart, it has no relation to

LV systolic function whereas patients having LBBB have significantly lower ejection fraction than non LBBB patients.[15]

Conclusion

Cardiovascular complications have become the main cause of death in DMD patient population. The onset of cardiomyopathy is inconspicuous, but the prognosis is poor once it progresses to heart failure. ECG abnormalities may more sensitively reflect myocardial involvement at early stage in DMD patients compared with echocardiography and even CMR imaging. Pathological Q, increased R wave amplitude with increased R/S ratio at lead V1, short PR interval, fragmented QRS were common abnormal ECG changes in children with DMD. ECG abnormalities increase with age and reduction in LV function. Conduction block, atrial and ventricular tachyarrhythmias mostly occur in older children and those who have established left ventricular dysfunction.

Abbreviations

DMD: Duchenne muscular dystrophy

ACE: Angiotensin converting Enzyme

LVEF: Left ventricular ejection fraction

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NOTES

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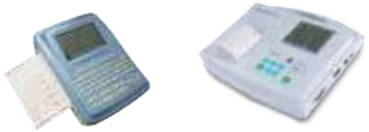

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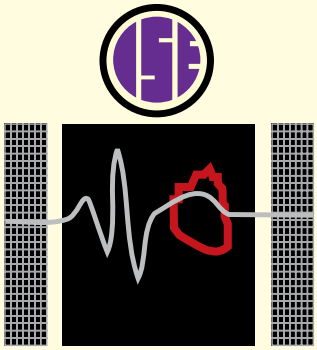

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