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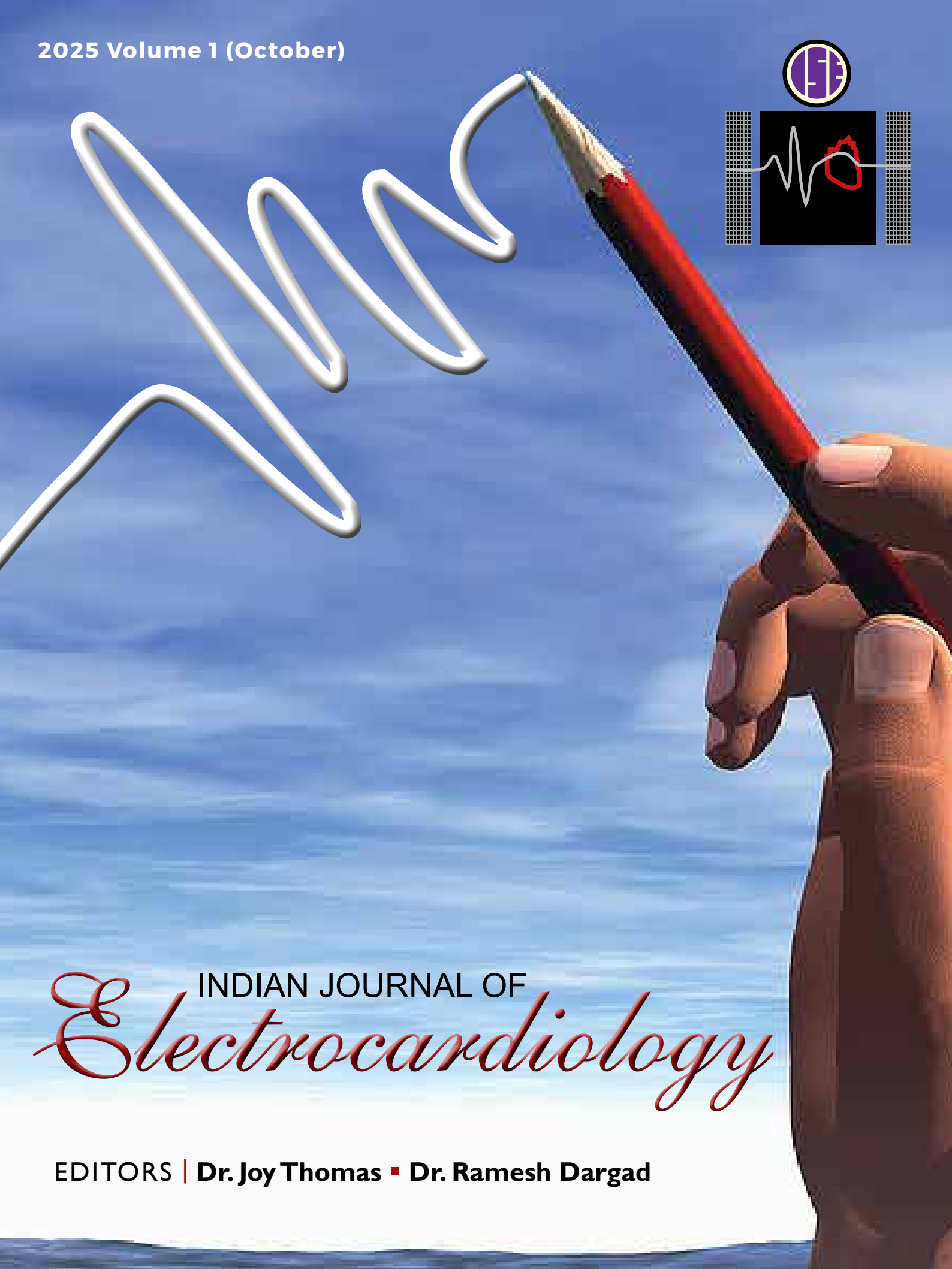

INDIAN JOURNAL OF Electrocardiology

EDITORS | Dr. Joy Thomas ▪ Dr. Ramesh Dargad

UPCOMING EVENT

ISECON 2026

27th-28th February-1st March 2026

Novetel Jaipur Exhibition and
Convention Centre (JECC), Jaipur

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Editorial

Ever since the Golden Jubilee edition of the Indian Journal of Electrocardiology was published in 2022 it has been a roller coaster ride for me and my various co-editors, due to the varying promptness with which the agreed upon articles finally land in our mail boxes. Amidst the busy practice, clinical and teaching responsibilities that the contributing authors face in day to day life the task of writing up an article is by no means an easy task, but yet again the contributors have fulfilled it in no small measure. My co-editor Dr Ramesh Dargad has gone about his task with great enthusiasm and cobbled together six very interesting and illuminating topics.

This issue begins with an article by the evergreen Dr Yash Lokhandwala, assisted by his colleagues Drs Chetan Mahajan and Suresh Joshi who have presented a case discussion on Catecholaminergic polymorphic VT with bradycardia in a child touching upon clear approach to the case and its management.

Drs Balamurugan and Anand have defined the various stages in the ischemic cascade with an illustration of the ECG changes that accompany it. Mistaken lead placements do happen amidst the busy schedule of an ECG technologist and Drs Srinivasan Ramadurai and T R Muralidharan have unravelled the changes it can cause.

Hypertension has been talked about for decades but the ECG changes peculiar to that has been brought out very comprehensively by Dr S B Gupta our patron.

Drs Vinod Vijan and Vikrant Vijan have discussed about the disturbances in the electrical system of the heart that can lead to a short QT and its serious consequences.

Fascicular blocks are quite often seen in ECGs and identifying them has implications in identifying the effects of various illness and the direction and scope of treatment. This has been done effectively by Drs Arun David Arul and Joy M Thomas.

Diagnosing myocardial infarction in a patient with chest discomfort needs urgency and when such patients present without the usual ECG changes it can be challenging situation and the article by Dr Arvind Ghongane throws abundant light on this topic.

Obesity is becoming an epidemic in our country and Drs Ramesh Dargad and Harish Tantia have succinctly brought out the special features in the ECG of these patients.

Present day publications will not be complete without a word about Artificial Intelligence (AI) and Dr Gurunath Parale will guide you through the complex web of AI and its application in ECG recording, diagnosing, storing, retrieval and most importantly more effective patient management.

Hope you enjoy this yet another publication from the Indian Society of Electrocardiography

Dr. Joy M Thomas
Editor

Dr. Ramesh Dargad
Co-Editor

From the Desk of Advisor

Dear Members,

It is indeed a great pleasure that Indian Society of Electrocardiology is bringing the new issue of Indian Journal of Electrocardiology, the Official Journal of Indian Society of Electrocardiology on the eve of ISE Mid-term Conference to be held at New Delhi on 4th-5th October 2025. Dr Joy Thomas and Dr Ramesh Dargad have really worked hard to get the articles who will be of help I day-to-day practice for the post-graduates, physicians, cardiologists and even by the electrophysiologists.

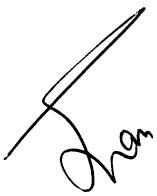
Current issue of Indian Journal of Electrocardiology has very useful articles like ECG in ischemic cascade, Understanding ECGs in lead misplacements, ECG in hypertensive heart disease, Short QT Syndrome, fascicular Blocks, MI without traditional ECG signs, ECG changes in obesity and current burning topic of Artificial Intelligence in ECG and a case report of Bradycardia and Polymorphic VT, The readers will enjoy reading them and be benefitted.

I would like to thank Dr Jitendra Makkar, President ISE, Dr Ashish Nabar, Treasurer ISE and Dr Ketan Mehta, Secretary ISE for their support.

My heartfelt thanks to the Journal Editors, Dr Joy Thomas and Dr Ramesh Dargad for their hard work to bring the IJE October 2025 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,

On behalf of Indian Society of Electrocardiology, I am delighted to present our esteemed journal. This platform showcases cutting-edge research and advancements in electrocardiology, fostering knowledge sharing and collaboration among professionals.

This journal aims to promote excellence in cardiac rhythm management, electrocardiography, and related fields. I request esteemed authors, researchers, and clinicians to keep contributing their work and share their expertise.

I extend my gratitude to the editorial team, reviewers, and contributors for their tireless efforts. Together, let's advance the field of electrocardiology and improve patient care.

Dr. Jitendra Makkar
President
Indian Society of Electrocardiology

A Child with Sinus Bradycardia and Catecholaminergic Ventricular Tachycardia

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Abstract

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an inherited arrhythmia syndrome characterized by exercise- or stress-induced ventricular tachyarrhythmias. Sinus node dysfunction is an unusual but clinically important association.

A 16-year-old boy presented with recurrent syncope and palpitations. Resting ECG revealed sinus bradycardia with junctional escape rhythms. Exercise testing induced bidirectional/polymorphic VT, and genetic testing confirmed a CASQ2 mutation. The patient was treated with an AAIR pacemaker, propranolol, and bilateral thoracic sympathectomy, leading to resolution of arrhythmias. At four months follow-up, he remained asymptomatic.

Conclusion: Sinus node dysfunction may coexist with CPVT, influencing both clinical presentation and therapeutic strategy. Recognition of this association can guide more tailored management approaches.

A 16-years-old boy presented with a history of palpitations with recurrent syncope for the last 5 years. During these episodes, he would feel a brief prodrome of rapid palpitations before passing out. Then there would be tonic posturing of limbs, no clonic movements, followed by prompt and complete recovery. There was no associated sweating, nausea or incontinence. There was no family history of cardiac disease or sudden death.

He was investigated in a local hospital for the same where sinus bradycardia with junctional escape was noted. He was started on orciprenaline and for a few months he was apparently better. However, because of another syncope at a family event where he had rushed on stage for a group photograph, he was sent to us for further evaluation and management. After arrival, he was afebrile with a pulse of 48/min, blood pressure 120/60 mmHg and a normal systemic examination. The ECG (Figure 1) revealed sinus bradycardia intermingling with junctional escape complexes in an isorhythmic AV dissociation pattern; there were isolated Tall T waves in lead V2.

The echocardiogram and routine biochemistry were normal. A treadmill test was performed. During moderate exercise he felt tired with rapid palpitations, bidirectional/polymorphic ventricular tachycardia was documented (Figure 2). A diagnosis of catecholaminergic polymorphic ventricular tachycardia (CPVT) was made.

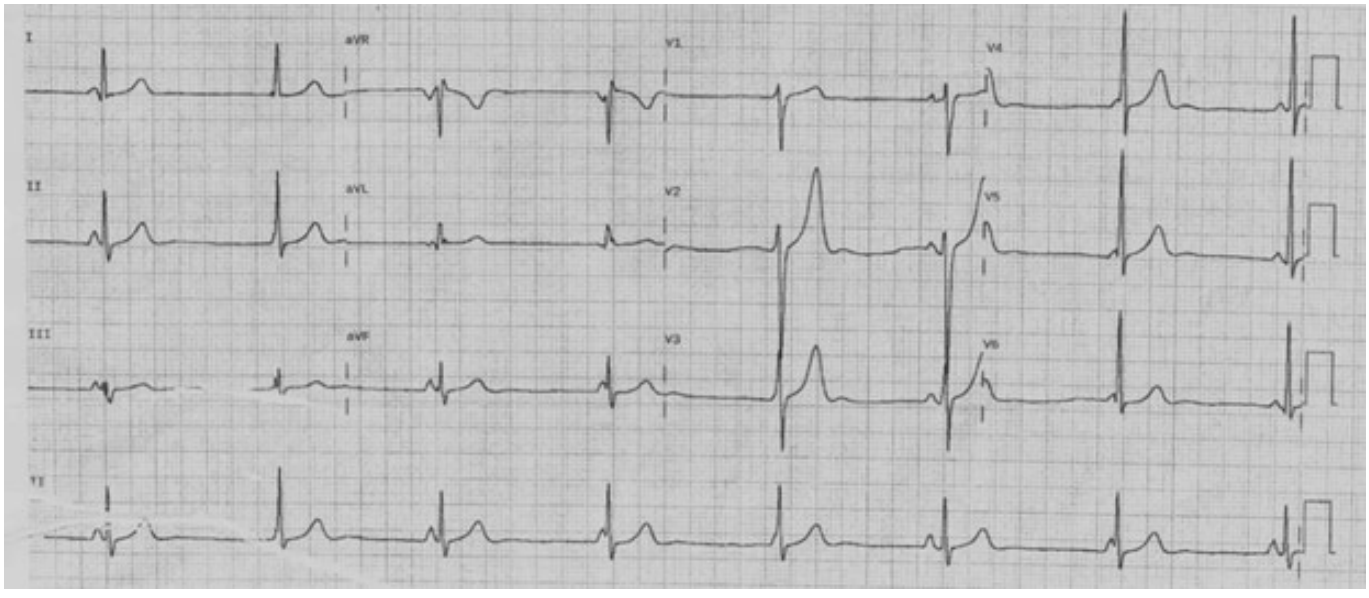

Figure 1: Sinus bradycardia, junctional escapes with isorhythmic AV dissociation.

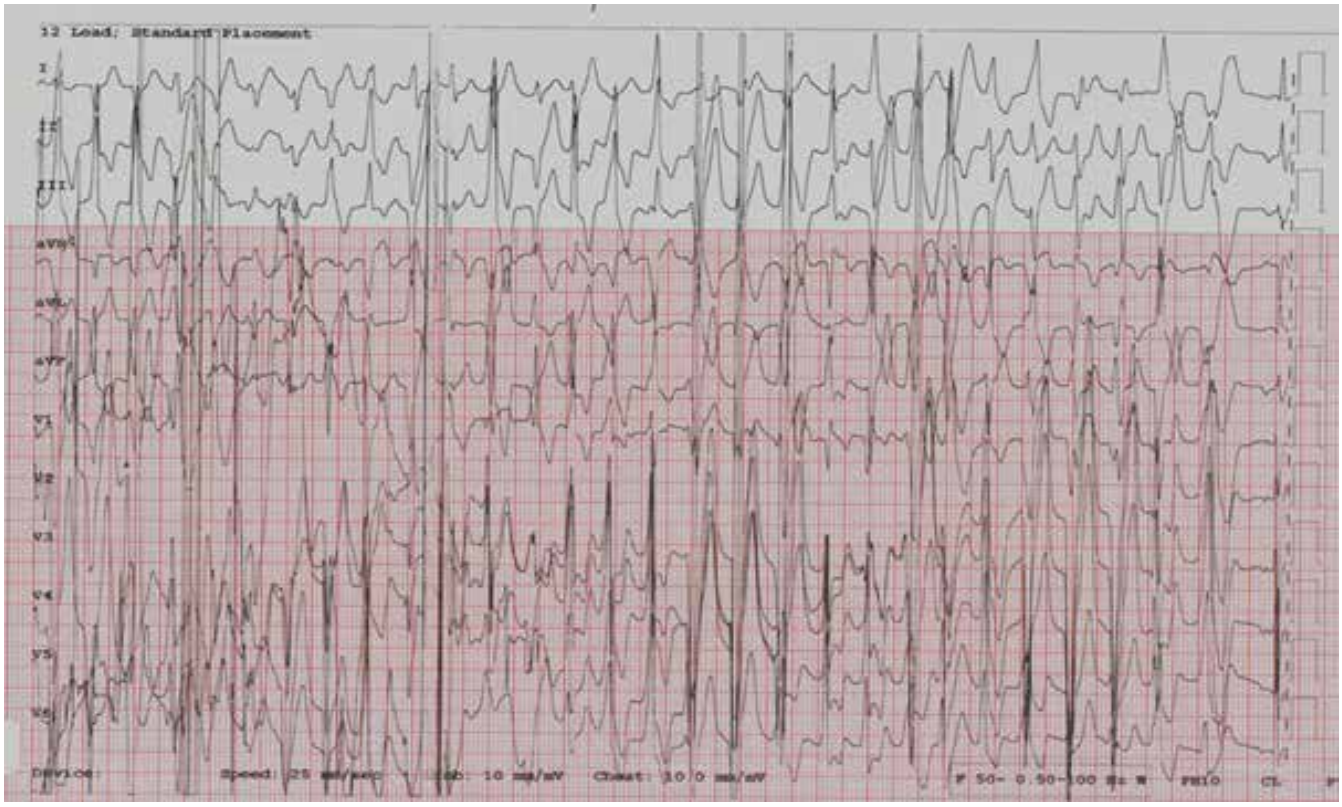

Figure 2: Rapid rate, broad bizarre QRS complexes with changing morphologies- positive, negative, biphasic.

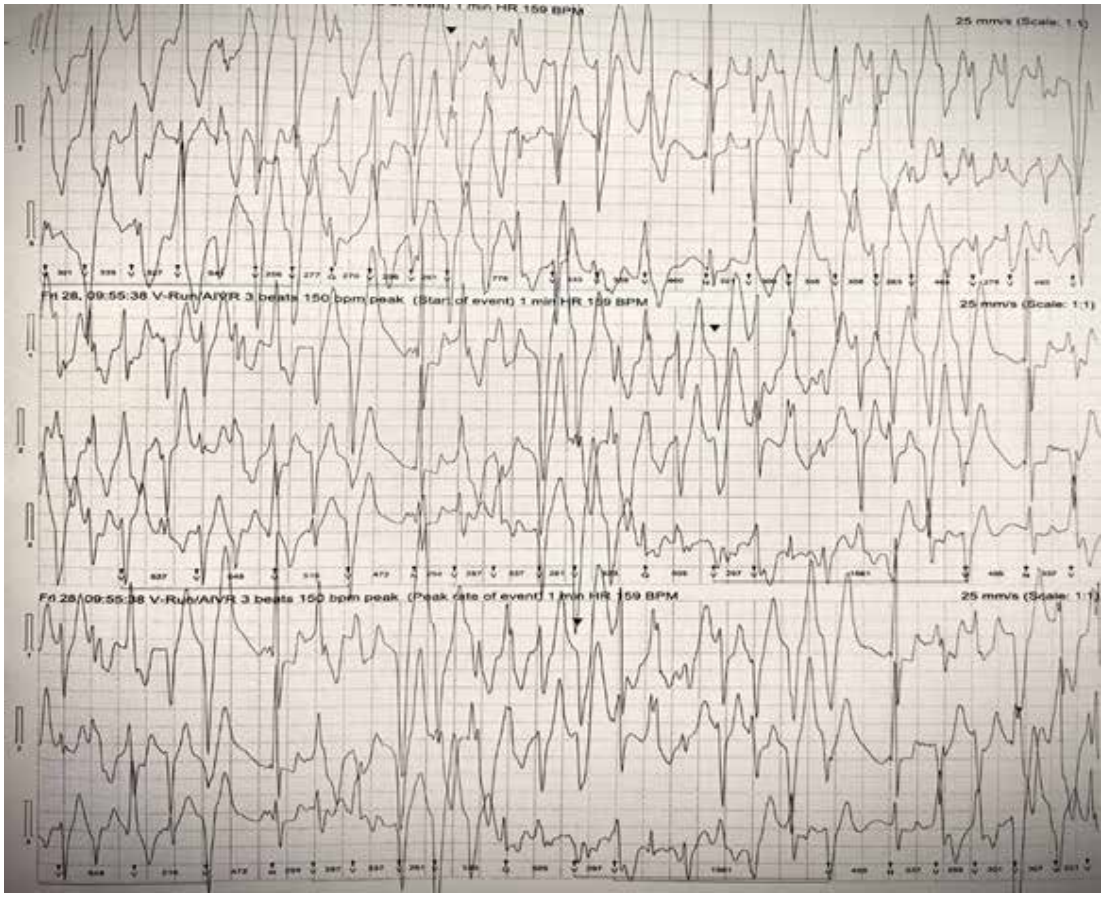

Figure 3: 24 hours Holter monitoring tracing which showed persistent exertional ventricular tachycardia.

The boy underwent an atrial-based (AAIR) pacemaker implantation and was then started on propranolol 20 mg twice a day. Three days later, a 24 hours Holter monitoring was performed, during which he was instructed to intermittently climb stairs and walk briskly. A review of the Holter showed that exertional ventricular tachycardia persisted (Figure 3).

Bilateral upper (T1-T4) thoracic sympathectomy was performed. Three days after this, another 24 hours Holter was performed, again with similar physical activity as before. This time, no arrhythmia was seen. Genetic testing was conducted which showed a CASQ2 mutation, confirming diagnosis of CPVT. At four months follow up, the boy is doing well.

Discussion

Catecholaminergic polymorphic ventricular tachycardia is an inherited rhythm disorder characterized by the occurrence of potentially life-threatening polymorphic ventricular tachyarrhythmias in conditions of physical or emotional stress in a structurally normal heart. The underlying cause is a dysregulation in intracellular calcium handling due to mutations in the gene encoding the sarcoplasmic reticulum calcium release channel (RYR2), or in genes encoding the RyR2 binding proteins cardiac calsequestrin (Casq2), triadin and calmodulin that regulate RyR2 channel openings.

In classic CPVT cases, release of catecholamines during exercise exacerbates sarcoplasmic reticulum dysfunction, beta adrenergic stimulation promotes calcium reuptake and increases RYR2 permeability to calcium. Moreover, the catecholamine-induced increase in heart rate further promotes myocyte calcium loading and the ensuing spontaneous calcium release triggers CPVT.

However, calcium handling abnormalities are not confined to ventricular myocytes but also extend to cells of the sinoatrial node. Sinus node dysfunction *independently* contributes to the emergence of ventricular arrhythmia during exercise via the following mechanism: prolongation of diastolic interval allows the occurrence of premature calcium release in diastole with sympathetic tone increasing automaticity and excitability of the ventricles.

Patients with CPVT typically present with syncope, presyncope usually triggered by exercise or emotional stress. A persistently low resting heart rate, particularly in younger individuals, should prompt consideration of CPVT in the differential diagnosis of unexplained syncope, especially if stress-induced symptoms are present.

The resting electrocardiogram (ECG) is often normal, a key diagnostic feature distinguishing CPVT from other arrhythmogenic syndromes like long QT Syndrome. An exercise stress test remains the cornerstone of CPVT diagnosis, revealing characteristic bidirectional or polymorphic VT during increasing heart rates.

Managing CPVT primarily focuses on preventing stress-induced ventricular arrhythmias, with beta-blockers being the cornerstone of therapy. Sodium channel blockers, especially flecainide, have an additive role in those not responding in beta blockade. For high-risk CPVT patients, especially those with recurrent arrhythmias on medication, cardiac sympathetic denervation should be considered.

While an implantable defibrillator is the most effective way to treat cardiac arrest, the discomfort of a shock can itself trigger an arrhythmic storm and hence this should be used only in patients refractory to the above measures.

Conclusion

- CPVT is not solely a ventricular disorder but involves broader cardiac electrical instability.
- The presence of sinus bradycardia can influence the clinical presentation, diagnosis, and particularly the therapeutic approach, necessitating a nuanced understanding of shared pathophysiological mechanisms.
- A comprehensive approach that integrates genetic insights, meticulous clinical evaluation and tailored therapeutic strategies is paramount to improving outcomes in this challenging and potentially lethal inherited arrhythmia syndrome.

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The Ischemic Cascade in ECG

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Introduction

Ischemic cascade was described in 1987 by Nesto RW and Kowalchuk GJ.[1] The ischemic cascade refers to sequence of events which occur when there is imbalance between the myocardial oxygen demand and supply. The term cascade refers to one event in the sequence leading to the next event and so on. The temporal sequence of events is not always uniform and varies from person to person and the various experiments have confirmed the same.[2] In this review article, we will discuss about the temporal sequence of events in ischemic cascade, metabolic alterations which occur, sequential ECG changes during acute myocardial infarction, studies which support and contradict the cascade and the clinical implications of the ischemic cascade.

Temporal sequence of events:

A transient reduction in myocardial blood flow due to increased myocardial demand (workload) across a fixed stenosis or increased stenosis with a fixed workload leads to sequence of events which is called ischemic cascade (Figure 1). Usually, it occurs in the following sequence: 1) metabolic alterations leading to increased lactate production, 2) diastolic dysfunction which has been demonstrated by echocardiography, angiocardiography or by nuclear echocardiography, 3) systolic dysfunction characterised by regional wall motion abnormality and later on global reduced systolic wall motion abnormalities and rarely fall in blood pressure, 4) electrocardiographic changes (ECG) such as ST segment depression and elevation (Figure 2) and finally 5) angina.[1]

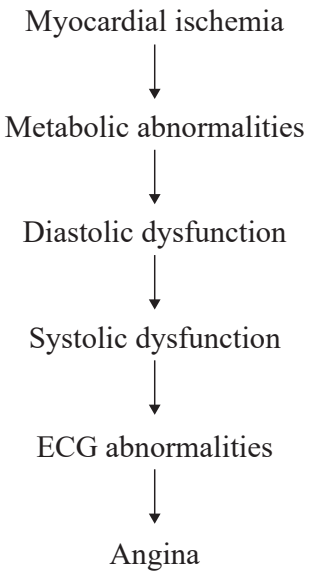

Figure 1: Ischemic Cascade

Metabolic alterations:

Myocyte metabolism changes from aerobic to anaerobic metabolism under ischemic conditions, leading to lactate accumulation and ATP depletion, impairing ion gradients, and cellular polarity.[3] At the individual cell level, a cascade is feasible, but not all cells will be in the same state of metabolism beyond a stenosis. The various metabolic changes which occur are 1) ATP Depletion and Ion Pump Failure: Impaired Na^+/K^+ ATPase function results in ionic imbalance, affecting membrane potentials and conduction, 2) Acidosis-Induced Conductivity Changes: Lactic acid accumulation reduces myocardial excitability, prolonging conduction time, 3) Gap Junction Dysfunction: Ischemia-induced uncoupling leads to slowed conduction velocity and arrhythmogenic potential, 4) Heterogeneous Repolarization: Ischemic and non-ischemic zones repolarize at differing rates, causing characteristic ST and T wave changes, 5) Severe Ischemia and Depolarization Delay: Extensive ischemia disturbs depolarization, manifesting as QRS changes on the ECG.

Sequential ECG Manifestations of myocardial ischemia:

The sequential ECG manifestations of myocardial ischemia are hyperacute T waves, ST segment elevation or depression, T wave inversion, pathological Q waves and finally resolution of ECG changes (Figure 3).

Hyperacute T waves: It is the harbinger of Ischemia. It is observed seconds to minutes post onset of ischemia. Ischemia rapidly depletes intracellular ATP, thereby impairing the function of sodium-potassium ATPase. This leads to extracellular potassium accumulation, abbreviating the action potential duration locally. The result is accelerated repolarisation within ischemia zones, reflected on ECG as hyperacute T waves. Hyperacute T waves are tall, broad, and symmetrical T waves, often exceeding normal amplitude in affected leads. Clinical implication: These waves are fleeting, appearing before overt ST-segment changes, making them critical early markers if recognised in time.

ST segment deviations: It is the electrophysiological signature of Ischemia. St segment elevation and depression can occur.

ST-Segment elevation (Transmural Ischemia): It is observed minutes after ischemia onset. Ischemic myocardium alters resting membrane potential and shortens action potential duration, creating injury currents between ischemic and healthy myocardium. These currents manifest as deviations in the ST segment baseline, proportional to the degree of transmural involvement. ECG Pattern: Convex or tombstone ST elevation, with reciprocal depression in opposite leads.

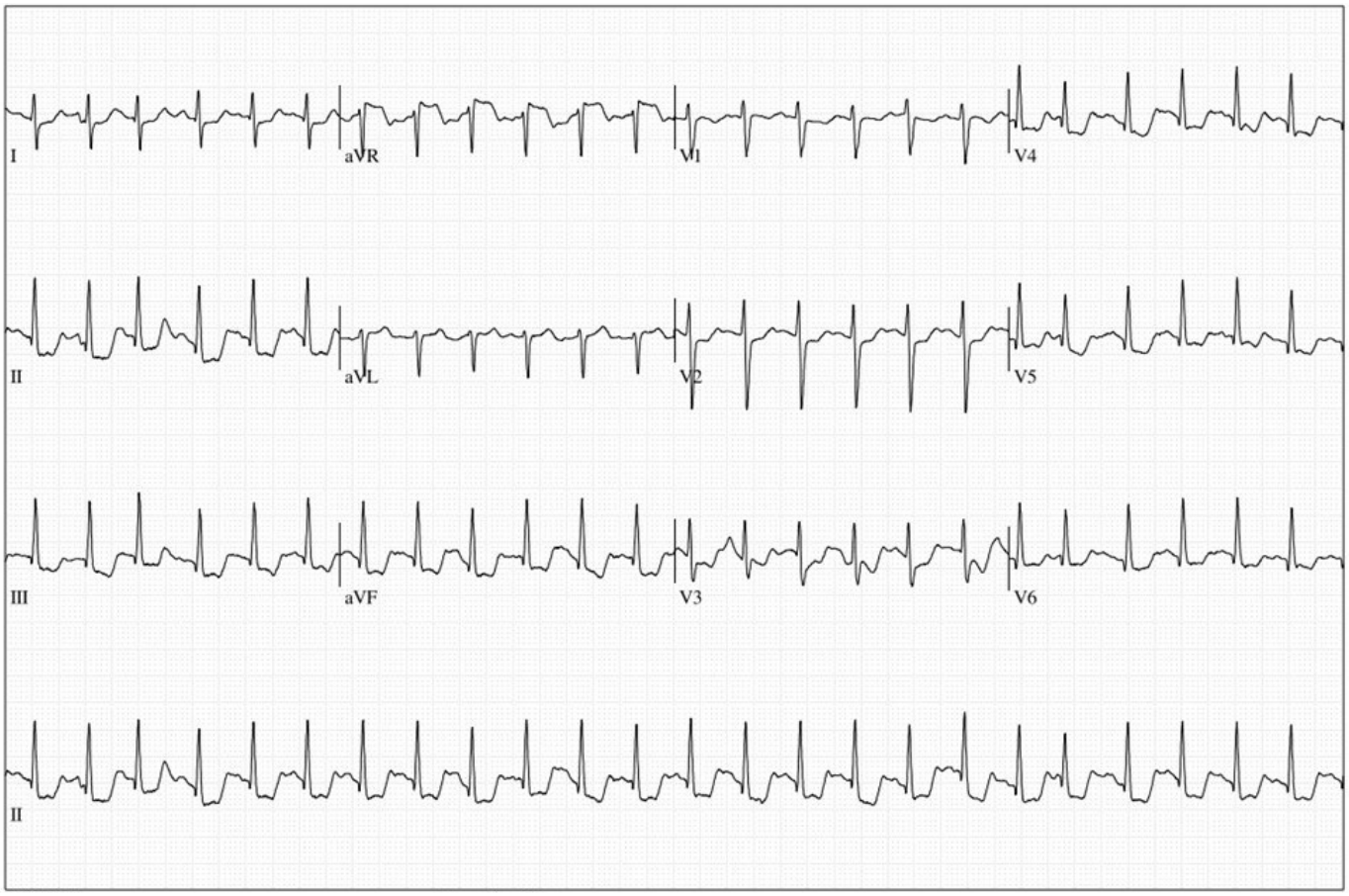

Figure 2: ST segment depression during exercise stress testing

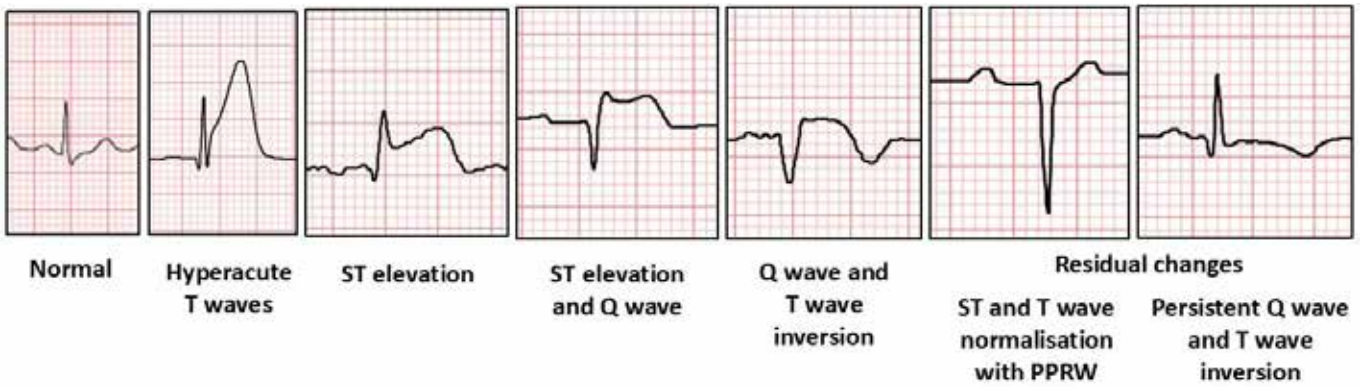

Figure 3: Sequential ECG changes in myocardial ischemia. [PPRW – poor progression of R wave]

ST Depression (Subendocardial ischemia): It is observed minutes after ischemia onset. ECG Pattern: Horizontal or down-sloping ST depression, sometimes with reciprocal posterior changes.

T wave inversions – Post Ischemic residue. It is observed hours to days following ischemia resolution or reperfusion. Even after reperfusion, repolarization heterogeneity persists due to residual myocardial stunning. The incomplete restoration of ionic gradients leads to T wave inversions, serving as a marker of recent ischemia or successful reperfusion. ECG Pattern: Deep, symmetrical T wave inversions in leads formerly showing ST elevation.

Pathological Q waves: Footprints of myocardial necrosis. It is observed several hours into sustained ischemia. Mechanism: Infarcted myocardium is electrically inert, causing opposing electrical vectors to predominate. This window effect results in the emergence of pathological Q waves. ECG Pattern: Q waves ≥ 40ms in width or ≥25% the height of the subsequent R wave, appearing in leads overlying the infarcted myocardium.

Resolution and residual changes: It is observed days to weeks after ischemic event. Mechanism: Myocardial recovery or fibrosis determines the persistence of ECG changes. ECG Pattern: ST normalisation, variable T wave persistence, permanent Q waves if infarction has occurred.

Birnbaum's Grading of Ischemia

Birnbaum et al proposed a three-grade system based on ECG morphology, providing a valuable prognostic framework.[3] Grade I ischemia – hyperacute T wave changes, Grade II ischemia – ST elevation without terminal QRS distortion and Grade III ischemia – ST elevation with distortion of terminal QRS complex. The Purkinje fibres are most resistant to ischemia than contracting myocytes. Hence, for an abnormality to occur in the terminal portion of the QRS, there should probably be a severe and prolonged ischemia that would affect the Purkinje fibres. The clinical implication of this grading is that patient with grade III ischemia have larger infarct size and thereby leading to poorer short term and long-term prognosis.

Variations in ischemic cascade

The cascade refers to a process where one event leads to another in a sequential order and can be envisioned to a waterfall where water falling to one level creates the potential for the next level down. If the events in a cascade do not occur in a sequential manner, the causal relationship or order of events cannot be secured. The original concept of ischemic cascade as proposed by Nesto RW and Kowalchuk GJ was based on experimental studies in humans. These studies induced myocardial supply - demand mismatch using one of the following manoeuvres: 1) coronary artery balloon occlusion during angioplasty, 2) atrial high-rate pacing, 3) exercise testing and 4) spontaneous angina.[1]

These studies did not show that anginal symptom and ECG changes consistently accompany ischemia. When the patients developed angina or ECG changes in these studies, the temporal sequence of events could not be verified. There are studies which contradict the temporal sequence of events in ischemic cascade. In the study by Levy et al, pulmonary artery diastolic pressure was measured along with ECG monitoring in patients with coronary artery disease during episodes of myocardial ischemia induced by treadmill exercise, atrial pacing, and unrestricted ambulant activity.[2] The measurement of pulmonary artery diastolic pressure during ischemia was a marker of diastolic dysfunction. 19 male patients were studied and there were 29 episodes of ST segment depression all of which were associated with increase in pulmonary artery diastolic pressure and angina. The ECG changes occurred before rise in pulmonary artery diastolic pressure in 11 episodes (38%) and simultaneous with the rise pulmonary artery diastolic pressure in 11 episodes (38%) and followed it in only 7 episodes (24%).

Gaspardone et al studied 21 patients with angina and isolated stenosis of left anterior descending artery.[2] Ischemia was induced by dipyridamole infusion and ECG, and echocardiography were continuously monitored. The temporal cascade was remarkably variable. 14 patients developed echocardiographic and ECG changes/angina. Of these 57% exhibited regional wall motion abnormalities first, while the remaining 43% exhibited ECG changes and or angina first.

Maznyczka A et al proposed the term ischemic constellation rather than ischemic cascade.[2] The ischemic constellation refers to collection of events in variable sequence which occur in ischemia. The clinical implications of this constellation model are relevant to the situation where in two diagnostic tests for ischemia show contradictory results. In a patient, one of the biological tests may have occurred at lower degrees of ischemia and the converse may be true in another patient. In such a scenario where in two tests show varying results in a patient, sophisticated approach is needed in determining the presence of myocardial ischemia. This constellation model further implies that a single gold standard test for ischaemia may not be possible.

The role of ECG in ischemic cascade:

ECG changes such as ST segment depression and elevation occur down the order in ischemic cascade after more subtle signs of ischemia, such as perfusion defects or functional deficits like diastolic dysfunction, and systolic dysfunction. Hence, this temporal sequence suggests that tests detecting ischemia at earlier stages of the cascade (e.g., myocardial perfusion imaging or stress echocardiography) may be more sensitive than a standard surface ECG for detecting ischemia.

Conclusion

The term ischemic cascade is a misnomer as the temporal sequence of events may vary from individual to individual. Hence, a single test for inducible ischemia may not be gold standard and an integrated approach is required when different tests show varying results. Tests which detect ischemia in the earlier stages of cascade such as perfusion imaging and stress echocardiography are more sensitive than the standard ECG based stress testing.

The ischemic cascade on ECG in acute myocardial infarction is predictable. It helps in early diagnosis and treatment of acute myocardial infarction and thereby aids in salvaging myocardium and improved outcomes.

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Understanding ECG Lead Misplacement

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Introduction

Electrocardiography (ECG) remains one of the most widely used, rapid, and non-invasive diagnostic tools in clinical practice, playing a central role in the evaluation of patients with suspected cardiac disease. Its utility spans from routine screening to the diagnosis of life-threatening arrhythmias, ischemia, infarction, conduction disturbances, and structural abnormalities. However, the accuracy of ECG interpretation critically depends on correct electrode placement. Misplacement of leads is not uncommon in real-world clinical settings, especially in emergency rooms (ER), intensive care units (ICUs), and general wards where ECGs are performed frequently under time pressure.

The reported incidence of ECG lead misplacement ranges between 0.4% and 4% globally, though the true prevalence may be underestimated due to recurrent under recognition.[1,2] Moreover, some studies suggest that over half of ECGs may contain some form of lead mispositioning, especially involving precordial electrodes applied by nursing staff, sometimes mispositioned in up to 64% of cases.[3]

Errors are more often committed by inexperienced physicians, nursing staff, or technicians unfamiliar with the characteristic patterns of misplacement. The most common error involves reversal of the right arm (RA) and left arm (LA) leads accounting for approximately 20% of all electrode misplacements,[4] but various combinations involving limb and chest leads have been documented. Importantly, these errors may produce electrocardiographic changes that closely mimic pathological conditions such as myocardial infarction, ischemia, chamber enlargement, conduction abnormalities, ectopic rhythms, or even technical failures of the ECG machine.[2,3,5]

Misinterpretation due to lead misplacement can have serious clinical consequences. Patients may be subjected to unnecessary investigations, thrombolysis, or even invasive procedures such as coronary angiography, with associated risks and costs. On the contrary, genuine cardiac pathology may be overlooked if abnormalities are attributed to technical error. Therefore, awareness of the characteristic ECG patterns produced by different types of lead misplacements is essential for clinicians.

The standard 12-lead ECG consists of six limb leads (I, II, III, aVR, aVL, aVF) and six precordial (chest) leads (V1–V6). While chest leads are less commonly interchanged due to their anatomical positioning, they may still be misplaced, particularly when applied in a hurry, leading to disruption of the expected R-wave progression across the precordium. Limb lead reversals, on the other hand, are more frequent because of the similarity of the electrodes and accidental swapping during placement. Once a pair of bipolar limb leads is misplaced, the augmented unipolar leads also change automatically, producing distinctive patterns that, if recognized, can help clinicians promptly identify the error.

This article aims to: (1) highlight the clinical implications of ECG lead misplacement through real case scenarios, (2) describe possible combinations of misplacements, and (3) provide practical recognition patterns to aid physicians and paramedical staff in distinguishing true pathology from artefactual changes. Early recognition of such errors is vital to ensure accurate diagnosis, prevent mismanagement, and improve patient safety.

Principles of Normal ECG

Electrocardiography (ECG) records the electrical activity of the heart as it propagates through specialized conduction tissue and myocardium, projecting this activity onto different axes through surface electrodes. Understanding the principles of normal ECG requires a clear grasp of the electrode placement, lead systems, and the normal waveforms they generate.

Bipolar Limb Leads (Einthoven's Triangle)

Willem Einthoven conceptualized the bipolar limb leads (I, II, III) as an equilateral triangle with the heart at its center. These leads record the potential difference between two limb electrodes:

- Lead I: RA → LA (0°)
- Lead II: RA → LL (60°)
- Lead III: LA → LL (120°)

This system provides information on the cardiac axis in the frontal plane.

Augmented Unipolar Limb Leads

Introduced by Goldberger in 1942, augmented leads (aVR, aVL, aVF) are unipolar and record the electrical potential of one limb relative to a central terminal (formed by averaging inputs from the other two limbs via Wilson's central terminal).

- aVR: Directed from the heart toward the right arm (–150°)
- aVL: Directed toward the left arm (–30°)
- aVF: Directed toward the left leg (+90°)

When superimposed on Einthoven's system, they form the hexaxial reference system, dividing the frontal plane into 30° increments, crucial for determining electrical axis and localizing pathology.

Chest (Precordial) Leads

Proposed by Wilson, chest leads (V1–V6) are unipolar and positioned across the anterior thorax to record electrical activity in the horizontal plane, closer to the myocardium than limb leads. Standard positions are:

- V1: 4th intercostal space, right sternal border
- V2: 4th intercostal space, left sternal border
- V3: Midway between V2 and V4
- V4: 5th intercostal space, midclavicular line
- V5: Level with V4, anterior axillary line
- V6: Level with V4, midaxillary line

A normal ECG demonstrates progressive R-wave progression from V1 (predominantly negative) to V6 (predominantly positive), reflecting ventricular depolarization.

Standard ECG Configuration

- A 12-lead ECG comprises:
- 6 limb leads (I, II, III, aVR, aVL, aVF) → frontal plane view
- 6 chest leads (V1–V6) → horizontal plane view

Together, they provide a three-dimensional electrical mapping of the heart.

Normal Waveforms and Intervals

- P wave: Atrial depolarization (upright in I, II, aVF; inverted in aVR).
- PR interval: AV nodal conduction (0.12–0.20 s).
- QRS complex: Ventricular depolarization (<0.12 s); normal axis between –30° and +90°.
- ST segment: Normally isoelectric; deviation suggests ischemia or infarction.
- T wave: Ventricular repolarization (upright in most leads, inverted in aVR).
- QT interval: Total ventricular depolarization and repolarization (rate-corrected QTc ≤440 ms in men, ≤460 ms in women).

Patterns of Lead Misplacement

Lead misplacement produces characteristic and reproducible changes on the electrocardiogram, which can be broadly grouped into misplacements involving limb leads and chest leads. Within these categories, specific reversal patterns generate distinct abnormalities that, when recognized, allow the clinician to differentiate artefact from true pathology.

Limb leads without neutral lead involvement:

This is the most common type of error and occurs when two active limb electrodes are interchanged, while the neutral electrode (right leg, RL) remains unaffected.

- **RA–LA reversal**: The right arm (RA) and left arm (LA) electrodes are swapped. This leads to inversion of Lead I and aVL, with an upright aVR. Clinically, it may mimic dextrocardia or lateral wall ischemia.
- **LA–LL reversal**: Exchanging the left arm (LA) and left leg (LL) electrodes results in an inverted Lead III with otherwise minimal changes, which may be mistaken for inferior wall ischemia.
- **RA–LL reversal**: When the right arm (RA) and left leg (LL) electrodes are interchanged, Leads I, II, III, and aVF all appear inverted, while aVR becomes positive. This can strikingly mimic an acute inferior myocardial infarction and is one of the most clinically misleading errors.

Limb leads with neutral lead involvement

In this group, the neutral electrode (RL) is interchanged with one of the active limb electrodes, leading to either flatline recordings or polarity changes.

- **RA–RL reversal**: The right arm electrode is placed on the right leg, and vice versa. Lead II shows a flatline due to identical potentials at both ends, while Leads I and aVL are inverted and aVR becomes positive.
- **LA–RL reversal**: The left arm electrode is placed on the right leg, producing a flatline in Lead III with a positive aVR, which may falsely suggest conduction abnormality.
- **LL–RL reversal**: Here, the left leg electrode is placed on the right leg. Because both lower limb electrodes generate nearly identical signals, there is no significant change, making this error harder to detect.

Misplacement of all four limb leads

This less frequent but more complex error involves simultaneous misplacement of all four limb electrodes, leading to marked distortions.

- **Bilateral arm–leg reversal**: When both arm and leg electrodes are exchanged (RA ↔ RL and LA ↔ LL), Lead I becomes flat, while Leads II and III show inversion. This can mimic technical malfunction if unrecognized.
- **Clockwise rotation of all four leads**: A systematic rotation of all electrodes produces flatline in Lead III with altered polarity elsewhere, but AVR remains unchanged.

- **Counterclockwise rotation of all four leads**: This produces inversions in Leads I, II, and aVL, with a positive aVR. Such a pattern can closely resemble lateral or inferior ischemia.

Chest lead misplacement

Precordial leads are particularly prone to error because of their close spacing and similarity in appearance.

- **Adjacent lead swaps (e.g., V1–V2, V3–V4, V5–V6)** disrupt the normal R-wave progression, producing apparent loss of transition or unexpected QRS morphologies. This can be misinterpreted as anterior or lateral ischemia.
- **Misplacement in incorrect intercostal spaces** (such as placing V1 and V2 in the second instead of the fourth intercostal space) can produce pseudo–right bundle branch block, septal Q waves, or ST-segment elevation, occasionally mimicking Brugada syndrome or acute myocardial infarction.

Summary

Electrocardiographic lead misplacement, though often under recognized, follows reproducible patterns that can be systematically identified. In total, nine key ECG patterns account for the majority of clinically relevant misplacements, including simple limb lead reversals, neutral lead exchanges, complex four-limb rotations, and precordial lead misplacements. Each produces distinctive features such as flat line tracings, global waveform inversion, or disrupted R-wave progression, which can closely mimic acute coronary syndromes, conduction disturbances, or chamber abnormalities.[6-8] Accurate recognition of these patterns is not merely academic it has direct implications for patient safety. Misinterpretation of pseudo-infarct patterns may result in unnecessary admissions, invasive coronary testing, or inappropriate fibrinolytic therapy, while true pathology may be overlooked if abnormalities are dismissed as technical error.[9,10] Training physicians, nurses, and technicians to identify these hallmark ECG misplacement patterns significantly reduces the risk of diagnostic error and improves clinical decision-making.[11]

Conclusion

ECG lead misplacement is both common and clinically significant, with reported incidence ranging from 0.4% to 4% of all ECGs performed.[6,12] Despite its frequency, it remains underdiagnosed, particularly by less experienced clinicians or in high-pressure environments such as emergency departments and intensive care units.8 Awareness of the characteristic ECG signatures associated with different types of misplacements, combined with a systematic approach to interpretation, is essential for safe practice. Prompt recognition not only prevents misdiagnosis but also spares patients from unnecessary and potentially harmful interventions, such as thrombolysis or invasive angiography.[13] Therefore, ensuring proper electrode placement and cultivating vigilance in detecting misplacements represent vital competencies in cardiology training and practice.[14]

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ECG in Hypertensive Heart Disease

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Introduction

In Clinical practice, the electrocardiogram (ECG) remains the cornerstone for the diagnosis of left ventricular hypertrophy (LVH) as it is easy to perform, widely available and quiet specific. LVH in hypertension is associated with increased cardiovascular morbidity and mortality. ECG in hypertension serves as an established prognostic tool and regression of LVH on ECG showing better outcomes prompt clinicians to actively look for the ECG-LVH and treat the patients effectively.

Hypertension is not only globally the most common non-communicable disease, but also the major contributor to cardiovascular events. In US, the disease burden is estimated to be as high as 30% in general population[1]. It is a major risk factor for heart failure (HF) and precedes in almost 3/4th of the patients. HF with preserved ejection fraction (HFpEF), hypertension is present in almost 60 to 89% of the patients, representing the main causative factor[2] and mortality rate secondary to mild diastolic impairment was 10% in a 5-year period compared to 25% in moderate to severe diastolic dysfunction (grade II and III)[3].

Hypertensive Heart Disease may present in following forms either singly or in combination :

a. Diastolic dysfunction

b. Left ventricular hypertrophy (LVH)

c. Systolic overload

d. Arrhythmias

e. Heart failure with reduced ejection fraction (HFrEF) or with preserved ejection fraction

Increased left ventricular mass (LVM), an adaptive response, a part of LVH is strong marker for cardiovascular disease (CVD) morbidity, including HF and mortality[2]. LVH happens as an adaptive response to increased haemodynamic load in hypertension, and is independently associated with increased risk of myocardial infarction, sudden cardiac death, HF and stroke in the general population.

LVH has been recognized as a powerful predictor of serious cardiovascular events, detected either by ECG or by echocardiography[4]. Haemodynamic factors such as BP, large artery structure and stiffness, and volume load are the important determinants for the development of LVH. Apart from these, there are non-haemodynamic mechanisms such as the influence of sympathetic nervous system, the renin-angiotensin-aldosterone system, and other neurohormonal mediators who play an important role in the development of LVH. Concentric LVH, a marker of severe pressure overload, is associated with worse outcome as compared to eccentric LVH or concentric remodelong[5].

Regression of LVH is beneficial beyond BP reduction and has been shown that CV events occur more commonly in patients in whom the LVH progresses than in those where it regresses[6]. In SPRINT (Systolic Blood Pressure Intervention Trial), having 8164 individuals with hypertension without diabetes mellitus, showed that intensive control of BP had lower risk of developing ECG-LVH by 46% and the same group were more likely to show regression of ECG-LVH[7].

Left Ventricular Hypertrophy : The ECG Criteria

Electrocardiogram (ECG), though a century old tool, widely available and very cost effective and may be the first cardiac investigation of choice, is of limited use to diagnose and risk stratify the asymptomatic hypertensive patients. Sensitivity for diagnosing LVH on standard ECG is grossly poor. Specificity for diagnosing LVH is good. In hypertensive heart disease, myocardium is hypertrophied and the electrical activation has to pass through the larger myocardial mass, thereby the amplitude of QRS complex representing ventricular depolarization, is increased and is also associated with QRS widening and increased intrinsicoid deflection or ventricular activation time. However, there is no correlation between QRS duration and left ventricular mass index (LVMI). And the repolarizing abnormalities in the same scenario represents as ST-T wave changes. Rodrigues et al reported ECG strain in hypertension is a marker of advanced LVH with increased interstitial fibrosis[8].

Though multiple criteria have been suggested in the literature to diagnose LVH on ECG, most commonly used criteria have been listed below :

Cornell Criteria

Voltage of R wave in aVL added to the voltage of the S wave in V_3 and if the sum is > 28 mm in males and > 20 mm in females, LVH is present.

Modified Cornell Criteria

If voltage of R wave in aVL is > 12 mm, LVH is present.

Cornell Voltage Duration Product

$(RaVL + SV_3) \times$ QRS duration = 2436 mm × ms

Sokolow-Lyon Criteria

Voltage of S wave in V_1 added to the voltage of R wave in V_5 or V_6 and if the sum is > 35 mm, LVH is present.

Romhilt-Estes LVH Point Scoring System

a. Amplitude of largest R or S wave in limb leads ≥ 20 mm = 3 points

b. Amplitude of S wave in V_1 or V_2 ≥ 30 mm = 3 points

c. Amplitude of R wave in V_5 or V_6 ≥ 30 mm = 3 points

d. ST and T wave changes opposite to QRS direction without digoxin = 3 points

e. ST and T wave changes opposite to QRS direction with digoxin = 1 point

f. Left atrial enlargement = 3 points

g. Left axis deviation = 2 points

h. QRS duration ≥ 90 msec = 1 point

i. Ventricular Activation Time (VAT) in V_5 or V_6 > 50 msec = 1 point

If score is 4, LVH is present (Sensitivity 30-54%) and if the score is ≥ 5, LVH is present with 83-97% specificity.

Various criteria have different sensitivity and specificity.

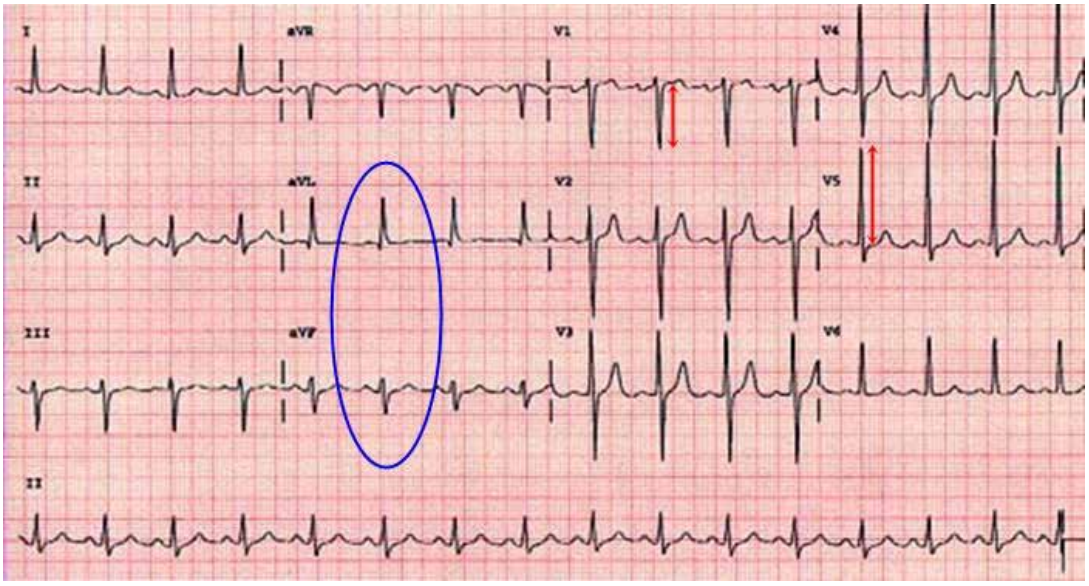

Figure 1: ECG-LVH by voltage criteria

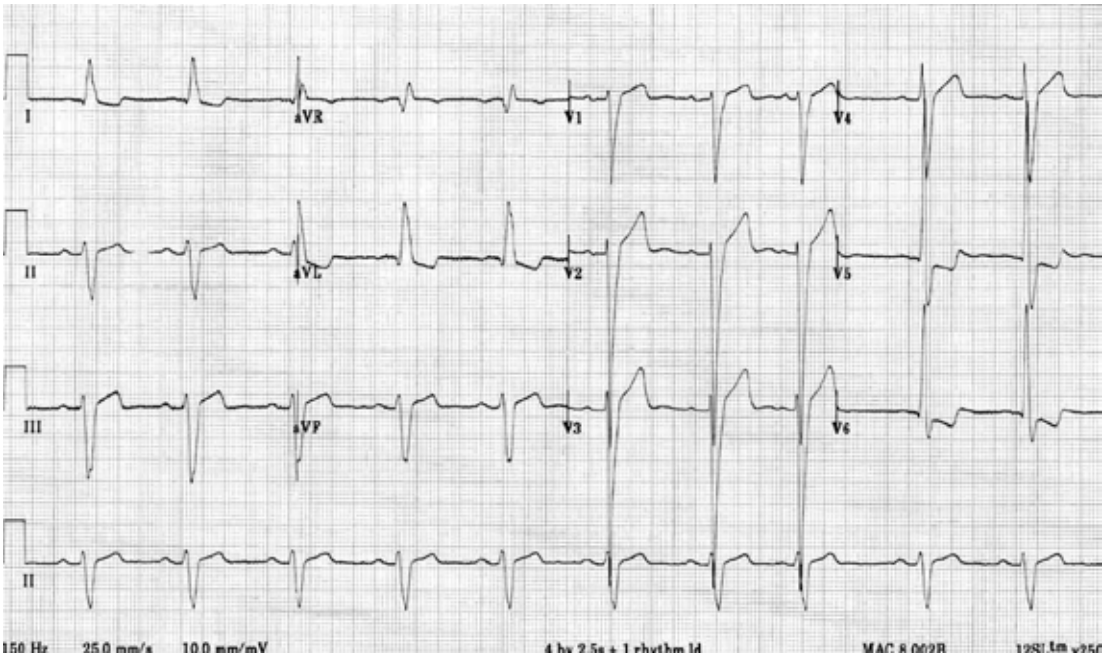

Figure 2: ECG-LVH showing ↑voltage + ST-T changes + ↑ QRS + ↑ VAT + LAD

Cornell voltage duration product as compared to Sokolow-Lyon criterion has shown to be more sensitive (51 vs 31%) maintaining same specificity. Still, Sokolow-Lyon criterion is the most commonly used criteria because of its simplicity.

ECG features of LV diastolic dysfunction

Ventricular activation time, P-wave terminal force in V_1 and P-wave dispersion on the ECG have been implicated as a reflection of LV diastolic dysfunction.

VAT

Ventricular Activation Time or intrinsicoid deflection is measured from the onset of QRS complex to the peak of R-wave and VAT of > 0.05 sec in V5 or V6 has been taken a scoring point in Romhilt-Estes Criteria for diagnosing LVH. VAT was prolonged in subjects with diastolic dysfunction (46.3±0.4 vs. 39.6±0.3 ms; $p<0.01$) in the study correlating high resolution ECG and echocardiographic assessment equipped with tissue Doppler imaging (TDI) capabilities[9].

P-wave terminal force in V_1 ($PTFV_1$)

$PTFV_1$ is being calculated as the product of the amplitude of terminal negative component of the P-wave in V_1 and its duration and $PTFV_1$ has emerged as novel ECG marker with a strong prognostic value for CV events. $PTFV_1$ of ≥ 40 mm/ms is considered positive and was a predictor of cardiac death or hospitalizations for heart failure. $PTFV_1$ has superior diagnostic value in assessing LV end-diastolic failure (LVEDP) and hence diastolic function than P-wave duration only[11]. Sensitivity and specificity for echo parameters for diastolic dysfunction and $PTFV_1$ of ≥ 40 mm/ms were 62% and 75% respectively and were statistically significant[11].

P-wave Dispersion (PWD)

PWD is defined as the difference in msecs between the longest and shortest P-wave duration on the 12 lead ECG. And this happens because of scar accumulation in the atria in hypertensive patients leading to heterogeneous propagation of electrical activity through the atria[12] and a significant correlation was observed with echo diastolic parameters such as TDI and transmitral Doppler and PWD ($p>0.002$)[11].

Tp-Te interval

Tp-Te interval (time interval between the peak and the end of T wave) is an index of transmural dispersion of repolarization and an emerging new marker for ventricular arrhythmogenesis and repolarization heterogeneity[13]. Karagaac and coworkers reported in their 70 newly diagnosed hypertensive patients that Tp-Te (91±12 ms 74±10 ms), Tp-Te/QT (0.24±0.02 vs 0.20±0.02), and Tp-Te/QTc (0.22±0.02 vs 0.18±0.02) were significantly higher in the nondipper than in the dipper group, respectively[14]. Ferrucci and coworkers described an association of prolonged Tp-Te interval and systemic hypertension, the index significantly higher (2.9±0.5 mm) in the hypertensive group than in the normotensive group (2.2±0.3 mm) and the interval emerged as the only independent correlate of hypertension, whereas BMI, LVMI, and Em/Am ratio failed to maintain a significant association[15]. Review on the study commented that as compared to the several conventional ECG parameters like, Sokolow-Lyon index, Cornell voltage index,

Cornell Voltage product index, P-wave dispersion, P-wave area and VAT, Tp-Te interval is a more sensitive biomarker of pressure overload and of early myocardial changes related to hypertension[13].

Combining ECG Criteria for diagnosis of LVH

Okin PM et al published an article, stating that combining ECG criteria for LVH improves risk prediction in patients with hypertension[16]. In 9193 patients with hypertension studied for a mean period of 4.8±0.9 years follow-up, ECG-LVH was diagnosed by Cornell product (CP) and/or Sokolow-Lyon (SL) Voltage criteria. Patients were categorized into 4 groups at baseline – No LVH, LVH by CP alone, LVH by SL alone and LVH by both CP and SL. Patients were followed up yearly during the study for the persistence or development of ECG-LVH. 960 patients (10.4%) had LVH by both criteria at baseline. Patients having LVH by both criteria has >3 fold increased risk of the events (Incident stroke, myocardial infarction, cardiovascular death, the composite of these outcomes, and all-cause mortality) as compared to patients with no LVH. Patients with LVH by any single criteria had the intermediate risk (45% to 140%).

Prognostic value of the ECG

LVH diagnosed on ECG is an ominous prognostic sign that predicts high rate of CV events. In the Framingham Heart Study, published more than 35 years ago, in middle aged persons with ECG-LVH as compared normal adults of similar age, the risk of fatal and non-fatal CV morbid events were 3-fold to 8-fold higher[17, 18]. Recently, the Heart Outcomes Prevention Evaluation (HOPE) study, having ECG-LVH defined solely by Sokolow-Lyon criterion, was an independent predictor of all cause deaths, CV deaths and heart failure[19]. In the PIUMA study, for predicting fatal CV morbid events among hypertensive subjects, the Sokolow-Lyon criterion yielded lowest hazard ratio (HR 1.19) in contrast to Cornell voltage (HR 1.34) and Romhilt-Estes score HR (2.63)[20]. In the LIFE study, a significant increase in both CV mortality and morbidity was observed with every 5 mm increase in the Sokolow-Lyon voltage[21]. Hsieh et al compared 17 different ECG methods of identifying LVH and demonstrated that a composite of ECG criteria (Framingham, Perugia and Romhilt-Estes point scores) was more strongly predictive of CV mortality as compared to voltage-only LVH[22].

The presence of LV strain pattern in addition to voltage criteria yielded an HR of 3.9 in a longitudinal study of 19434 male patients with a mean follow up of 7 ± 4 years[22]. In Framingham Heart Study also, LVH with strain was the most important predictor of future CV events and the same was demonstrated in LIFE study too that LVH with strain was a

significant predictor of CV death, non-fatal MI or stroke and were at increased risk for developing chronic heart failure.

In the LIFE study, increased QRS duration have been shown as an independent predictor of both CV and all-cause mortality[23]. In the PIUMA study, prolonged QTc was associated with a 2-fold increased risk of coronary events and CV deaths[24].

Presence of ECG-LVH in women put them at a higher risk of dying from CV causes as compared to men.

ECG-LVH , ECHO-LVH and CMR-LVH

ECHO is more sensitive than ECG in diagnosing LVH and may help in more precise stratification. However, this modality is more time-consuming and requires considerable skill to perform. Further, enormous cost implications, considering the vast number of hypertensive patients, ECHO for assessment of LVH is not being advocated routinely[25].

In the second Strong Heart Study, the presence of both echo LVH and ECG ST depression was associated with 6.3-fold increased CV death[26]. Cuspidi and co-workers, in the PAMELA study, also showed that the association of ECG LVH and ECHO LVH significantly increased the risk of CV mortality[27].

Cardiac Magnetic Resonance (CMR) is now the criterion standard for LVM Measurement. Multi-Ethnic Study of Atherosclerosis (MESA), having 4748 participants, described ECG-LVH and CMR-LVH in 6.7% and 10.5% of the participants respectively. ECG-LVH alone was predictive of the events, but ECG-LVH and CMR-LVH had the strongest link and both taken together were predictive of HF[28].

As on date, evaluation of LVH by routine imaging is not recommended as there is very limited data regarding the cost-effectiveness for risk stratification and management[2]. In fact routine echocardiogram in hypertension received a score of 3 on the Appropriate Use Criteria from 2011, considering it inappropriate. However, in young patients with secondary hypertension, uncontrolled hypertension or HF symptoms, imaging can be helpful[2].

Conclusion

ECG is an important tool for detection of LVH in hypertensive patients as its prognostic value seems to surpass its limited ability to detect LVH. For diagnosing LVH in patients with hypertension, ECG remains the first choice because it is widely available, easy to perform, specific, inexpensive, reproducible, and of established prognostic value. Serial ECGs showing regression of LVH have shown better cardiovascular outcomes. Combing ECG criteria can increase the sensitivity of ECG in detecting LVH and prognosticating the individual. Newer markers can help in risk stratification. In selected individuals, ECHO and CMR can work as complimentary modality in detection of LVH and better prognostication.

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Short QT Syndrome: Disturbance of the Heart's Electrical System

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Introduction

In recent years, numerous underlying cardiac pathologies have been recognized, including structural abnormalities like hypertrophic cardiomyopathy and non-structural electrical disorders that cause unstable heart rhythms, potentially resulting in sudden cardiac death.[1]

Short QT Syndrome (SQTS) is a rare, inheritable primary electrical disorder of the heart, characterized by abnormally short QT intervals on the electrocardiogram (ECG) and an increased risk of developing both atrial and ventricular tachyarrhythmias specifically in the absence of structural heart disease which can lead to syncope or sudden cardiac death.[2-4]

It is a relatively recent addition to the group of inherited channelopathies implicated in sudden cardiac death (SCD) among individuals with structurally normal hearts.

Definition and Diagnosis

This inherited cardiac channelopathy is typically characterized by a QTc interval of less than 340 ms in adults and less than 320 ms in children, or less than 360 ms measured via electrocardiogram when accompanied by one or more of the following criteria: [5-7] (Table 1)

- a history of cardiac arrest, syncope
- a family history of Short QT Syndrome
- sudden cardiac death (SCD) before the age of 40

Since its initial recognition in 1999, substantial advances have been made in understanding the clinical presentation, genetic and ionic underpinnings of the disease, as well as in developing therapeutic strategies.[8] Other ECG criteria include high familial penetration, autosomal dominance.

Table 1: QTC Formula Guide

Formula	Equation	Advantage
Bazett	QTc = QT / √RR	Widely used, accurate at HR 60–100 bpm
Fridericia	QTc = QT / RR^(1/3)	Better at extreme heart rates
Framingham	QTc = QT + 0.154 × (1 – RR) ms	Linear correction; robust at extremes
Hodges	QTc = QT + 1.75 × (HR – 60) ms	Another linear approach

By definition, a diagnosis of SQTS should only be considered after excluding secondary causes of a shortened QT interval, such as hyperkalemia, acidosis, hypercalcemia, hyperthermia, the effects of drugs like digitalis, the influence of acetylcholine or catecholamines, and QT shortening due to activation of the K_{ATP} current. A rare yet intriguing paradoxical ECG phenomenon known as deceleration-dependent QT interval shortening—where the QT interval shortens as the heart rate decreases—should also be considered in the differential diagnosis.[9,10]

Genetic counseling is essential in Short QT Syndrome (SQTS) to help families understand the disorder, its mode of inheritance, and associated risks. Genetic testing can also confirm diagnosis, as there are three genes encoding potassium channels where Gain-of-function mutations that have definitive pathogenic variants: KCNQ1, KCNH2, and KCNJ2.[5] For loss-of-function mutations the genes responsible are CACNA1C, CACNB2, CACNA2D1 (Ca2+ channels).[11]

Measuring the QT interval:[12]

- Identify QT interval: Measure from the start of the QRS complex to the end of the T wave (return to baseline), ideally in lead II or V5/V6.
- Use ECG scale: On 25 mm/s paper, 1 small box = 40 ms; manually count QT duration in milliseconds.
- Correct for heart rate: Use Bazett's formula: QTc = QT / √RR (RR in seconds)
- QT; normal QTc < 440 ms (men), < 460 ms (women).
- Clinical significance: QTc > 500 ms increases risk for Torsades de Pointes; confirm abnormal values manually, even if machine-calculated. (Figure 1)

The guidelines from the Joint Steering Committees of UCARE and IVF-US[13] recommend a comprehensive battery of tests to exclude organic heart disease. These include, but are not limited to, resting ECG, exercise stress testing, echocardiography, 24-hour Holter monitoring, and cardiac MRI. A diagnosis of Short QT Syndrome (SQTS) should be strongly considered in young individuals presenting with a shortened QT interval on a 12-lead ECG, particularly when accompanied by arrhythmic symptoms, isolated atrial fibrillation (AF), primary or resuscitated ventricular fibrillation (VF), or a significant family history of arrhythmic events, including SCD. Even in the absence of arrhythmogenic complications, the detection of a short QT interval on ECG necessitates further evaluation to exclude SQTS (Figure 2).

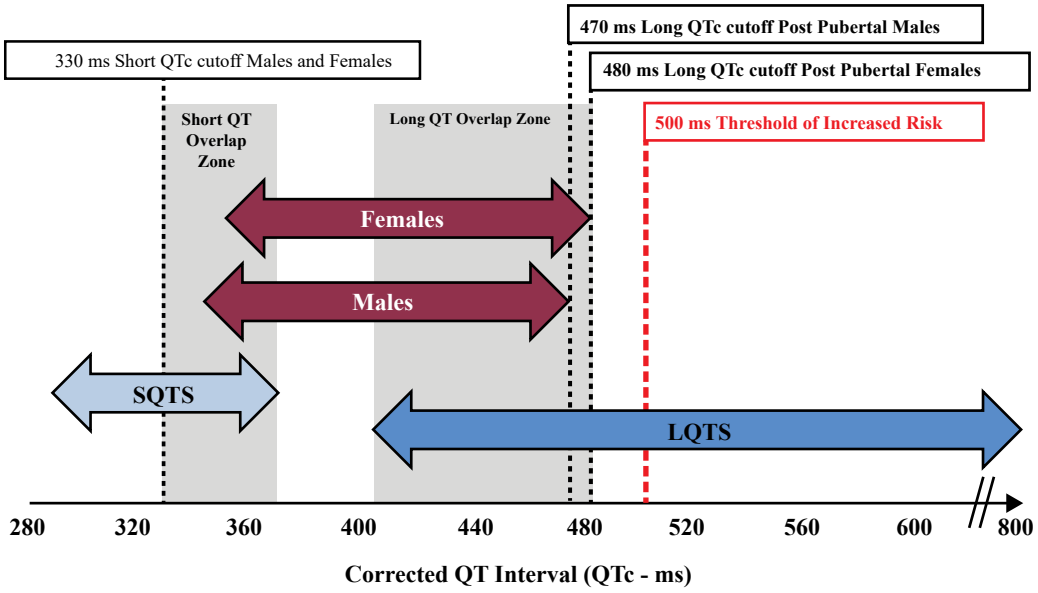

Figure 1: A range of QT interval covering SQTS and LQTS

Criterion	Points
QTc, ms	
<370	1
<350	2
<330	3
Jpoint - Tpeak interval < 120 ms	1
Clinical history	
History of sudden cardiac arrest	2
Documented polymorphic VT or VF	2
Unexplained syncope	1
Atrial fibrillation	1
Family history	
First- or second- degree relative with high-probability SQTS	2
First- or second- degree relative with autopsy-negative sudden cardiac death	1
Suffrn infant death syndrome	1
Genotype	
Genotype positive	2
Mutation of undetermined significance in a culprit gene	1

QTc: corrected QT; SQTS: short QT syndrome; VF: vetricular fibrillation; VT: ventricular tachycardia. High-probability SQTS: 4 points; intermediate-probability SQTS: 3 points; low-probability SQTS: 2 points.
Adapted from Gollob et al.[1]

Figure 2: Proposed Diagnostic criteria for SQTS

When SQTS is suspected, a resting 12-lead ECG should be obtained while the heart rate is within normal limits. The QT interval should ideally be measured at a heart rate below 100 bpm, and preferably under 80 bpm. In individuals with SQTS, the QT-RR relationship typically shows minimal rate dependence, displaying a flatter slope than normal.

It is crucial to recognize that a short QTc interval alone should not be used as the primary or exclusive criterion for diagnosing SQTS. A thorough evaluation of ECG characteristics (Figure 3), along with detailed clinical and family history, is essential for an accurate differential diagnosis.

The Pathophysiology of SQTS:

The myocardium consists of three distinct layers: the epicardium, the endocardium, and a middle layer composed primarily of M cells. These cells vary in their ion channel composition across the layers, leading to differences in electrophysiological properties. In certain syndromes, these variations can cause the layers to repolarize at different rates, resulting in heterogeneous **refractoriness**. This disparity in repolarization can create a substrate for **reentry circuits**, which are a common mechanism underlying arrhythmias.

In short QT syndrome, it is believed that endocardial and M cells repolarize more rapidly than epicardial cells, creating a substrate for reentry and arrhythmias. This enhanced **transmural dispersion of repolarization** contributes to arrhythmogenesis not only in short QT syndrome but also in long QT and Brugada syndromes. The key difference among these conditions lies in the myocardial region most affected by the underlying channelopathy—M cells in long QT syndrome, the right ventricular epicardium in Brugada syndrome, and a combination of endocardial and M cells in short QT syndrome.[14]

SQTS is associated with a range of inherited mutations that alter the function of ion channels responsible for regulating cardiac action potentials.[6] SQTS has been associated with both gain-of-function mutations in potassium channel genes and loss-of-function mutations in calcium channel genes[7]

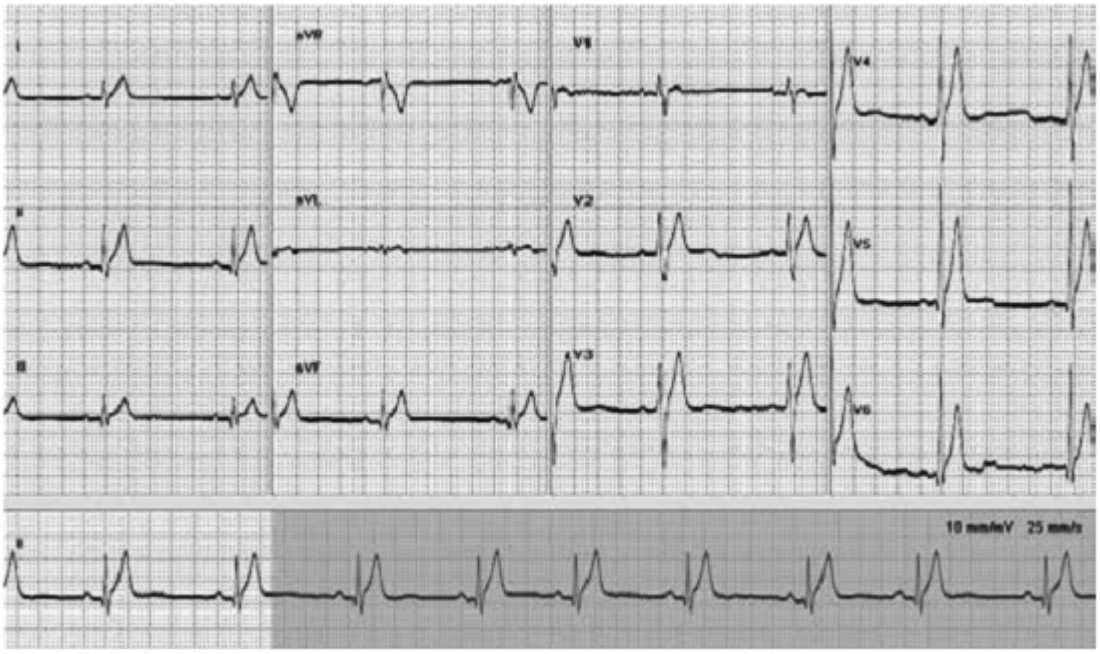

Figure 3: ECG with short QT interval (QTc=300 ms) and tall peaked T waves

Case study description

A 28-year-old male with a known history of **SQTS**, previously complicated by **cardiac arrest** and **ventricular fibrillation**, status post **Boston Scientific subcutaneous implantable cardioverter-defibrillator (ICD) placement and cardiac sympathectomy**, presented with complaints of **increased palpitations** and **recent ICD shocks**.

Prior to admission, the patient reported experiencing three episodes of ICD discharges since the initiation of atenolol. He has been compliant with quinidine therapy without missing any doses and had previously only experienced ICD firing when doses were missed. The patient attributes the recent increase in ICD discharges to the addition of atenolol.

On physical examination, the patient reported only mild chest pain without any other associated symptoms. **ECG** showed sinus bradycardia with a QT/QTc interval of 422/407 ms. **Chest X-ray** revealed no evidence of acute cardiopulmonary disease. **Laboratory tests** including troponin, complete blood count (CBC), comprehensive metabolic panel (CMP), magnesium, phosphorus, and thyroid-stimulating hormone (TSH) were all within normal limits. **Atenolol was discontinued**, and the patient was admitted for observation.

Discussion

Sudden cardiac death (SCD) is a leading cause of mortality in the developing world, with **ventricular arrhythmias** recognized as a primary underlying mechanism. While these arrhythmias most commonly arise in the context of **structural heart disease**, they can also result from **acute events** or **inherited abnormalities** that predispose cardiac tissue to life-threatening electrical disturbances.

The first-line treatment is an ICD; however, several reports have documented inappropriate shocks triggered by sinus tachycardia, atrial fibrillation, and oversensing of tall, narrow T waves.[6] Pharmacological options include Quinidine or hydroxyquinidine, where device is not advised. These drugs effectively prolong the QT interval and ventricular refractory periods, making it a viable alternative or adjunctive pharmacological therapy to ICD placement.[6]

The initiation of Atenolol in this patient with short QT syndrome (SQTS) and a history of cardiac arrest and ventricular fibrillation was associated with increased ICD discharges and QTc prolongation. This raises the possibility of a proarrhythmic effect of Atenolol in individuals with SQTS. While beta-blockers are routinely used to manage various cardiac arrhythmias, including tachyarrhythmias, their use in SQTS—marked by a shortened QT interval and heightened risk of ventricular arrhythmias—warrants cautious evaluation. An ICD is recommended as the first-line treatment for secondary prevention of SCD and may also be beneficial for primary prevention. Among pharmacologic options, hydroxyquinidine and quinidine has thus far demonstrated the greatest efficacy.

Future Ahead: Among the causes of sudden cardiac death in young individuals, SQTS is a relatively newly recognized entity. In contrast to its mirror-image disorder, Long QT Syndrome (LQTS), there is a notable scarcity of data regarding SQTS—particularly concerning its clinical presentation, diagnostic criteria, genotype-phenotype correlations, risk stratification, and therapeutic strategies. Its identification has enabled the reclassification of cases that were previously labelled as idiopathic ventricular fibrillation. Individualized therapy, close monitoring, and a multidisciplinary approach—incorporating electrophysiologists, cardiologists, and

pharmacologists—are essential for the effective management of these patients.

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Fascicular Blocks: Contemporary Understanding in Electrophysiology

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Introduction

Fascicular blocks also known as hemiblocks , first described by Rosenbaum and his coworkers in 1968, represent conduction delay or interruption within one of the major fascicles of the left bundle branch system.[1]

While traditionally described as benign ECG findings in otherwise healthy individuals, they can indicate underlying anatomical or functional disturbance in the left bundle fascicles which act as markers of diffuse His–Purkinje disease or structural myocardial pathology.

This article aims to provide basic diagnostic criteria, management as well as latest updates on this topic in the world of electrophysiology.

Anatomical Considerations

The left bundle branch divides into:

1. Left anterior fascicle (LAF) supplies the anterolateral wall via its Purkinje fibres.
2. Left posterior fascicle (LPF) supplies the inferoposterior wall.
3. In 5-10% of population a third centroseptal fascicle aka median fascicle supplies the mid-septum.[2]

Key properties

The LAF is thin and long supplied by septal perforators of the LAD which are end arteries hence are more vulnerable to injury due to ischemia, fibrosis, surgical trauma.

The LPF is broad, short, and richly supplied with dual blood supply i.e. PDA and LAD septals, hence LPFB is very rare in otherwise healthy individuals and almost always indicates an underlying pathology.

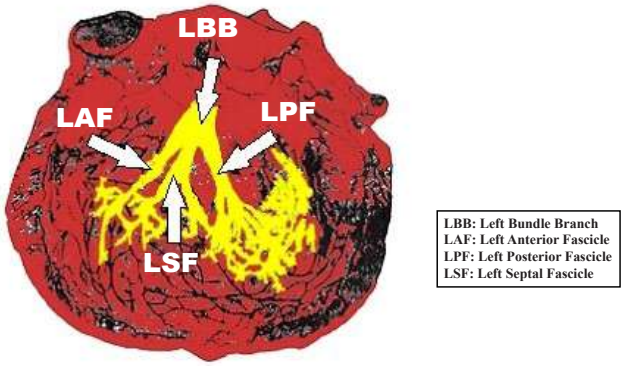


Figure 1: Showing Left bundle dividing into three fascicles i.e. left anterior, left posterior, and the left septal fascicles.

Centroseptal Fascicle is supplied by proximal septal perforators of the LAD and may be involved in septal MI or during septal myectomy/ablation.

Etiology

1. **Ischemic heart disease**:
   - LAFB often associated with anterior MI
   - LPFB occasionally seen with inferior MI but usually with concomitant RBBB
2. **Fibrotic/degenerative conduction disease**: Lenègre and Lev disease
3. **Infiltrative cardiomyopathies**: Sarcoidosis, amyloidosis, Chagas disease
4. **Congenital heart disease**: Fascicular blocks are common post-surgical repair like TOF, VSD closure
5. **Iatrogenic**: Post-TAVI, EP ablation near LVOT, septal myectomy or alcohol septal ablation[3]

Left Anterior Fascicular Block (LAFB):

LAFB with a prevalence of 5-7% of elderly population is due to the disruption in left anterior fascicle, conducts readily via the posterior fascicle producing a small r wave in inferior leads (II, III, aVF) and a small q in lateral leads I and aVL, slowly the leftward forces of the LV myocardium takes over producing a deep S in inferior leads (II, III, aVF) and a corresponding tall R in leads I and aVL. V5-V6 also show qR complexes. All this put together causes a Left axis deviation (–45° to –90°) and delay in QRS duration but usually less than 120msec.

One should look for anteroseptal MI which may mimic LAFB, inferior wall MI q waves maybe masked by LAFB, also rule out LVH, pre-excitation or paced rhythms that mimic LAFB.

ECG criteria:

1. **Axis**: Left axis deviation (–45° to –90°)
2. **QRS morphology**:
   - Small r with deep S in inferior leads (II, III, aVF)
   - Small q with tall R in leads I and aVL

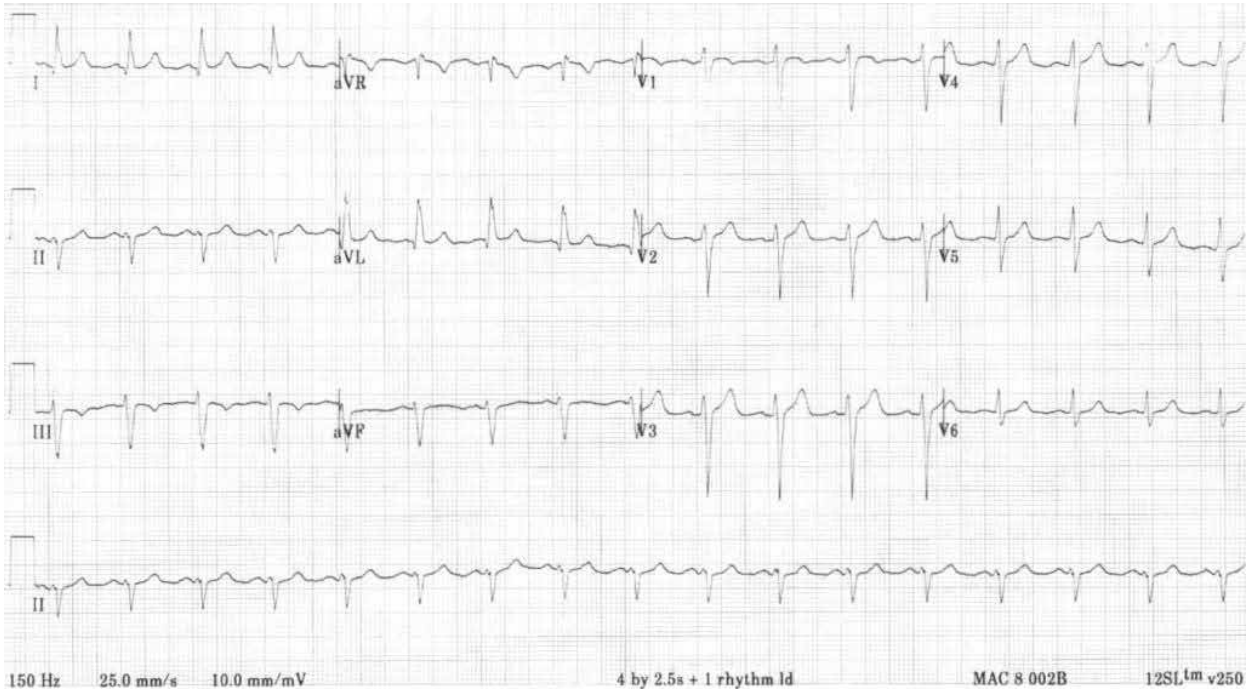


Figure 2: Showing LAFB with Left axis deviation, qR complexes in I, aVL; rS complexes in inferior leads, Prolonged R wave peak time in aVL, Slightly prolonged QRS duration. *Image derived from https://litfl.com*

- Prolonged R wave peak time in aVL > 45ms

3. **QRS duration**: Normal or slightly prolonged (80-110 msec)

Left Posterior Fascicular Block (LPFB)

Isolated LPFB is very rare (<0.1%) and is due to disruption in the left posterior fascicle leading to preferential activation via the intact left anterior fascicle. This directs the initial depolarization vector superiorly and leftward, producing a small r wave in leads I and aVL and a corresponding deep S wave in these same leads. In the inferior leads (II, III, aVF), the activation reaches later via the anterior fascicle, generating an initial small q followed by a tall R complex (qR pattern). Lateral precordial leads (V5–V6) may show a predominant R pattern with absent or minimal q. All this results in a Right axis deviation (+90° to +180°), with QRS duration usually normal or only mildly prolonged (<120 ms).

Differential considerations include right ventricular hypertrophy, lateral wall MI, vertical heart position, or pre-excitation that can mimic LPFB. Isolated LPFB is rare due to its dual blood supply (LAD septals + PDA); when present, it often implies significant underlying structural heart disease, usually in combination with RBBB (bifascicular block).

ECG criteria

1. **Axis**: Right axis deviation (+90° to +180°) without other causes
2. **QRS morphology**:
   - rS in leads I and aVL
   - qR in II, III, aVF
   - Prolonged R wave peak time in aVF
3. **QRS duration**: Usually <120 msec

Bifascicular Block

Bifascicular block is defined as conduction impairment involving two of the three fascicles i.e. the right bundle branch, left anterior fascicle, or left posterior fascicle. The most frequent combination is RBBB with LAFB, while RBBB with LPFB occurs less often. On the ECG, it presents as a typical RBBB pattern accompanied by axis deviation (leftward or rightward depending on the affected fascicle). Its presence suggests significant disease of the His–Purkinje system and is clinically important because of the potential to progress to complete heart block.[4]

ECG: Typical RBBB pattern (rsR′ in V1, broad S in I and V6) with axis deviation pattern of LAFB or LPFB.

Trifascicular Block:

Trifascicular block manifests with impairment across all three fascicles. On ECG, it is commonly described as a bifascicular block accompanied by first-degree AV block (prolonged PR interval) but this can be a pseudotrifascicular block which indicates proximal conduction slowing and not a diffuse distal His–Purkinje disease.

On the other hand an alternating fascicular pattern with intermittent higher-grade AV block with the above ECG changes will point towards a diffuse distal His-Purkinje

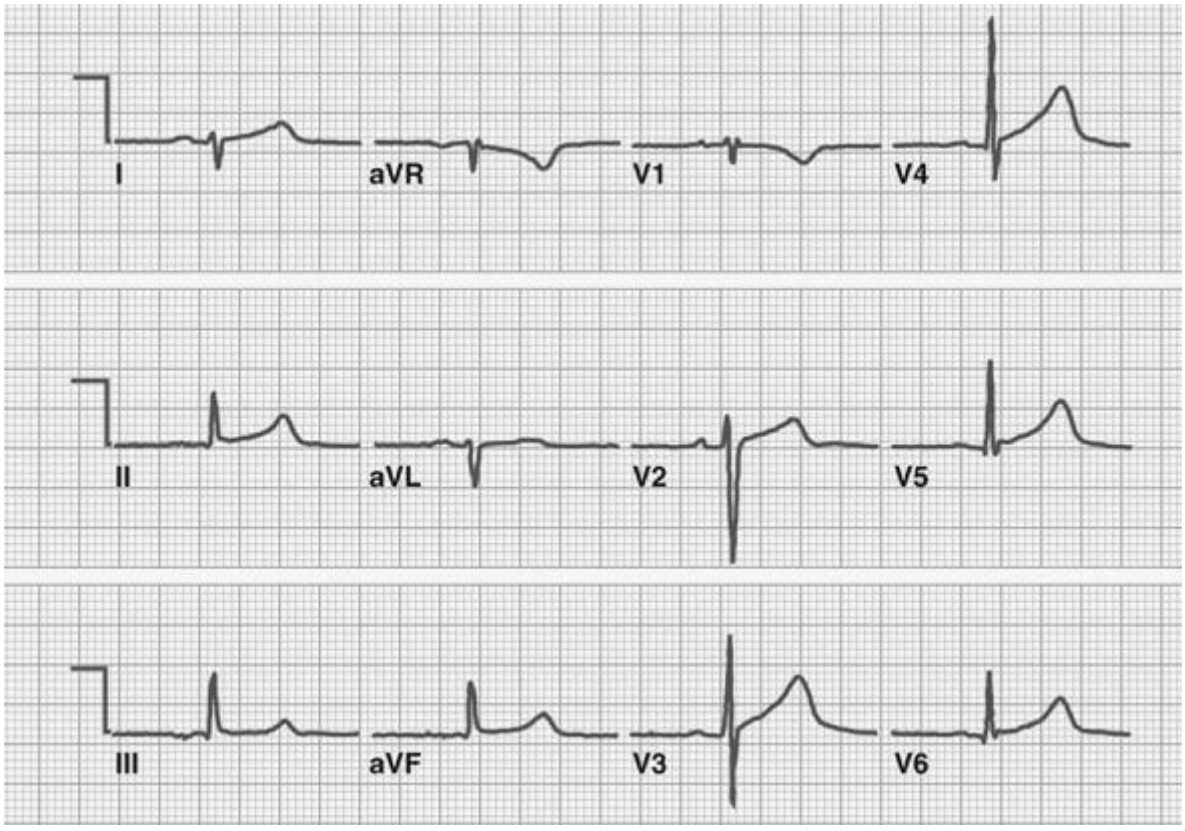


Figure 3: Isolated LPFB with rS in leads I and aVL, qR in II, III, aVF, Prolonged R wave peak time in aVF. *Image derived from https://litfl.com*

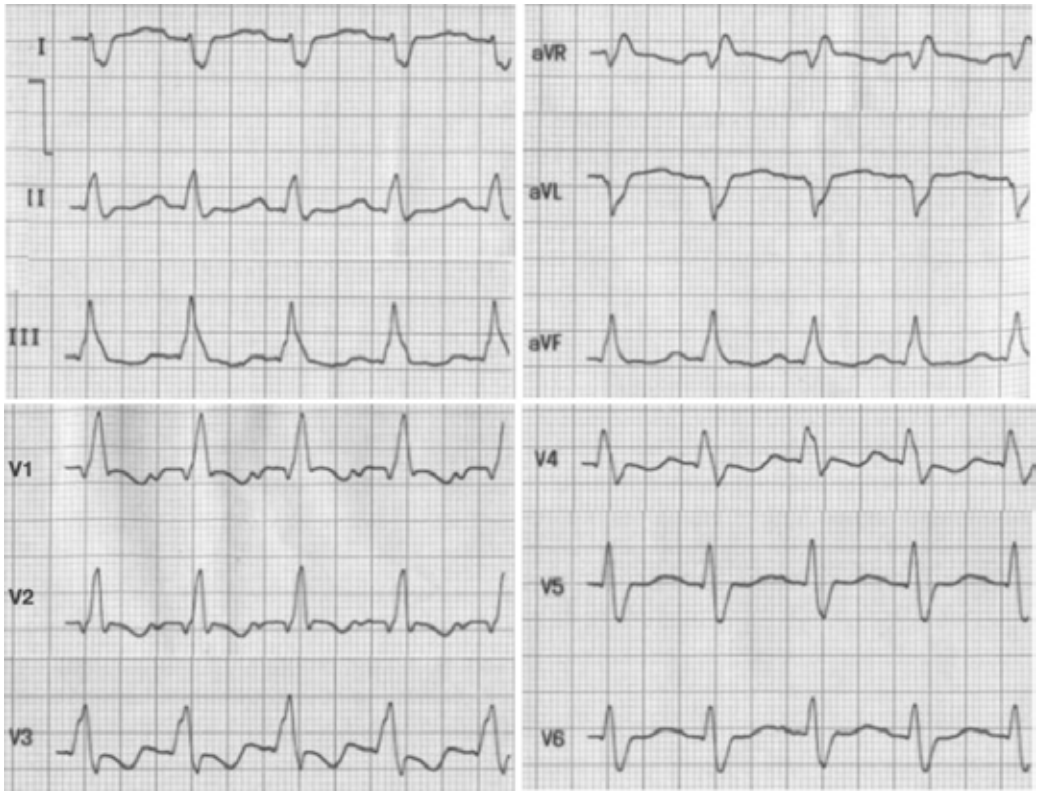
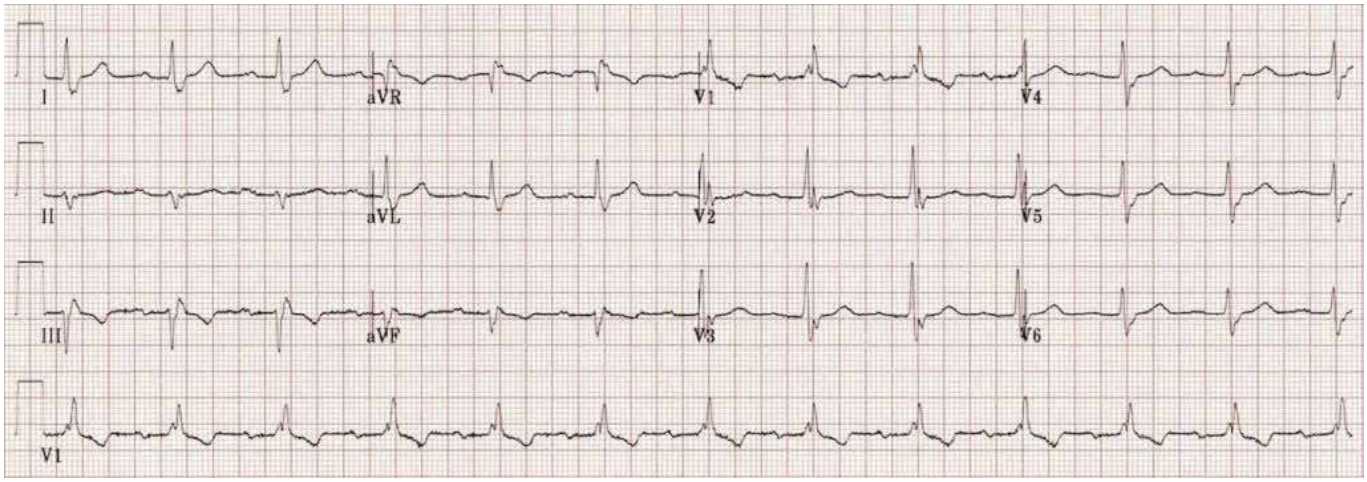


Figure 4: RBBB with LPFB with slurred S wave in lead I and slurred R in V1, Right axis deviation (dominant negative deflection in leads I and aVl) with dominant positive deflection in aVF along with rS pattern in lead I and qR pattern leads III and aVF. *Image derived from https://litfl.com*



k + prolonged PR interval.

Figure 5a: Pseudotrifascicular block showing RBBB+LAFB and 1st Degree AV block. *Image derived from https://litfl.com*

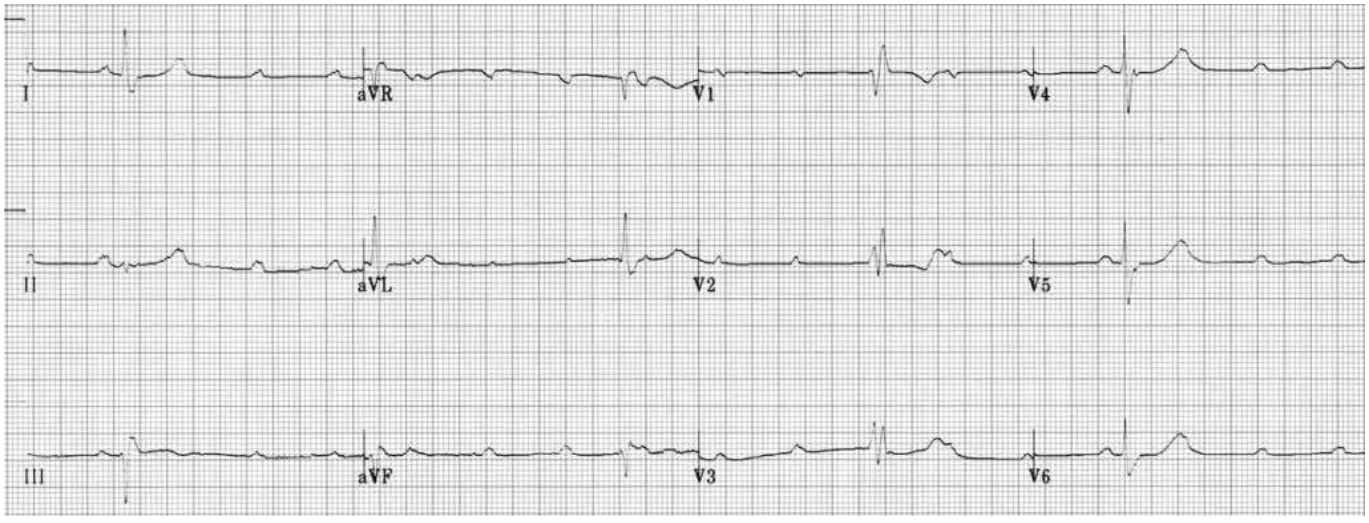


Figure 5b: True trifascicular block showing Right bundle branch block with Left anterior fascicular block and Third degree heart block. *Image derived from https://litfl.com*

disease. This can be confirmed by a electrophysiological study showing a prolonged H-V interval(>55msec).[5]

Clinically, it indicates a significant risk of progression to advanced AV block and warrants pacing when symptoms or high-grade conduction disturbances are documented.

ECG: Bifascicular block and prolonged PR interval

Electrophysiological Perspectives

Mapping studies suggest that left anterior fascicular block (LAFB) often represents slowed conduction with altered activation patterns rather than a complete interruption. The resulting QRS axis deviation does not necessarily indicate isolated fascicular pathology but instead reflects the net effect of shifted depolarization vectors. Invasive electrophysiologic testing is rarely needed for isolated fascicular blocks, unless patients present with unexplained syncope or advanced atrioventricular conduction abnormalities. Importantly, a prolonged HV interval (>55 ms) in the presence of bifascicular block points toward more widespread His–Purkinje system disease.[6]

Clinical Implications

1. **Isolated LAFB**: Mostly benign, but can indicate a prior silent anterior wall myocardial infarction or other structural disease as discussed.
2. **Isolated LPFB**: Almost always pathological and should prompt search for ischemic heart disease or structural abnormalities.
3. **Bifascicular block**:
    - Asymptomatic patients need to be on regular follow up, pacemaker is not indicated.
    - In symptomatic individuals with syncope with intermittent AV block, pacemaker is indicated.

- o In those with syncope/presyncope consider EP study to detect any HV prolongation and confirm infra-Hisian block.

4. Trifascicular block:

- o Asymptomatic individuals without intermittent high grade AV block pacing is not indicated.
- o Alternating bundle branch block or symptomatic trifascicular block with intermittent grade AV block (or prolonged HV interval) pacing indicated.

Evolving Insights:

- Septal fascicular blocks are described, but is not reliably identified on surface ECG.
- New onset fascicular blocks during acute MI may indicate extensive myocardial involvement and predict higher mortality.
- Among the post-TAVI conduction disturbances, LAFB is a common precursor to AV block. Hence it is debated whether these individuals need prophylactic pacing.
- Distinguishing true fascicular block from secondary axis shifts remains challenging.

Conclusion

Fascicular blocks which were previously regarded as minor ECG curiosities, are now regarded as indicators of conduction system integrity and an valuable marker for underlying myocardial diseases. They serve as surrogate of His–Purkinje function and help to diagnose patients who are at risk for progression to advanced atrioventricular block or complications in structural heart disease. Their evaluation must always consider clinical context, associated conduction abnormalities and progression over time to provide only appropriate treatment without wasting valuable medical resources.

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Obstructive Myocardial Infarction Without Traditional ECG Signs

Arvind Ghongane
Consultant Physician

Traditional Acute Coronary Syndrome definition carries several limitations, most notably its reliance on

'ST-segment elevation' for emergent reperfusion therapy. This approach fails to identify a substantial proportion of patients with acute coronary occlusion (ACO) who present without diagnostic ST- elevation, often resulting in delayed treatment. Approximately 30% of ACO cases are missed when relying solely on standard STEMI criteria, leading to delays in reperfusion and worse clinical outcomes.

OMI (Obstructive Myocardial Infarction) is defined by acute coronary occlusion causing transmural ischemia and requiring urgent reperfusion, even in the absence of classic ST-elevation. OMI criteria- including - De Winter T-waves, Wellens syndrome, posterior infarction signs, and terminal QRS distortion are better compared to standard STEMI criteria. Observational data suggest that up to 30% of patients initially classified as NSTEMI have an unrecognized OMI, with comparable infarct size and mortality to STEMI patients but significantly delayed treatment.

Several machine learning models for ECG interpretation have been developed to differentiate between OMI and NOMI, reflecting the growing clinical demand for more accurate and timely identification of acute coronary occlusion (Al-Zaiti et al, 2023).

Shortcomings of the 12-lead ECG

The standard 12-lead ECG is suboptimal for detecting OMI in several scenarios. The sensitivity is poor for OMI affecting the posterior wall of the left ventricle, the right ventricle and left circumflex artery infarctions. Moreover, some patients exhibit non-specific changes, e.g. new bundle branch block, or ST elevations that are subtle and do not meet the formal voltage criteria. Thus, relying solely on millivolt thresholds for ST-segment elevation oversimplifies the complex and dynamic ECG changes occurring during acute coronary occlusion.

OMI-guided diagnosis facilitates earlier and more appropriate intervention, potentially improving outcomes in high-risk patients who would otherwise be misclassified.

Additionally, emerging artificial intelligence (Al) tools show promise in enhancing ECG interpretation and decision-making under the OMI framework. While these findings are compelling, randomized clinical trials are needed to validate the OMI approach and support its integration into routine clinical practice.

OMI is a pathophysiological and clinical diagnosis, not solely an ECG-defined entity. The diagnosis of OMI integrates the ischemic symptoms, hemodynamic status, comprehensive ECG findings (including subtle signs beyond classic ST-elevation), cardiac biomarker levels, and point-of-care echocardiography.

Angiographic evidence of a culprit artery occlusion (e.g. TIMI flow grade 0, 1, or 2) is a key confirmatory feature. Even TIMI 3 flow on angiography can be consistent with a recently reperfused OMI if associated with very high cardiac troponin levels, indicating a significant preceding ischemic event due to occlusion. The fundament of the OMI approach is that the presence of an ACO is the determinant for emergent reperfusion, regardless of whether traditional STEMI ECG criteria are fulfilled.

Occlusion Myocardial Infarction (OMI) is defined as an acute myocardial infarction resulting from the acute total or near-total occlusion of a coronary artery, where there is insufficient collateral circulation to prevent ongoing transmural myocardial ischemia and active infarction. Emergent reperfusion therapy is required to limit infarct size and minimize the risk of malignant ventricular arrhythmias.

Diagnosing Occlusion Myocardial Infarction

Diagnosing OMI requires a multifaceted approach that integrates advanced ECG interpretation skills, clinical judgment, appropriate use of cardiac biomarkers, and the incorporation of emerging technologies like artificial intelligence (Al) and point-of-care echocardiography.

Patients classified under the NOMI category do not derive benefit from emergent reperfusion strategies (i.e., interventions aiming for reperfusion within <2 hours of presentation) in the same way OMI patients do. However, urgent coronary angiography (e.g., within 24-72 hours) is frequently indicated for NOMI patients based on their overall ischemic risk profile and to definitively characterize their coronary anatomy and underlying pathology.

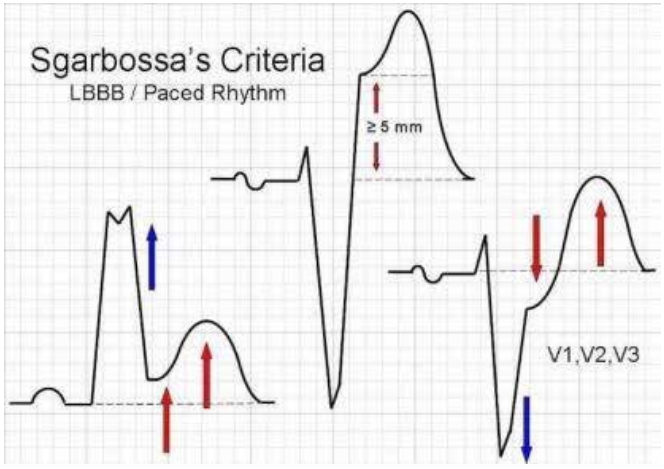
Non-Occlusion Myocardial Infarction (NOMI) encompasses myocardial infarctions where there is no acute, persistent thrombotic occlusion of the culprit coronary artery, or where collateral circulation is sufficient to prevent progressing transmural infarction. This category can include MIs resulting from plaque rupture with a non-occlusive thrombus, instances where spontaneous reperfusion (by means of endogenous thrombolysis) of an occluded artery has occurred prior to presentation, myocardial infarction with non-obstructive coronary arteries (MINOCA) due to various mechanisms (e.g., coronary spasm, microvascular dysfunction, embolism), or Type 2 MI secondary to a profound supply-

demand mismatch (e.g., severe anemia, tachyarrhythmia in the setting of stable coronary disease).

Patients classified under the NOMI category do not derive benefit from emergent reperfusion strategies (i.e., interventions aiming for reperfusion within <2 hours of presentation) in the same way OMI patients do. However, urgent coronary angiography (e.g., within 24-72 hours) is frequently indicated for NOMI patients based on their overall ischemic risk profile and to definitively characterize their coronary anatomy and underlying pathology.

Sgarbossa Criteria and Smith-Modified Criteria

The Sgarbossa criteria, and the more accurate Smith-Modified Sgarbossa criteria, are the most widely used criteria to detect ongoing ischemia in patients with LBBB. Key features include concordant ST-segment elevation (ST elevation in leads with a positive QRS complex) or excessively discordant ST changes (e.g., ST elevation ≥1 mm and ≥25% of the preceding S-wave depth in leads with a negative QRS).



Smith-Modified Sgarbossa Criteria

Concordant ST elevation ⩾ 1 mm in ⩾ 1 lead Concordant ST depression ⩾ 1 mm in ⩾ 1 lead of V1-V3

Excessive discordant STE in ⩾ 1 lead anywhere with ⩾ 1 mm STE, as defined by ⩾ 25% of the depth of the preceding S-wave

De Winter's sign

Up-sloping ST- depression at the J-point in leads V1-V6. Tall, symmetrical T-waves in precordial leads.

Possible slight ST elevation in lead aVR. Clinical correlation

Indicates acute occlusion of the proximal left anterior descending (LAD) Represents approximately 2% of anterior myocardial infarctions. Requires immediate reperfusion therapy despite absence of classic ST-elevation.

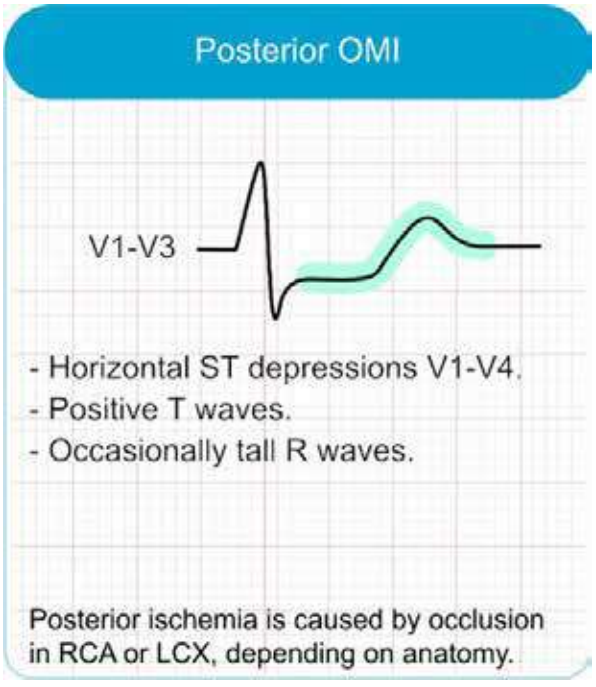
Posterior OMI

Horizontal ST-segment depression in leads V1-V3. Tall R-waves and upright T-waves in V1-V3.

ST elevation in posterior leads V7-V9 if recorded. Clinical correlation

Often due to occlusion of the left circumflex artery or right coronary artery.

May occur in isolation or with inferior/lateral MI. Easily missed; requires posterior lead placement for confirmation.



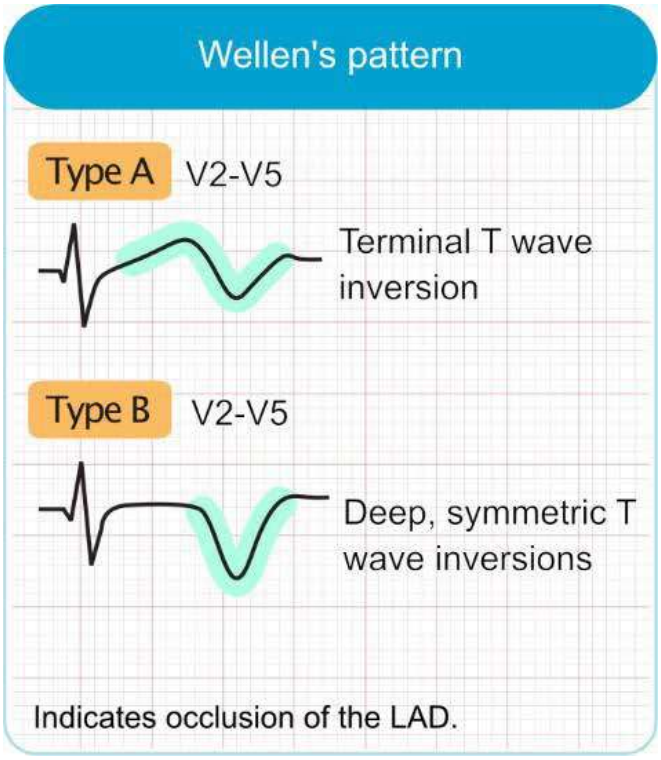
Wellens Syndrome (Wellens ECG pattern)

Biphasic or deeply inverted T-waves in leads V2-V3 (may be V1-V6). Minimal or no ST-segment elevation.

No pathological Q waves; normal R-wave progression. Clinical correlation

Signifies critical stenosis of the proximal LAD artery. Typically observed in pain-free state after recent angina.

High risk for extensive anterior wall myocardial infarction if left untreated. Subtle ST-Segment Elevation



New RBBB + LAFB

ST-segment elevation that does not meet standard STEMI criteria (e.g., <1 mm in limb leads or <2 mm in precordial leads).

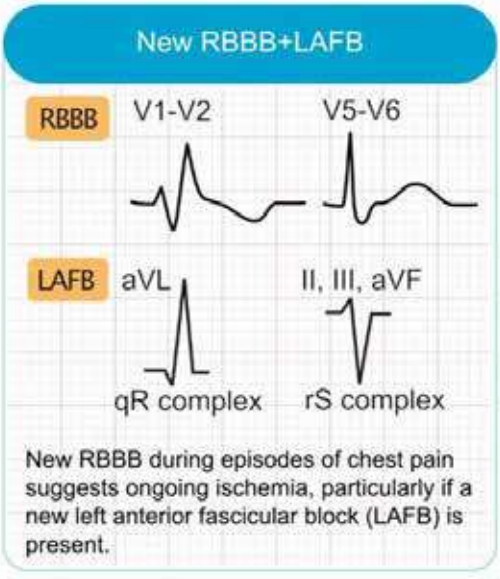
Elevation may appear disproportionately large relative to a low-amplitude QRS complex.

Often accompanied by dynamic changes, such as evolving T- wave morphology or reciprocal ST-segment depression, Clinical correlation

Commonly associated with acute occlusion of the left anterior descending (LAD) artery.

Patients may present with significant myocardial ischemia despite not meeting traditional STEMI criteria.

Prompt recognition is crucial, as these patients benefit from immediate reperfusion therapy.



Northan OMI

ST-segment elevation in leads aVR and aVL, often accompanied by negative T-waves.

ST-segment depression in inferior leads (II, III, AVF) and lateral precordial leads (V4-V6), with positive or biphasic T-waves.

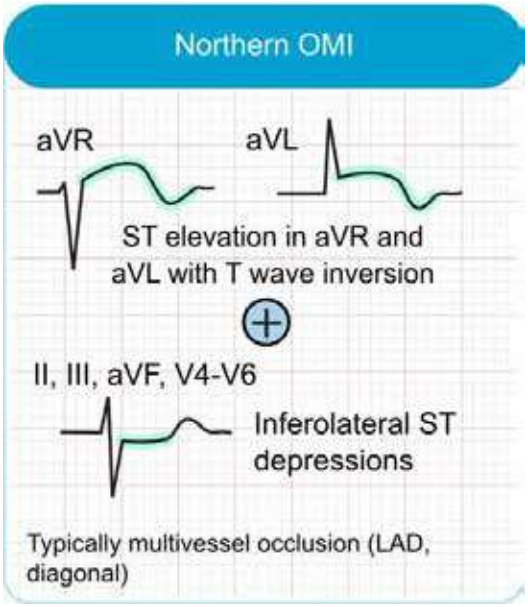
Clinical Correlation:

Suggests acute occlusion of the left main coronary artery or proximal left anterior descending (LAD) artery.

Represents a high-risk pattern associated with extensive myocardial ischemia.

Terminal QRS distortion

These criteria are applied in suspicion of anterior wall infarction.



Terminal QRS Distortion in V2 – V4

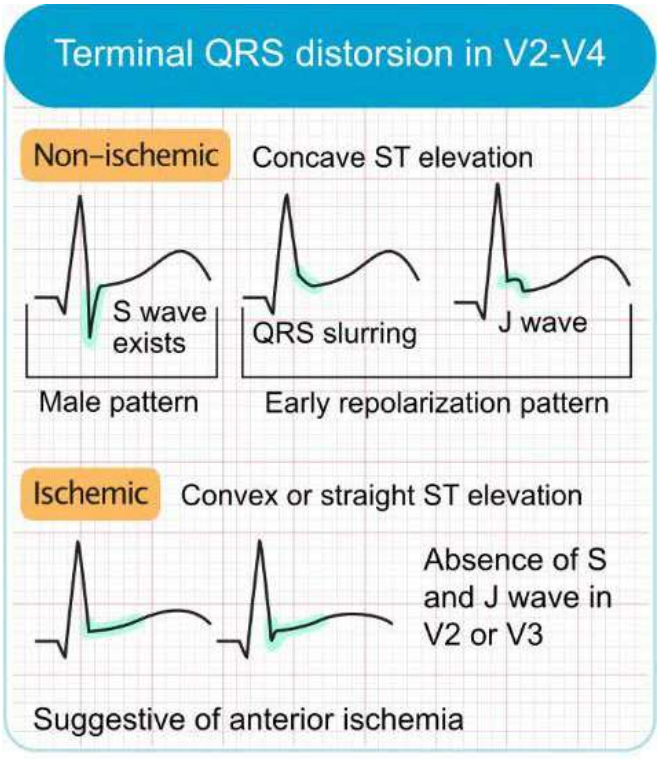
Absence of both the S-wave and J-wave in leads V2 and/or V3.

In leads with an Rs configuration (e.g., V2-V3), the S-wave is absent.

In leads with a qR configuration (e.g., V4-V6), the J-point is elevated to ≥50% of the R-wave amplitude.

This pattern is distinct from early repolarization. Clinical correlation

Highly specific for acute anterior myocardial infarction, particularly involving proximal left anterior descending (LAD) artery occlusion.



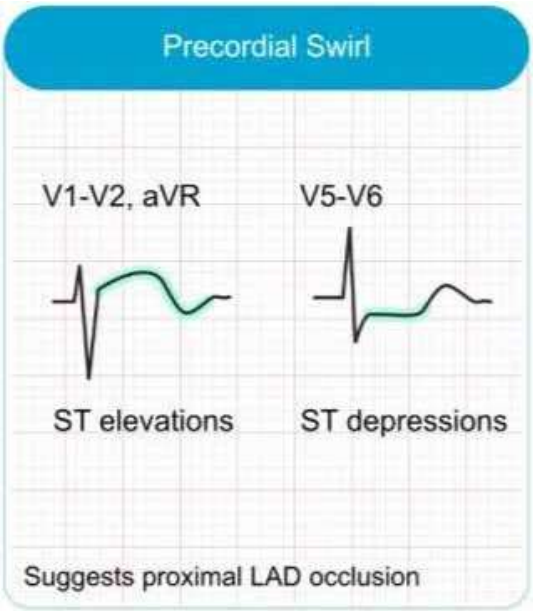
Precordial Swirl

ST-segment elevation in leads V1 and/or aVR.

Reciprocal ST-segment depression in leads V5 and/or V6. Hyperacute or disproportionately tall T-waves in leads V1-V2. Absence of left ventricular hypertrophy (LVH) or wide QRS complexes.

Clinical correlation

Suggests acute occlusion of the proximal left anterior descending (LAD) artery, typically before the first septal perforator. Associated with septal, anterior wall, and apical myocardial ischemia. Often missed by standard STEMI criteria; early recognition is crucial for timely reperfusion therapy.



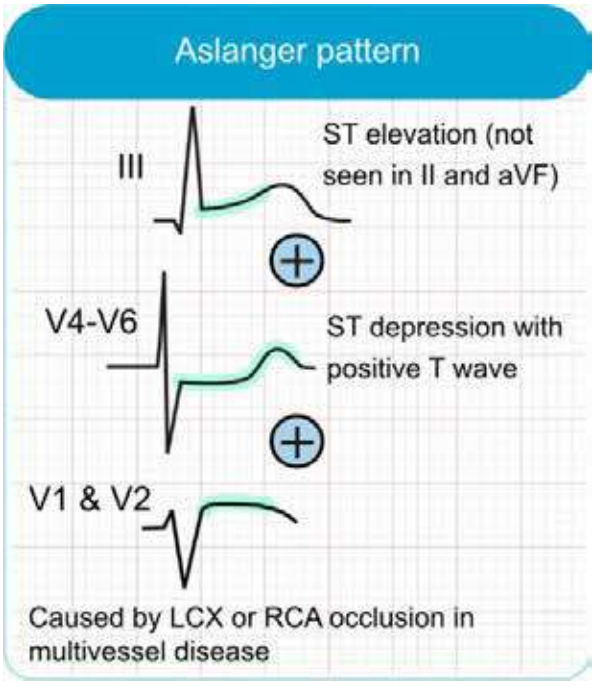
Aslanger Pattern

ST-segment elevation isolated to lead III, without elevation in leads II or aVF.

ST-segment depression in leads V4-V6 (but not in V2), accompanied by positive or terminally positive T-waves. ST-segment elevation in lead V1 greater than in lead V2. Clinical Correlation:

Suggests acute inferior myocardial infarction, often due to occlusion of the left circumflex artery, in the context of multivessel coronary artery disease.

Associated with larger infarct size and higher mortality rates, comparable to those seen in STEMI patients.



Occlusion of the left circumflex artery

Occlusion of the left circumflex artery (LCx) is challenging to detect using standard 12-lead ECG. This difficulty arises because the LCx supplies the lateral and posterior walls of the left ventricle, areas that are less directly represented in the conventional ECG leads. Consequently, LCx occlusions often lack the classic ST-segment elevation patterns seen with occlusions in LAD and RCA, leading to underdiagnosis and potential delays in treatment.

The following should lead to suspicion of occlusion in a left circumflex artery not supplying the infero- basal wall (i.e. not resulting. in posterior wall ECG pattern):

- ST-segment elevation in lateral leads (I, aVL, V5-V6): Elevation in these leads suggests lateral wall involvement, even if changes are subtle.
- Reciprocal ST-segment depression in inferior leads (III and aVF): This finding can support the diagnosis of lateral wall ischemia, even if changes are subtle.

High clinical suspicion: In patients presenting with severe chest pain and an inconclusive ECG, a high suspicion for LCx occlusion should be maintained.

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Changes in Electrocardiogram in Obesity

Ramesh R Dargad[1], Harsh Tantia[2]

[1]Cardiologist, Lilavati Hospital, Mumbai, Seven Hills Hospital, Mumbai, [2]Associate Professor in Radiology, D.Y. Patil Medical College, Navi Mumbai.

Abstract

Cardiovascular disease (CVD) remains the leading cause of global mortality, with hypertension and obesity as pivotal, interconnected risk factors. Recent research and clinical updates, particularly from the ACC 2025 Scientific Sessions and several peer-reviewed studies, emphasize not only the diagnostic limitations in obese hypertensive populations but also emerging pharmacological advances. This review synthesizes the latest evidence on the electrocardiographic (ECG) detection of left ventricular hypertrophy (LVH) in the context of obesity, evaluates repolarization abnormalities, and explores new therapeutic options such as SGLT2 inhibitors and novel anticoagulants.

Introduction

The global burden of cardiovascular disease is intensifying, partly fuelled by the rising prevalence of obesity and hypertension. Electrocardiography (ECG) remains a frontline tool for cardiovascular risk stratification, particularly for identifying left ventricular hypertrophy (LVH), a known precursor to adverse cardiac events. However, accumulating evidence suggests that standard ECG criteria are less effective in obese patients, necessitating the re-evaluation of diagnostic thresholds and integration of novel biomarkers and therapeutics.

Ecg and Obesity: Diagnostic Limitations

Several studies, including those by *Dykiert et al. (2024)*[1] and *Rodrigues et al. (2016)*[2], demonstrate how obesity significantly impacts ECG readings. Dykiert's study, involving 181 adults with overweight or class 1 obesity, revealed prolonged repolarization parameters—such as the T_{peak}-T_{end} interval and JT_{peak}-JT_{end} dispersion—among obese participants compared to controls. These prolongations are clinically significant, as they correlate with increased risk of ventricular arrhythmias and sudden cardiac death.

Similarly, *Rodrigues et al.* recalibrated traditional ECG LVH criteria against cardiac magnetic resonance imaging (CMR), the gold standard for LV mass assessment. The study found that obesity reduced both the sensitivity and specificity of Sokolow–Lyon and Cornell criteria. Obese patients exhibited attenuated QRS voltages, leading to underdiagnosis when using standard thresholds.

The LIFE study by *Okin et al.*[3] further confirmed that Sokolow–Lyon voltage underestimates LVH prevalence in obese individuals, whereas the Cornell voltage-duration product remains a more reliable diagnostic marker. Obese patients had a >2-fold increased risk of LVH when assessed via the Cornell product, but a 4-fold lower risk using Sokolow–Lyon criteria.

The combined evidence underscores the need for BMI-adjusted ECG thresholds and alternative imaging in high-risk populations, a theme supported across multiple reviews and clinical presentations.[4,5]

Therapeutic Developments: Insights From ACC 2025

The 76th Annual Scientific Sessions of the American College of Cardiology (ACC 2025) highlighted several groundbreaking trials aimed at improving outcomes in patients with obesity, diabetes, and cardiovascular comorbidities.[6] A selection of key findings is discussed below:

1. **DapaTAVI Trial: Dapagliflozin in TAVI Patients**

   This randomized controlled trial explored the efficacy of dapagliflozin in patients with aortic stenosis undergoing transcatheter aortic-valve implantation (TAVI). The use of dapagliflozin significantly reduced the composite endpoint of death or worsening heart failure compared to standard care (15.0% vs. 20.1%, HR 0.72, p=0.02).[6]

2. **Empagliflozin vs. Dapagliflozin: Meta-Analysis in Type 2 Diabetes**

   A meta-analysis involving over 370,000 patients compared cardiovascular outcomes between empagliflozin and dapagliflozin. No significant differences were observed across major endpoints, including CV mortality, MACE, and heart failure hospitalization.[6]

3. **API-CAT Trial: Extended-Dose Apixaban in Cancer-Associated VTE**

   The API-CAT trial assessed extended anticoagulation in cancer patients using reduced-dose apixaban. It demonstrated noninferiority to full-dose regimens in preventing recurrent thromboembolism and showed a lower bleeding risk (12.1% vs. 15.6%, p=0.03).[6]

4. **RIVAWAR Trial: Rivaroxaban vs. Warfarin Post-MI**

   In patients with left ventricular thrombus post-myocardial infarction, rivaroxaban was as effective as warfarin for thrombus resolution, with no significant differences in adverse outcomes. Given the ease of use and consistent

anticoagulation with DOACs, rivaroxaban presents a viable alternative for post-MI management.[6]

5. **SMART-CHOICE 3 Trial: Clopidogrel vs. Aspirin Monotherapy**

   In high-risk patients post-PCI, clopidogrel monotherapy was superior to aspirin in reducing the composite of death, MI, and stroke (4.4% vs. 6.6% over 3 years, HR 0.71, p=0.013) without increasing bleeding risk. This challenges the long-standing preference for aspirin and suggests a shift toward personalized antiplatelet strategies.[6]

Integration of Diagnostics and Therapy: A Clinical Perspective

The convergence of diagnostic and therapeutic innovations underscores a critical evolution in cardiovascular medicine. Standard ECG, though affordable and widely accessible, faces serious limitations in detecting LVH among obese hypertensive patients. Adjusting ECG interpretation by incorporating BMI, waist circumference, and comorbid conditions (like type 2 diabetes and atrial fibrillation) enhances its diagnostic utility.

Simultaneously, emerging therapies are not only targeting glycaemic control or thrombosis prevention but also showing cardiovascular benefit in diverse populations—including those with valvular disease, obesity, or cancer. The parallel refinement of diagnostics and pharmacologic options enables precision medicine that was not feasible even a decade ago.

Future Directions

1. **Standardization of BMI-adjusted ECG criteria**
   Validation of obesity-specific thresholds—such as modified Cornell product cutoffs—should be integrated into guideline-directed ECG interpretation frameworks.

2. **Hybrid imaging protocols**
   When ECG fails to provide reliable data, echocardiography or cardiac MRI should be promptly employed, particularly in high-risk populations.

3. **Broader implementation of cardiometabolic drugs**
   Expanding indications for SGLT2 inhibitors and GLP-1 receptor agonists into non-diabetic but high-risk patients (e.g., post-TAVI) could redefine treatment paradigms.

4. **Personalized antiplatelet therapy**
   The SMART-CHOICE 3 data support a tailored approach to monotherapy post-PCI, factoring in genetic, clinical, and lifestyle variables.

Conclusion

The interplay between obesity, hypertension, and cardiovascular disease creates complex diagnostic and therapeutic challenges. Recent research underscores the need to recalibrate diagnostic tools like the ECG to account for body composition. In parallel, landmark trials from ACC 2025 signal a new era in therapeutics—where treatments not only control risk factors but also actively reduce cardiovascular events across a spectrum of patient profiles. As both diagnostics and therapies evolve, clinicians are better equipped to deliver truly individualized care.

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Artificial Intelligence in Electrocardiography: Transforming Diagnosis and Prognosis

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Abstract

The integration of Artificial Intelligence (AI) into electrocardiography (ECG) is redefining the landscape of cardiovascular care. Leveraging machine learning (ML) and deep learning (DL), AI-ECG systems offer superhuman accuracy in diagnosing arrhythmias, detecting silent myocardial infarction (MI), predicting heart failure, and even identifying non-cardiac diseases. This article comprehensively reviews recent advancements, real-world applications, key randomized trials, and emerging challenges in AI-powered ECG, drawing from over 100 peer-reviewed studies and multi-center trials.

Introduction

Evolution of Electrocardiography

Since Willem Einthoven's pioneering work in the early 1900s, ECG has remained a cornerstone of cardiovascular diagnosis. Traditional interpretation is dependent on clinician expertise and prone to variability. Despite advances in digital ECGs, diagnostic accuracy has plateaued — until the recent emergence of AI.

Emergence of AI in Cardiology

AI technologies, particularly DL models like convolutional neural networks (CNNs) and transformer architectures, can detect minute, complex patterns in ECG signals beyond human capability. These tools hold potential not only for diagnosis but also for risk stratification and longitudinal disease monitoring.

Technical Foundations of AI in ECG

AI Methodologies

Method	Strengths	Limitations
Traditional ML	Interpretable	Requires manual feature engineering
CNNs	Learns raw features	Often black-box models
Transformers	Captures long-range dependencies	Computationally intensive
Hybrid Models	Combines strengths of various models	Complex implementation

Notable datasets like PhysioNet, CODE-ECG, and Mayo Clinic's repositories have driven model development.

Data Preprocessing Essentials

Typical steps include:

- Noise filtering
- Beat segmentation
- Signal normalization
- Feature extraction (e.g., RR intervals)

These ensure model robustness, particularly for real-world wearable device signals.

Diagnostic Applications

Arrhythmia Detection

AI models demonstrate cardiologist-level accuracy:

- **Hannun et al. (2019)**: DNN classifying 12 arrhythmias (AUC 0.97)
- **Fiorina et al. (2024)**: Improved AF detection via smartwatch ECGs
- **PED-ECG AI study (2023)**: 94% sensitivity for pediatric arrhythmias
- **VITAL-AF (2023)**: 68% faster VT detection in ICUs

Myocardial Infarction (MI)

AI enables early and accurate MI detection:

- **Makimoto et al. (2020)**: CNN outperforming physicians
- **AI-EMERGE Trial (2023)**: 32% reduction in false STEMI activations
- **Zhou et al. (2023)**: AUC 0.889 for occlusion MI

Structural Heart Disease

AI can diagnose subtle and silent structural changes:

- **Attia et al. (2019)**: LVEF < 35% with AUC 0.93
- **Siontis et al. (2023)**: Track HCM progression longitudinally
- **Grogan et al. (2023)**: Amyloidosis screening (AUC 0.91)
- **Pulmonary Hypertension**: AI predicts mean pulmonary artery pressure

Valvular Heart Disease

AI models detect aortic stenosis and mitral regurgitation:

- **Kwon et al. (2020)**: AUC 0.88 from 12-lead ECG
- **Ulloa-Cerna et al. (2023)**: Transformer model with 90% sensitivity

Non-Cardiac Conditions

- **Hyperkalemia**: Predicted 48 hours earlier (Gallagher et al., 2023)
- **Sepsis**: ICU monitoring with 92% sensitivity
- **Neurologic Diseases**: Early Parkinson's detection from ECG
- **COVID-19**: ECG indicators for cardiac strain and mortality prediction

Prognostic Applications

Mortality Prediction

- **ECG-MORT (2023)**: AUC 0.82 vs. ASCVD score (AUC 0.74)
- **Imperial College Model**: Predicts early mortality from ECG

Heart Failure Prediction

- **PREVENT-HF Trial (2023)**: 27% reduction in HF hospitalizations using AI guidance
- **Anumana Model**: Early prediction of LV systolic dysfunction

Stroke Risk & AF Prediction

- **DIGITAL-AF (2023)**: 3.1x more AF detection via smartwatch AI
- **LA strain analysis**: Predicts LAA thrombus for stroke prevention

ICU and Critical Illness

- **QCG Platform (Korea)**: AI predicts ICU admission, ventilation need, and mortality

Real-World Deployment & Devices

AI Integration in Devices

Smartwatches (Apple, Samsung, Fitbit), AliveCor, and clinical-grade patches use AI to monitor arrhythmias continuously, particularly AF.

EMR & Hospital Workflow Integration

AI algorithms now work within EHR systems, enabling:

- Population-level screening
- Automated triage alerts
- Risk prediction embedded in daily workflow

Workplace & Community Screening

- **Toyota Program**: Detected 14% previously undiagnosed CVD in workers
- **AI-HEART Study (2023)**: Reduced CV events by 18% in community screening

Challenges and Ethical Considerations

Data Diversity and Bias

Most models are trained on Western datasets. They may underperform in underrepresented populations (e.g., Obermeyer et al., 2023).

Interpretability and Trust

Black-box models hinder clinical adoption. Tools like:

- SHAP values
- Saliency maps are aiding transparency.

Regulatory and Legal Issues

Only 8 AI-ECG tools were FDA-approved as of 2023. Questions remain around:

- Informed consent
- Liability
- Updating AI post-deployment

Real-World Reliability

- **Model drift**: Performance may degrade over time
- **Resistance**: 42% of physicians are skeptical of AI guidance (AMA survey, 2023)

Future Directions

Explainable AI (XAI)

Efforts are underway to make predictions understandable by:

- Visualizing model focus areas on ECG
- Providing confidence scores and rationale

Multimodal Integration

Combining ECG with:

- Echo
- Lab data
- Imaging
- Genomics

This enhances diagnostic precision and enables **truly personalized medicine**.

Generative AI for ECG

Synthetic ECG generation (e.g., GANs) helps:

- Augment rare disease datasets
- Improve model generalizability

Global Health Impact

Smartphone-based, AI-enabled ECGs can bring cardiology expertise to underserved regions.

Conclusion

AI has firmly established its role in revolutionizing ECG interpretation. From diagnosing arrhythmias and myocardial infarction to predicting sudden cardiac death and heart failure, AI-ECG tools are not only matching but often surpassing human performance. However, true clinical integration requires tackling biases, improving explainability, and aligning regulatory frameworks. With continued innovation, AI in ECG is poised to democratize cardiovascular care, reduce disease burden, and improve outcomes globally

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*As compared to Top 5 Branded Generics as per IQVIA Rx MAT April 2025(Prices as per 1mg : as on 30.05.2025)
#For Empagliflozin: No Dose Adjustment recommended for use in individuals with an estimated glomerular filtration rate (eGFR) of 20 mL/min/1.73 m2 or higher
Data on file

ECG Summit Schedule 2025-2026 (Ongoing Program)

Date	Topic	Recap	Speaker	Moderator
27.04.2025	Generation of normal electrocardiogram		Dr S B Gupta	Dr Aditya Kapoor
25.05.2025	QRS goes tall, wide or bizarre-Chamber Hypertrophy, Bundel Branch and Fascicular Bocks	Dr S B Gupta	Dr Aditya Kapoor	Dr Amit Vora
29.06.2025	Paroxysmal SVT-AV Node Dependence, Asmptomatic Pre-excitation	Dr Aditya Kapoor	Dr Amit Vora	Dr Ajay Naik
03.08.2025	Atrial Arrhythmias : Atrial Tachycardia, Atrial Flutter and Atrial Fibrillation	Dr Amit Vora	Dr Ajay Naik	Dr Jitendra Makkar
31.08.2025	Ventricular Arrhythmias : Monomorphic, Pleomorphic or Polymorphic	Dr Ajay Naik	Dr Jitendra Makkar	Dr Hygriv Rao
28.09.2025	Bradyarrhythmias : SND or AV Block	Dr Jitendra Makkar	Dr Hygriv Rao	Dr Daljeet Kaur Saggu
26.10.2025	ECG in ACS : NSTEMI & STEMI. Other Variants.	Dr Hygriv Rao	Dr Daljeet Kaur Saggu	Dr Joy Thomas
30.11.2025	ECG in STEMI : Culprit Vessel Localization, Judging Recanalization	Dr Daljeet Kaur Saggu	Dr Joy Thomas	Dr Ketan Mehta
28.12.2025	ECG Mimics of MI and Artifacts	Dr Joy Thomas	Dr Ketan Mehta	Dr Gurunath Parale
18.01.2026	ECG Abnormalties of drugs, toxicity and electrolyte imbalance : Case Studies	Dr Ketan Mehta	Dr Gurunath Parale	Dr Aparna Jaswal
22.02.2026	Channelopathies (Long and short QT, Brugada, ER, CPVT	Dr Gurunath Parale	Dr Aparna Jaswal	Dr Ashish Nabar
29.03.2026	Pacemaker ECG What a physician should know?	Dr Aparna Jaswal	Dr Ashish Nabar	Dr Yash Lokhandwala

ECG Clinician's Angiography

Scientific Agenda 2025-2026 (Ongoing Program)

Request the faculties to note their respective date and time slot.

Date	Topic	Speaker	Moderator
9th July, 2025	ECG in ACS	Dr. Amit Vora	Dr. T.R. Muralidharan
13th August, 2025	Localisation of Culprit Vessel in ACS	Dr. Aditya Kapoor	Dr. Uday Jadhav
10th September, 2025	ST Mimics in ACS	Dr. S.B. Gupta	Dr. Gurunath Parale
8th October, 2025	Arrythmias in ACS	Dr. Jitendra Makkar	Dr. Yash Lokhandwala
12th November, 2025	Guideline Directed Therapy in ACS Post Hospitalisation	Dr. Ashish Nabar	Dr. Aparna Jaswal
10th December, 2025	PQRST in HF	Dr. B. Hygriv Rao	Dr. Dayanand Kumbla
14th January, 2026	Guideline Directed Therapy in Chronic Heart Failure	Dr. Ketan Mehta	Dr. Joy Thomas

Time: 8.30 pm to 9.30 pm

USV-ISE ECG Master Class Schedule 2025-2026 (Ongoing Program)

Date	Time	Speaker 1	Speaker 2	Moderator	Hotel Name
17.05.25	8.30 PM	Dr Ajit Bhagwat	Dr Rajkumar gumare		Rama International
26.05.25	8:00 PM	Dr. Y. Rama Kishore		Dr. Buchi Babu	Hotel Capital
29.05.25	7.30PM	Dr Manjappa		Dr C B Keshavmurthy	Hotel Regenta Mysore
30.05.25	8:00 PM	Dr Niti Chadha			
30.05.25	8:00 PM	Dr Saurabh Deahpande	Dr Ameya Udayavar		
06.06.25	8.30 PM	Dr Rajesh Badani	Dr Chandrakant Chavan	Dr Rohan Kate / Dr Amit Walimbe	Hotel Pride Pune
06.06.25	8:00 PM	Dr. Somashekar Ghanta		Dr. Guru Prasad Pyla	Hotel Murali furtune
07.06.25	9:00 PM	Dr Anirudh Vyas	Dr Rajkumar Agrawal		Hotel Sayaji
13.06.25	9.00 PM	Dr Rituparna Shinde	Dr Rajesh Dhopeshwarkar	Dr Sayiprasad	Sayaji Grand Kolhapur
15.06.25	11.30AM	Dr Ashish Nabar	Dr Santosh Dora		
22.06.25	11:30 AM	Dr Aditya Kapoor	Dr SK Dwivedi	Dr Umeshwar Pandey and Team	
24.06.25	8:00 PM	VK Ajith Kumar	Anees Thajudeen		
29.06.25	11:30 AM	Dr SB Gupta	Dr Ketan Mehta		
30.06.25	8:00 PM	Ajith Thachil	Yet to finalise	Dr Jabir Abdullakutty	
02.07.25	8:00 PM	Arun Gopi	Girish PV		
05.07.25	8.30 PM	Dr Gurunath parale	Dr Basavraj sutar	Dr Suhas Karkamkar	Hotel Balaji Sarovar
19.07.25	8:00 PM	Dr Joy M Thomas		M Jaiganesh	Hotel GRT Grand, T Nagar
26.07.25	8.30 PM	Dr Vinesh Jain	Dr Himanshu		Hotel PL Palace
13.08.25	8:00 PM	Ajay Kumar Sinha		Ajay Kumar Sinha	
23.08.25	8.30 PM	Dr SK Pal	Dr Deepak Gupta		Hotel Le Lac Sarovar Portico
20/09/25	8.30 PM	Dr Prashant Jagtap	Dr Chetan Rathi		
TBD		Dr Smit Srivastava	Dr CK Das		
TBD		L Sathish Kumar	Dr Deepak Padmanathan		
TBD		Dr Daljit Saggu			
TBD		Dr Arun Kumar Chopra			
TBD		Dr Rajat Sharma			
TBD		Dr Aparna			
TBD		Dr Kush kumar Bhagat	Dr Jitendra Singh Makkar		
TBD		Dr Aparna	Dr Rakesh Yadav		
TBD		Dr Aditya Kapoor	Dr SK Dwivedi	Dr Ajay Pandey and Team	
TBD		Dr Shomu Vohra			
TBD		Dr Anoop Gupta			
TBD		P Shanumgasundaram			
TBD		B Ramprakash			
TBD		Dr Sadiq Azam		Dr Hygriv Rao	

INDIAN SOCIETY OF ELECTROCARDIOLOGY

APPLICATION FORM FOR LIFE MEMBERSHIP/FELLOWSHIP

SECRETARIAT
Prof. Dr. Ketan K. Mehta
Indian Society of Electrocardiology
Health Harmony, 2-Dattani Chambers, S V Road, Malad (W), Mumbai 400064
Mobile : 91-98200 51849 • e-mail : drketanmehta@yahoo.com • www.iseindia.org

Dear Sir,

I wish to become the Life Member* / Fellow** of the Indian Society of Electrocardiology. I promise to abide by the rules and regulations of the Society.

My particulars are as follows:

Name in full (Surname first) _____

Qualifications _____

University (Post-Graduate obtained) _____

Year of obtaining first Post-Graduation _____

Mailing Address _____

_____

Tel. No. Hospital _____ Clinic _____ Residence _____

Fax _____ E-Mail _____

Enclose cheque/draft for Life Membership: Rs. 4720/- or Fellowship: Rs. 7080/- towards Membership of the

Society _____ Dated _____

of _____

(Bank), drawn in favour of "Indian Society of Electrocardiology", payable at Mumbai.

Thanking you,

Yours sincerely

Signature of the Applicant

Proposed by (the Member of the Society)

Name _____

Address _____

Signature _____

FOR OFFICE USE ONLY

Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No. or Fellowship No.

_____

Hon. Secretary, ISE

RULES/REGULATIONS OF THE SOCIETY REGARDING ADMISSION OF LIFE MEMBERS/FELLOWSHIP

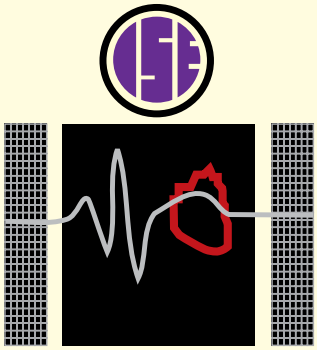
***Life Members :**

1. Person should be a Post-Graduate in Medicine/ Pediatrics/Anaesthesia/ Physiology or other allied subjects from an University recognised by Medical Council of India, with interest in Cardiology / Electrocardiology.
2. Candidates are requested to submit self-attested **Xerox** copies of the PG Certificate and Medical Council of India Registration Certificate alongwith Application Form.
3. Life Membership Fees: Rs.4,000/- + 18% GST. Total Rs. 4720/- only. In case, Life Membership is not approved by the Credential Committee, the cheque / draft will be returned.

****Fellowship:**

1. Person should be a Member of the Society.
2. He/She should be of atleast 7 years of standing after Post-Graduation.
3. He/She should have minimum 3 publications In Cardiology In Indexed Journals (Not Abstracts)
4. List of Publications to be submitted for the Fellowship.
5. Fellowship Fees: Rs.6,000/- + 18% GST. Total Rs. 7080/- only. In case, fellowship is not approved by the Credential Committee, the cheque / draft will be returned.

*Subject to approval of the Executive Body of the Society
**Subject to the approval of the Credential Committee of the Society.



SECRETARIAT
Ketan K. Mehta
HON. GENERAL-SECRETARY

Indian Society of Electrocardiology
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Mobile : +91-98200 51849 • e-mail : drketanmehta@yahoo.com • www.iseindia.org

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