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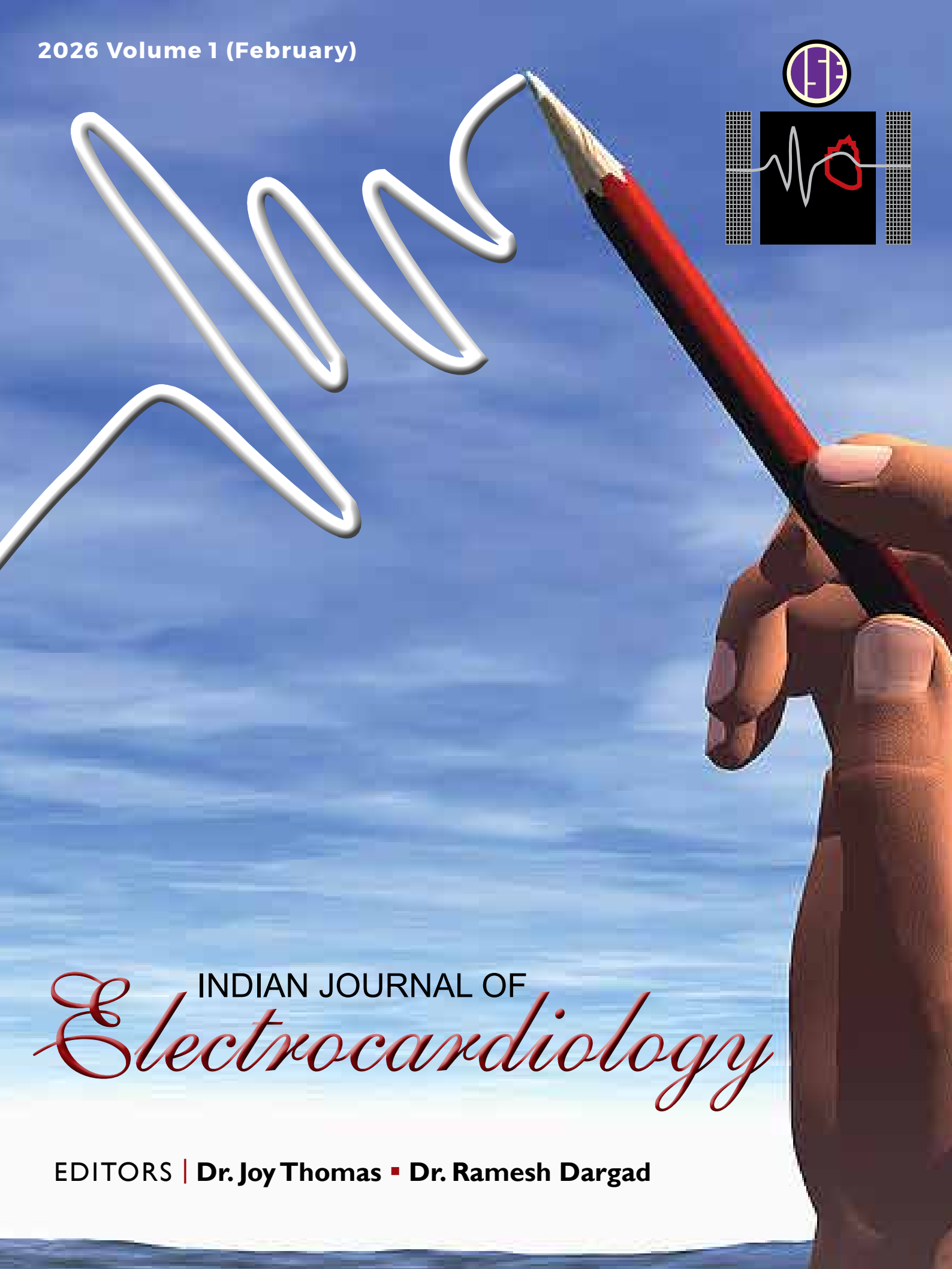

INDIAN JOURNAL OF Electrocardiology

EDITORS | Dr. Joy Thomas ▪ Dr. Ramesh Dargad

C O N T E N T S

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Editorial

Ever since I took on the responsibility of hemming the editorship with my co-editor for the Indian Journal of Electrocardiology it has been a roller coaster ride peppered with hope, hype, disappointments, regret but finally ending with satisfaction once the work is bundled up. The last few years has been overshadowed by AI (Artificial Intelligence) which has permeated our life in ways that seem to make it a daily necessity – right from the new gen cars to the washing machine- they all must have it. Our last edition had an article on AI in the electrocardiogram.

Use of conduction system pacing (CSP) in patients of heart failure who are not primarily affected by bradycardia has been debatable. This edition has discussed a few cases where the revolutionary CSP has shown very good improvement in patients with heart failure and offers food for thought in real live cases.

The commonest cause of bradycardia indicated pacemaker implantation has been for sick sinus syndrome (SSS) and the various features of this disease and its management with pacemakers has been discussed.

The “P” wave that heralds each cardiac cycle can give us information that can educate about the physical condition of the heart that can unravel the status of the valves and the rhythm of the heart. The latter is aided by studying the morphology and width of the “P” which could point which of the patients are likely to develop atrial fibrillation.

Heart transplant recipients are faced with the prospect of allograft rejection if proper antirejection medications are not adhered to, graft versus host reactions and immunocompromised infections. The ECG provides a simple non invasive way of obtaining subtle hints about these problems at follow-up.

The common problem in the ER while assessing patients with chest pain is to identify the ECG that confirms a patient who is having a myocardial infraction. Identifying De Winter’s ECG pattern identifies one.

Toxic effects come from drug overdose or from various animal or plant toxins. These have effects on the ECG that aid in diagnosing the severity and the management of these patients. We hope that these topics will help you in your clinical work

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 another issue of Indian Journal of Electrocardiology, the Official Journal of Indian Society of Electrocardiology on the eve of ISE Annual Conference to be held at Jaipur on 27th, 28th February and 1st March 2026. Dr Joy Thomas and Dr Ramesh Dargad have really worked hard to get the articles related to ECG who will be of help in the day-to-day practice for the post-graduates, physicians, cardiologists and even for the electrophysiologists.

Current issue of Indian Journal of Electrocardiology has very useful articles on the recent subject like conduction system pacing in heart failure and other common issues like sick sinus syndrome, "P" waves in ECG, ECG in cardiac transplant recipients, in common non-drug poisoning and in anemia and De Winter T waves which will enrich the knowledge of the readers.

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 February 2026 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,

As the President of the Indian Society of Electrocardiology, it is my privilege to welcome you to our esteemed journal. Electrocardiology is one of the oldest science and has come a long way, and our society has been at the forefront of promoting excellence in this field. Our journal aims to disseminate cutting-edge knowledge, share innovative research, and foster collaboration among professionals.

I am proud to lead an organization that is dedicated to advancing the understanding the art of ECG. Our journal provides a platform for experts to share their insights, experiences, and breakthroughs, ultimately benefiting patients and the medical community.

I would like to extend my gratitude to our editorial team, reviewers, and contributors for their tireless efforts. Together, we can make a significant impact in the field of electrocardiology and improve patient care.

I look forward to the journal's success and the society's continued growth.

Dr. Jitendra Makkar
President
Indian Society of Electrocardiology

Conduction System Pacing in Heart Failure – A Knight in Shining Armour!!

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Abstract

Cardiac resynchronization therapy (CRT) has become an accepted and fairly beneficial therapy in patients with wide QRS and poor left ventricular dysfunction. Recent developments of conduction system pacing has provided an alternate method of pacing to treat wide QRS cases of heart failure. This article provides an anthology of six cases where this method is effectively used to help different clinical scenarios.

Introduction

Cardiac Resynchronization therapy (CRT), involving biventricular pacing, using the right ventricular (RV) endocardial lead and quadripolar coronary sinus lead (CS), is a trial-proven contemporary therapy for patients with advanced symptomatic heart failure (HF) with left ventricular ejection fraction (LVEF) <35% and QRS width ≥130 ms, preferably with a left bundle branch block (LBBB) – type QRS morphology. Following a shared decision-making, patients with nonischemic cardiomyopathy (NICM) and multiple limiting comorbid conditions may receive CRT-pacemaker, vis-à-vis patients with ischemic cardiomyopathy (ICM) are more likely to be recommended a CRT-defibrillator. While narrowing the QRS by ensuring >95% biventricular capture may accrue responder status in patients with CRT therapy, the ultimate QRS narrowing achieved is influenced by the pathology causing the wide QRS. The latter may be due to bundle branch block, myocardial scar, hypertrophy or LV dilatation. Consequently, for different reasons, 1/3rd of patients receiving CRT are non-responders. Conduction system pacing (CSP), involving pacing the His bundle (HB) or left bundle (LB), could as well result in a narrow paced-QRS complex and prevent, reverse or treat HFrEF (HF with reduced ejection fraction). LB pacing involves drilling the lumenless or stylet-drive lead, using a proprietary delivery sheath, from the RV endocardial site and through the interventricular septum (IVS) to reach the LV subendocardium (See Figure 1).

From this site there may be a selective / non-selective capture of the LB or deep septal (myocardial) pacing, any of which may achieve a similar clinical result. Hence this method of pacing is best referred to as LB-area pacing (LBBAP). We describe exemplary cases of HFrEF where LBBAP could help to better clinical status.

Case 1: A 65-year man, hypertensive and diabetic, had recurrent presyncope with ECG showing RBBB with right QRS axis. His echocardiogram showed inferior wall motion abnormalities and LVEF 40%. His external loop recorder showed intermittent advanced AV block. Coronary

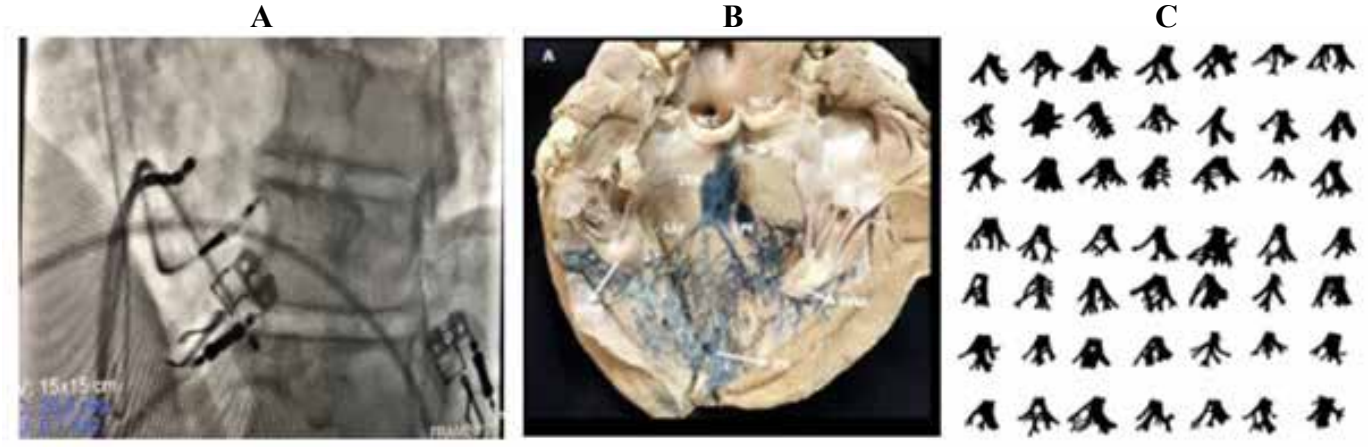

Figure 1: A. Screw- in lead is drilled with sheath-support from the RV endocardial site to reach LV subendocardium to achieve LBBAP. B. LBBAP can best achieved by pacing the trunk of LB which is anatomically below the right and non-coronary cusp junction. C. Multiple attempts at lead positioning may be required due to variations in the anatomy of the LB system.

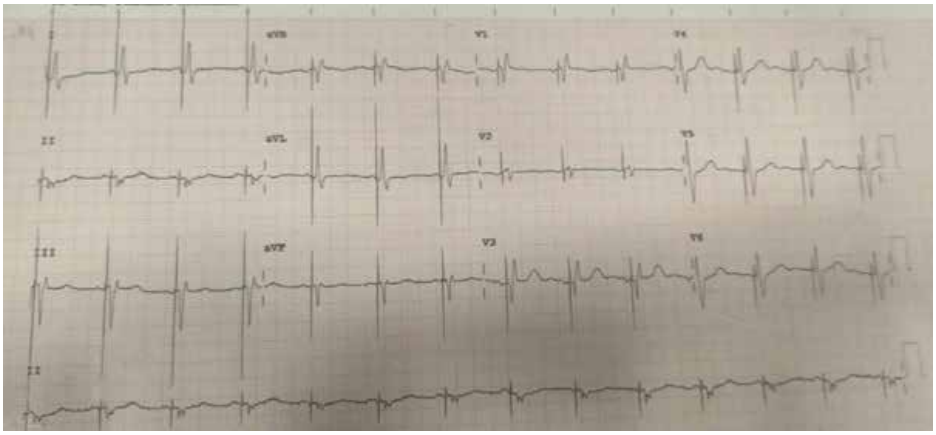

Figure 2: Patient described in case 1. Selective LBBAP was achieved with QRS width 110 ms and paced QRS morphology suggestive of left posterior fascicular capture

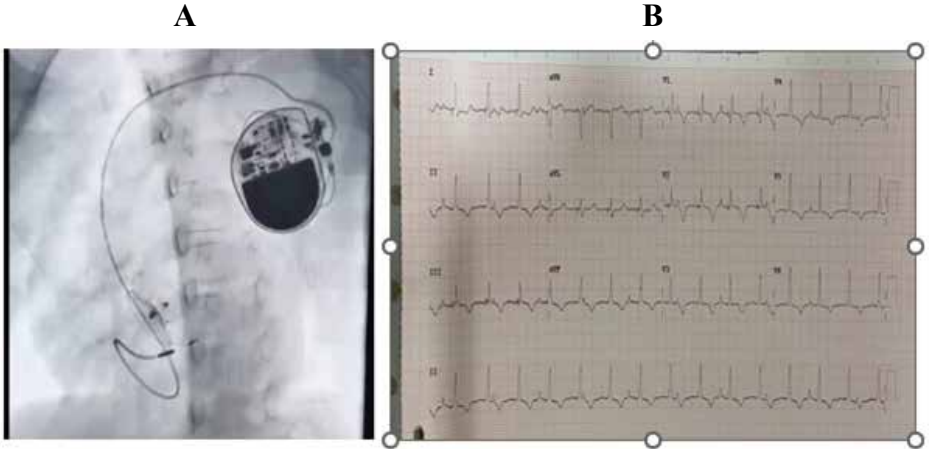

Figure 3: Patient described in case 2. A. SelectSecure 3830 fixed helix lead is used to achieve LBBAP in a child status post ASD device closure and BPV. Note the alpha loop of the lead. The ASD closure device is seen. B. Selective LBBAP was achieved. Note the qR pattern in lead V1. The paced QRS width is 100 ms and morphology (positive in all inferior leads) is suggestive of left anterior fascicular capture

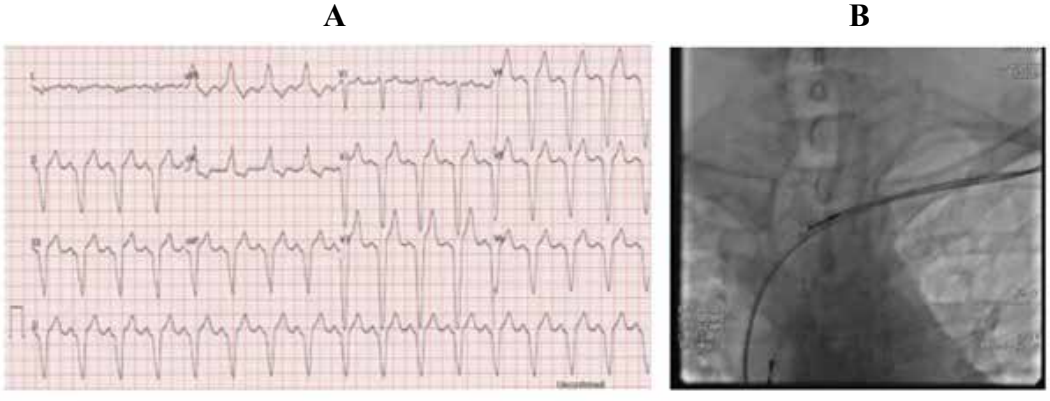

Figure 4: Patient described in case 3. A. Following a dual chamber pacemaker implant for CHB, the RV apical pacing results in a wide QRS = 160 ms. B. 3 months post implant, the RV lead was easily explanted.

angiography showed significant lesions in obtuse marginal and proximal right coronary artery. An electrophysiological study done prior to device implant revealed a prolonged HV (90 ms) and no inducible ventricular tachycardia. He was counselled for dual chamber pacemaker with LBBAP. A selective LBBAP was achieved (See Figure 2).

Comment: LBBAP in patients with mild LV systolic dysfunction requiring bradypacing may prevent further

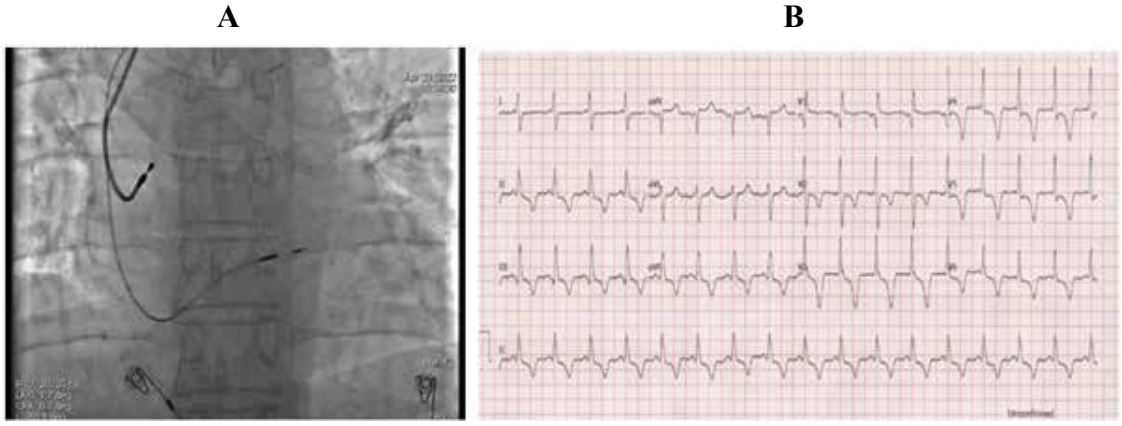

Figure 5: Patient described in case 3. A. Note that the dual chamber pacemaker is now done by implanting the RV lead using LBBAP technique. B. 12-lead QRS morphology confirms LBBAP, the QRS width is 100 ms

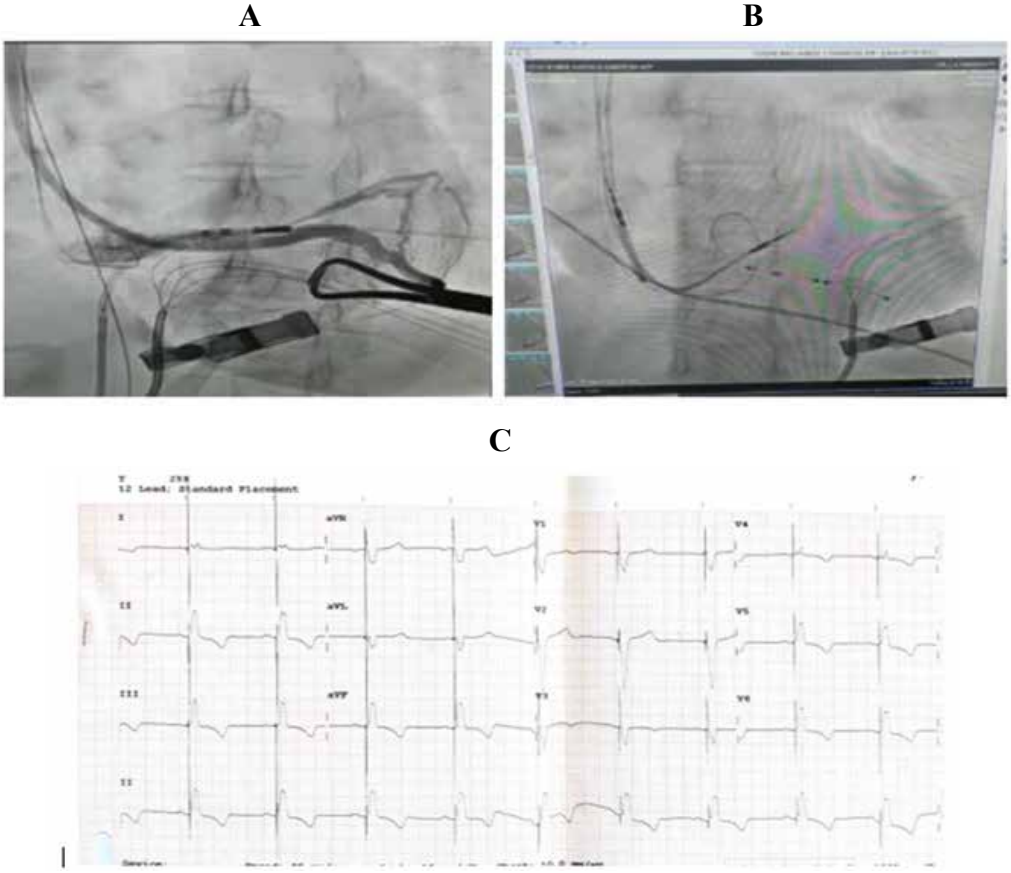

Figure 6: A. Note the large and roomy posterolateral CS branch. B. The quadripolar CS lead, despite introducing deep into the vein, would repeatedly slip out while withdrawing the sheath. C. Deep septal pacing was achieved

deterioration of LVEF by providing electrical synchrony (narrow paced QRS).

Case 2: A 10 year-old child who had previously undergone balloon pulmonary valvotomy (BPV) followed by atrial septal defect (ASD) closure has a congenital complete heart block (CHB). We are often restricted to single chamber VVI pacing due to both, short-term and long-term considerations, associated with dual chamber pacing in pediatric population. Single chamber LBBAP could possibly prevent pacing-induced cardiomyopathy (PICM) by avoiding VV dyssynchrony. See figure 3.

Comment: The 4.1F lumenless-lead 3830 (Medtronic Inc) is more suited for a long-term freedom from lead malfunction and the lead length (≥59 cm) allows an alpha-loop. It is FDA approved for LBBAP.

Case 3: A 56 year-old man with diabetes and hypertension underwent dual chamber pacemaker for CHB in June 2021. Active fixation lead was placed at RV apex. 3 months later he presented with new LV systolic dysfunction. Coronary angiogram showed nonincriminatory single-vessel circumflex stenosis. PICM was diagnosed and RV lead explanted followed by LBBAP (see Figures 4 and 5). 3 months later the LVEF had recovered.

Comment: RV apical pacing causing PICM is under-recognized unless symptomatic and with significant reduction in LVEF. LBBAP for every bradypacing indication, are we there yet? Await a randomized trial.

Case 4: A 43 year-old lady presented with HF. She had sinus rhythm with LBBB-type QRS with QRS width = 150 ms. Echocardiogram suggested NICM with LVEF 25% and a moderate mitral regurgitation. She was planned for a conventional CRT. Quadripolar CS lead was easily introduced in the posterolateral CS branch. However, the CS lead was displaced 5 times due to the wide caliber of the CS branch. We switched to LBBAP as a bail out strategy. A deep septal pacing was achieved; however, the NYHA class, LVEF and MR grade improved in the early follow up. See figure 6.

Comment: Planning the conventional CRT procedure should include the possibility to switch to LBBAP, if required. Suitable hardware should be kept standby even if conventional CRT is primarily planned.

Case 5: A 63 year-old gentleman had HF NYHA III with NICM (LVEF 30%) and ECG with LBBB-type QRS. He was planned for CRT-D implantation. Despite easy CS cannulation with a CRD-1 catheter, the CPS 115^0 CS sheath dissected the CS endocardium and the previously visible target branch was no longer seen to enable passing of the guidewire. After ruling out pericardial effusion, the plan was changed to do LBBAP. This was achieved using the Agilis HisPro steerable sheath and the stylet driven Tendril STS Model 2088TC lead (Abbott Inc) (see figure 7). DF4 lead and RA were implanted as usual. All 3 leads connected to the CRT-D device with bipolar LV port. Patient was responder at the intermediate 6-month follow up.

Comment: CS dissection is a rare complication during implantation of the LV lead. If the dissection involves the ostium of the target CS branch, it would no longer be possible to pass the guide wire into this branch. Instead of abandoning the procedure to a later date, LBBAP can be done for resynchronization. LBBAP can be considered even for non-LBBB wide QRS.

Case 6: A 51 year-old lady with NICM and LBBB QRS with NYHA III HF was planned to receive a CRT-P implantation. A quadripolar CS lead was implanted into the posterolateral vein and demonstrated acceptable thresholds without diaphragmatic pacing through all the tested bipoles. RV endocardial lead was implanted in mid-septal site. The best biventricular paced QRS width was 140 ms and deemed unacceptable for a future

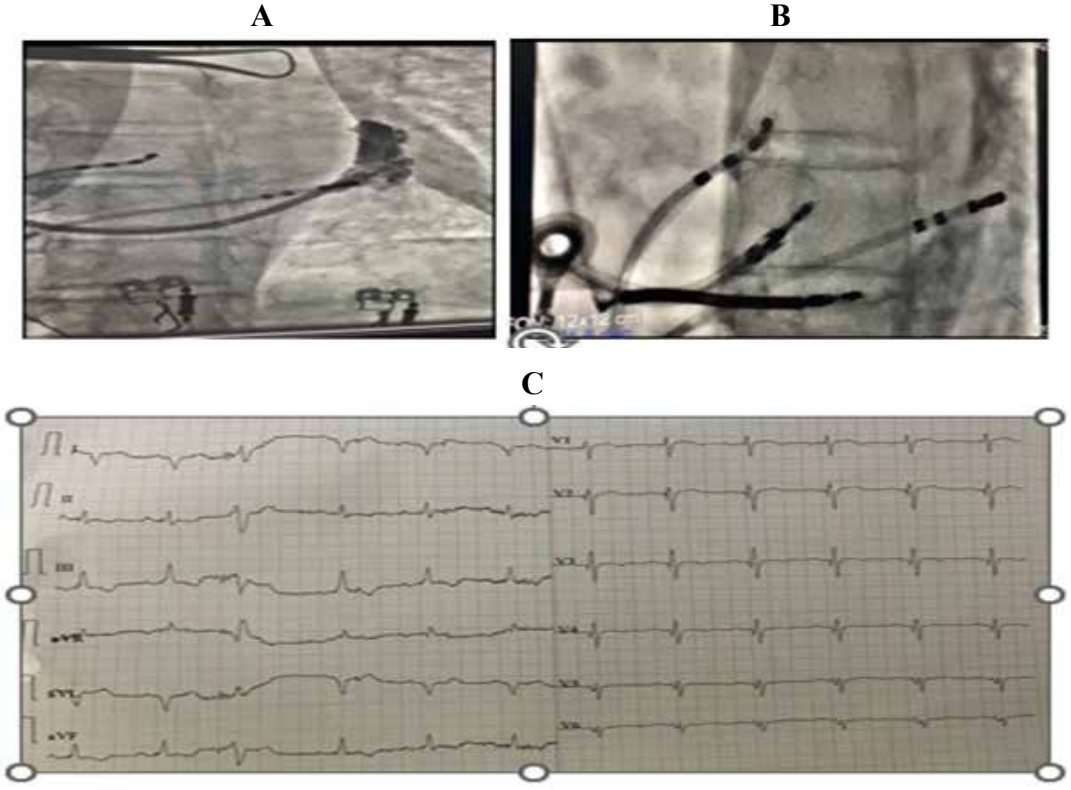

Figure 7: A. CS dissection occurred while introducing the sheath over the CRD-1 catheter, following which the previously noted target branch was occluded at the ostium by the dissection flap and no longer visible. B. RV pacing lead was implanted by LBBAP technique. C. Electrical synchrony was achieved, note that the paced QRS width was <120 ms.

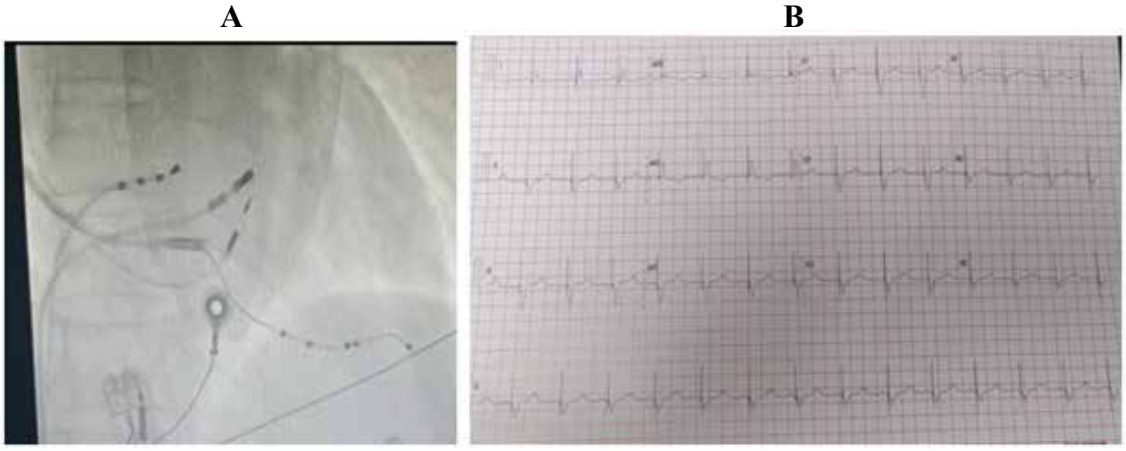

Figure 8. A. Shows biventricular pacing achieved by RV pacing through LBBAP lead and LV pacing through quadripolar CS lead. B. Note the narrowest biventricular paced QRS = 100 ms.

responder status. Hence it was decided to implant the RV lead by LBBAP technique. The biventricular pacing, combining LBBAP (bipolar) with LV3-4 bipoles of the quadripolar lead resulted in the narrowest QRS = 100 ms. See Figure 8.

Comment: Achieving narrowest biventricular paced QRS is considered equivalent to reversing electrical dyssynchrony and equated with improvement in mechanical synchrony with subsequent responder status. Every attempt should be made during the implant to achieve the narrowest QRS. In our case "Cardiac Physiologic Pacing" was obtained by LBBAP-LV (CS) pacing. This is referred to as LB optimized CRT (LOT-CRT).

The advantages of LBBAP over HBP are that a larger target area is available to position the lead with correction of distal conduction disease, low capture thresholds and good sensing parameters and a consistent back-up myocardial capture is possible. The biggest worry is that these leads are not primarily designed for penetration into the myocardium and whether they disrupt at the insertion site in the long-term? Although in HF, conventional CRT is preferred, LBBAP has been given a class IIA indication. For bradypacing, LBBAP may be selectively used.

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Sick Sinus Syndrome

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Abstract

Sick sinus syndrome (SSS) is a syndrome characterised by various manifestations resulting from impaired impulse generation and/or propagation from the sinoatrial (SA) node (sinus node dysfunction).[1] Patients with sick sinus syndrome can present with both brady and tachyarrhythmia. In this article, we present a brief clinical case scenario followed by a focused discussion on the pathophysiology, clinical and ECG manifestations, and management of sick sinus syndrome.

Case Scenario

A 48-year-old lady presented with paroxysmal palpitation and recurrent episodes of presyncope for 3 years duration. Her symptoms were unrelated to exertion. She had no other comorbidities. ECG taken at the time of presentation showed sinus arrhythmia. Echocardiography done showed normal LV systolic function with biatrial enlargement. Holter showed episodes of atrial fibrillation terminating in long pauses with maximum duration of 6.3 seconds resulting in presyncope and most of these episodes occurred during wake hours. Hence, she was diagnosed with tachy-brady syndrome. She underwent permanent pacemaker implantation in view of bradycardia (termination pause of 6.3 seconds) and was treated with betablockers for tachycardia (atrial fibrillation). She was started on oral anticoagulation based on CHA_2DS_2VASc score. In view of significant biatrial enlargement, amyloidosis workup was done which was negative. She is doing well on regular follow-up.

Introduction

The sinoatrial (SA) node serves as the primary pacemaker of the heart, ensuring appropriate heart rate modulation in response to physiological demands. Dysfunction of SA node and/or the surrounding atrial myocardium can lead to a constellation of rhythm disturbances collectively termed sick sinus syndrome (SSS). First described in the mid-20th century, SSS has since been recognized as an increasingly prevalent condition due to enhanced survival of the aging population. This condition carries significant morbidity due to palpitations, syncope, falls, heart failure, and thromboembolic complications related to atrial fibrillation.

Sick sinus syndrome is primarily a disorder of older adults. The prevalence is estimated at approximately 1 in 600 individuals over the age of 60 years, with a median age at diagnosis of around 75 years. Both sexes are affected equally. Although predominantly seen in the elderly, SSS can occur in younger

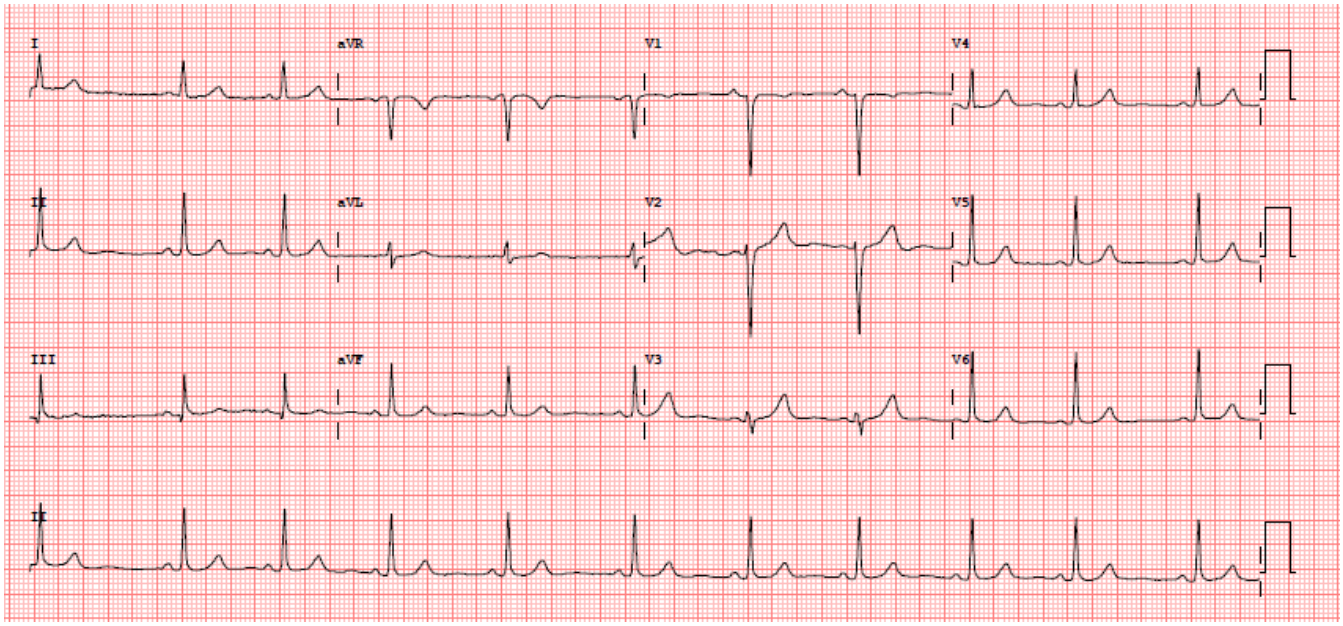

Figure 1: ECG at presentation

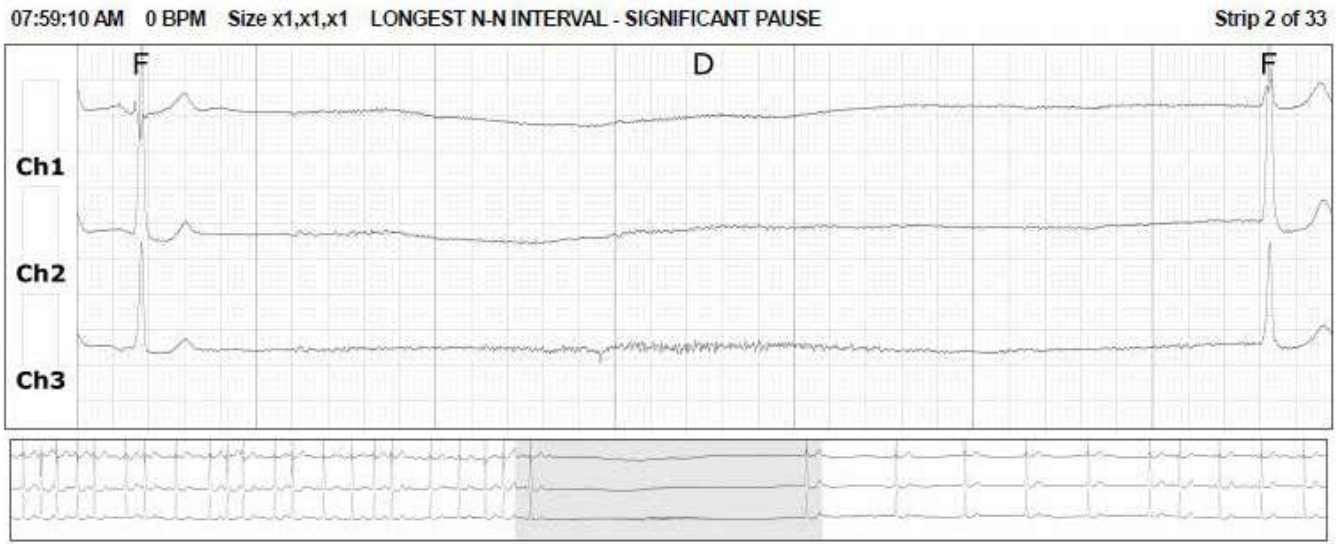

Figure 2: Holter tracing showing atrial fibrillation terminating in a long pause

patients, particularly those with a history of congenital heart disease surgery, infiltrative myocardial disorders, or genetic causes.

Pathophysiology

The most common underlying mechanism in elderly patients is age-related degenerative interstitial fibrosis of the SA node and surrounding atrial myocardium. This fibrotic remodelling disrupts impulse generation and conduction, leading to reduced automaticity and impaired sinoatrial conduction, respectively.[2] Aging is associated with a reduction in intrinsic heart rate, prolongation of sinoatrial conduction time and structural and molecular remodelling of atrial tissue. These changes contribute to the heterogeneous clinical expression of SND. Chronic ischemia is also proposed to be one of the factors in SA node dysfunction.

In younger individuals, SSS may result from direct injury to the SA node following cardiac surgery for congenital heart disease, genetic variants in the genes such as HCN4, SCN5a and idiopathic atrial myopathy. Several infiltrative and inflammatory conditions are associated with SSS, including sarcoidosis, hemochromatosis, connective tissue disorders, myocarditis, cardiomyopathies, and neoplastic infiltration.

Aetiology

The aetiology of SSS can be classified as primary or secondary. Primary causes are degenerative fibrosis of the SA node, genetic or inherited channelopathies and infiltrative atrial myopathies. Secondary (potentially reversible) causes are hypothyroidism, intracranial hypertension, electrolyte disturbances and drug-induced (beta-blockers, calcium channel blockers, antiarrhythmics). Identification and correction of reversible causes are mandatory before establishing a diagnosis of intrinsic SND.

Clinical Manifestations

The clinical presentation of SSS is variable and depends on the severity of bradycardia, the presence of pauses, and associated atrial tachyarrhythmias. Symptoms such as fatigue, exercise intolerance, giddiness, presyncope, syncope, or dyspnoea are due to reduced cardiac output leading to insufficient blood supply to the brain and other body parts. Few patients may complain of palpitations which may be due to tachyarrhythmias such as atrial tachycardia or atrial fibrillation or forceful heartbeat following a pause. Few patients may remain asymptomatic, with SND detected incidentally during routine ECG or ambulatory monitoring.

Electrocardiographic Features

Electrocardiography remains central to the diagnosis of SSS, although findings are often intermittent or paroxysmal in nature. Manifestations include one or more of the following:

1. Persistent, inappropriate sinus bradycardia, particularly when not explained by medications or athletic conditioning.
2. Sinus arrest or sinus pause, characterized by pauses of 3 seconds or more without P-wave activity. The pause can be terminated by sinus beat or an escape beat which may be atrial or junctional in origin.
3. Atrial fibrillation:
    - Long standing persistent AF due to complete SA node failure.
    - Paroxysmal AF caused by intermittent sinus arrest. These episodes are often associated with slow ventricular response due to coexisting atrioventricular (AV) nodal disease (binodal disease).

4. Failure to resume sinus rhythm after cardioversion for atrial fibrillation, manifesting as prolonged sinus pause.
5. Sinoatrial exit block, unrelated to drug therapy.
6. Tachy-brady syndrome – episodes of sinus bradycardia alternating with atrial tachyarrhythmia predominantly AF.
7. Chronotropic incompetence - defined as inappropriate bradycardia resulting in an inability to meet the metabolic demands.

Among these, tachy–brady syndrome, characterized by alternating atrial tachyarrhythmias and profound bradycardia, is particularly common and clinically significant.

When to Suspect Sick Sinus Syndrome

The presence of sinus bradycardia in symptomatic patient should prompt evaluation for SSS. A critical concept is that not all bradycardia is benign. Bradycardia due to SSS is typically present during both daytime and nighttime, observed in the absence of rate-limiting medications and if it is associated with minimal heart rate variability during physical activity, reflecting chronotropic incompetence.

Natural history

The sick sinus syndrome in its chronic form runs an erratic course with periods of normal sinus node function alternating with abnormal behaviour. The sinus node function gradually deteriorates over 5 -10 years. The escape rhythms, at first only periodic rescuers, eventually become the basic rhythm, particularly atrial fibrillation. The onset of atrial fibrillation may lead to an apparent improvement or disappearance of bradycardia-related symptoms in patients with sick sinus syndrome. This phenomenon has historically been described as "sick sinus syndrome being cured by atrial fibrillation." However, this is a functional rather than the true resolution of sinus node disease.

Diagnostic Evaluation

Reversible causes for sinus node dysfunction should be excluded before making a diagnosis of sinus node dysfunction. This includes ruling out electrolyte disturbances, endocrine disorders, and drugs causing bradycardia. Diagnosis relies on correlating rhythm abnormalities with patient's symptoms. 12 lead ECG assessment is often inadequate in view of episodic nature of the disease. Prolonged monitoring of the patient's rhythm is required in the form of Holter monitoring which may be 24-hours, 3 days, 7 days or extended monitoring depending on the frequency of symptoms. Implantable loop recorders (ILR) may be required if symptoms are infrequent and extended Holter monitoring did not show any abnormalities. Exercise ECG may be required to look for chronotropic competence.

Management

General principles

Permanent Pacemaker Therapy

Permanent pacemaker implantation is the definitive treatment for symptomatic SSS. Pacemaker is indicated for patients with documented symptomatic bradycardia, symptomatic frequent sinus pauses, symptomatic sinus bradycardia caused by medications required for other medical conditions, and symptomatic tachy-brady syndrome in order to correct bradyarrhythmias and enable pharmacological treatment for tachycardia. Pacing is considered for symptomatic patients with documented heart rate <40 bpm without clear symptom–rhythm correlation and unexplained syncope with SA node dysfunction demonstrated during electrophysiological studies. It is important to remember that pacemaker therapy effectively alleviates symptoms but does not halt disease progression or prevent atrial arrhythmias.

Special Considerations

- In patients with tachy–brady syndrome and atrial fibrillation, anticoagulation should be initiated based on the CHA_2DS_2-VASc score, irrespective of pacemaker implantation.
- In pacemaker recipients with SSS, continuous surveillance for atrial fibrillation is crucial. Early detection and appropriate anticoagulation significantly reduce the risk of thromboembolic complications.
- Patients with longstanding persistent AF often have latent or overt SND that is masked by the arrhythmia.

Conclusion

Sick sinus syndrome is a common yet frequently underrecognized cause of bradyarrhythmia, particularly in the elderly. Its clinical presentation is diverse, and diagnosis requires careful identification and correlation of intermittent ECG findings with patient symptoms while excluding reversible causes. Permanent pacemaker implantation remains the cornerstone of management in symptomatic individuals. Importantly, clinicians must recognize that not all bradycardia is benign and maintain a high index of suspicion for SSS to prevent morbidity related to syncope and thromboembolism.

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"P" waves on ECG

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Abstract

The P wave represents atrial depolarization and is the first electrical event recorded on the surface electrocardiogram (ECG). Despite its small amplitude, the P wave provides crucial information regarding atrial size, conduction, rhythm, and the presence of atrial cardiomyopathy. In routine clinical practice, P-wave analysis is often cursory, leading to missed diagnostic and prognostic opportunities. This teaching review revisits the electrophysiological basis, normal characteristics, and clinically relevant abnormalities of the P wave, with emphasis on practical interpretation, common pitfalls, and its role in predicting atrial arrhythmias and stroke.[1,2]

Introduction

The surface ECG remains a cornerstone of cardiovascular evaluation in India because of its availability, affordability, and diagnostic value. While considerable attention is devoted to QRS complexes and ST–T changes, the P wave—representing atrial depolarization—often receives inadequate scrutiny. Classical electrocardiographic texts have long emphasized the diagnostic importance of the P wave, particularly in atrial enlargement and rhythm disorders.[1,2]

A systematic understanding of P-wave physiology and morphology can enhance diagnostic accuracy, guide further investigations, and assist in risk stratification for atrial arrhythmias and thromboembolic events.[5,8]

Electrophysiological Basis of the P Wave

The P wave reflects depolarization of the atrial myocardium. The electrical impulse originates in the sinoatrial (SA) node, located in the high right atrium. Depolarization spreads through the right atrium and then to the left atrium primarily via Bachmann's bundle, with additional interatrial connections near the fossa ovalis and coronary sinus.[1,4] (Figure 1)

Because atrial myocardium has less mass and slower

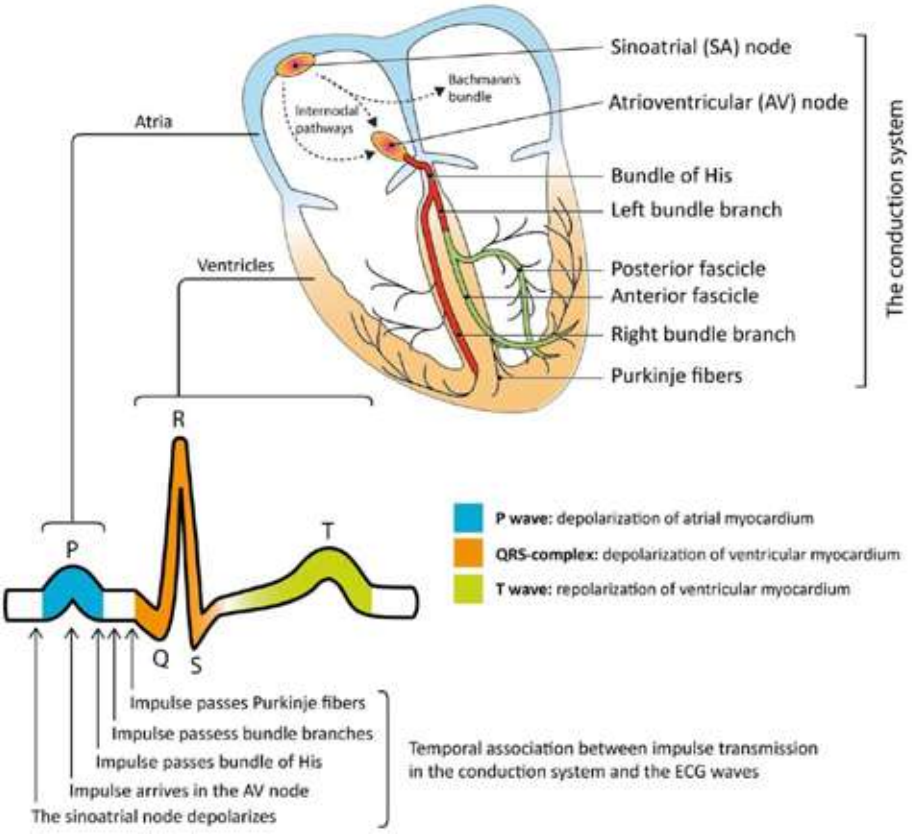

Figure 1: The temporal sequence of Depolarisation spreading from the sinus node, colour coded with corresponding time of transmission in the conduction system and chambers

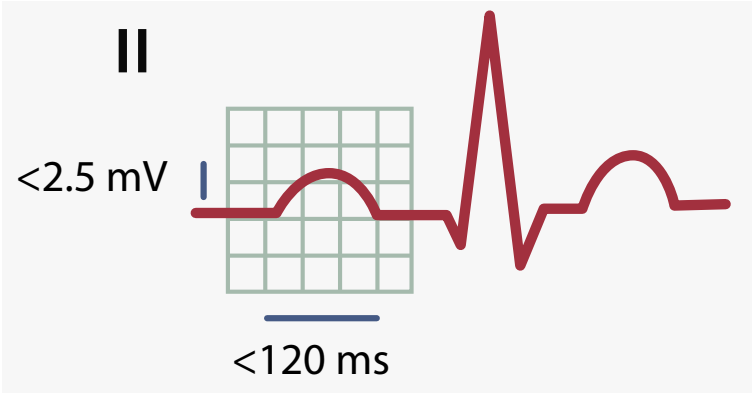

Figure 2: The Normal P wave

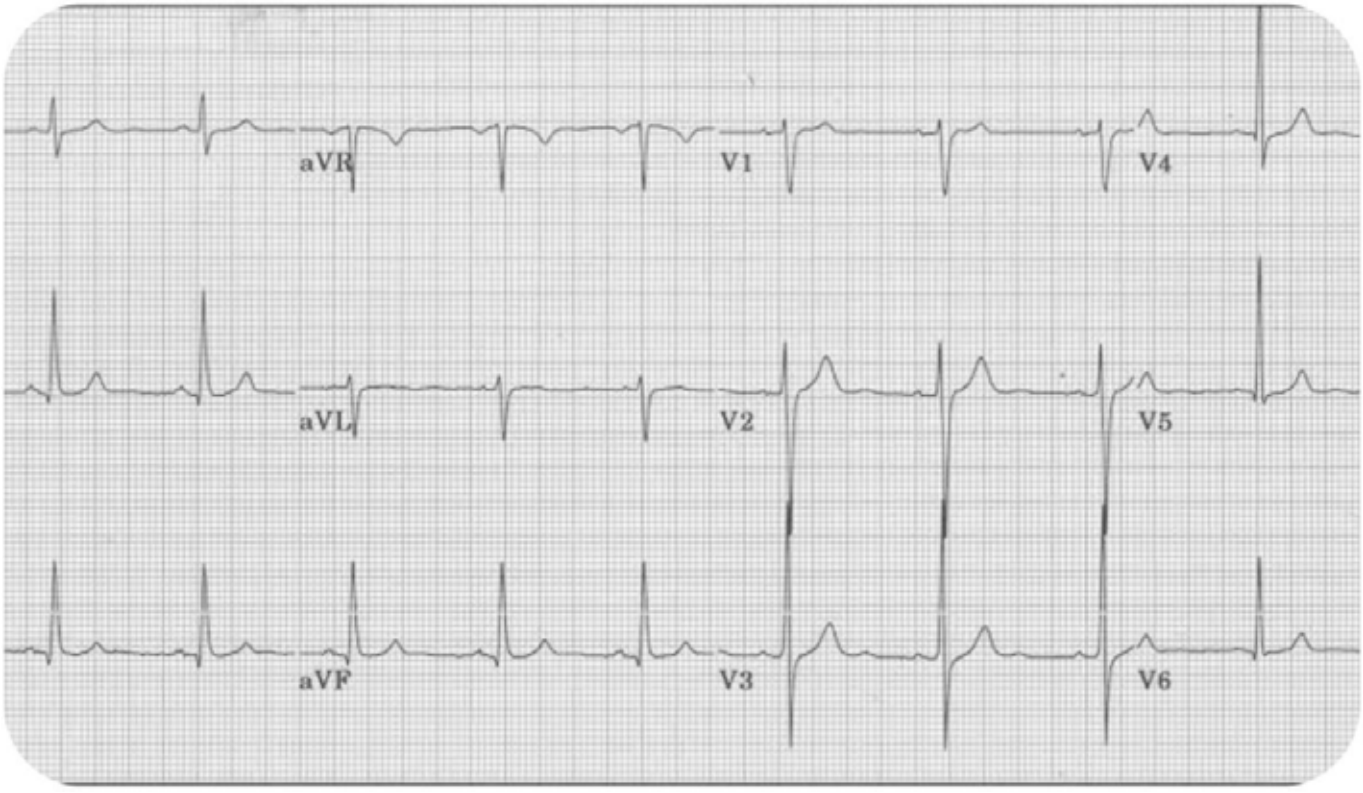

Figure 3: The normal ECG with normal P waves

conduction compared with ventricles, the resulting electrical signal is of low amplitude and longer duration. The surface P wave therefore represents the summation of right and left atrial activation.[1,2]

Normal P-Wave Characteristics

In sinus rhythm, each P wave precedes a QRS complex with a constant PR interval.

Normal parameters in adults include:

- Duration ≤120 ms
- Amplitude ≤2.5 mm in limb leads
- Axis between 0° and +75°

The P wave is upright in leads I, II, and aVF, inverted in aVR, and often biphasic in V1. These features reflect sequential right and left atrial depolarization.[2,6] (Figures 2,3,4)

P-Wave Abnormalities and Atrial Enlargement (Figure 4)

Right Atrial Abnormality (RAE)

Right atrial enlargement increases early atrial forces, producing tall, peaked P waves (>2.5 mm) in inferior leads with relatively normal duration. This classical pattern, historically termed P pulmonale, is described in pulmonary hypertension, chronic

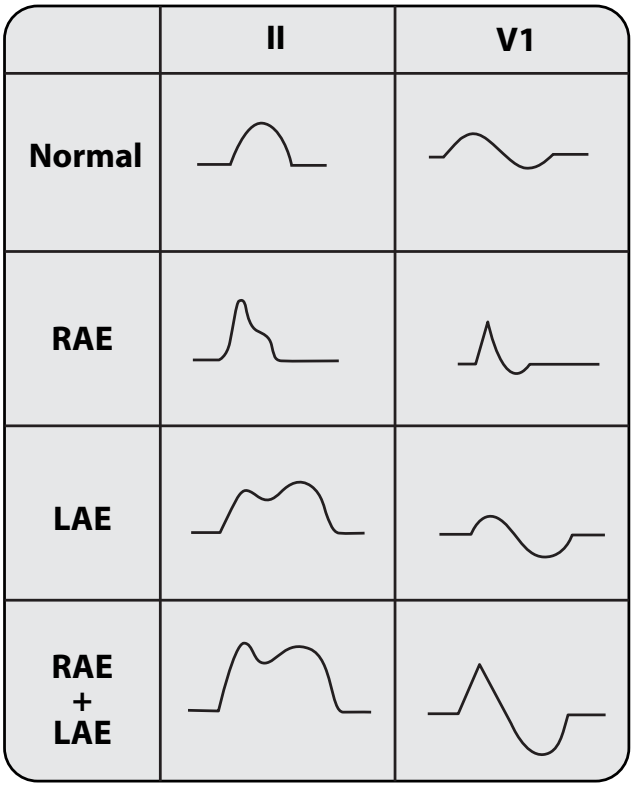

Figure 4: Morphology of the P wave in various chamber enlargement

lung disease, and tricuspid valve pathology.[1,7] (Figures 2 and 5)

Left Atrial Abnormality

Left atrial enlargement primarily prolongs atrial depolarization.

ECG features include:

- P-wave duration >120 ms
- Notched or bifid P wave in lead II
- Prominent negative terminal P wave in V1

These changes, collectively referred to as P mitrale, are seen in mitral valve disease, systemic hypertension, and cardiomyopathies.[1,2,7] (Figures 3,6)

Biatrial Abnormality

Biatrial enlargement manifests as P waves that are both tall and prolonged, often combining features of right and left atrial abnormality.[2] (Figures 5, 6)

Interatrial Conduction Delay and Block

Interatrial conduction abnormalities result from delayed or blocked conduction between the atria. Advanced interatrial block (Bayés syndrome) is characterized by prolonged

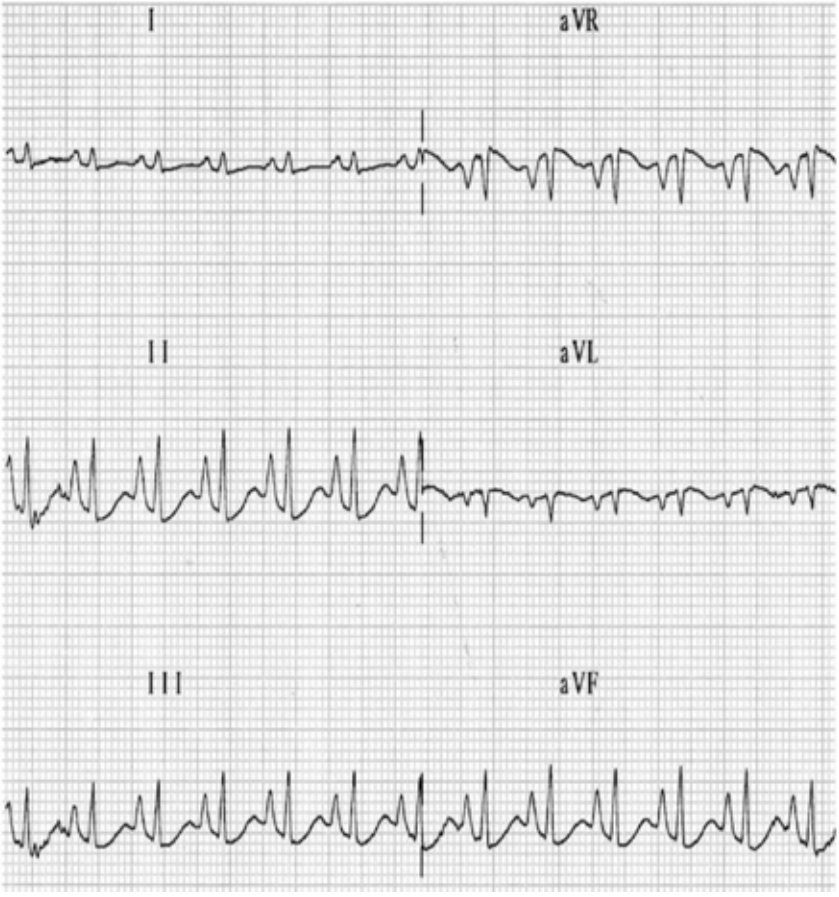

Figure 5: Right Atrial enlargement

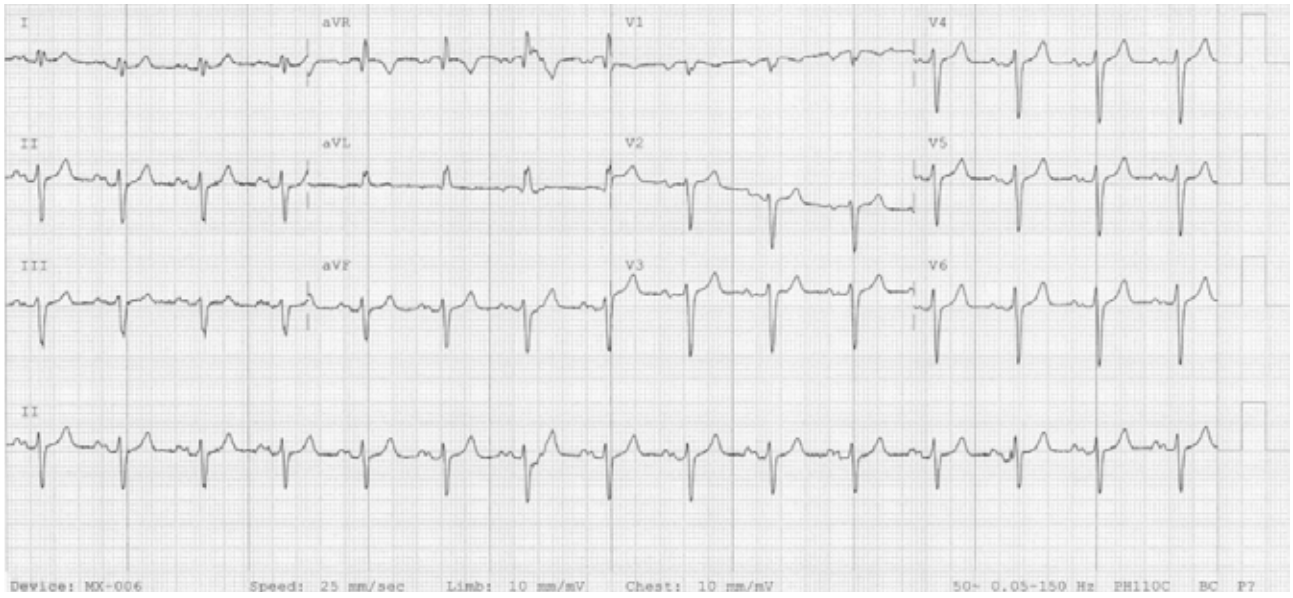

Figure 6: Left atrial Enlargement

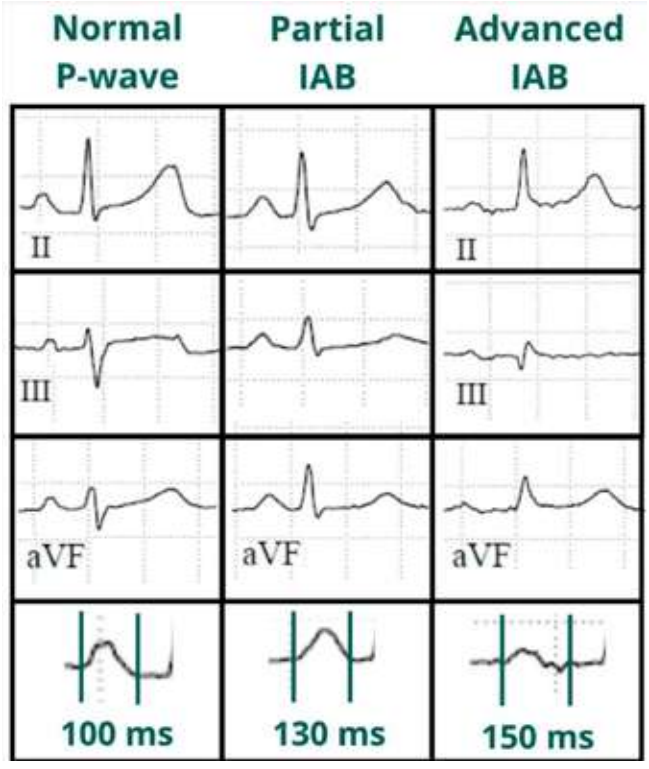

Figure 7: The P wave in varying conditions of intra atrial conduction block (IAB)

P-wave duration (>120 ms) with biphasic (positive–negative) morphology in inferior leads. (Figure 7)

This entity has been shown to correlate strongly with atrial fibrillation, ischemic stroke, and atrial cardiomyopathy.[3]

P Wave in Atrial Arrhythmias

P-wave analysis is essential in diagnosing atrial arrhythmias.

- Atrial fibrillation: Absence of discrete P waves
- Atrial flutter: Regular flutter waves with saw-tooth pattern
- Ectopic atrial rhythm: Altered P-wave morphology and axis
- Multifocal atrial tachycardia: Three or more distinct P-wave morphologies

Standard ECG interpretation guidelines emphasize careful P-wave analysis in rhythm evaluation.[6]

Advanced P-Wave Indices and Prognostic Implications

Quantitative P-wave indices such as P-wave dispersion, P-terminal force in V1, and signal-averaged P waves reflect atrial conduction heterogeneity. Increased P-wave dispersion and abnormal terminal force have been associated with incident atrial fibrillation and stroke risk.[5,8]

These findings support the concept of atrial cardiomyopathy as an underlying substrate for thromboembolism, even in the absence of documented atrial fibrillation.[8]

Clinical Relevance in the Indian Context

In India, rheumatic heart disease, poorly controlled hypertension, and delayed presentation of structural heart disease remain prevalent. Early detection of atrial abnormalities through systematic P-wave analysis can prompt timely echocardiography, rhythm monitoring, and preventive strategies.[1,2,8]

Greater emphasis on P-wave interpretation during undergraduate and postgraduate medical training can significantly improve bedside ECG skills.

Conclusion

The P wave is a valuable yet underutilized component of the ECG. Careful evaluation of its morphology, duration,

amplitude, and axis provides insights into atrial size, conduction, and rhythm disorders. Even in the era of advanced imaging, meticulous P-wave analysis remains a simple, inexpensive, and powerful clinical tool.[1,2,5]

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ECG in Cardiac Transplant Recipients

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Abstract

Heart transplantation remains the most effective treatment in patients of end stage heart failure. The ECG is a simple non invasive means to identify quite early the gathering problem of inflammation and possible rejection and provides a simple tool for follow-up.

Introduction

Orthotopic heart transplantation remains the definitive therapy for selected patients with end-stage heart failure. Advances in surgical technique, immunosuppression, and post-transplant surveillance have significantly improved long-term outcomes. Despite the availability of advanced imaging and invasive diagnostics, the 12-lead ECG remains one of the most accessible and frequently used investigations in transplant follow-up.

ECG interpretation in heart transplant recipients differs fundamentally from that in non-transplanted patients. Surgical anastomoses alter atrial anatomy and conduction pathways, while cardiac denervation modifies heart rate control and ischaemic symptomatology. Consequently, ECG findings that would typically be considered abnormal may represent expected post-transplant physiology, whereas subtle new changes may indicate serious pathology. This review aims to provide a structured approach to ECG interpretation in cardiac transplant recipients, focusing on the influence of surgical technique and the identification of complications.

Biatrial Anastomosis and Dual Sinus Node Physiology

The biatrial anastomosis technique, historically the most common approach, involves retaining cuffs of the recipient's right and left atria, including the native sinus node, which are sutured to the donor atria. Although this approach simplifies implantation, it results in enlarged atrial chambers and discontinuity between donor and recipient atrial myocardium.

Electrophysiologically, the recipient sinus node may continue to generate electrical impulses, but these impulses are electrically isolated from the donor atrium and ventricles.

Table 1: Surgical Techniques and Their Electrocardiographic Implications

Feature	Bi-atrial anastomosis	Bi-caval anastomosis
Sinus node activity	Dual (recipient + donor)	Single (donor only)
P wave morphology	Two P wave morphologies possible	Single uniform P wave
AV conduction	Only donor atrium conducts	Normal donor AV conduction
Atrial size	Enlarged atrial remnants	Near normal atrial geometry
Atrial arrhythmias	More frequent	Less frequent
ECG interpretation	More complex	More straightforward

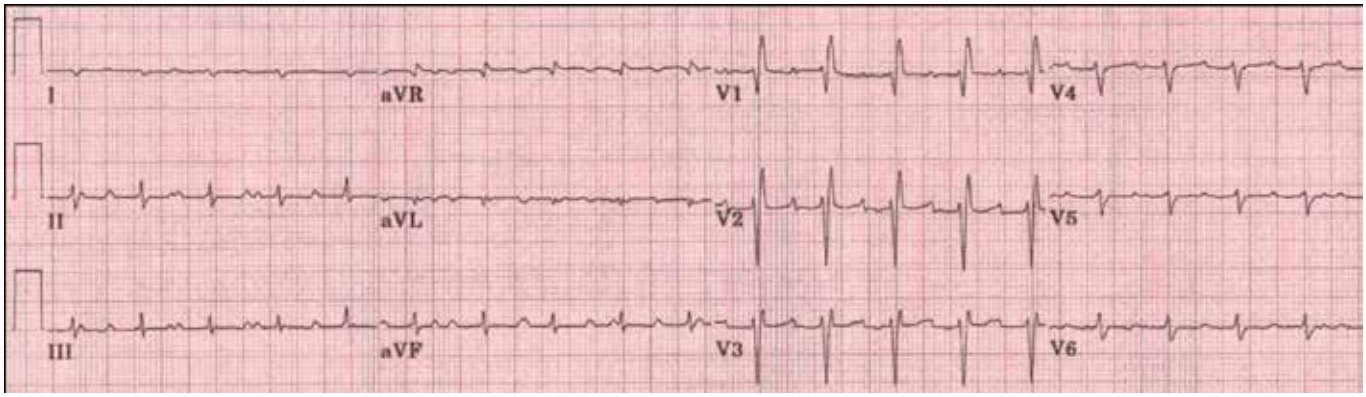

Figure 1: ECG demonstrating two distinctive P waves post biatrial cardiac transplant.

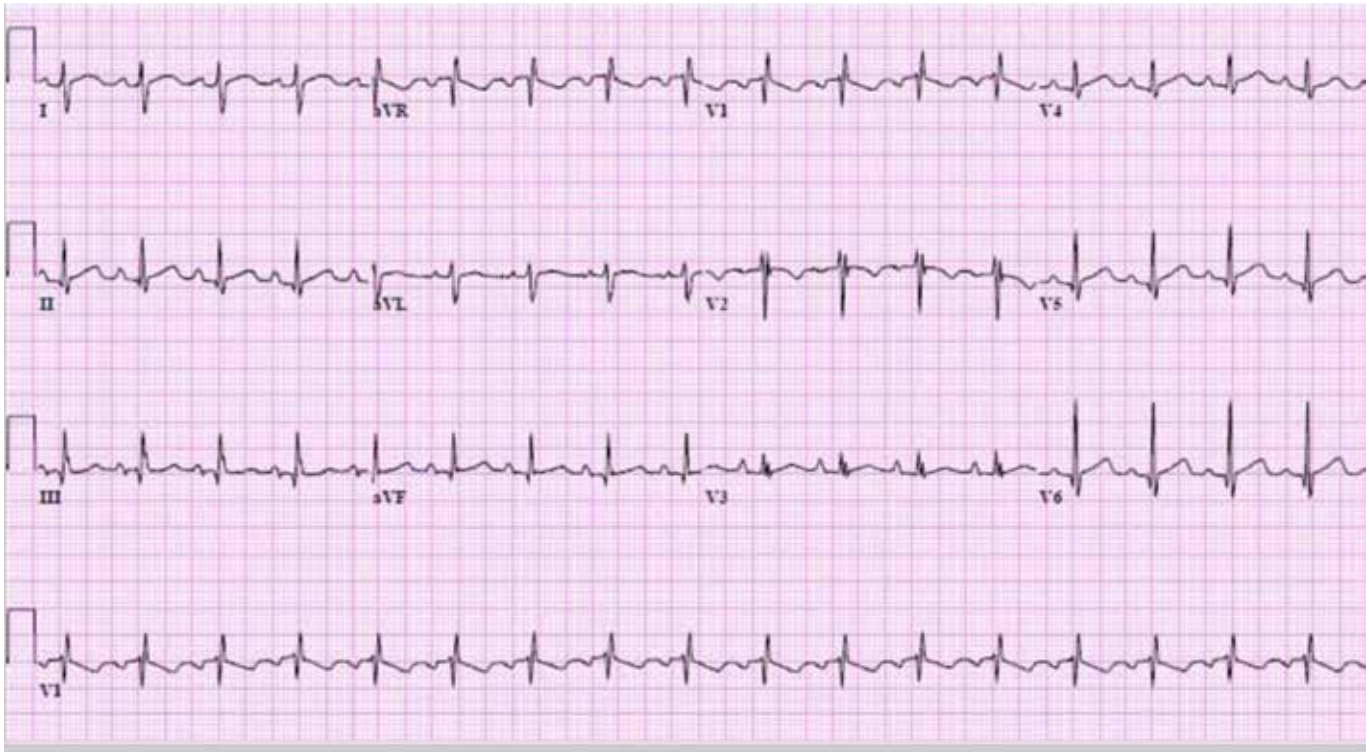

Figure 2: ECG showing single consistent P wave morphology with stable AV conduction *(Pickham D, Hickey K, Doering L, Chen B, Castillo C, Drew BJ. Electrocardiographic abnormalities in the first year after heart transplantation. J Electrocardiol. 2014 Mar-Apr;47(2):135-9. doi: 10.1016/j.jelectrocard.2013.09.006. Epub 2013 Oct 10. PMID: 24119878; PMCID: PMC3951586.)*

ECG characteristics include:

- Two distinct P-wave morphologies
- Recipient P waves that are not followed by QRS complexes
- Donor P waves with consistent atrioventricular (AV) conduction

This phenomenon is referred to as atrial electrical dissociation and must be distinguished from atrial arrhythmias or AV block.

Bicaval Anastomosis and Single Sinus Node Physiology

The bicaval technique preserves the donor right atrium and sinus node while directly anastomosing the superior and inferior vena cavae. This approach maintains more normal atrial geometry and conduction.

ECG features typically include:

- A single P-wave morphology
- Stable sinus rhythm originating from the donor sinus node
- Lower incidence of atrial arrhythmias

Because of these advantages, the bicaval technique has become the preferred surgical approach in many transplant centres.

Expected ECG Findings After Cardiac Transplantation

Several ECG findings are commonly observed in stable heart transplant recipients and reflect denervation, surgical manipulation, or medication effects.

Sinus Tachycardia and Reduced Heart Rate Variability

Loss of parasympathetic innervation results in a resting heart rate typically between 90 and 110 beats per minute, with diminished circadian and exercise-related variability.

Conduction Abnormalities

Right bundle branch block, complete and incomplete, seen in 83% of patients, left anterior hemi-block, seen in 25% patients, and nonspecific intra-ventricular conduction delay are frequently observed, often related to surgical trauma or altered ventricular geometry.

Repolarization Abnormalities

Nonspecific ST-segment and T-wave changes are common and often stable over time. Some patients may develop diffuse ST-segment elevation followed by evolutionary changes of the ST segment and T wave changes consistent with acute pericarditis in the early post-operative period. Mild QT prolongation may occur due to immunosuppressive or antiarrhythmic medications.

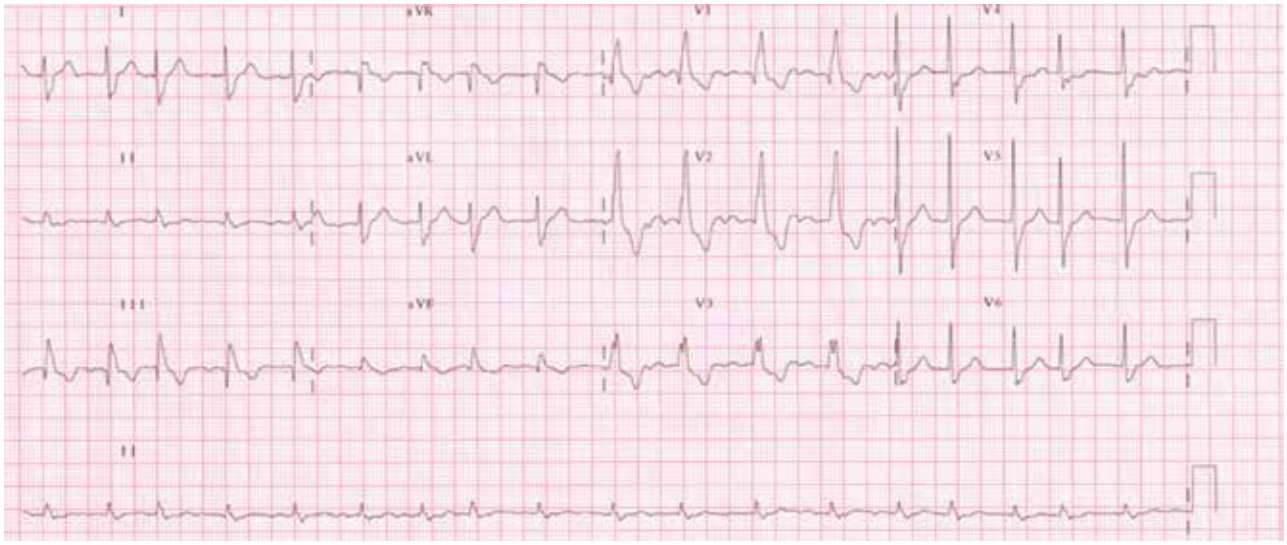

Figure 3: Graft rejection patient presenting with AF with underlying RBBB

ECG Manifestations of Post-Transplant Complications

Acute Rejection

Acute cellular or antibody-mediated rejection may affect myocardial conduction and contractility. ECG changes are nonspecific but clinically relevant when new or progressive.

- Decrease in QRS voltage
- New ST-segment or T-wave abnormalities
- Sinus bradycardia
- New AV block or bundle branch block

Serial ECG comparison is essential, as absolute findings may be subtle.

Sinus Node Dysfunction

Sinus node dysfunction may occur early post-transplant or later due to ischaemia, rejection, or fibrosis. Various presentations which include:

- Inappropriate sinus bradycardia
- Sinus pauses or arrest
- Junctional escape rhythms

Persistent symptomatic bradycardia may necessitate permanent pacemaker implantation.

Atrial Arrhythmias

Atrial arrhythmias are more common following biatrial transplantation due to atrial enlargement and suture-line reentry circuits. Typical arrhythmias include atypical atrial flutter and macro–reentrant atrial tachycardias. Approximately 18% of transplant patients develop AF or flutter. These rhythm disturbances become more common when rejection occurs. AF may be treated with amiodarone or cardioversion. Digoxin is ineffective. Circulating catecholamines are used by the graft to increase cardiac output, so beta blockers should be used with caution. Ablation may also be considered.

New-onset atrial arrhythmias should prompt evaluation for rejection or structural atrial disease.

Ventricular arrhythmias

Rare in the early period following transplant. Commonly occurs after using anti-arrhythmic drugs for atrial arrhythmia. Graft coronary disease may cause VT and VF in the late period after transplant. They may present as sudden cardiac death.

AV block

Less common than sinus node dysfunction following transplantation. In biatrial heart transplant patient, should be distinguished from the dissociated P wave from the recipient heart. It is more common in late onset bradyarrhythmias and there may be underlying rejection or graft vasculopathy. A patient with AV block should therefore be further investigated with angiography and biopsy. If the patient is haemodynamically compromised, a permanent pacemaker should be inserted. Atropine is usually ineffective in treating transplant associated bradyarrhythmias.

Coronary Allograft Vasculopathy

Coronary allograft vasculopathy (CAV) is a leading cause of late graft failure. Due to denervation, ischaemia is frequently silent. ECG findings may include:

- ST-segment depression
- T-wave inversion
- Pathologic Q waves in advanced disease

However, ECG lacks sensitivity for early CAV and should not be used as a screening tool.

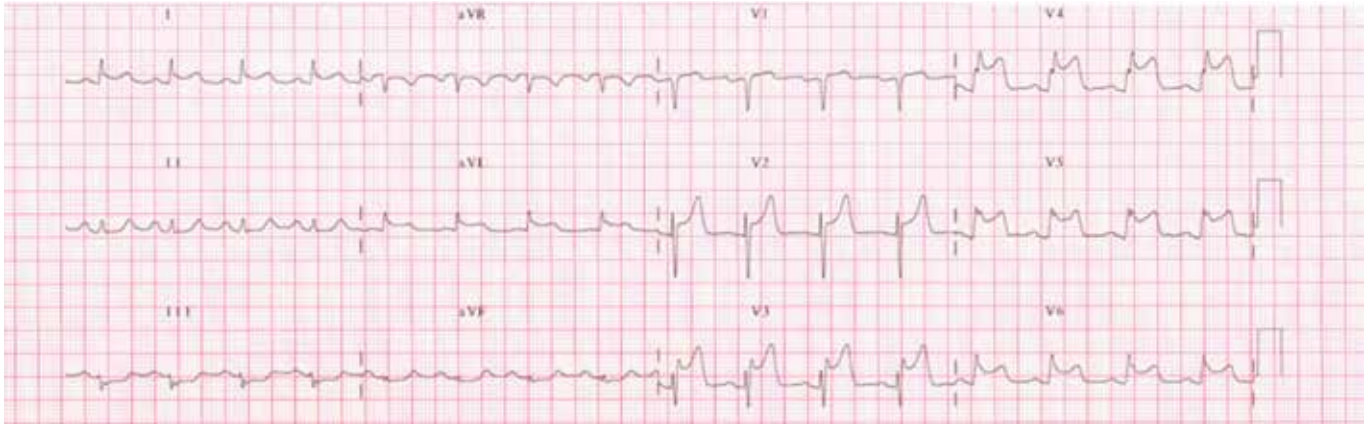

Figure 4: Extensive anterior wall STEMI in a transplant patient

Ischaemia may produce frank ST elevation and a working diagnosis of infarction should be made. Due to absence of pain, it will be difficult to establish a time course of the infarction.

Practical Approach to ECG Interpretation in Transplant Recipients

A structured approach improves diagnostic accuracy:

1. Identify surgical technique
2. Assess heart rate and rhythm
3. Examine P-wave morphology and AV relationship
4. Compare with prior ECGs
5. Correlate findings with clinical status and transplant timeline

Conclusion

ECG interpretation in heart transplant recipients requires familiarity with surgical anatomy and post-transplant physiology. Dual P-wave activity following biatrial transplantation is a benign finding, whereas new conduction abnormalities, arrhythmias, or voltage changes warrant further investigation. When used longitudinally and in clinical context, the ECG remains an invaluable tool in transplant care.

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De Winter's T Waves to Normal ECG: - The Physician's Dilemma

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Abstract

De Winters signs, often referred to as a "STEMI equivalent" is a subtle yet life threatening ECG finding that can easily be overlooked. Unlike the classic ST elevation this sign hides in plain sight and missing it can delay reperfusion therapy with devastating consequences. For physicians working in busy emergency settings and peripheral centers, early recognition of De Winters signs is a game changer- it can make the difference between life and death. Its rarity and deceptive appearance pose a diagnostic dilemma, but awareness is key to reducing mortality and morbidity in Acute Myocardial Infarction

Keywords: De Winter, LAD Occlusion, pseudo-normalization, reperfusion, repolarization vector, hyperacute T waves

Introduction

De winter sign was described in 2008 and it is defined as upsloping ST depression with hyperacute T waves. Although the incidence is only 2%,[1] this ECG sign is considered as a STEMI Equivalent and is suggestive of critical LAD Occlusion[2] EMBASE and COCHRANE databases and screened for bias using QUADAS-2. First, measurements were recorded from every ECG reported in the literature and aggregated. Second, diagnostic accuracy data from eligible cohort studies were extracted. The primary outcome was defined as at least 70% angiographic stenosis of a major epicardial vessel. Thirteen papers reported data relevant to question 1 and three papers reported data relevant to question 2. All ECGs showed maximal up-sloping ST depression in lead V3 with a median amplitude of 0.3 mV (interquartile range: 0.2–4 mV. In this case report we look at an intoxicated patient who presented to our emergency with complaints of epigastric pain

Summary of Case

45-year-old male with no previous co-morbidities presented to our ER in with excruciating chest pain. Patient had a history of binge drinking the previous night (12 units of whiskey). He had diffuse constricting type of rest pain in the lower chest and epigastrium at 0400 hrs. in the morning. This was associated

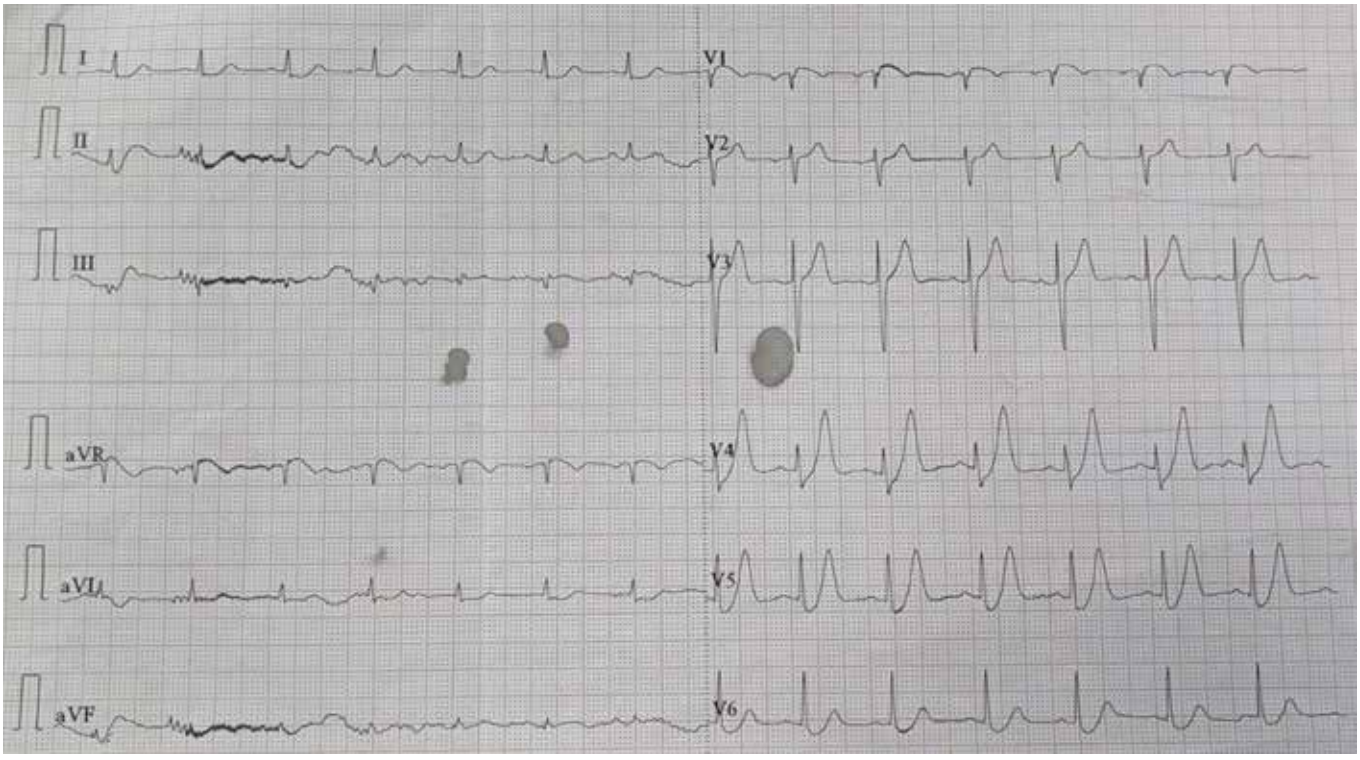

Figure 1: 12 lead ECG showing Upsloping ST depression with tall, peaked t waves- De Winter's T wave- Taken at 0430 hrs

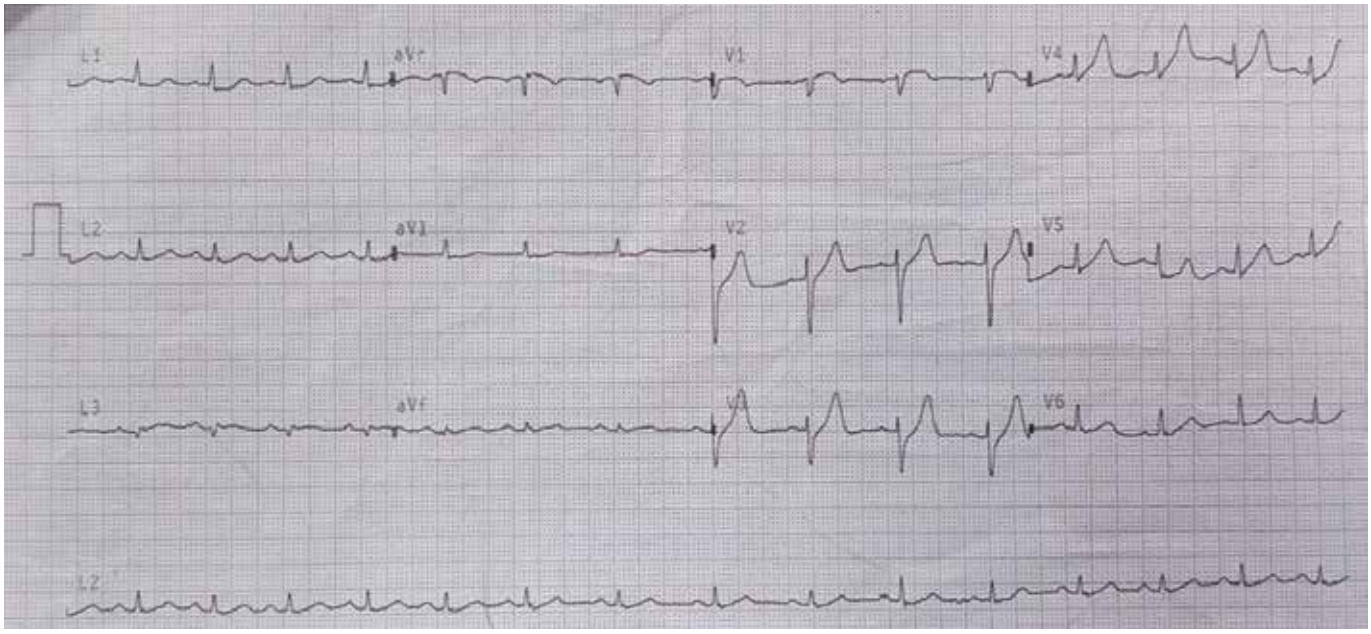

Figure 2: 12 lead ECG which was taken at 0700 hrs. Shows normalization of the T waves and ST depression

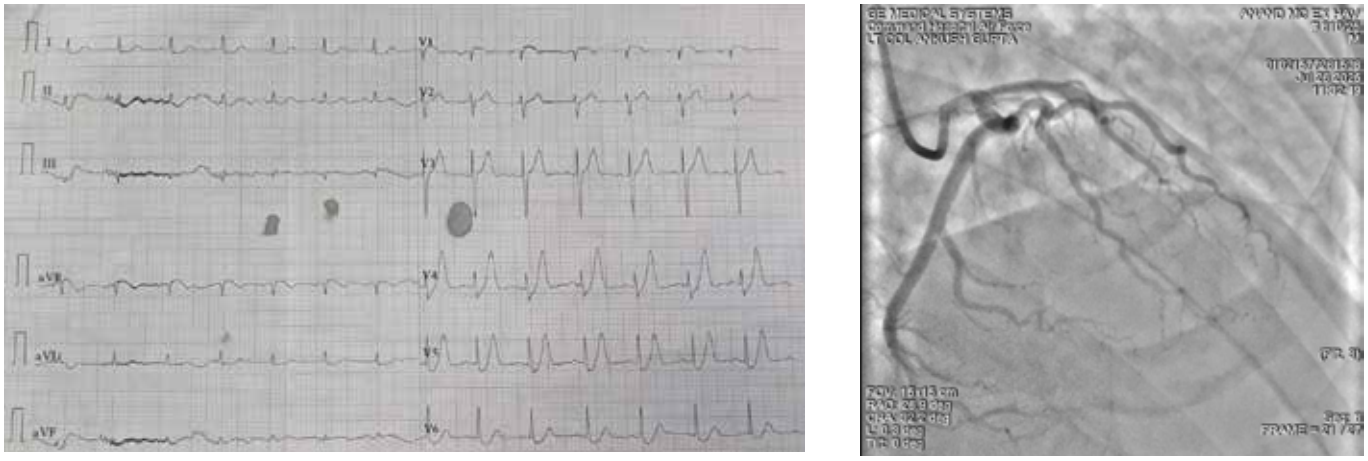

Figure 3: Comparison of initial ECG with angiogram image showing obstruction at level of mid LAD.

with 3 episodes of non-bilious, non-blood stained and non-projectile vomiting. He was first taken to a civil hospital where the ECG was suggestive of ST depression with tall T waves in V4-V6 (Figure 1). He was given loading dose of Statins, Dual Anti-platelets, and Anti Coagulants. Patient was brought to our center at 0730 hrs and on presentation he was in an intoxicated state. Patient continued to complain of pain in the bilateral lower coastal margin. It was difficult to elicit the character and nature of pain owing to the intoxicated state. There was also significant costochondral tenderness in the left side. However, the ECG taken at our center (Figure 2) was within Normal Limits. The Troponin T Card test done was negative. Initial POCUS ECHO examination showed no evidence of RWMA. The history of alcohol intake the previous night, the nature of symptoms, state of acute intoxication, normal ECG pattern and normal levels of Cardiac Biomarkers put the treating team in a dilemma as to whether we should proceed for a Primary PCI. The team decided to proceed with a PCI owing to the De Winter T wave noted in the initial ECG. Emergency Coronary Angiography was done, and it was suggestive of complete occlusion of the mid LAD (Figure 3) and hence PCI was done to LAD with a Drug Eluting Stent. Post procedure angiogram was suggestive of complete recanalization of LAD (Figure 4). The patient had relief from pain post procedure.

Discussion

Dressler in 1947 presented 27 cases of acute myocardial infarction where the initial EKG revealed tall T waves with one case presenting with de Winter's sign pattern of EKG.[3] De Winter described an EKG sign which was characterized by upsloping ST segment depression >1 mm at the J-point in the precordial leads, tall, prominent, symmetric T waves in the precordial leads with absence of ST elevation in the precordial leads (Table 1), and ST segment elevation (0.5–1 mm) in AVR(Figure 5). Based on the analysis of database of PPCI,

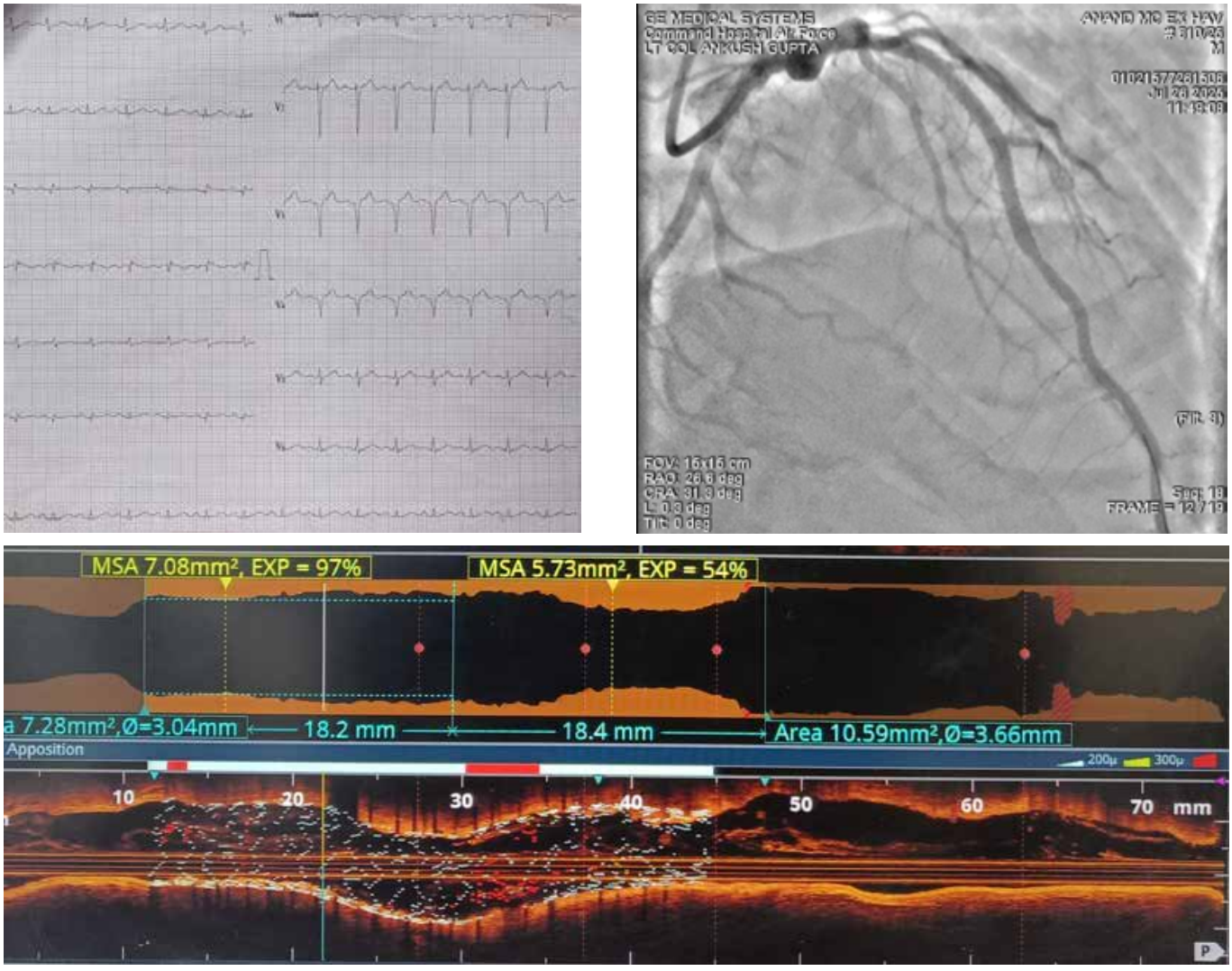

Figure 4: Post PCI to LAD. Post Procedure ECG and Angiogram image shows recanalized LAD. OCT Image of the stented LAD

Table 1 : Features of De Winter pattern and classical STEMI

Feature	De Winter Pattern	Classic Anterior STEMI
ST segment	Upsloping ST depression (1-3 mm) in V1-V6	ST elevation (convex, "tombstone") in V1-V6
T Waves	Tall, symmetric, hyperacute T waves	Initially tall, then inverted T waves with evolution
J Point	Depressed below baseline	Elevated above baseline
aVR Lead	Slight ST elevation	ST elevation (often reciprocal changes)
Culprit Artery	Proximal LAD (sometimes left main)	LAD (any segment)
Clinical Interpretation	STEMI equivalent – urgent PCI	STEMI – urgent PCI

De Winter, found 30 out of 1,532 (2%) patients with acute proximal LAD occlusion on cardiac catheterization presenting with this sign. Verouden's retrospective analysis also found the prevalence to be at 2%.[4]

Electrophysiological Basis of De Winter Sign

During Acute Phases of Ischemia there is depletion of ATP in the cardiac tissues. Hence there is failure of the Na-K ATPase pump. This causes the potassium ions to leak out through the K ATP channels, resulting in a state of membrane depolarisation in the ischemic tissue due to increase in potassium ions outside the membrane. There is a difference in membrane potentials between the ischemic myocardium and the adjacent normal myocardium. The epicardium has a higher concentration of K ATP channels as compared to the myocardium. Hence there is more K+ efflux in the epicardium. This shortens the action potential duration. This electrical heterogeneity between the epicardium and myocardium causes the injury currents which is manifested as ST elevation in Myocardial Infarction.[5]

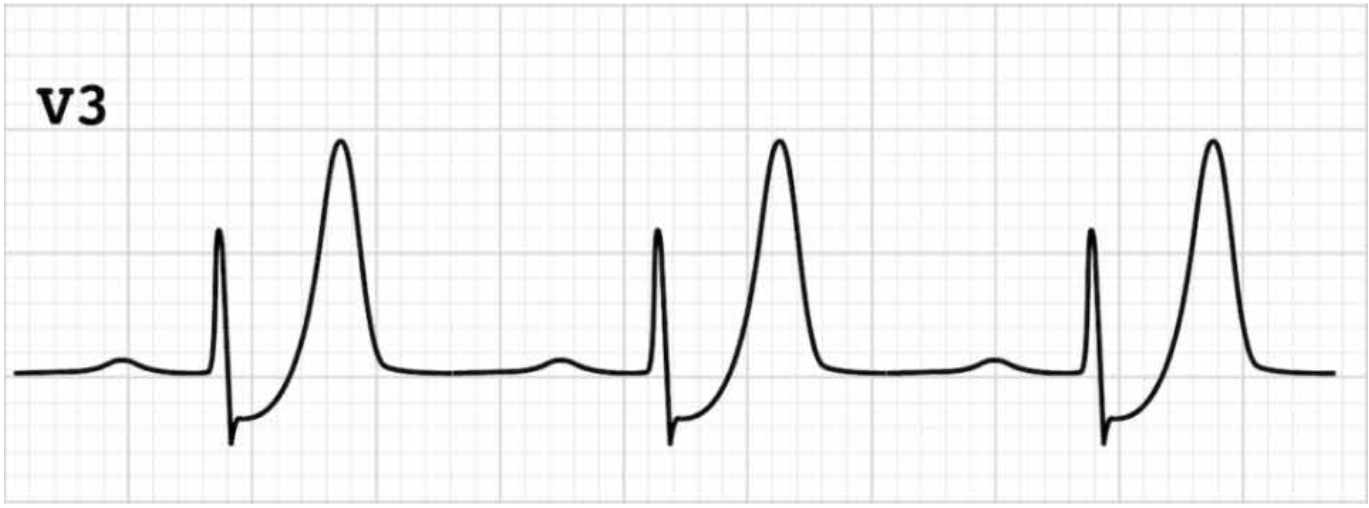

Figure 5: De Winter T waves. Upsloping ST depression with hyperacute T waves in Chest leads

However in Acute LAD Occlusion there is transmural ischemia and the epicardium is highly ischemic. Due to the high concentration of K ATP channels in the epicardium, it is in a state of membrane depolarisation. Although the myocardium is also ischemic and is in a state of depolarisation, the epicardium undergoes a quicker repolarisation. The potential difference between the epicardium and myocardium is lowered due to abnormal repolarisation of the epicardium. This causes absence of a typical ST elevation. We can safely say that the quicker repolarisation of the epicardium as compared to the myocardium leads to a reduced potential difference and hence the absence of ST elevation in De Winter pattern. This strong repolarising vector manifests as a Tall T wave in the ECG

Other authors have attributed the De Winter pattern to residual flow owing to subtotal occlusion, thrombotic cascade phases, variations in coronary anatomy, collateral recruitment, and recurrent ischemic episodes with preconditioning.[6]

Why did the ECG Normalise in our case

In our case in the initial ECG taken at 0430 hrs there was an upsloping ST depression with Tall T waves in leads V4 to V6 with no ST elevations noted in any of the leads (Figure 1). He was given loading dose at this juncture. The ECG taken at our center at 0730 hours was normal (Figure 2). This phenomenon is called pseudo-normalisation. This signifies on going ischemic insult. Continued ischemic insult leads to electrical stunning of the the myocytes. This causes slowing of the action potential and reduction in amplitude of the repolarization vector. The Tall T waves shrink and comes back to "normal". The pitfall was that it was initially misinterpreted as normal ECG. Only when we saw the previous ECG we could recognise the De Winter pattern.

Conclusion

The case is discussed in this forum as it was a Myocardial Infarction that presented with De Winter pattern followed by Pseudo-normalisation. The case could have been misdiagnosed due to the clinical history, patient presentation and the ECG progression**.** The de Winter ECG pattern is associated with acute coronary artery occlusion, most notably the LAD. As such, early recognition and treatment allocation are critical to allow adequate reperfusion and improve the substantial morbidity and mortality associated with a large anterior MI. Hence it is imperative this acute change is picked up in the peripheral hospitals and necessary reperfusion strategy may be initiated.

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The Electrocardiogram in Common Non drug Toxin Poisoning in India

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Abstract

Intentional and accidental poisoning with various vegetable, animal and insect poisoning and pesticides is not uncommon among ER admissions. The effects of these toxins on the ionic channels of the heart, the neural and circulatory system and the consequent effects on the action potential cause various changes in the ECG. These can help in the diagnosis of unknown poisoning and further management.

Introduction

Poisoning, which is defined as exposure to any drug, chemical, or toxin that leading to cardiotoxicity resulting from poisoning is a significant contributor to mortality. Therefore, it is essential for clinicians to swiftly identify signs of cardiotoxicity and be ready to make management decisions that necessitate immediate action, even when clinical data is limited.

While poisoning is a rare cause of cardiac arrest in older adults, it is the primary cause of cardiac arrest in individuals under the age of 40. The approach to emergency cardiovascular care for myocardial injury should be adjusted in both diagnostic assessment and treatment if the patient has a history of drug or toxin exposure. This review of ECGs of poisoning is limited to accidental or suicidal ingestion of plant and other toxins commonly encountered in India

Management of Poisoned Patients: Signs, Symptoms and Ecgs

The preliminary assessment of a poisoned patient relies on a combination of vital signs, symptoms, and findings from the physical examination known as the potential toxic syndrome, or 'toxidrome'.[1] Identifying toxidromes facilitates a more logical approach to treating the poisoned individual. Incorporating ECG interpretation into the initial assessment can yield vital information to inform management decisions.

Role of the ECG in Poisoning

Due to its extensive application, accessibility, affordability, and non-invasive characteristics, and the rapid availability of electrocardiogram (ECG) which can be obtained within minutes it is the first investigation available to the beleaguered emergency physician. The ECG is crucial for assessing poisoning cases to identify or rule out cardiotoxicity, as well as to implement essential initial management steps. Comparison with a prior ECG is always useful to identify suspected fresh ECG changes. Repeated serial ECGs should be continuously monitored in an appropriate setting until toxicity resolves.

Specific Toxic Effects on Myocardium

Drug cardiotoxicity may reflect disturbances on the delicate balance of the myocardial membrane potential, as these have effects on specific ion-channels (particularly sodium, calcium, and potassium) that produce important changes in the action potential as well as resting potential.

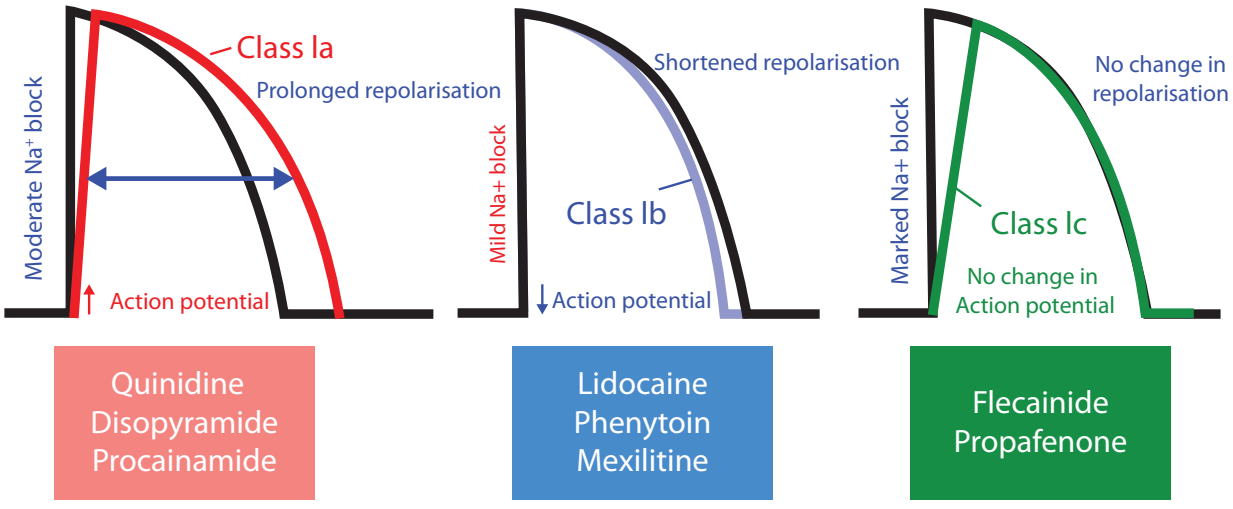


Figure 1: Sodium channel blockade effects on action potential. Reproduced from Vetscraft

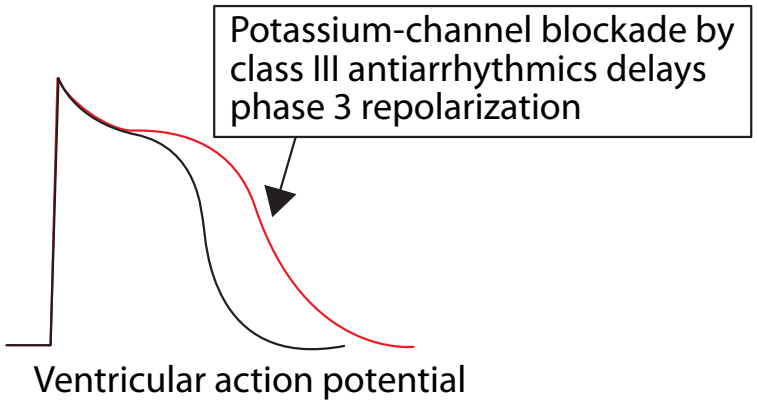


Figure 2: Potassium channel blockade. Taken from JaypeeDigital e book reader

Sodium Channel Blockade

The upward stroke of the myocardial action potential is a result of the conformational change and opening of voltage dependent sodium channels in response to an electrical stimulus from adjacent cells, thus causing depolarization. This phase 0 of the action potential is delayed, demonstrating a less steep slope of depolarization.

Potassium Efflux Blockade

Myocardial membrane permeability to potassium efflux is responsible for repolarization, or the return to myocyte resting membrane potential. Potassium channel blockade that interrupts the rectifying potassium current produces an increased duration of phase 2 and phase 3 of the myocardial action potential and translates to the ECG primarily as a prolonged QT interval. Blockade of the rectifying potassium channels may also cause T-wave abnormalities or the presence of U-waves. The presence of a long QT represents slowed repolarization, which produces the myocardial substrate for the development of polymorphic ventricular tachycardia, or torsades de pointes (TdP).

Common Toxins Encountered in Clinical Practice

Some of the common toxins that are encountered in poisoning cases in India are:

Vegetable Toxins

Datura poisoning

Oleander poisoning

Foxglove poisoning

Cannabis abuse and overdose

Animal attacks

Snake bite

Scorpion sting

Bee sting

Chemical Toxins

Insecticides

Rodenticides

Datura poisoning

In Southern India suicidal attempts by ingesting the crushed seeds with milk. The clinical features are remembered by the 9 D's:- 1 Dry & hot Skin, 2 Dilated pupils, 3 Dilated vessels, 4 Delirium, 5 Dermatitis, 6 Dysphagia, 7 Diplopia, 8 Delusions 9 Dry mouth & difficulty in speaking. The active ingredients are atropine, scopolamine and hyoscyamine. Unintentional poisoning occurs when in the pursuit of the hallucinogenic effects of Datura the individual lands in a poisoned state. The ECG in such cases show atropine like effects with Sinus Tachycardia, Right bundle branch block but with normal ST segments.

Figure 3: The Datura plant

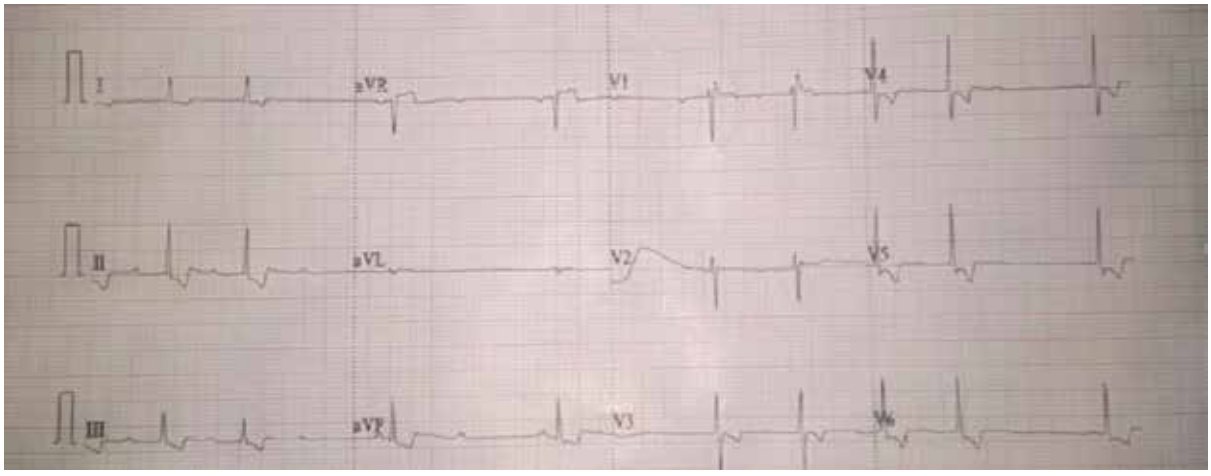
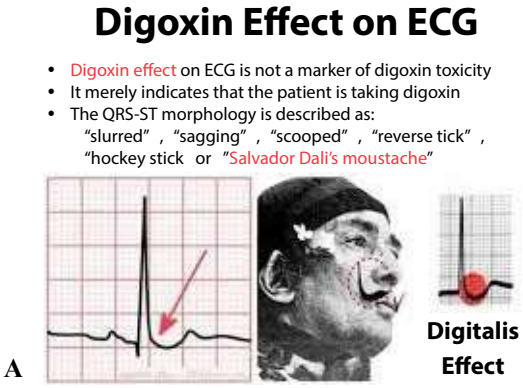


Figure 4: ECG of patient with Oleander poisoning and serum K of 10.1 surprisingly showing no typical features of hyperkalemia. There is variable AV block and a short QT interval (QTc 228). Taken from Guru S et al. EJCRIM, 2021 (8).[2]



Reproduced from the Hearts Post

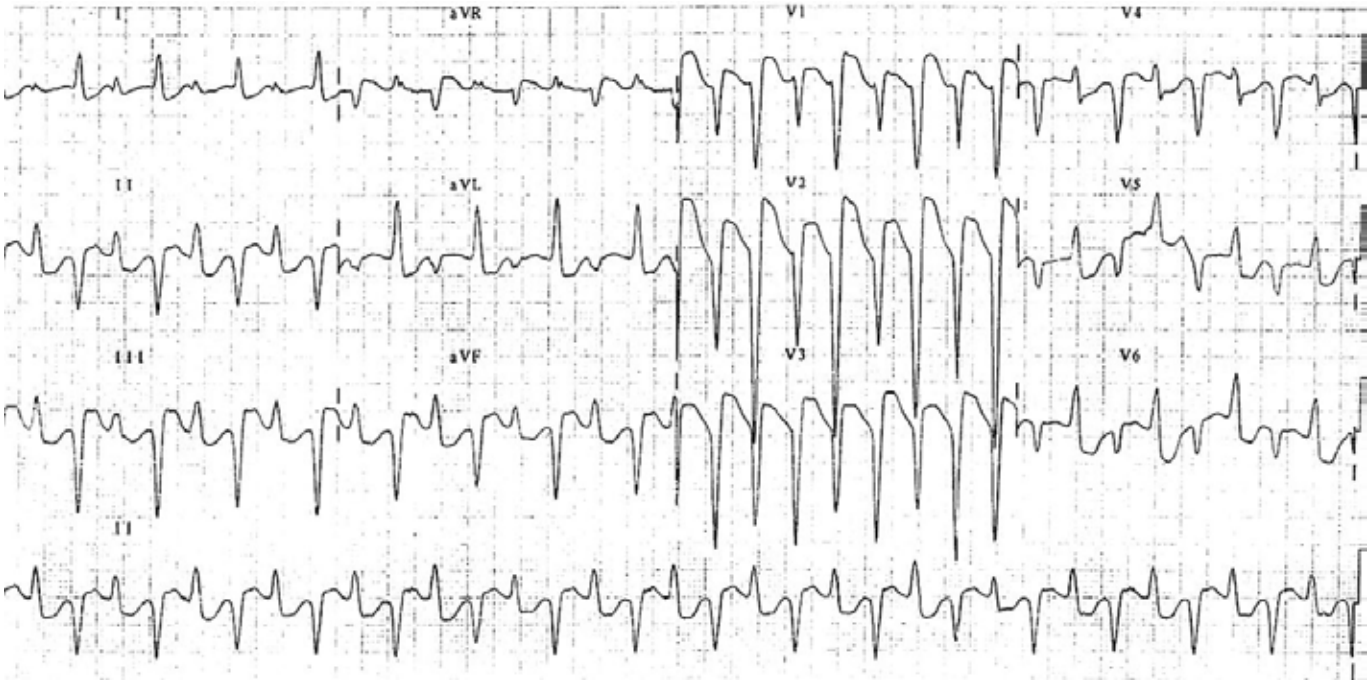


Figure 5: Top Panel, Digitalis effect on the ECG showing the sagging st segment named the Salvadore Dali effect. Bottom Panel. Bidirectional tachycardia. ECG Library Toxicology

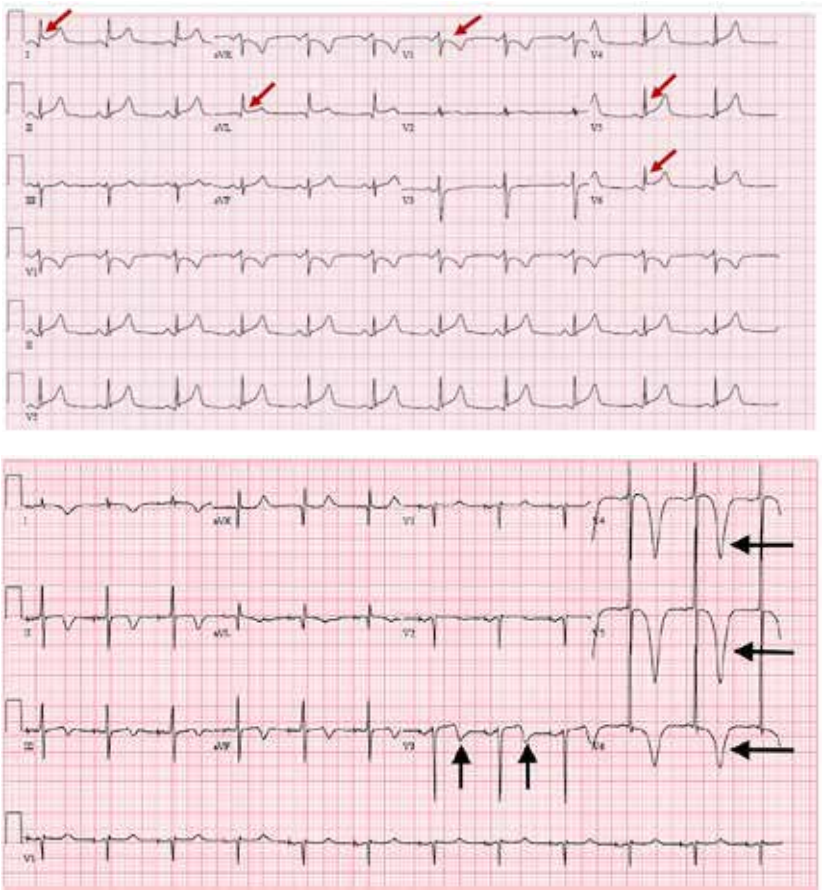


Figure 6: Cannabis induced ECG situations. Top panel. Marijuana induced STEMI. Reproduced from Sundeep Kumar et. BMJ 2018. https://doi.org/10.1136/bcr-2018-226894. Bottom panel. Marijuana induced Pseudo Wellens syndrome Reproduced from Kandah F et al[3]

Oleander poisoning

Oleander poisoning can be caused by ingesting the seeds or the leaves of the plant. It has an non digoxin cardiac glycoside effect caused by inhibition of the Na/K ATPase exchange pump leading to hyperkalemia and a vagotonia effect.

Foxglove (Digitalis) poisoning

The Digitalis lanata plant is the source of the drug digoxin. The cardioactive steroids found in this plant influence electrolyte homeostasis during phase 4 of the action potential through inhibition of the Na/K ATPase exchanger. The subsequent increased intracellular calcium concentrations are responsible for digoxin's positive inotropic effects. The action of cardioactive steroids on the vagus nerve also produces direct suppression of impulses from the sinoatrial (SA) and atrioventricular (AV) nodes.

Cannabis sativa

Cannabis both inhalational and ingestion has been used as a psychoactive drug. Overdose can cause various types of ECG abnormalities – STEMI to deep T wave inversions are seen depending on the active ingredient -tetrahydrocannabinol (THC) or tetrahydrocannabindiol. Representive ECGs are given below.

Animal toxins are encountered in snake bites, scorpion stings and bees sting.

Snake bites, particularly venomous ones, commonly cause electrocardiographic (ECG) changes in 39–42% of cases, due to direct cardiotoxicity or secondary systemic issues. The most common findings are

1. sinus tachycardia (17–25%), followed by
2. sinus bradycardia (9–11%),
3. non-specific ST-T changes (5–6%), and
4. AV blocks.

Severe cases may show atrial fibrillation, Bundle Branch Block, or acute myocardial infarction.[4]

Scorpion Sting ECG changes often result from an "autonomic storm" (excessive catecholamine release), causing abnormalities in up to 88% of severe cases. The most common findings include sinus tachycardia, premature ventricular contractions (PVCs), ST-segment/T-wave changes, and AV

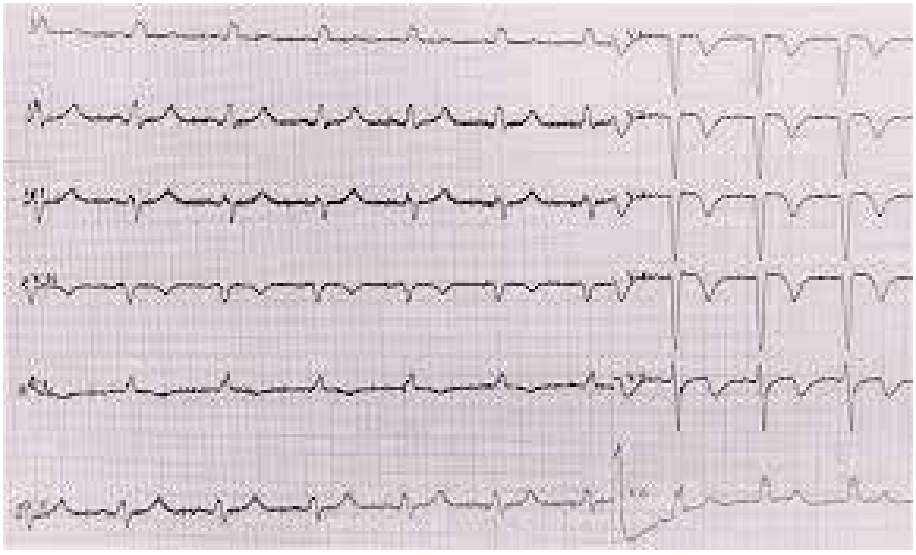
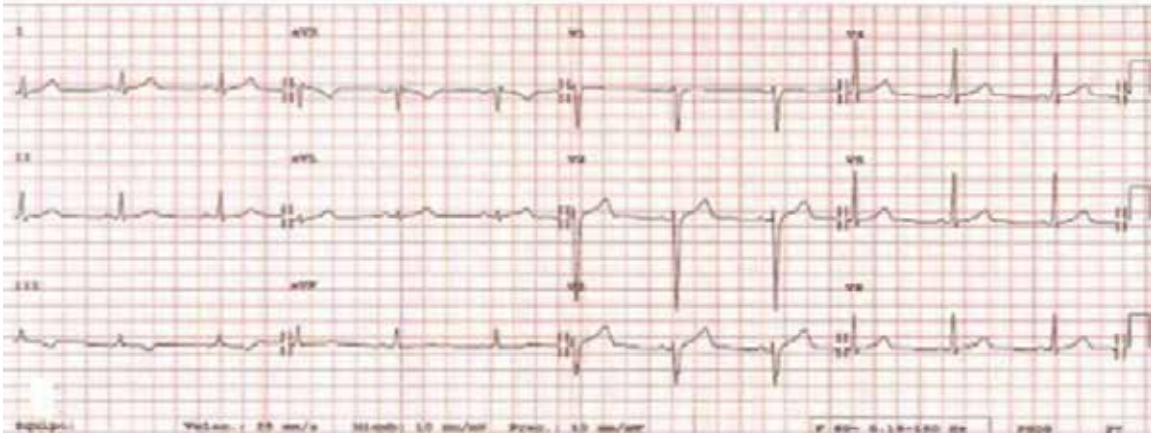


Figure 7: Scorpion sting causing Extensive anterior wall MI.Reproduced from Banait S, et al[6]



Figue 8: Idioventricular rhythm and shock after a honeybee sting. Desiree Franco Lugo et al[8]

blocks. Severe cases may show myocardial infarction-like patterns, QT prolongation, or reduced ejection fraction.[5]

Bees Sting can trigger acute cardiovascular reactions known as Kounis syndrome, causing ECG changes like ST-segment elevation or depression, T-wave inversion, and arrhythmias. These changes result from allergic-induced coronary vasospasm or direct toxic effects of the venom leading to myocardial infarction.[7]

Chemical poisons – The insectcides and rodenticides

Insecticide poisoning, particularly with organophosphates, frequently causes serious electrocardiographic (ECG) changes due to autonomic instability, including sinus tachycardia, sinus bradycardia, QTc prolongation, and ST-T segment abnormalities. These abnormalities, often appearing within hours to days, can lead to life-threatening arrhythmias, such as ventricular fibrillation or tachycardia, and require immediate cardiac monitoring and treatment.[10]

Common ECG Changes by Insecticide Type

Organophosphates (OPs)

The most common findings include sinus tachycardia or bradycardia, QTc prolongation (very common), ST-T segment changes, first-degree AV block, and ventricular arrhythmias (premature ventricular complexes).

Carbamates

Similar to organophosphates, they induce QTc prolongation, tachycardia, bradycardia, and ST-T changes.

Organochlorines

Often cause ventricular tachycardia, premature ventricular complexes (PVCs), and ST segment depression.

Pyrethroids

Can cause QT prolongation, conduction block, or transient complete heart block.

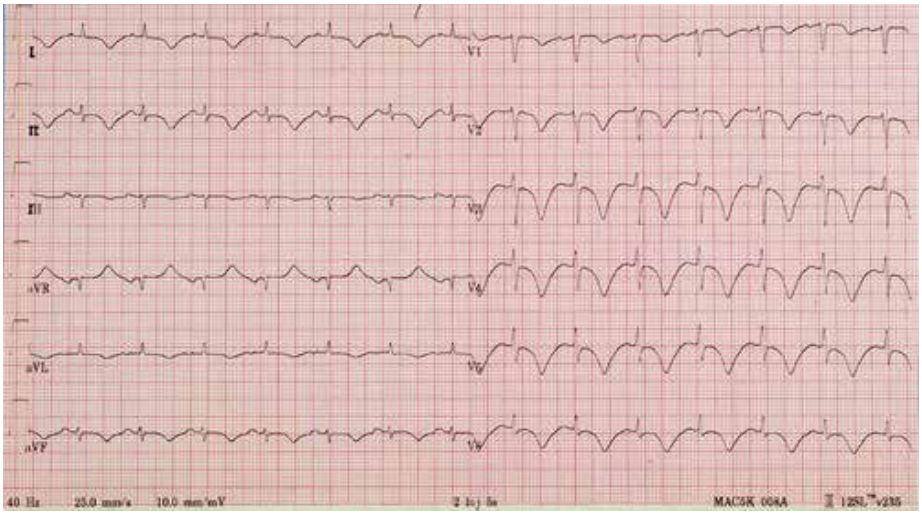


Figure 9: ECG of organophosphate poisoning Jorens, P. G., Robert, et al[9]

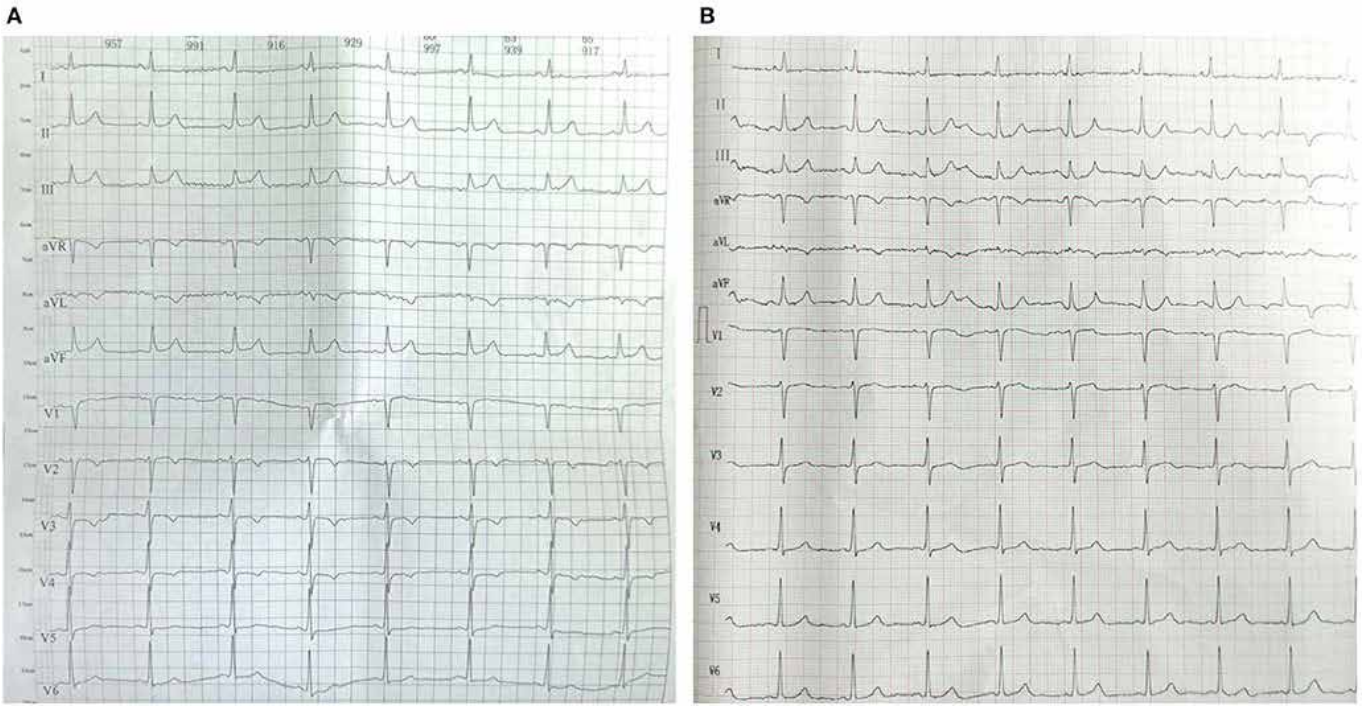


Figure 10: Chlorpyrifos poisoning leading to NSTEMI Reproduced from Ye C, et al[11]

Neonicotinoids

Known to cause tachycardia, mild QT prolongation, and cardiac arrest.[10]

Conclusions

The recognition of ECG changes after a toxin poisoning requires a systematic approach to recognising various abnormalities in the ECG- by analysing the rhythm, the morphological changes in the various components of the ECG and a quick recognition of the causative toxin and its prompt treatment.

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Electrocardiographic Changes in Anaemia: Pathophysiology, Clinical Findings

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Abstract

Anaemia causes systemic hypoxia that significantly influences cardiac electrophysiology. Electrocardiography (ECG) demonstrates characteristic changes in anaemic patients, including sinus tachycardia, ST-segment depression, T-wave abnormalities, low QRS voltage, and QT interval prolongation, often correlating with haemoglobin severity. Many of these changes are reversible following correction of anaemia, highlighting their functional nature. This review summarizes the pathophysiological basis, clinical ECG findings, severity correlation, and reversibility of changes in anaemia, and discusses emerging artificial intelligence–based ECG tools for non-invasive detection and screening..

1. Introduction

Anaemia is a global health issue affecting billions of individuals across all age groups specially in India. Reduced haemoglobin decreases oxygen delivery to tissues, including the myocardium, leading to compensatory and pathological cardiovascular responses. Although ECG is primarily used for cardiac evaluation, evidence suggests it can reveal distinctive changes in the context of anaemia. This review focuses on summarizing known ECG findings in anaemia and their clinical significance.

2. Pathophysiology of ECG Changes in Anaemia

2.1 Mechanisms

In anaemia, hypoxic stress on the myocardium and compensatory circulatory adjustments (e.g., increased heart rate, increased stroke volume, hyperdynamic circulation) influence cardiac conduction and repolarization. Limited oxygen supply relative to myocardial demand can lead to subendocardial ischemia, altered ion channel function, and changes in depolarization and repolarization behavior. These contribute to observable ECG changes such as ST-segment and T-wave abnormalities.

3. Common ECG Changes in Anaemia

3.1 Heart Rate and Rhythm

Sinus tachycardia is the most frequently reported ECG change in anaemia, reflecting compensatory increase in heart rate to maintain cardiac output in the face of reduced oxygen content. This has been observed across multiple cohorts and correlates inversely with haemoglobin levels.[1]

3.2 ST-Segment Changes

ST-segment depression is commonly reported in severe anaemia and is believed to represent subendocardial ischemia due to oxygen supply-demand mismatch. The extent of ST depression correlates with haemoglobin severity, being most prominent when Hb is <5 g/dL.[2]

3.3 T-Wave Abnormalities

T-Wave inversion or flattening are frequent in anaemic patients and are often reversible upon correction of anaemia. These reflect repolarization disturbances secondary to hypoxia and metabolic shifts.[3]

3.4 QRS Voltage Changes

Low QRS voltage (reduced amplitude) is another common finding in severe anaemia and may indicate decreased myocardial electrical activity due to hypoxia or associated circulatory changes. Studies have demonstrated diffuse low voltage across limb and precordial leads in patients with haemoglobin ≤5 g/dL.[4]

3.5 QT Interval and Dispersion

Prolonged QT interval and increased QT dispersion have been noted, particularly in sickle cell anaemia and other chronic anaemic states, suggesting altered ventricular repolarization that may predispose to arrhythmia.[5]

3.6 Novel ECG Indicators

Research on novel ECG parameters—such as the **frontal QRS-T angle**—suggests these may also be altered in anaemia and correlate with severity, potentially serving as additional electrophysiological markers in future risk stratification.

4. Correlation with Severity of Anaemia

Several studies demonstrate an inverse relation between haemoglobin levels and the frequency or severity of ECG abnormalities. In individuals with moderate to severe anaemia (Hb ≤7 g/dL), ECG changes are significantly more common compared to mild anaemia.[6]

5. Reversibility of ECG Changes

5.1 Correction Improves ECG

ECG abnormalities in anaemia are largely **reversible** upon treatment, particularly after blood transfusion or correction of haemoglobin levels. ST changes, T-wave inversions, and low QRS voltages often normalize post-treatment, underscoring that many of these ECG changes reflect functional rather than structural alterations.[7]

5.2 Clinical Case Illustrations

Case reports have documented dramatic ECG changes in severe anaemia that mimic myocardial infarction, which resolve within hours after transfusion. Such findings highlight the risk of misdiagnosis and the importance of considering anaemia in differential ECG interpretation.[8]

6. Clinical Implications and Recommendations

6.1 ECG as a Screening Tool

Although ECG should not replace haemoglobin testing, it can provide immediate suspect signs of anaemia in emergency and resource-limited settings, especially when combined with clinical context.

6.2 Interpretation Guidance

Clinicians should be cautious not to misinterpret reversible ECG changes due to anaemia as primary myocardial pathology. Integration with clinical findings and haemoglobin measurements is essential.[9]

6.3. Computational and AI-Based Detection of Anaemia from ECG

Beyond classical interpretation, cutting-edge research has shown that deep learning models applied to conventional ECG signals can accurately detect anaemia. A multicentre retrospective cohort demonstrated that raw ECG data processed through convolutional neural networks could differentiate anaemic patients (Hb ≤10 g/dL) with high accuracy (AUROC ~0.90+), suggesting potential for automated screening. This study reveals that a DLA, a powerful tool of artificial intelligence, can elucidate subtle ECG changes that distinguish patients with anaemia. Many studies confirmed that anaemia is independently associated with adverse outcomes in patients with congestive heart, chronic kidney disease, acute coronary syndrome, and sepsis. DLA focused especially on the QRS complex to decide the presence of anaemia. The DLA also focused on the T-wave for deciding the presence of anaemia. A previous study showed that several electrocardiographic changes had been identified in patients with severe anaemia, including a small QRS amplitude, prolonged QT interval, T-wave inversion, and rightward T-wave axis deviation.10 In this study, patients with severe anaemia also had a small QRS amplitude, rightward T-wave axis deviation, and longer corrected QT interval. In one case, a patient with anaemia showed a changed ECG after transfusion . After transfusing a five-pack of red blood cells, the QRS amplitude increased, and the QT interval shortened. When the patient had a status of severe anaemia, bradycardia was present; after transfusion, tachycardia occurred[10]

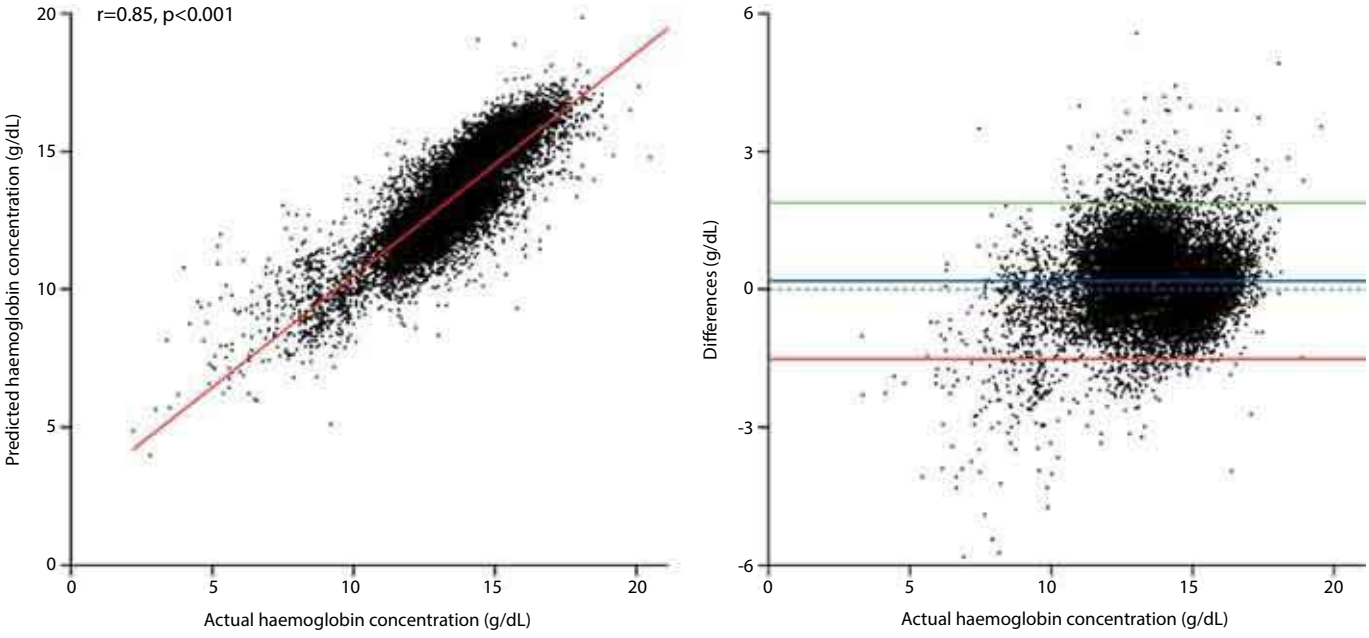

Figure 1: Predicted and actual haemoglobin values

The red line is the linear regression line. The blue line is the mean difference, and the red and green lines are the 1·96 SD value from the mean difference.[10]

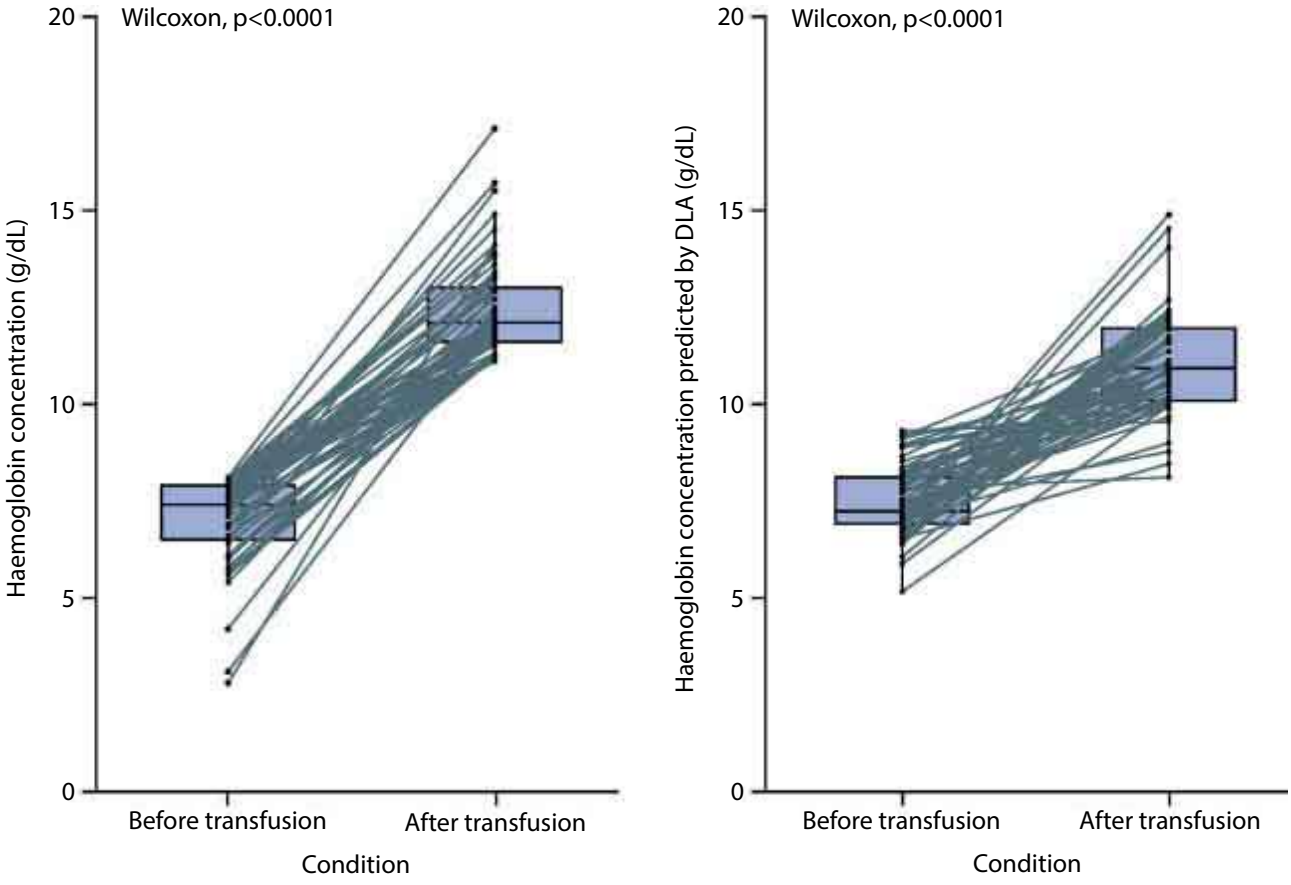

Figure 2 : Changes in haemoglobin values and predicted haemoglobin values after transfusion[10]

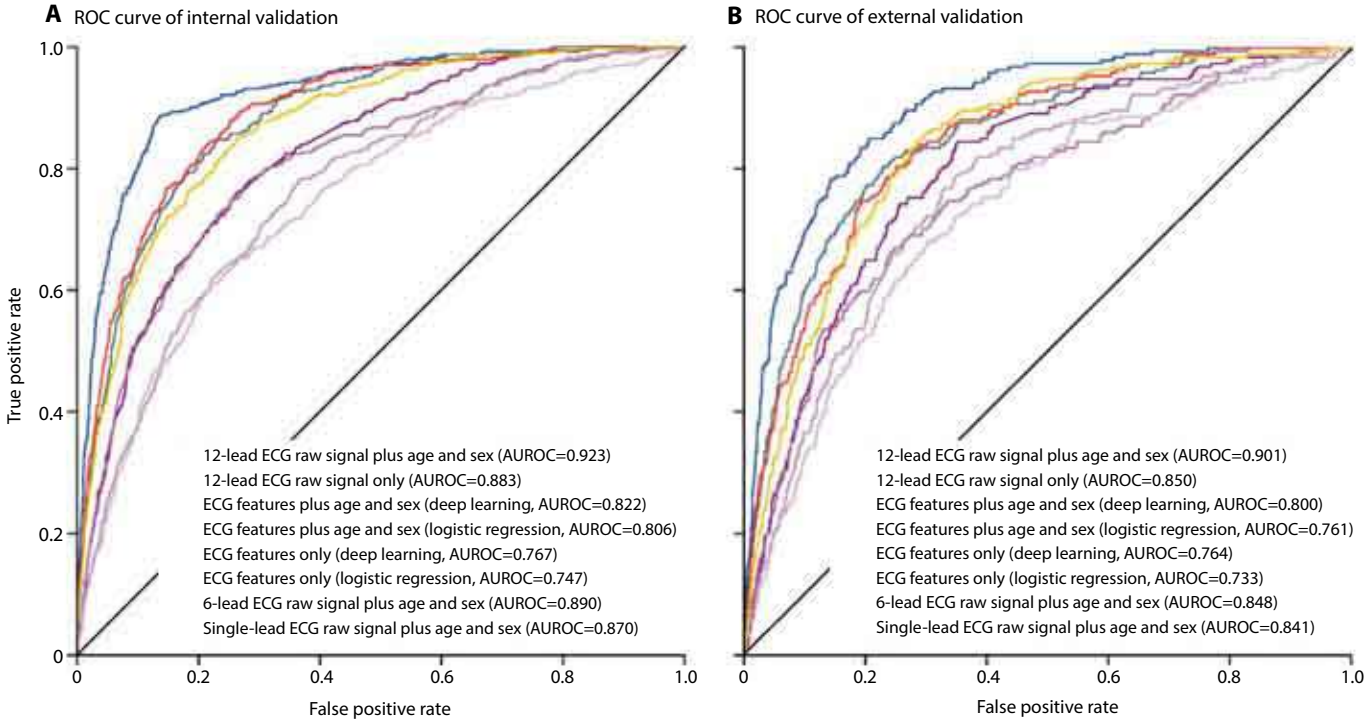

Figure 3: Performance of the deep learning algorithm for detecting anaemia

ROC=receiver operating characteristics. AUROC=area under the receiver operating characteristic curve.[10]

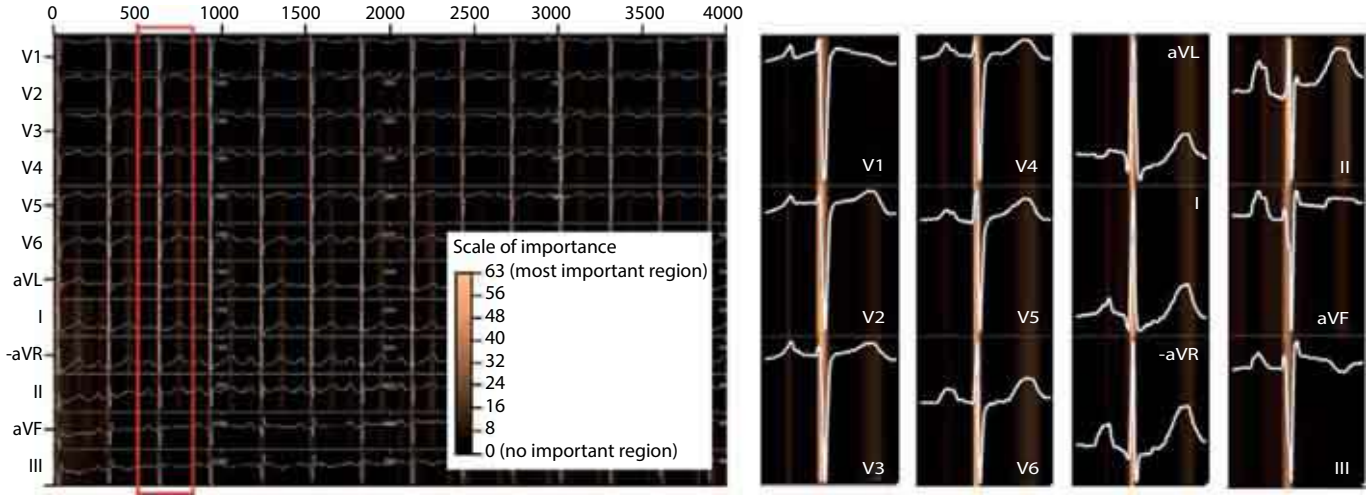

Figure 4: Sensitivity map of a deep learning algorithm for detecting anaemia.

The sensitivity map shows the region in which the convolutional neural network algorithm focused attention for deciding the presence of anaemia. The most important region is in orange and the least important region is in black. As the number of filters of the first convolutional layer was 63, the sensitivity map described the region of importance for deciding the presence of anaemia using a 63-grade scale. We visualised grade 0 as black and 63 as orange. V=precordial lead.[10]

7. Conclusion

Anaemia produces discernible ECG alterations that reflect myocardial oxygenation status and compensatory cardiac responses. Frequent abnormalities include sinus tachycardia, ST-segment depression, T-wave changes, and low voltage QRS complexes. Many of these changes correlate with severity and are reversible with haemoglobin correction. Emerging AI-based ECG analysis tools hold promise for non-invasive anaemia screening, offering future avenues for early detection and monitoring. Many of these changes (small QRS amplitude, prolonged QT interval, T-wave inversion) are nonspecific and may be in seen in other medical conditions like congestive heart failure, COPD, etc and needs clinical correlation.

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Abbreviations

BP: Bopp Pressure

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68.4% **of obese participants** achieved BP control in **5** years.[1]

8.2% reduction in LVM[2,3]

21%[4] Reduction in CV Events in patients in Prior MI and Stroke

Abbreviations

LVVE Left Verticu ar Mars

References:

1. The Antihypertensive and ic's-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), JAMA, 288(23), 2981-2907
2. Sagarad, Suresh V., et al. Journal of Clinical and Diagnostic Research: CDR 7.4 (2013): 687 8 Roush, George C., et al.
3. The Journal of Clinical Hypertension 20.10(2018) 1507-1415. N Fagl. Mod 2022,387:2401-10.

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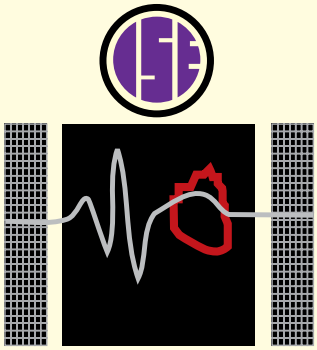
*As compared to Top 5 Branded Generics as per IQVIA Rx MAT April 2025(Prices as per 1mg : as on 30.05.2025)
#For Empagliflozin: No Dose Adjustment recommended for use in individuals with an estimated glomerular filtration rate (eGFR) of 20 mL/min/1.73 m2 or higher
Data on file



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