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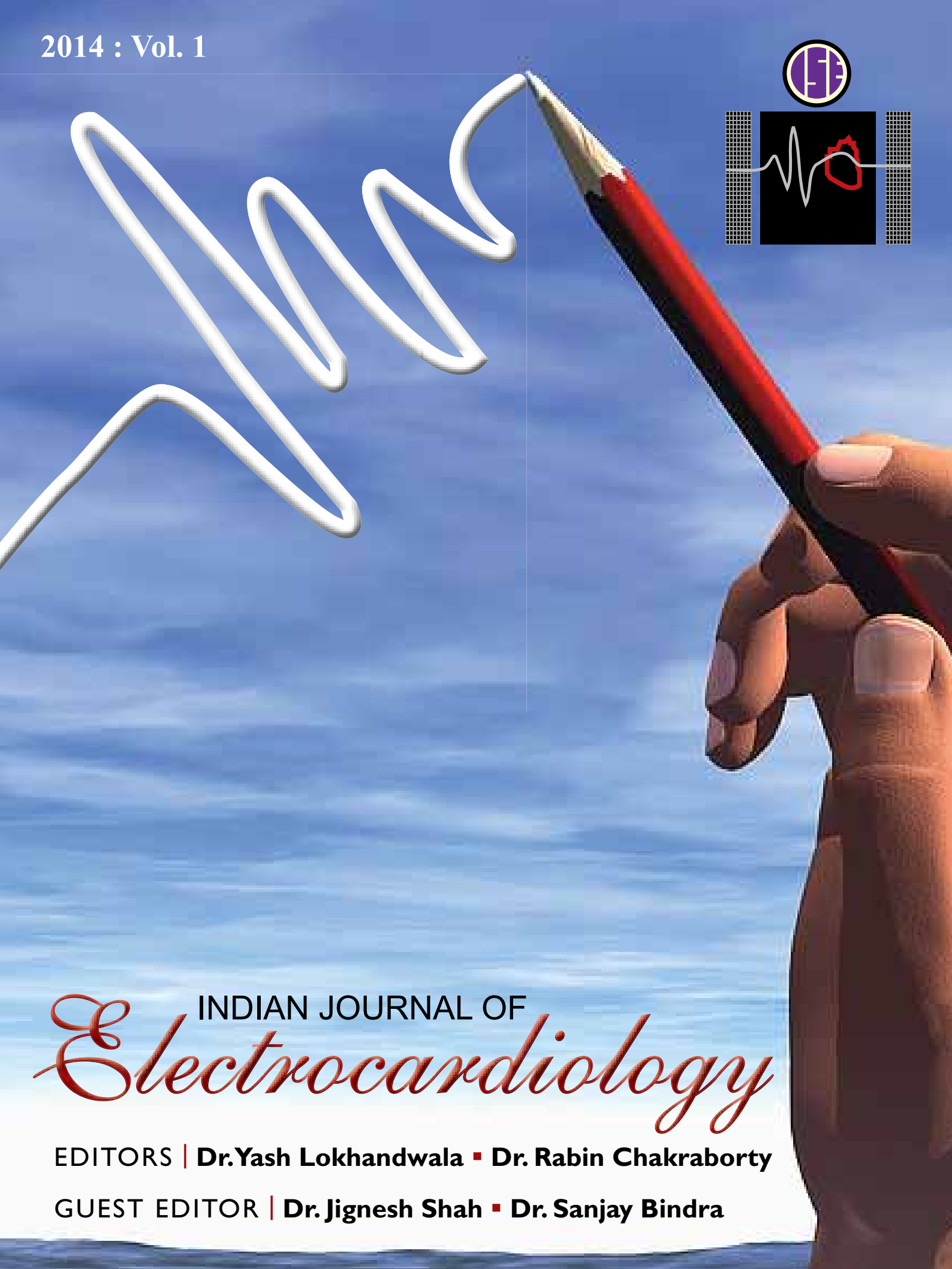

INDIAN JOURNAL OF Electrocardiology

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Editorial

Dear Friends,

As we release this issue of the IJE, we are at the threshold of ISECON Lucknow. The scientific committee has prepared an excellent arrhythmia course encompassing interesting topics relevant in day to day clinical practice as well as some rare clinical scenarios. I am sure their teachings and your interest will create the right mix for a good understanding of pathophysiology from the perspective of cardiac conduction system. We are very excited to bring you the current issue of IJE since it includes interesting ECG related manuscripts as well as clinically relevant review articles.

We have been fortunate to have a number of clinicians contributing excellent review articles to this issue of IJE. Dr. Ghogare and colleagues present an review of LBBB tachycardia. All of us have been perplexed by wide complex tachycardias. The authors present a comprehensive review of the algorithms for differentiating SVT vs VT in LBBB tachycardia and then go on to describe the management of various etiologies of LBBB tachycardias. All clinicians will benefit from this review article.

The use of MRI for various neurological and musculoskeletal pathologies continues to rise and so does the use of pacemakers and ICDs. So far, patients with cardiac devices have been considered ineligible for MRI. Dr. Mollerus is a leading authority on the subject and has performed several hundred MRIs on patients with cardiac devices. He shares his expertise and experience in the review article related to MRI safety of cardiac devices.

Dr. Taksande presents an interesting case or Torsades de pointes after pacemaker implant and review the etiologies and management of this life threatening condition. As always, the ECG Quiz by Dr. Lokhandwala continues to be the star feature of IJE.

Happy reading and we hope to have more contributions from you for future issues.

Jignesh Shah **Sanjay Bindra**

From President's Desk

Dear Members,

It is our great pleasure in bringing out the 1st issue of Indian Journal of Electrocardiology of the year 2014 on the eve of Annual Conference of Indian Society of Electrocardiology at Lucknow.

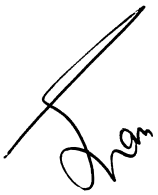
ISECON 2013 – The Annual Conference of Indian Society of Electrocardiology was organized by Dr Balbir Singh and the team at New Delhi on 6th and 7th April 2013. It was a great scientific feast! Our heartiest congratulations to Dr Balbir Singh and his team.

Mid-Term Conference of Indian Society of Electrocardiology was organized by Dr. H. Prabhakar and his team at Mangalore on 28th – 29th September 2013 and a very well attended meeting with excellent scientific material. Dr H Prabhakar and his team needs worthy praise.

ISE has initiated Pacemaker/CRT/ICD Survey. The results will be published soon in one of the leading journals.

My sincere thanks to Dr Yash Lokhandwala, Dr Rabin Chakraborty, Dr Jignesh Shah, Dr Sanjay Bindra and the Editorial Team for bringing out the ISE Journal – 2014, 1st Volume.

Long Live Indian Society of Electrocardiology



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

Review Article

Tachycardia with Left Bundle Branch Block (LBBB) Morphology – Approach, Differential Diagnosis and Management

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Abstract

Evaluating a patient with wide complex tachycardia has always been a physician's worst nightmare. Such tachycardias have variety of etiologies broadly categorized into supraventricular and ventricular origin. Algorithms with varying sensitivity and specificity have been proposed to differentiate wide complex tachycardia, to reach a reasonable diagnosis and practical decision making. However, these algorithms are not self-evident unless the underlying mechanism is well understood. Therefore, in this review we describe the approach, mechanism, clinical manifestation, electrocardiographic (ECG) features and management of one such category of wide complex tachycardia with LBBB morphology.

Introduction

Evaluating a patient with wide complex tachycardia (WCT) has always posed diagnostic and therapeutic dilemma.[1] The process of clinical heuristics to come to definitive diagnosis is cumbersome and confusing. Presence of atrio-ventricular (AV) dissociation though highly suggestive of ventricular origin of tachycardia is seldom present on ECG or is difficult to discern during rapid tachycardia. Hemodynamic instability during tachycardia is helpful but not sacrosanct of its ventricular origin. Hence, many diagnostic algorithms are proposed over time to help differentiating WCT (Table 1).[2-6] The algorithms are complex, confusing and with varying sensitivities and specificities. However, committing to these algorithms to memory is challenging and overreliance on them may impede cardiologists from understanding the underlying tachycardia mechanism. Most of these algorithms subcategorize WCT based on QRS morphology into right bundle branch block (RBBB) or left bundle branch block (LBBB) morphology. The current review focuses upon the subcategory of WCT with LBBB morphology and assessing its mechanism, clinical presentation, diagnostic and therapeutic implications.

Definitions and Pathophysiology

LBBB is defined as a prolonged QRS duration (≥ 120 ms) with broad monophasic R waves in leads I, V5 and V6 that are usually notched or slurred. There is delayed intrinsicoid deflection (the beginning of the QRS to the peak of the R wave is > 50 ms) in leads I, V5 and V6. In typical, or "classic" LBBB, lead V1 will demonstrate either an rS or QS complex, and, more importantly, Q waves will be absent from the left lateral leads[3]. The secondary ST and T-wave changes are in the opposite direction of the major QRS deflection.[7]

Typically, there is left to right interventricular septum activation followed by activation of the left ventricle via the left bundle branch. However, during LBBB, there is activation of the His Purkinje system and right bundle, followed by right to left interventricular septum activation and the rest of the left ventricle is depolarized in a cell to cell activation sequence (slow conduction). This leading to an absence of Q waves in lateral lead (reversal of interventricular septal activation from left to right to right to left in LBBB) and prolonged QRS duration (slow cell to cell conduction).

In some instances, if both the bundle branches are significantly diseased, the conduction system may not be involved in LV activation and the impulse originates from the ventricular myocardium travelling by cell to cell conduction and may also have a rightward vector of depolarization leading to further prolonged QRS duration and a presence of Q wave on lateral leads of surface ECG leading to WCT with LBBB like morphology.[8]

LBBB Tachycardia SVT or VT

Differential diagnosis of WCT with ***typical*** LBBB morphology encompasses following five clinical entities:

- Supraventricular tachycardia (SVT) with functional aberrancy,
- SVT with fixed aberrancy,
- Antidromic AV re-entrant tachycardia (AVRT) across the atrio-fascicular pathway,
- SVT with aberrancy with bystander atrio-fascicular pathway
- Tachycardia arising from bundle branch re-entry.

Table 1 : Electrocardiographic QRS morphology criteria to differentiate ventricular tachycardia from supraventricular tachycardia in WCT

Study author	Year of publication	Morphology of Tachycardia	Criteria to diagnose VT
Wellens *et al*	1978	RBBB like	Monophasic R in V1
			qR, QS, RS in V1
			rS, QS, qR in V6
			R/S < 1 in V6 (S > R or QS in V6)
			Left axis deviation
			QRS width > 140 ms
Kindwall *et al*	1988	LBBB like	R in V1 or V2 > 30 ms
			Any Q wave in V6
			Onset of QRS to nadir of S ≥ 60 ms in V1 or V2
			Notching of downstroke of S in V1 or V2
Akhtar *et al*	1988	LBBB like	Positive QRS concordance across the precordium
			Extreme left axis deviation (-90° to ± 180°)
			Right axis deviation
			QRS > 160 ms
		RBBB like	QRS > 140 ms
Brugada *et al*	1991		Absence of RS complex in all precordial leads
			R to S interval > 100 ms in ≥ one precordial lead
			Wellens' morphologic criteria in leads V1 or V6
Vereckei *et al*	2008		Initial R wave in lead aVR
			Initial r or q wave > 40 ms in lead aVR
			Notch on descending limb of negative onset, predominantly negative QRS in lead aVR
			vi/vt ≤ 1

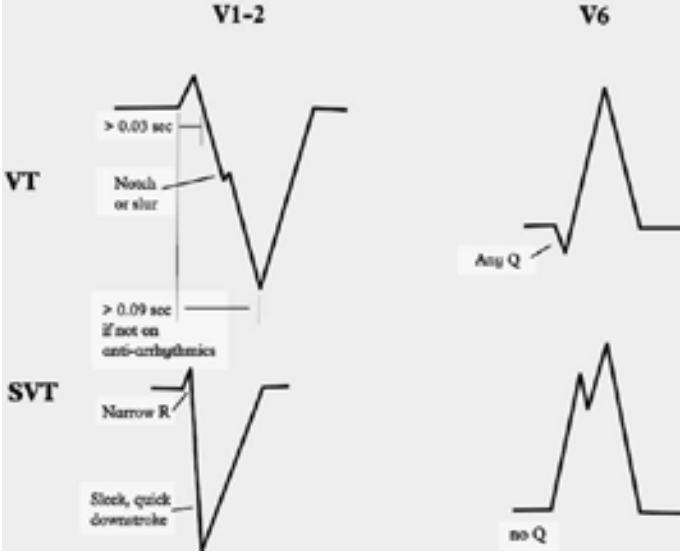

Figure 1 : Morphological criteria based on lead V1-2 and V6 to differentiate between VT and SVT proposed by Kindwall et al in 1988

Tachycardia not fulfilling the criteria for "typical" LBBB is termed as LBBB like tachycardia and encompasses entities like:

- Antidromic tachycardia using right sided WPW
- Right ventricular outflow tract (RVOT) VT,
- VT occurring in Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC),
- Tricuspid annular VT
- VT originating in scarred right ventricle.

Kindwall et. al in 1988 first described the following morphological features on ECG to differentiate tachycardia with LBBB morphology of ventricular origin from that of supraventricular origin (Figure 1). (1) R wave in lead V1 or V2 > 30 ms; (2) any Q wave in V6; (3) a duration of ≥ 60 ms from the onset of the QRS to the nadir of the S wave in V1 or V2; and (4) notching of the downstroke of the S wave in V1 or V2.[3] Various other algorithms described to differentiate SVT vs VT in WCT are summarized in Table 1.

SVT with Fixed or Functional LBBB

Any form of SVT or even sinus tachycardia in patients with pre-existing LBBB will demonstrate a typical LBBB tachycardia on surface ECG. The diagnosis of a pre-existing LBBB can be made by assessing their baseline ECGs. Alternatively a patient with narrow QRS rhythm at baseline who develops a broad complex rhythm during tachycardia may be related to the function aberrancy caused in the left bundle because of the encroachment of impulse travelling from the His bundle on to the refractory period of left bundle.[9] Left bundle can also remain refractory to impulse propagation by following mechanisms: (1) rapidity of antegrade impulse bombardment encroaching upon the refractory period of left bundle caused by previous antegrade impulse[10] (2) retrograde conducted ventricular premature complex (VPC) across the left bundle (3) transseptal activation of left bundle via the impulse travelling through the contralateral bundle (Figure 2).

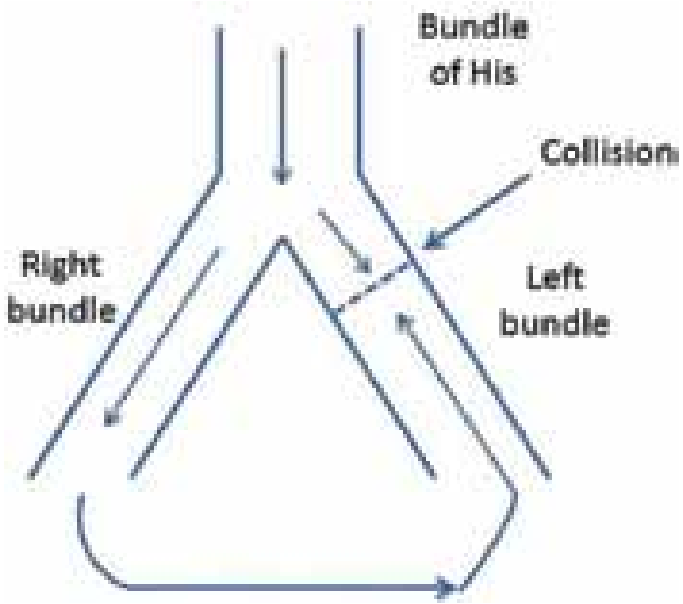

Figure 2 : Schematic representation demonstrating mechanism underlying functional LBBB caused by Repetitive transseptal retrograde concealed penetration of impulses conducting antegrade via the right bundle.

SVT with aberrancy accounts for upto 20% of patients with WCT and LBBB. This is also fairly common in young population <35 years.[11] It is also commonly observed in orthodromic AVRT with left lateral pathway.[12,13]

Atriofascicular Accessory ("Mahaim") Pathway and SVT with a Bystander Atriofascicular Pathway

Atriofasicular pathway originates in the atrial myocardium and bypasses the AV node and His bundle to get inserted over the distal right bundle or the adjoining myocardium. Peculiarity of this pathway is that they have conduction properties similar to the AV node ie. they demonstrate decremental conduction and Wenkebach phenomenon on rapid atrial pacing. These pathways are adenosine-sensitive. Antidromic AVRT results from antegrade conduction down the bypass tract and retrograde propagation *via* the normal conduction system (Figure 3 and 4).[15] Other right sided accessory pathway participating in

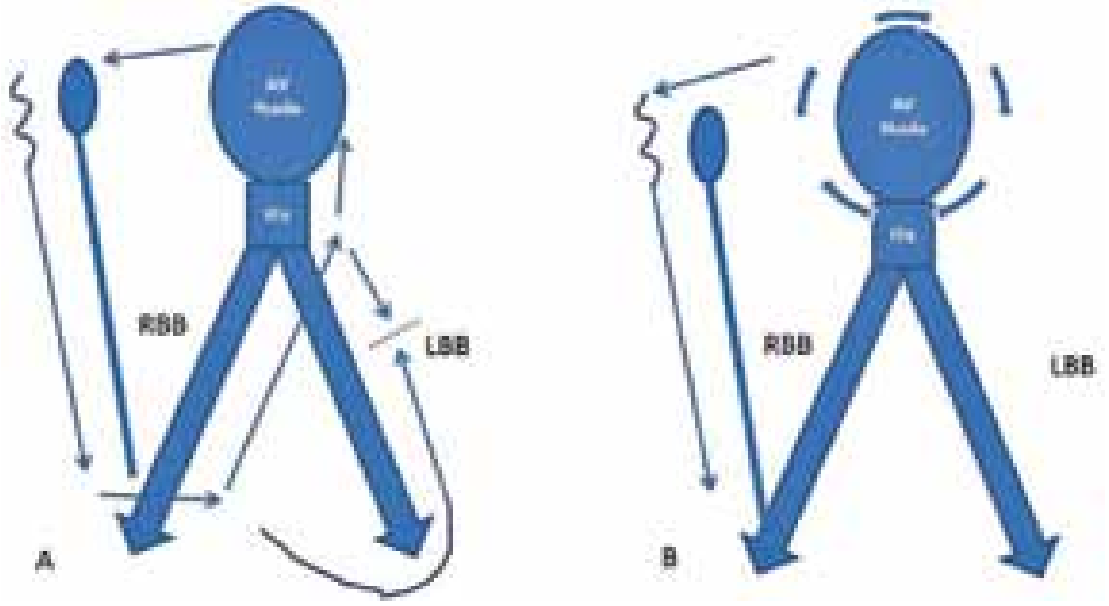

Figure 3 : Pre-excitation via atriofascicular ("Mahaim") bypass tracts. A: Antidromic AV reentry tachycardia. The tachycardia circuit conducts antegrade down the Mahaim fiber and inserts into the distal right bundle branch (RBB). It propagates retrograde back to the atrium via the more proximal portion of the RBB; B: Atrioventricular nodal reentrant tachycardia with a "bystander" Mahaim fiber present. The bypass tract is not part of the tachycardia circuit, but contributes to ventricular activation. A wide QRS complex will be present with a left bundle branch block configuration, but ablation of the bypass tract will not eliminate the tachycardia.

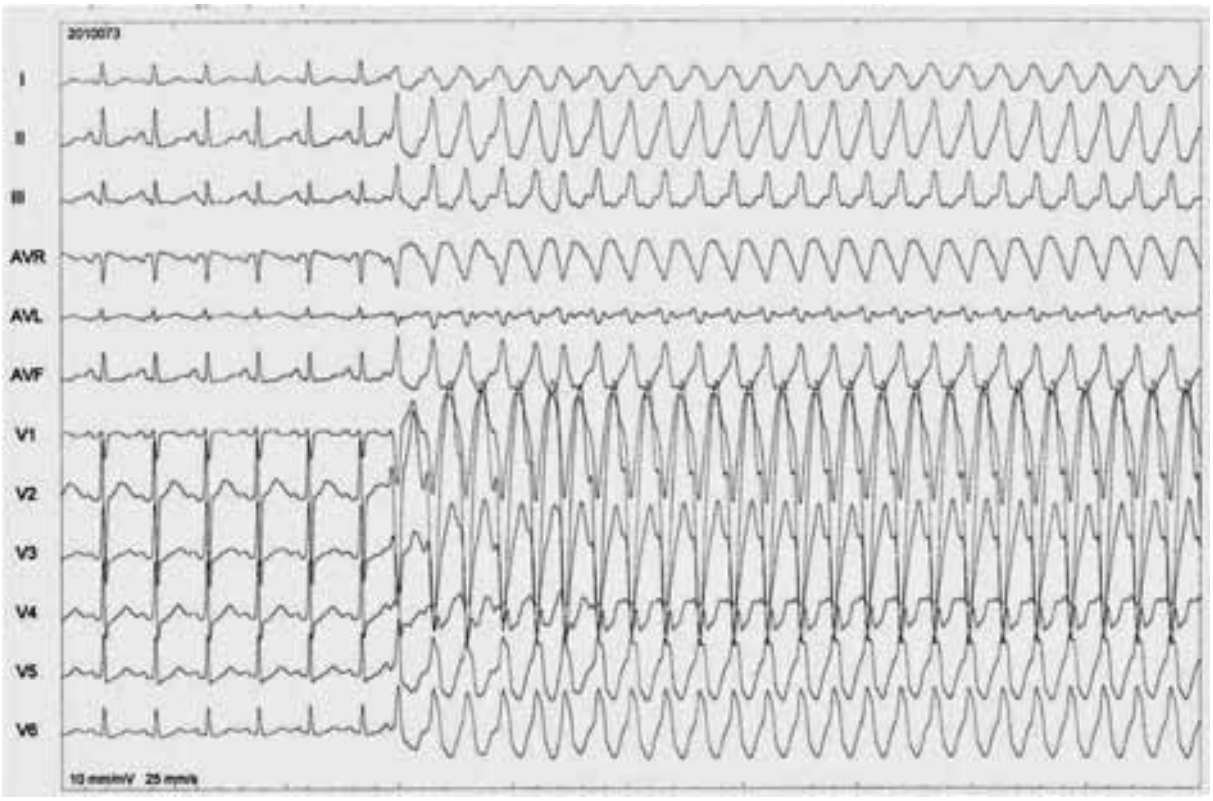

Figure 4 : ECG demonstrating conversion of sinus rhythm into WCT with LBBB because of antidromic AVRT involving atriofascicular ("Mahaim") tract as antegrade limb.

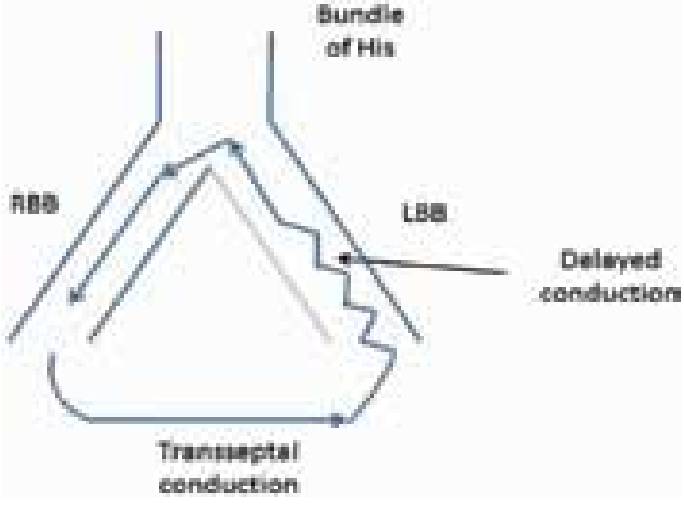

Figure 5 : Mechanism of bundle branch reentrant ventricular tachycardia. The right bundle branch (RBB) is the antegrade limb of the circuit, with retrograde conduction via the slowly conducting left bundle branch. This allows the RBB to recover and be capable of reactivation, thereby perpetuating the reentrant circuit. LBB: Left bundle branch.

antidromic AVRT do not demonstrate "typical" LBBB pattern as the distal limb is not inserted into the right bundle.

Radiofrequency ablation for Mahaim pathways is curative. The site where atriofascicular bypass tracts cross the tricuspid annulus is the preferred target for radiofrequency ablation. Acute success rates have been reported in the range of 90% to 100%.[17-19]

Bundle Branch Reentrant VT (BBRVT)

Bundle branch re-entrant VT is a variety of macroreenrant tachycardia occurring within the bundle branch. It was first described in 1989 by Caceres et. al.[20] They are seen in persons with cardiomyopathy and constitute approximately 6% of all monomorphic VT's. In cases of non-ischemic dilated cardiomyopathy with VT, BBRVT is the predominant underlying mechanism and is seen in upto 45% of patients.[21] It is also seen in patients with ischemic and valvular heart disease but with relatively low frequency (6-40%). Clinical presentation of this subgroup of patients is presyncope, syncope or sudden cardiac arrest.

The macro-reentrant circuit involves right bundle acting as the antegrade limb during the tachycardia circuit and any of the two fascicles of left bundle providing the retrograde limb. It results in a typical LBBB morphology tachycardia. Inciting event for the tachycardia is a VPB or a paced beat which finds right bundle refractory to conduct retrogradely and thus conducts retrogradely and slowly across the left bundle branch and then find the right bundle recovered and capable of antegrade

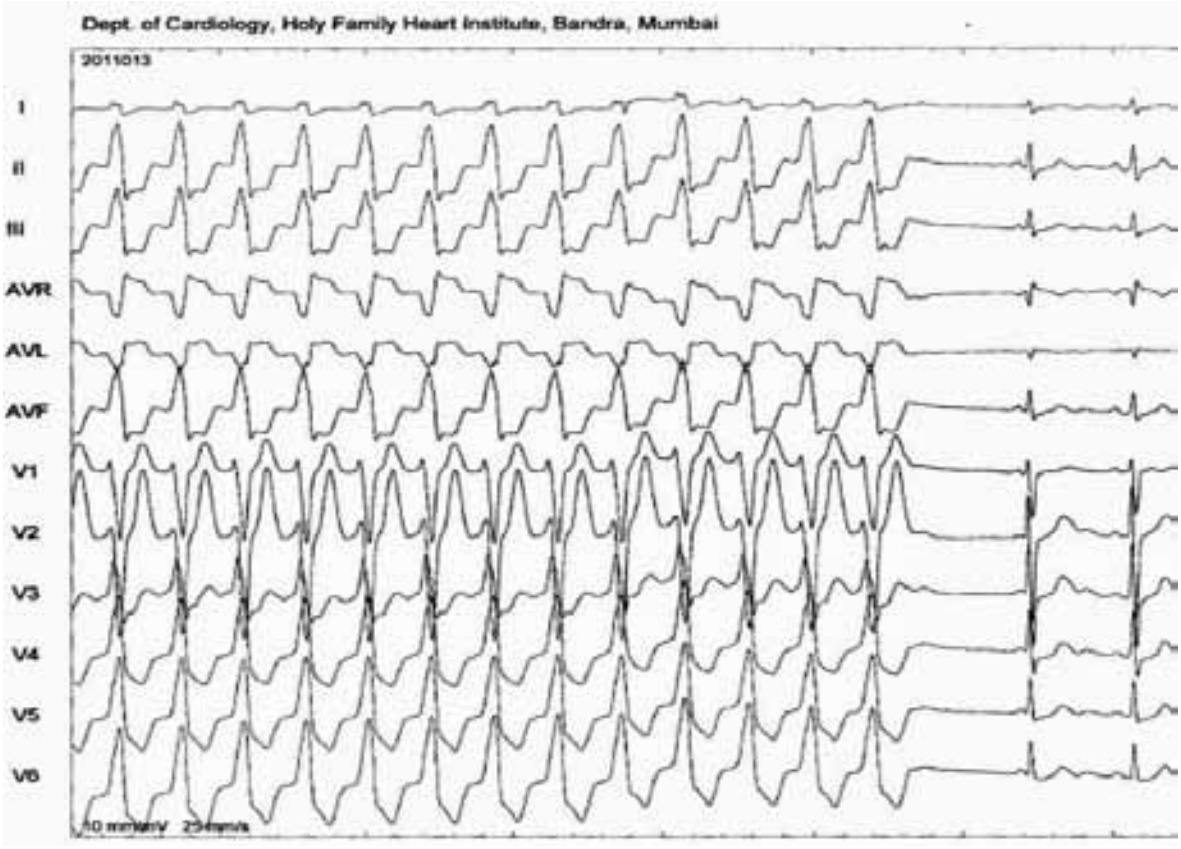

Figure 6. : ECG showing tachycardia with LBBB morphology and inferior axis. QRS transition in chest leads is in lead V3 which is typical of RVOT VT. It also shows spontaneous conversion of VT into sinus rhythm.

conduction. Subsequent retrograde left bundle conduction may perpetuate the sequence resulting in sustained tachycardia (Figure 5).

Sinus rhythm ECG in these patients demonstrates distal conduction system abnormalities in the form of prolonged PR and non-specific intra-ventricular conduction defect (IVCD). On electrophysiologic testing, conduction through the His-Purkinje system (baseline HV interval) is typically prolonged, averaging 80 ms (normal 35-55 ms) compatible with infra-hisian conduction disease. Management strategy for this tachycardia is radiofrequency ablation of antegrade right bundle, but with a definite risk of developing complete heart block if person has already existing left bundle disease. In view of coexistent structural heart disease these patients may have coexistent other VT's and thus ICD may be an option in well selected patients. Antiarrhythmic agents are usually ineffective in these patients.

RVOT VT

It is a form of idiopathic VT characterized by monomorphic VT arising from the right ventricular outflow tract in the absence of structural heart disease or coronary artery disease. The mechanism of VT is cyclic adenosine monophosphate triggered activity resulting from early or delayed after-depolarizations. RVOT VT has LBBB pattern in lead V1 with inferior axis (Figure 6). Stress, exercise, premature stimulation or isoproterenol infusion tends to initiate this tachycardia whereas adenosine and increased vagal activity can terminate the episodes of VT. While paroxysmal form of RVOT VT is related to exercise and stress, repetitive monomorphic type occurs at rest and is unrelated to exercise. LBBB morphology tachycardia can also arise from inflow portion or apex of the right ventricle. RVOT VT is usually well tolerated, but degeneration into rapid tachycardia and ventricular fibrillation is not uncommon. The prognosis of RVOT VT is good. While antiarrythmic drugs are effective, radiofrequency catheter ablation effectively eliminates this tachycardia with a success rate of >90%.

Tachycardia in ARVC

Patients with Arrhythmogenic right ventricular cardiomyopathy have VT that generally has a LBBB like morphology (because the tachycardia arises in the right ventricle), often with right-axis deviation and T waves inverted over the right precordial leads. The VT may be caused by reentry. ARVC can be an important cause of VT in children and young adults with apparently normal hearts, as well as in older patients. Initial findings can be subtle and often mimic those of outflow tract VT that is, manifested only by tachycardia and no symptoms

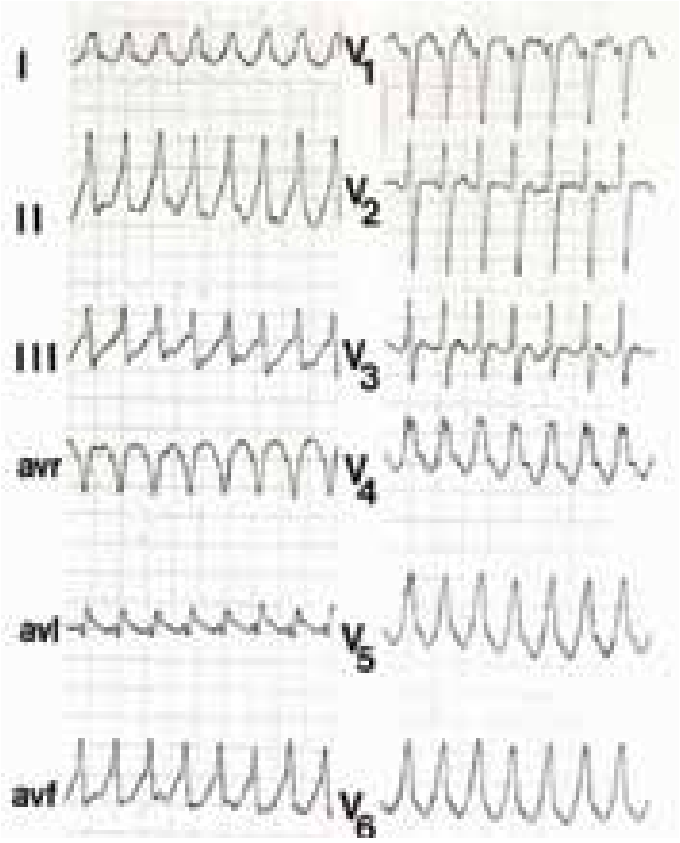

Figure 7 : ECG showing scar ventricular tachycardia with typical LBBB morphology

of right-sided heart failure. The ECG during sinus rhythm can exhibit complete or incomplete RBBB and T wave inversions in V_1 to V_3. A terminal notch in the QRS, called an epsilon wave, can be present as a result of slowed intraventricular conduction. ICDs are generally preferable to pharmacological approaches because of the progressive nature of the disease and poor prognosis, particularly if the patients have poorly tolerated VT, resulting in syncope or sudden cardiac death. Radiofrequency catheter ablation can be tried but is often not successful because of the multiple morphologies of VT and the progressive nature of the disease.

Tricuspid Annular Ventricular Tachycardia

Idiopathic VT can also arise from tricuspid annulus in small proportion of patients. Tricuspid annular tachycardia usually arises in the septal region and manifests as LBBB pattern i.e. Qs in lead V1 with narrow QRS and early transition in precordial leads.[14] Treatment and prognosis for annular VT is similar to outflow tract VT and radiofrequency catheter ablation is effective modality for treating the arrythmogenic foci.

Scar Ventricular Tachycardia

Previous myocardial infarction is independent risk factor for the development of VT due to infarct scar. Deranged conduction leads to reentry particularly at the border zones of the scar and manifests typically as monomorphic VT (Figure 7). At times it can be polymorphic VT resulting from different exit sites from the same circuit leading to different activation patterns of the rest of the left ventricle. Patients with compromised left ventricular function benefit from implantable cardioverter-defibrillator (ICD). Monomorphic VT in this setting often responds to antitachycardia pacing via ICD. In hemodynamically stable patients with normal LV systolic function antiarrythmic drugs, radiofrequency ablation and/or ICD implantation are the different options. Refractory cases may benefit from endocardial resection of the scarred area.

Management of WCT with LBBB Morphology

Patients with WCT are usually a source of anxiety to the treating physicians. But if the presenting ECG is of typical LBBB morphology the diagnostic possibilities are restricted to five entities mentioned above. This makes life simple and thus allows early and appropriate therapeutic strategy. If a prior ECG is available, it should be reviewed for the possible clues. LBBB on the resting ECG is highly suggestive of the WCT being SVT with fixed aberrancy. When LBBB is present on a resting ECG during sinus rhythm the QRS morphology during SVT with fixed aberrancy typically matches precisely.[22]

Patients with fixed LBBB may show electrical alternans during SVT as compared to sinus rhythm. However this alternans is rate related and does not help differentiate SVT from VT.[23] Those with BBRVT have evidence of severe conduction disease in the form of PR prolongation, IVCD or trifascicular block on baseline surface ECG. In patients with atrio-fascicular bypass tract baseline ECG shows morphology caused by fusion of impulse travelling across the tract and antegrade conduction across the node. However, during antidromic AVRT the morphology is of typical LBBB as the antegrade limb is through the bypass tract. Other features suggestive of atriofascicular preexcitation include a short PR interval (during sinus rhythm) and late (after lead V4) transition of the QRS complex.

In a hemodynamically stable patient with ***typical*** LBBB morphology WCT it may be worth attempting a rapid AV blocking measure in the form of intravenous adenosine or quick vagal manoeuvre as an initial strategy, as many of the etiologies respond to it very well. However, DC cardioversion facility should always be on a standby if conduction propagated along the aberrant pathway leading to VF. The safety and efficacy of this strategy has been demonstrated by Marill et.al in 2009.[1] AV nodal blocking agents have both diagnostic and therapeutic implications as they would treat AVNRT and AVRT by blocking the node and will also demonstrate the supraventricular rhythm in cases with sinus tachycardia, automatic atrial tachycardia and atrial fibrillation with fixed LBBB.

BBRVT should be suspected in patients with typical LBBB tachycardia and significant structural heart disease.[24] BBRVT

is the only one of these clinical entities that usually exhibits no response to adenosine. Although administration of adenosine occasionally may result in VA dissociation during hemodynamically stable VT, it should be noted that BBRVT is usually rapid and hemodynamically unstable, making DC cardioversion the initial option of choice. For any wide complex rhythm with hemodynamic instability DC cardioversion should always be the initial strategy.

Long-Term Management

In patients with SVT and LBBB, treatment of the SVT is required. Catheter ablation is the preferred treatment for AVNRT and AVRT involving atrio-fascicular pathway.[25,26] Atial flutter and atrial tachycardia are also amenable to catheter ablation. Treatment of cause is required in patients with sinus tachycardia. BBRVT can also treated with catheter ablation of right bundle, however with a definite risk of developing complete heart block if there is conduction abnormality affecting the left bundle. Patients with previous MI and scar related VT leading to WCT will benefit from ICD.

Conclusion

In patients with WCT with LBBB morphology, it is important to differentiate between those with typical LBBB and LBBB like tachycardia. The clinical features and electrophysiologic characteristics of tachycardia with a typical LBBB and LBBB like pattern have been outlined above. A clear understanding of their mechanisms should facilitate tachyarrhythmia management. Administration of adenosine is usually safe in the presence of hemodynamic stable WCT of ***typical*** LBBB morphology and may aid in making the correct diagnosis. Long-term management strategies usually require referral to an electrophysiologist for catheter ablation or ICD implantation.

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Case Vignette : Torsade's De Pointes Post Permanent Pacemaker Implantation

Anup Taksande[a], Ajay Mahajan[b], Yash Lokhandwala[c]
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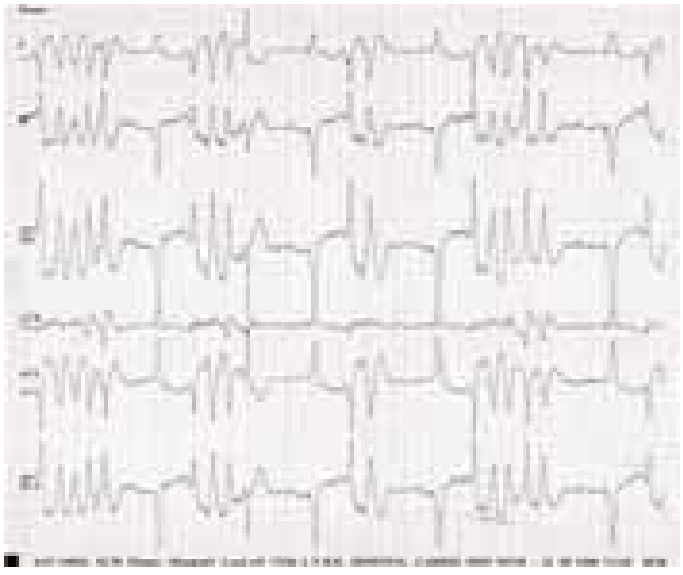
Clinical scenario

This is a 60 year old lady, diagnosed case of hypertension since 4 years on sporadic treatment, presented with history of giddiness and blackouts since last 3 months. Patient presented to Emergency room with repeated episodes of syncope over the past 24 hours. ECG performed in the emergency room revealed complete heart block with an atrial rate of 80/min with broad ventricular escape Rate of 32/minute. Patient was hemodynamically unstable and was immediately paced using a right internal jugular venous access. Workup for any reversible causes for CHB was negative. Renal Function Tests, Serum K+, Cardiac Biomarkers, Thyroid Function Tests were negative. There was no prolonged history of use of beta blockers or any other AV nodal blockers. 2 D Echo revealed briskly contracting LV with LVEF of ~ 60-65% and Mild Pulmonary Hypertension.

After pre-operative testing, a permanent pacemaker implant was performed using left subclavian approach; per patient preference, single chamber VVI Rate Responsive (lower rate of 70 bpm/min) pacemaker was placed. Procedure was uneventful, patient was shifted for observation.

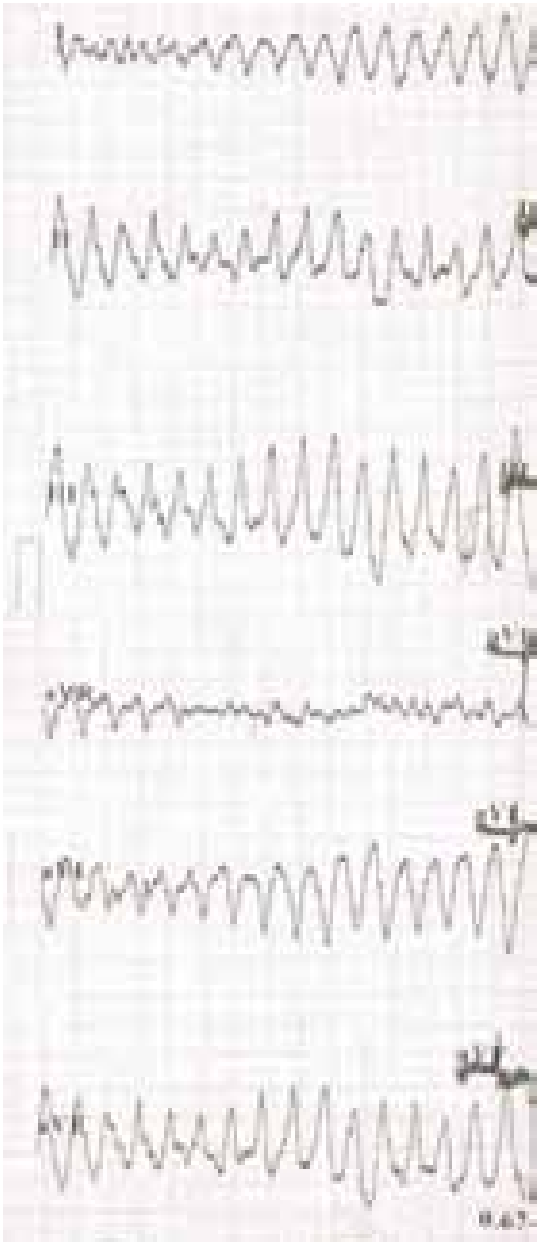
Patient remained asymptomatic for next 24 hours. Day 2 patient had recurrence of symptoms with abnormal rhythm on telemonitor.



Though various possibilities could be considered, it is clear that pacing function is maintained as and when it is needed. However, there are runs of non-sustained VT which appears to be Torsades de pointes.

Patient had multiple episodes of cardiac arrest which required external defibrillation.

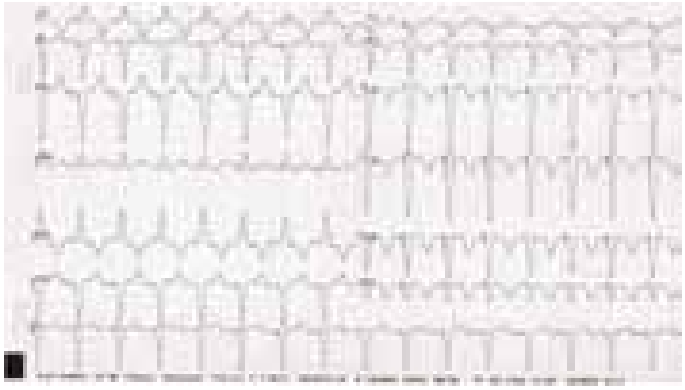
The ECG at the time of defibrillation is shown:



There was no evidence of ischemia and hence this was not an ischemia related polymorphic VT. This makes use of Lidocaine futile. Ideal choice of drug at this time was IV Magnesium. The

patient was treated with 2gm Mg in 100ml D5W as loading dose over 10 minutes.

Patient immediately reverted back to Paced rhythm with no further episodes of ectopy. There were no further episodes of Torsades after IV Mg 2+.



Magnesium is the drug of choice for suppressing EADs and terminating the arrhythmia. Magnesium achieves this by decreasing the influx of calcium, thus lowering the amplitude of EADs (1).

Based on the ACC/AHA/HRS guideline, there IV Mg has Class IIA indication (Level of Evidence B). Lidocaine is only useful in cases of polymorphic VT specifically associated with acute myocardial ischemia or infarction (Class IIb Level of Evidence: C). Role of pacing in treatment of Torsades has been established only when it is associated with heart block and symptomatic bradycardia. (Class I Level of Evidence: A), or with frequent rest pauses (Class II a , Level of Evidence B).

Review

Torsade de pointes is an uncommon and distinctive form of polymorphic ventricular tachycardia characterized by a gradual change in the amplitude and twisting of the QRS complexes around the isoelectric line.

This was originally termed torsade de pointes, or "twisting of the point" about the isoelectric axis, because it reminded the authors of the torsade de pointes movement in ballet.

Torsade de pointes, often referred to as torsade, is associated with a prolonged QT interval, which may be congenital or acquired.

Etiology

- Congenital long QT syndrome
  - Six genetic variants: LQT1 and LQT2: Slow K channel mutation.

    LQT3 Na channel mutation
- Acquired long QT syndrome :
  - Drugs (Antiarrythmics Class 1A, 1C and Class 3, Antipsychotics, newer Antihistaminics, Antibiotics, Antifungals etc.)
  - Hypokalemia
  - Hypomagnesemia
  - Ischemia/ infarction related
- Bradycardia: Bradyarrythmias with R on T phenomenon as in Complete Heart Block.

Pathophysiology

A variety of changes in ionic current can result in the common effect of decreased repolarizing current, reflected in a long QT, and these changes can secondarily lead to subsequent depolarizing currents termed *afterdepolarizations*. This leads to a further delay in repolarization and causes early afterdepolarization (EAD), the triggering event for torsade.

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MRI Safety of ICDs and Pacemakers

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Introduction

MRI scanning is now the imaging modality of choice for a number of neurological, vascular, or musculoskeletal conditions. Some authors have estimated that up to 50-75% of patients with either a pacemaker or implantable cardioverter-defibrillator (ICD) would have an indication for an MRI scan.[1] Until several years ago, exposure of patients with pacemakers or ICDs to an MRI environment was considered contraindicated by medical experts[2] and the U.S Food and Drug Administration.[3] Several series have suggested that appropriately selected and monitored patients with pacemakers or ICDs may undergo an MRI scan with low risk.[4-6] Medical societies in both Europe and the United States have acknowledged these findings with publications of expert opinion and guidelines on scanning patients with implanted cardiac devices.[7,8] In 2010, the safety of the first MRI conditional pacemaker was published.[9] The purpose of the current manuscript is to review the data on the risks to patients with implanted pacemaker or ICDs exposed to an MRI scan, and to review steps to be considered if scanning a patient with either an implanted pacemaker or ICD.

MRI Physics Overview

Magnetic resonance imaging (MRI) is a diagnostic technique that images body parts by producing a static magnetic field, followed by rapidly changing magnetic and electromagnetic fields to excite hydrogen atoms. The three main components of MRI imaging are the static magnetic field, radiofrequency energy (RF), and gradient currents, each of which can affect a cardiac implantable electronic device (CIED).

The static magnetic field is called the B_0 field. The Net Magnetization Vector (NMV) is the relative balance between spin-up and spin-down nuclei as the magnetic moments of the hydrogen nuclei orient themselves parallel or anti-parallel to the B_0 field. The relation between the NMV and static magnetic field is the basis for MRI. As the magnetic field strength increases, the NMV also increases, resulting in improved magnetic signal.[10] The strength of the static magnetic field is expressed as Tesla (T). One Tesla is equal to 10,000 Gauss (G). The earth's magnetic field is approximately 0.6 G. Most MRI scans performed today use a static magnetic field strength of 1.5 T or 3 T.

The static B_0 field produces an additional spin (wobble) of the magnetic moment of the hydrogen nuclei around B_0. The speed of the "wobble" around B_0 is the "precessional" frequency. "Resonance" occurs when external energy is applied that has a frequency close to its frequency of oscillation. "Excitation" is the application of energy that causes resonance to occur, such that the nuclei gain energy as an increase in amplitude of its oscillation at its natural frequency. The precessional frequency is defined by the Larmor equation.[10]

$$\Omega_0 = B_0 \cdot \lambda$$

(λ = gyro-magnetic constant = 42.57 Mhz/T for ^{1}H)

In clinical MRI, the precessional frequency of ^{1}H at all clinically relevant B_0 corresponds to the RF band of the electromagnetic spectrum.

RF exposure is measured as the Specific Absorption Rate (SAR). SAR is the rate of energy deposition and is expressed as the power absorbed per unit mass (W/kg). SAR can be thought of as a rate of heating from energy deposition where a SAR of 1 W/kg over 1 hour would result in a temperature rise of 1˚C. For a sphere of tissue of radius r, SAR is proportional to the square of the B_0 field.

$$SAR \propto \sigma r^2 B_0^2 \alpha^2 D$$

σ = conductivity

α = flip angle

D = pulse duty cycle

Comparing SAR measurements between various manufacturers can be problematic.[11,12] During a 1.5 T MRI scan, the RF wavelength approximates the length of a standard pacemaker lead.[13]

Gradient fields are used in MRI pulse sequences in the process of spatial encoding that determines the spatial locations of RF signals. These fields are created using electromagnetic coils that are turned on and off rapidly. This rapid cycling can potentially induce currents in conductive wires that potentially may lead to cardiac stimulation.

Electromagnetic interference (EMI) consists of a source, receiver, and path. Electromagnetic radiation source interacts with another receiver device in an unintended way over a prescribed path. Interference may be conducted (direct contact) or radiated. The frequency of the EMI determines the resulting effect on the receiver device. EMI can be prevented by modifying one of the three components: source, receiver, or path.

Risks for an MRI Scan

Because of these strong fields, potential adverse interactions between MRI scanners and devices may occur, including tissue heating, induction of ventricular fibrillation, rapid atrial pacing, rapid ventricular pacing, reed switch malfunction, asynchronous

pacing, pacemaker inhibition, damage to pacemaker circuitry, and device movement. Many of the studies suggesting adverse interaction between pacemakers and MRI scanners were performed with older devices and circuitry.[14,15] More recent trials suggest that the year of manufacture is important for device safety, especially for implantable cardioverter-defibrillators implanted before 2000.[16]

Tissue heating from exposure of the leads to the RF fields remains a significant concern for patients with implanted devices exposed to an MRI scan. Both phantom gel *in vitro* and *in vivo* animal models have shown tissue heating to greater than 20°C.[11,16,17,18] Leads may conduct energy generated from the RF fields that can lead to resistive tissue heating at the lead-tip interface. *In vitro* studies have shown that temperature changes correlate with SAR levels as well as position of the device and lead inside the MRI bore.[19] Necropsy studies of animals with implanted devices exposed to worse-case scenario scans showed no local tissue necrosis following the MRI scans.[16,19] Blood flow may have a cooling effect and limit some of the heating effects. Lead design may influence amount of tissue heating. Transient or permanent changes in device thresholds may occur: capture threshold, sensed amplitude, and lead impedance. Significant pacing threshold changes are very uncommon. Despite the concerns about local tissue heating, no correlation has been seen between threshold changes and peak SAR in a clinical setting.[5]

Fontaine, *et al*. first reported an episode of rapid ventricular pacing associated with RF pulses during an MRI scan despite the fact that the device was programmed to subthreshold pacing.[20] Runaway pacemaker is a theoretical risk that may occur with device circuitry damage, but is very unlikely to occur with modern circuitry design and shielding in devices manufactured after 2000. This behavior is better described in the radiotherapy environment.

The rapid cycling of magnetic fields used to generate gradient may potentially generate currents that can lead to cardiac stimulation. Tandri, *et al*. showed that gradient field-induced currents were less than or equal to 0.5 mA, and that current strength is related to the effective loop area of the lead.[21] In our own experience, most ventricular ectopy seen was from noise reversion mode pacing behavior at the lower pacing rate from excessive noise detected on the pacemaker that had not been programmed to subthreshold pacing output.[22]

The excessive noise detected by a pacemaker or ICD can lead to variable device behavior including noise reversion behavior, upper tracking behavior, mode switching, and power on reset behavior. Patients who are pacemaker dependent can have failure of appropriate pacing function if the pacing inhibition occurs because of excessive noise detected by the pacing system. In patients with an ICD, noise during an MRI scan can be interpreted by the ICD as ventricular fibrillation leading to inappropriate device charging that in turn can cause generator damage. Some pacemaker models are prone to noise revision or power on reset behavior when exposed to excessive noise coming from strong EMI sources. Frequently, the pacing modality in these back-up pacing modes is VVI, which can lead to potentially catastrophic device pacing inhibition in pacemaker dependent patients.[23] Though magnets are frequently used to program devices to asynchronous pacing behavior during surgery, reed switch behavior is unpredictable in the static magnetic field of current MRI systems and may be open or closed.[24]

The effect of an MRI on battery longevity, especially of implantable cardioverter-defibrillators, remains unclear. Naehle, *et al*. reported a significant decrease in reported battery status,[25] while Buendía, *et al*. reported no significant change.[26] The assessment of battery status is limited by the devices' methods of reporting battery longevity, and is frequently only an indirect measurement of battery longevity.

Clinical Experience

A number of series have reported on the relative safety of scanning patients with cardiac pacemakers or ICDs. In the first large series published, Martin, *et al*. reported on 54 patients undergoing 62 scans.[27] In their series, 9.4% of patients had significant pacing threshold changes, of which 1.9% required device reprogramming. This study though dichotomized changes as "any change/no change" and "any significant change/no significant change" and did not report impedance or sensed amplitude changes. In a series reported by Sommer, et al., 82 patients underwent 115 scans. An increase in pacing thresholds was seen in 3.1% of leads and there was a significant fall in lead impedance.[4]

We reported our experience in patients undergoing an MRI scan without SAR restriction.[5] In that series, 103 patients underwent a total of 127 scans without any significant change in pacing thresholds immediately post-scan, though small changes in sensed amplitude and pacing impedances were seen. SAR did not predict threshold changes. In our unpublished experience, small changes in sensed amplitude and pacing impedances recover within an after the scan. The most significant change in pacing threshold amplitude occurred in an atrial lead following a brain scan from 1.2 V at 0.5 msec at baseline to 3.1 V at 0.5 msec one hour later. At three month follow-up, the threshold remained 3.1 V at 0.5 msec. No lead or generator has required surgical revision in our experience.

In the largest series published to date, Nazarian, et al. reported on the results from 438 patients who underwent 555 scans.[6] In this series, immediately post scan statistically significant changes were seen for right ventricular sensed amplitudes and pacing impedances, but no pacing threshold changes were seen. At long-term follow-up, statistically significant changes were found for right ventricular sensed amplitude, lead impedance and pacing capture threshold. No patient, though, required system revision. Correlation between immediate post-scan and

long-term follow-up was not reported, and there was no control population.

In 2011, the first study of the safety of a MR-conditional pacemaker was published.[9] A MR-conditional device is one that that has no known risks in a specified environment and condition of use. Design changes of the pacemaker for the MRI environment included reduction of ferromagnetic components, improvement in internal circuitry, use of a Hall sensor instead of reed switch, and redesign of the pacemaker lead including reduction of inner coil filars from four to two. In this study, 456 patients receiving the device were randomized between those undergoing and MRI scan and those who did not. Scans were limited to 2 W/kg, and had to be above spinal level C1 and below T12. In this study, no significant complications including induced arrhythmias, inappropriate device behavior, or significant threshold changes.

Clinical Protocol

At our facility, all patients with an implanted device who need MRI undergo a pre-MRI screen. They should have a chest x-ray within 6 months of the scan to evaluate for lead fractures or retained leads. At the pre-MRI screen clinic visit, patients are assessed for device dependency, which we define as a native ventricular rhythm less than 40 bpm or symptomatic bradycardia if the native rhythm is less than 60 bpm. Battery longevity, device model, and device features are noted in light of the patient's clinical status. If the patient is a candidate for an MRI scan, risks are reviewed with the patient, written consent is obtained, and a prescription is written for our pacemaker nurses to follow during the scan. All scans are ordered by a referring provider.

Exclusion criteria included retained or fractured leads, specific generator with known problems in a MRI scanner, ICD models without asynchronous pacing modality in patients that are device dependent, device generators at ERI or EOL, or inability to provide informed consent. No restriction is placed on body landmark. The limit on SAR in most countries is 4 W/kg for a 1.5 T scanner.

During a scan, a pacemaker nurse is present in the MRI suite during the scan, programming the device as directed in the prescription. If the patient is device dependent, a physician is also present in the MRI suite. An ACLS trained person is available for all scans, and resuscitation equipment is present in the suite including transcutaneous pacing. If a patient is device dependent, the device is programmed to DOO/VOO modality at maximum output. If the patient is not dependent, the device is programmed to ODO/OVO modality or to DDI/VVI with subthreshold pacing. ICD tachy-therapy is disabled. All logs and histograms are retrieved and printed out before the scan. Thresholds of all leads are evaluated pre- and immediately post-scan. If a patient is device dependent, thresholds are repeated an hour later.

Conclusion

MRI scans can be performed safely in patients with contemporary CIED devices if specific protocols and evaluations are performed. The focus should be on the management of the patient, not the device: What does the patient require to get through the scan without damage, not what the device needs to get through the scan without damage

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Culprit Vessel Localization in Coronary Artery Disease and Acute Myocardial Infarction : Importance of ECG and Stress ECG Test

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Abstract

Coronary artery disease (CAD) is still single greatest cause of death of men and women in India. Studies by the National Commission for Macroeconomics and Health, Government of India, suggest that the number of patients with coronary artery disease is set to increase to over 60 million by 2015, which would represent about 7.6% of the adult population[1].

Exercise EKG Testing remains an important tool for diagnosis of chest pain to provide clues for Coronary Artery Disease[2]. However, it does not provide much information regarding the Culprit Vessel Localization. ST elevations on exercise in Anterior or Inferior Leads with no underlying Q waves point towards the Culprit Vessel.

Culprit vessel localization is of paramount importance in any case of acute myocardial infarction (AMI) as it has diagnostic and prognostic implication. In general, the more proximal the site of occlusion, the prognosis is less favourable. Exercise stress testing is an important diagnostic tool for the evaluation and finding out the culprit vessel responsible for the infraction. Despite the diffuse nature of atherosclerosis, culprit coronary lesions responsible for ST-segment elevation myocardial infarctions (STEMIs) are known to cluster in proximal coronary arteries[3,4].

Exercise testing has been validated and adopted for risk stratification before discharge in patients with acute coronary syndromes for several years[5,6]. In addition to providing important prognostic information, exercise testing has also been used to select high-risk patients with provocable myocardial ischaemia (MI) suitable for revascularization[7].

The role and timing of exercise stress testing in myocardial infarction also plays important role in management of patients with AMI. The first step in management of these patients is to classify them by using following criteria for risk stratification[8,9].

Key words : Acute Coronary Syndrome, Acute Myocardial Infarction, Culprit Vessel Localization, Electrocardiogram (ECG), Stress ECG Test.

Culprit Vessel Localization in AMI on ECG[10]

Culprit Vessel Localization can be predicted in AMI settings quiet accurately. Anterior Wall MI occurs because of the occlusion of Left Anterior Descending Artery (LAD) and Inferior Wall MI occurs because of the occlusion of either Right Coronary Artery (RCA) or Left Circumflex Artery (LCx).

Anterior Wall Myocardial Infarction (MI)[10]

In Anterior Wall MI, the vessel involved is predicted from precordial leads and the site of occlusion can be assessed from the limb leads. Patient presenting with typical symptoms and ECG showing ST segment elevations in the precordial leads, point towards LAD occlusion.

If occlusion is proximal to first septal and first diagonal (or left main), there is ST segment elevation in aVR and aVL, as the basal myocardium is involved and the vector is facing towards the above leads.

If LAD gives first diagonal (D1) first and then first septal (S1) and the occlusion is between D1 and S1, there is ST elevation in aVR and if LAD gives S1 first and then D1, and the occlusion is between S1 and D1, then ST elevation is seen in aVL. If the occlusion is distal to Diagonal and Septal, then there will be associated ST elevation in inferior leads (Lead II, III and aVF).

Occasionally, Left Main or proximal LAD occlusion will show generalized ST segment depressions and ST elevation only in aVR.

Further, in Anterior Wall MI, Right Bundle Branch Block (RBBB) again points towards proximal LAD occlusion.

Left Bundle Branch Block (LBBB) in Anterior Wall MI setting is very rare as posterior fascicle of Left Bundle Branch has dual blood supply.

AV Block and Bundle Branch Blocks in AMI settings and its implications will be discussed more in detail.

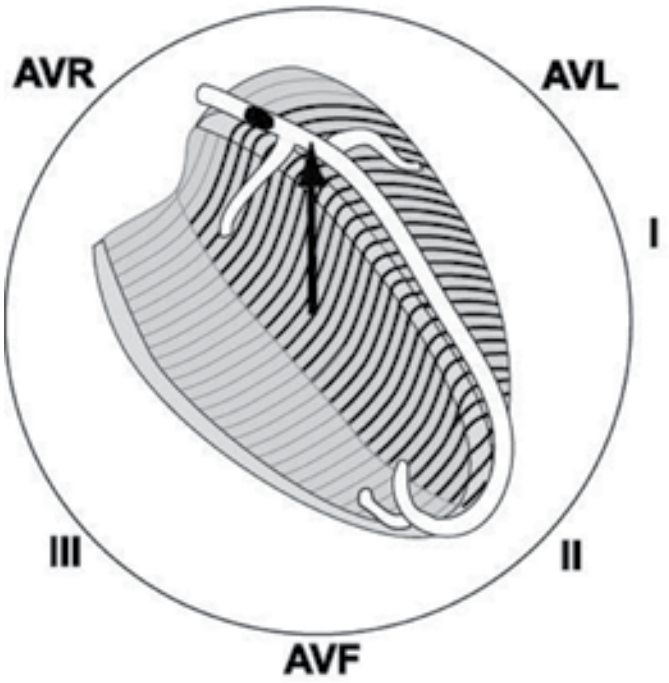


Figure 1 : Showing LAD occlusion proximal to S1 and D1

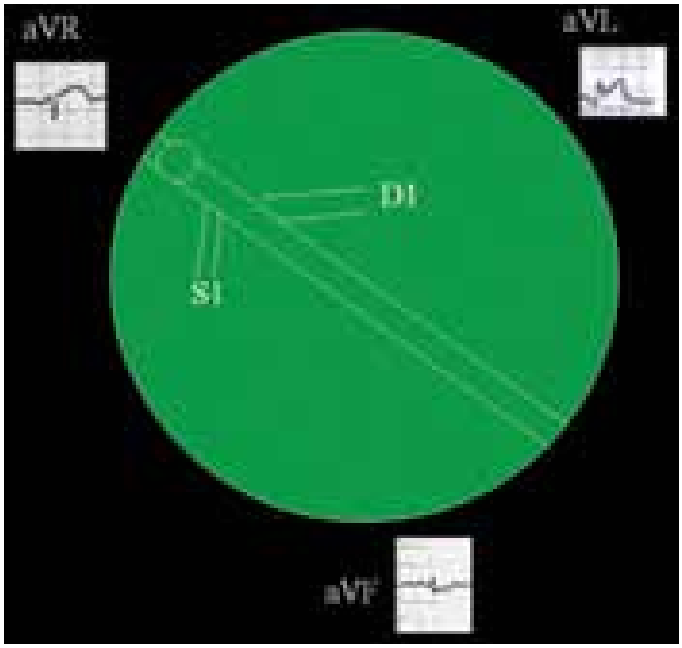


Figure 2 : Showing LAD occlusion proximal to S1 and D1

If ECG shows inverted T waves in the absence of pain and the same become upright during pain, it prompts Critical Proximal LAD Occlusion.

Inferior Wall MI[10]

In Inferior Wall MI (IWMI), culprit vessel is predicted from limb leads and site of involvement is predicted from precordial leads. ECG in Inferior Wall MI shows ST segment elevation in

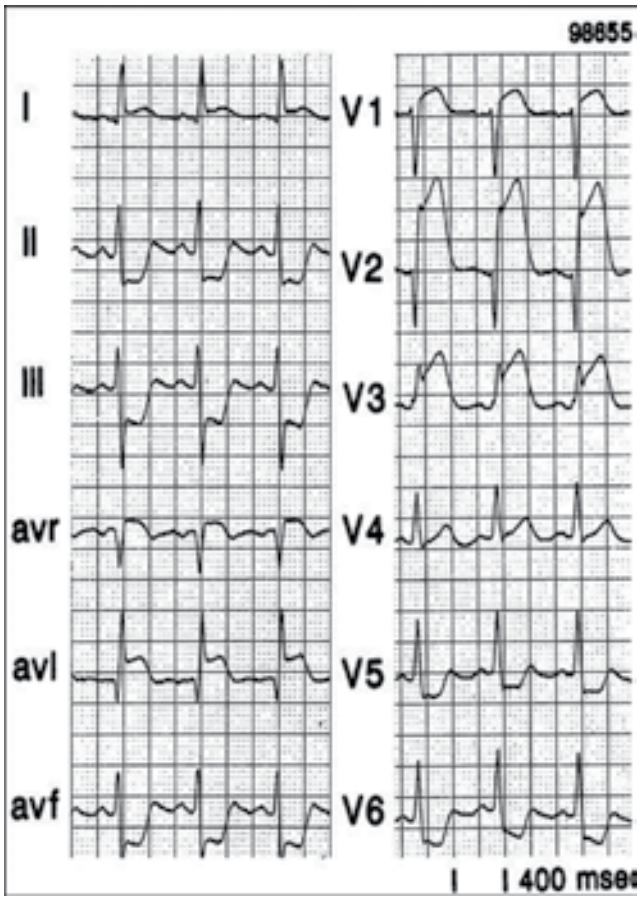


Figure 3 : ECG in Anterior Wall MI due to proximal occlusion of LAD

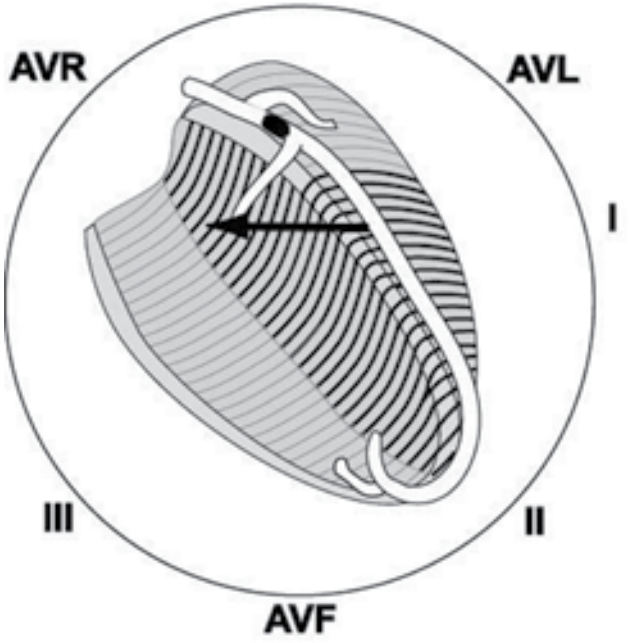


Figure 4 : Showing LAD occlusion distal to D1 and proximal to S1

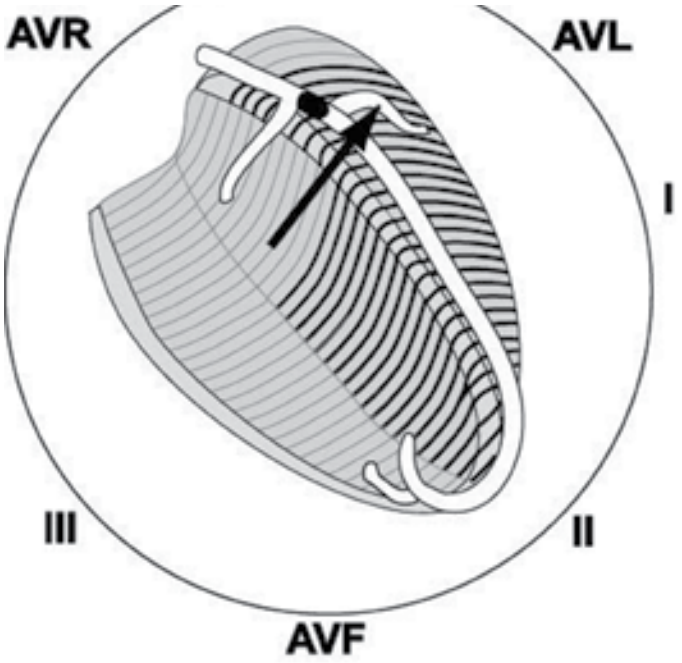


Figure 5 : Showing LAD occlusion distal to S1 and proximal to D1

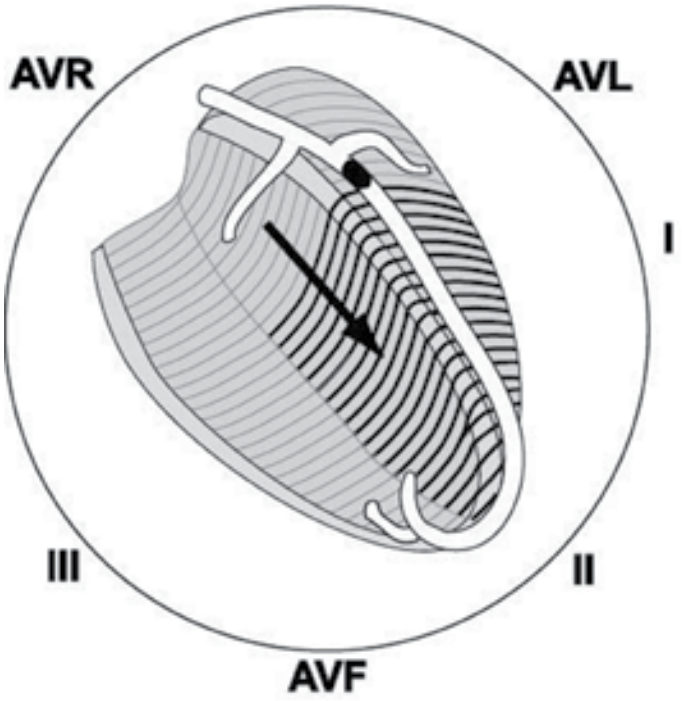


Figure 6 : Showing LAD occlusion distal to S1 and D1

LII, LIII and aVF. The culprit vessel in IWMI is either RCA or LCx. The vector of myocardial damage in RCA is towards LIII and in LCx is towards LII. In IWMI due to RCA involvement the ST segment elevation is more in LIII as compared to LII and in LCx involvement, ST segment elevation is more in LII as compared to LIII.

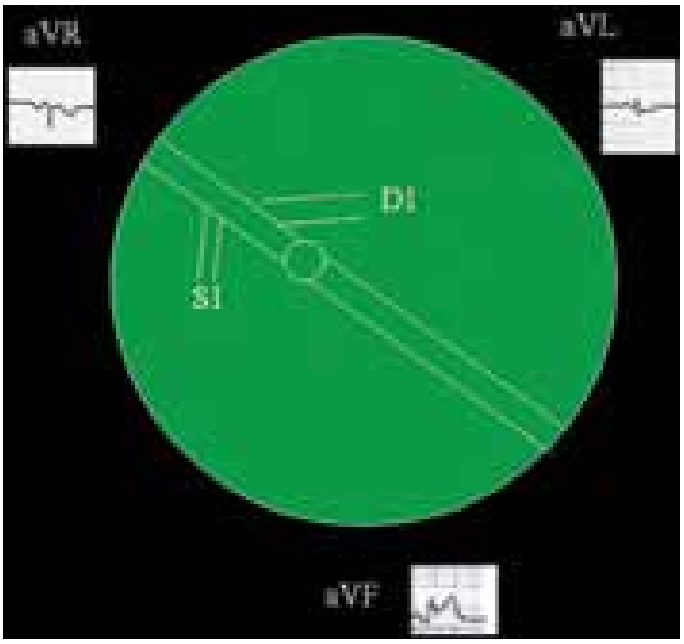


Figure 7 : Showing LAD occlusion distal to S1 and D1

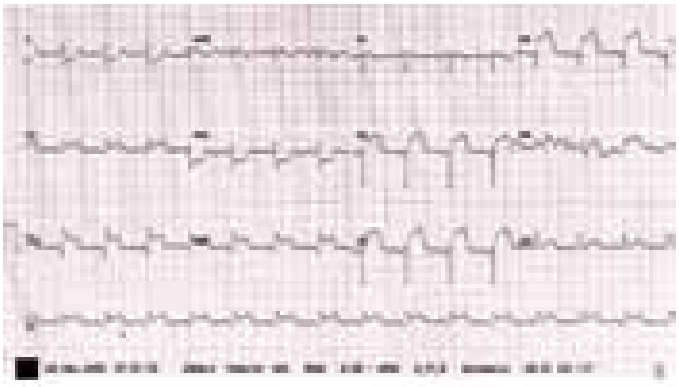
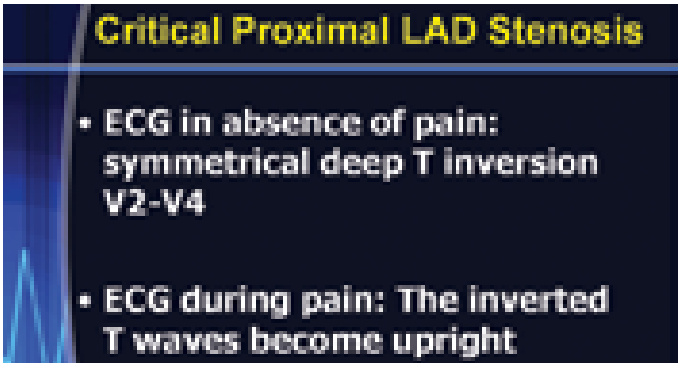


Figure 8 : ECG in Acute Anterior Wall MI with LAD occlusion distal to S1 and D1



Lead V4R in IWMI gives good information about the site of obstruction. However, V4R shall be recorded early in the course of AMI as the changes are short lasting and may not be seen after 3-4 hours. ST elevation in V4R is seen in proximal occlusion of RCA and not seen in distal RCA occlusion. While

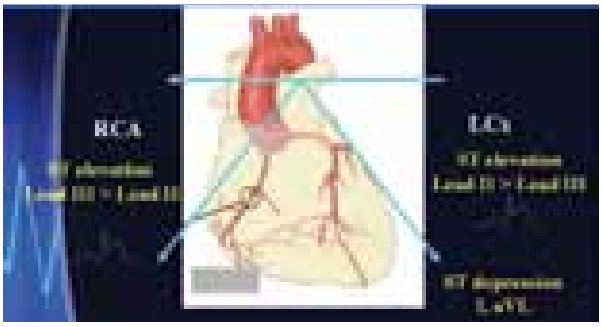


Table 1 : Showing localization of Culprit Vessel in IWMI

	Inferior Wall MI	
	RCA	LCx
ST elevation	Lead 3 > Lead 2	Lead 2 > Lead 3
T Wave	Lead 3 > Lead 2	Lead 2 > Lead 3
V4R	Upright T Wave	T Wave inverted

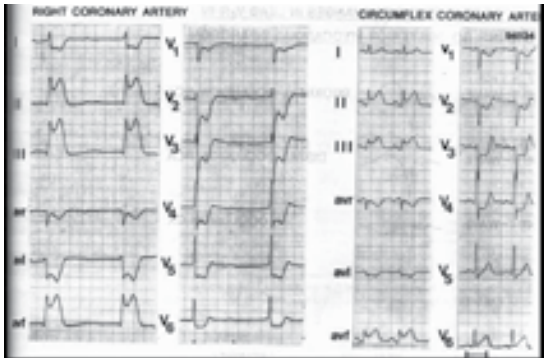
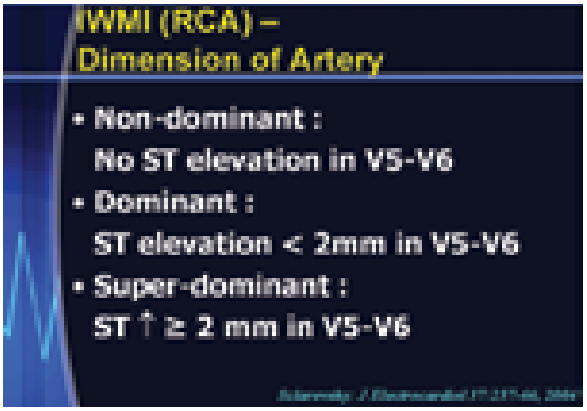


Figure 9 : ECG showing Culprit Vessel in IWMI

in LCx occlusion, ST may be depressed and T wave is inverted.

In IWMI precordial leads shows ST segment depressions. If the ST segment depression is noted in V1 to V3, it usually represents reciprocal changes. However ST segment depression is noted in V4 to V6, it usually represents poor prognosis. However, if ST segment depression is noted from V1 to V6, but in decreasing order, it has a favourable prognosis as compared to the previous case.

In IWMI, if ST elevations are noted in lateral leads (V5, V6), it predicts the dominance of RCA as shown below:



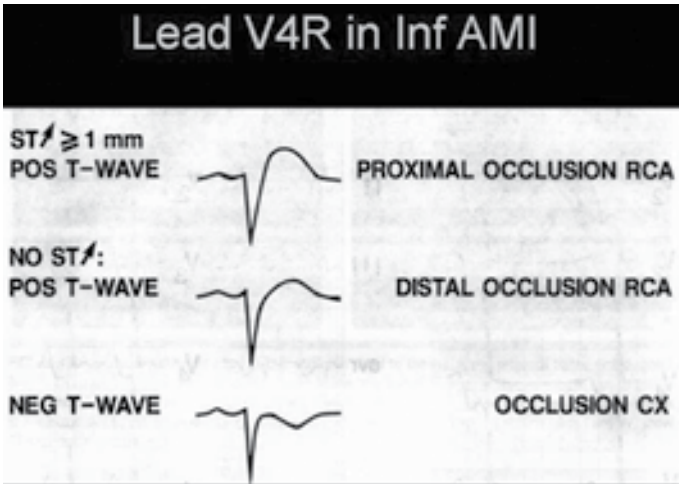


Figure 10 : Showing importance of V4R in IWMI

Table 2 : Showing importance of Precordial Leads in IWMI

Inferior Wall MI	Precordial Lead Changes
ST Depression in	
V4 to V6	Poor Prognosis (Triple Vessel Disease)
V1 to V6	Decreasing pattern – Prognosis better than 1
V1 to V3	Reciprocal changes

RV Infarction[10]

ST segment elevation in V4R of ≥ 1mm has a very high sensitivity and specificity for the diagnosis of RV infarction. It pinpoints the occlusion of proximal RCA also. There is probability of such patients going into AV blocks (approx. 45%). All patients of Acute Inferior Wall MI must have ECG recording in V4R on presentation to diagnose associated RV infarction The elevation in V4R is transient so there is importance of early ECG recording otherwise late recording or not recording of V4R will miss the diagnosis of RV Infarction.

Conduction Blocks in AMI[10]

LAD supplies to Right bundle branch and to anterior fascicle of Left bundle branch. Posterior fascicle of Left bundle branch has dual supply. RCA supplies the AV node. SA node is supplied by RCA or LCx. Right Bundle Branch Block (RBBB) is commonest block noticed in Anterior Wall MI settings. Left Anterior Fascicular Block (LAFB) is also quiet commonly seen in Anterior Wall MI. Left Bundle Branch Block (LBBB) is usually not a common occurrence in Anterior Wall MI. AV Nodal Blocks seen in IWMI prompts towards to proximal involvement of RCA.

Importance of aVR[10]

aVR is considered as orphan lead and not due importance is given to aVR. However, one must see the aVR in Acute

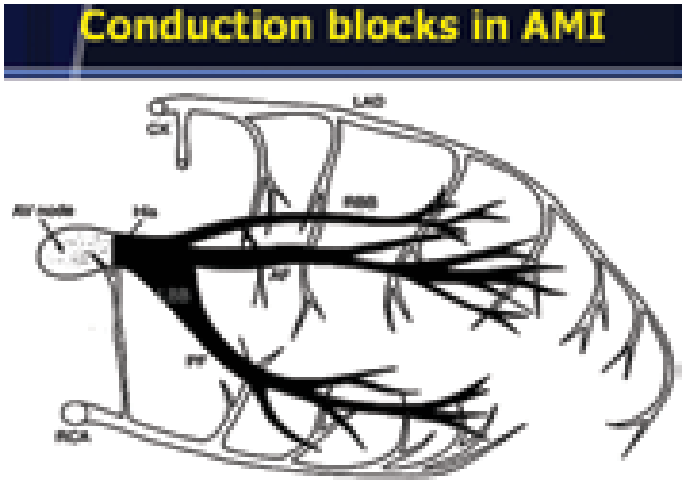
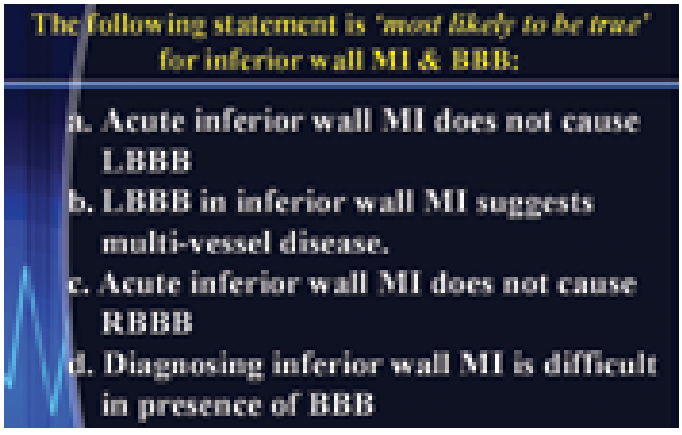


Figure 11 : Showing blood supply of the conduction system of the Heart



Coronary Syndrome (ACS). There may be isolated ST segment elevation in aVR in ACS and rest of the leads may be showing ST segment depression and if aVR is not seen, one may label the ECG as Non-ST segment Elevation MI (NSTEMI), while ECG belongs to Critical Left Main or Critical Proximal LAD involvement and signifies poor prognosis and needs aggressive management.

Culprit Vessel Localization on Stress ECG Test

Typical ischemic response on Stress ECG Testing is horizontal or downsloping ST segment depression. Few patients exhibit ST segment elevation of ≥ 1 mm on exercise in the leads without evidence of prior infarction. Reported incidence of such a finding is 0.2 to 1.7 %[11,12,13,14,15,16,17].

ST segment depression on exercise in any lead do not direct towards any vessel. Many authors have tried to localize the culprit vessel based on ST segment depressions in various leads, but of no value[18,19,20,21,22,23,24,25]. The interpretations were as follows :

- ST depression in V1 – almost always LCx disease – Sensitivity 67% Specificity 1000%

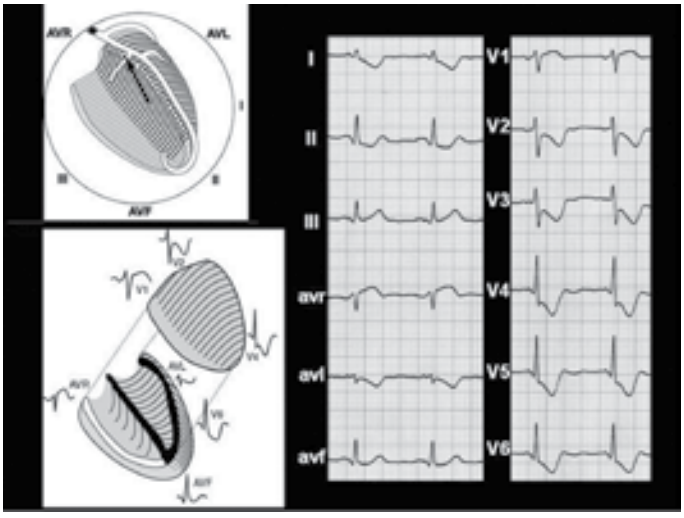


Figure 12 : Importance of aVR – ECG in Critical Proximal LAD involvement

- V2 – V6 – 40% Sensitive for any vessel involved
- LAD – ST segment depression wide spread
- Lead II, III, V4 – V6 – 60% Sensitive for LAD disease
- RCA – ST depression wide spread – 30 – 70% Sensitive in Leads II, III, aVF and V6 and 15 – 30% Sensitive in V2 – V5.

Study performed by Neeti Chauhan and S B Gupta[26] to localize Culprit Vessel based on ST segment depression. In this study of 100 patients, who were positive for ischaemia on Stress ECG test and were subjected for Coronary Angiography (CAG). They could not establish any criteria for Culprit Vessel Localization by ST Segment depression on the basis of leads. However, presence of chest pain, Stress test positivity at lower work load, at lower heart rate, ST depression in multiple leads (≥ 5 leads), longer recovery, severity of ST segment depression, fall in blood pressure and presence of complex arrhythmias reflected severe CAD (either critical triple vessel disease, left main disease or critical proximal LAD or LAD+RCA) (Unpublished data).

However, ST segment elevation on exercise has quiet a good correlation with the culprit vessel involvement. ST segment elevation in precordial leads favours LAD involvement and ST segment elevation in inferior leads implies involvement of RCA.

Many authors have reported the importance of ST elevation leads aVR, aVL and VI and have correlated with LAD involvement[27,28,29,30]. In study performed by Aditi Pandit and S B Gupta[31] to localize culprit vessel on the basis of ST elevation in aVR and V1, they observed ST elevation in aVR (≥ 2 mm) and V1 always predicts LAD involvement and ST elevation in aVR (≥ 2 mm) in males predicted LAD involvement with positive predictive value of 100% and 73% respectively (unpublished data).

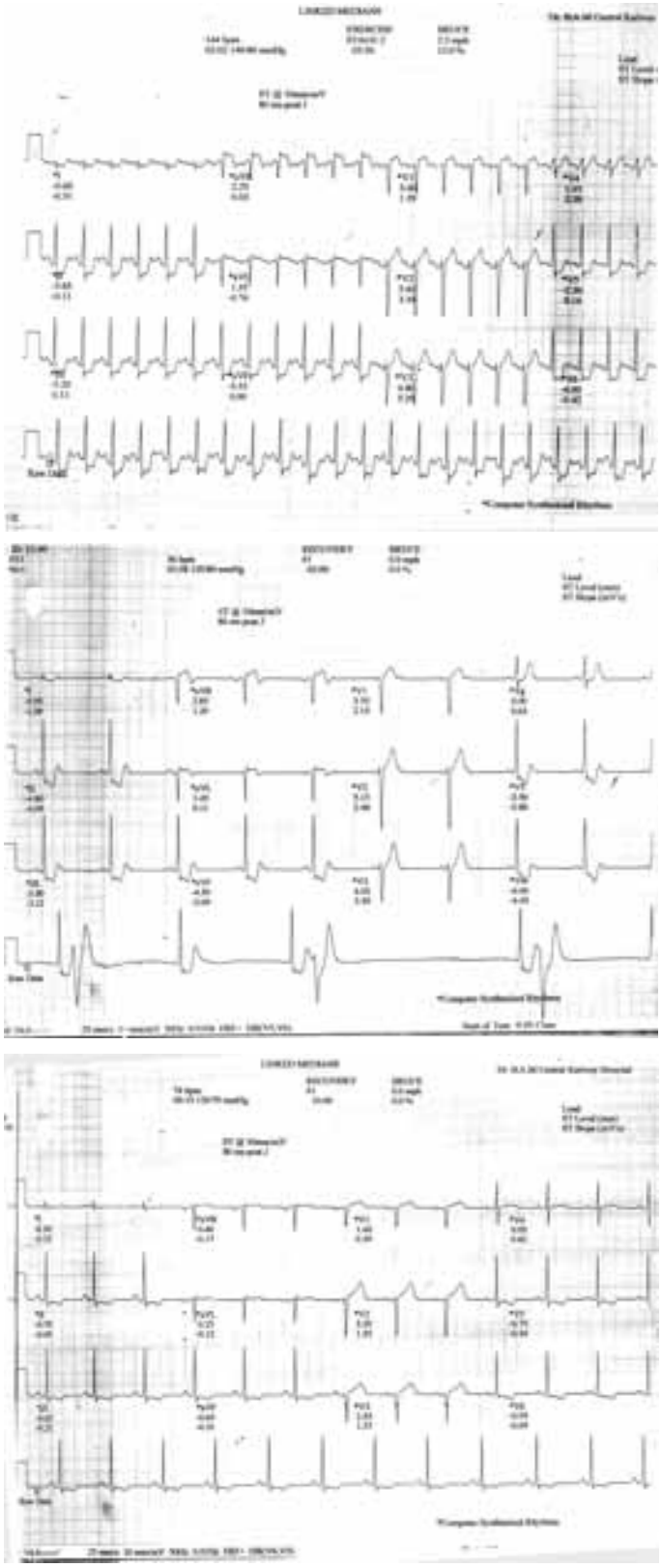


Figure 13 : Heart Rate Response on Peak Exercise, then sudden slowing of Heart Rate during Immediate Recovery and increases again during post-exercise period

S B Gupta[32] reported a novel finding on Stress ECG Test suggesting involvement of RCA/LCx. He observed sudden slowing of Heart Rate during immediate recovery and later the Heart Rate again rises. He hypothesized that this happens because of SA Node ischaemia due to proximal RCA/LCx involvement.

Conclusion

Stress ECG Test in evaluation of CAD and in evaluation, prognostication and rehabilitation in AMI is very useful tool. Localization of culprit vessel on Stress ECG Test is possible in certain situations and can help the treating physician to take the decision for sending the patient for an early intervention[33].

Acknowledgements

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J Wave Syndromes

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Introduction

J wave is a hump like deflection at the end of QRS complex in 12-lead surface ECGs, first described in 1951 by J Osborn in patients with hypothermia[1]. It is characterized by elevation of J point with a prominent notch or slur at the end of the QRS complex, followed up by an upward ST segment concavity, and a positive T wave. Early repolarization (ER) is a well-recognized pattern characterized by presence of J waves with J-point elevation of ≥0.1 mV, in two or more contiguous leads of the 12 lead ECG[2,3,4]. This ECG pattern is quite common in healthy adult population especially in young males and black populations[5,6]. Early repolarization had been generally regarded as a benign variant with limited clinical significance and could be mistaken for other serious conditions like acute myocardial infarction, pericarditis or intraventricular conduction defects[5,6]. The identification of inferolateral J waves in patients with idiopathic ventricular fibrillation (VF) has sparked great interest of medical community in the early repolarization. Recent studies by Haïssaguerre et al and Tikkanen et al have identified a link between early repolarization and the risk of malignant ventricular arrhythmias and sudden cardiac death in apparent healthy individuals without structural heart disease[7,8]. There is much debate on the definition of what truly constitutes the pathological or malignant form of early repolarization and about specific ECG subtypes that may be potential harbingers of ventricular arrhythmias. In fact the malignant form of early repolarization have been termed as the early repolarization syndrome. Early repolarization syndrome has been defined as early repolarization in the presence of J-point elevation ≥1 mm in ≥2 contiguous inferior and/or lateral leads of a standard 12-lead ECG in a patient resuscitated from otherwise unexplained VF or polymorphic VT[9].

Because of the similarity in ECG findings in the form of amplification of Ito-mediated J waves, a marker of increased dispersion of repolarization and substrate for re-entrant arrhythmias (alongwith risk factors like young age, male gender and clinical outcomes in the form of tachy-arrhythmias and sudden cardiac death), the Brugada syndrome and early

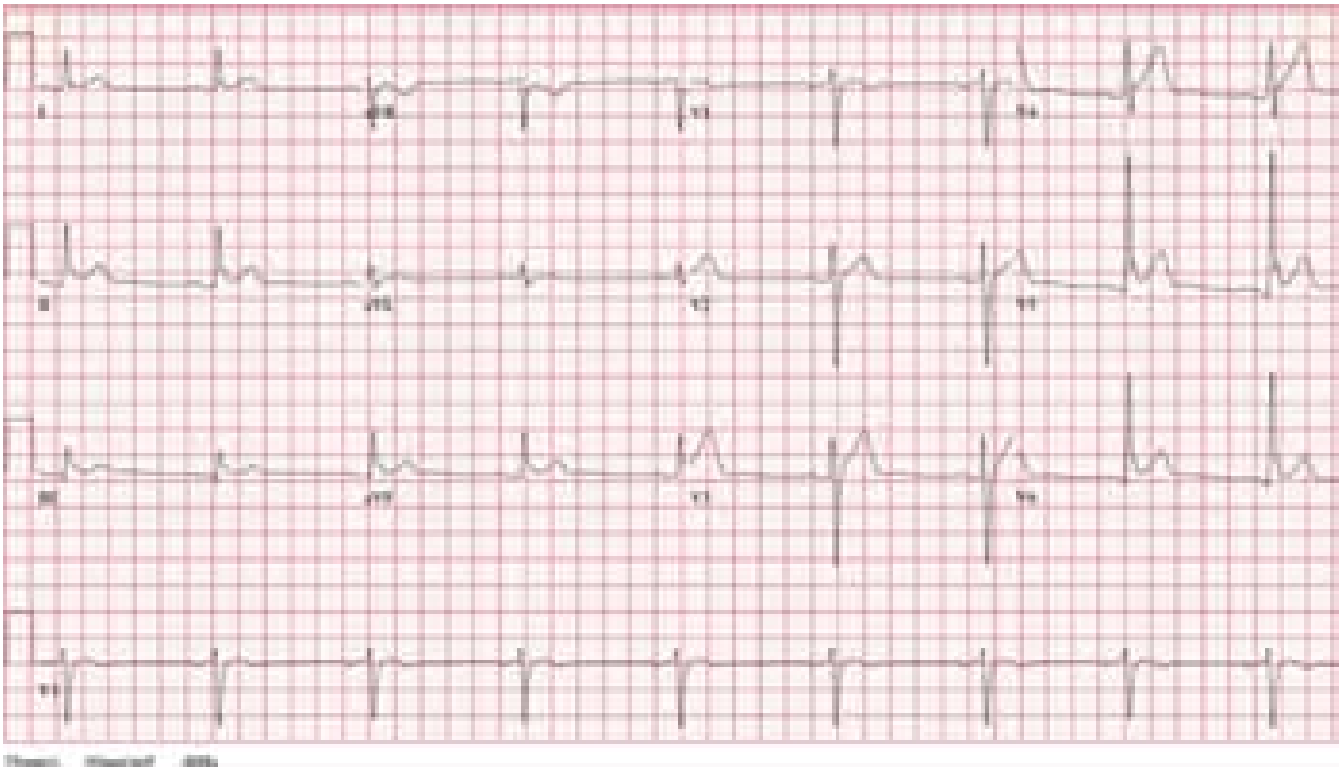

Figure 1 : Early Repolarization

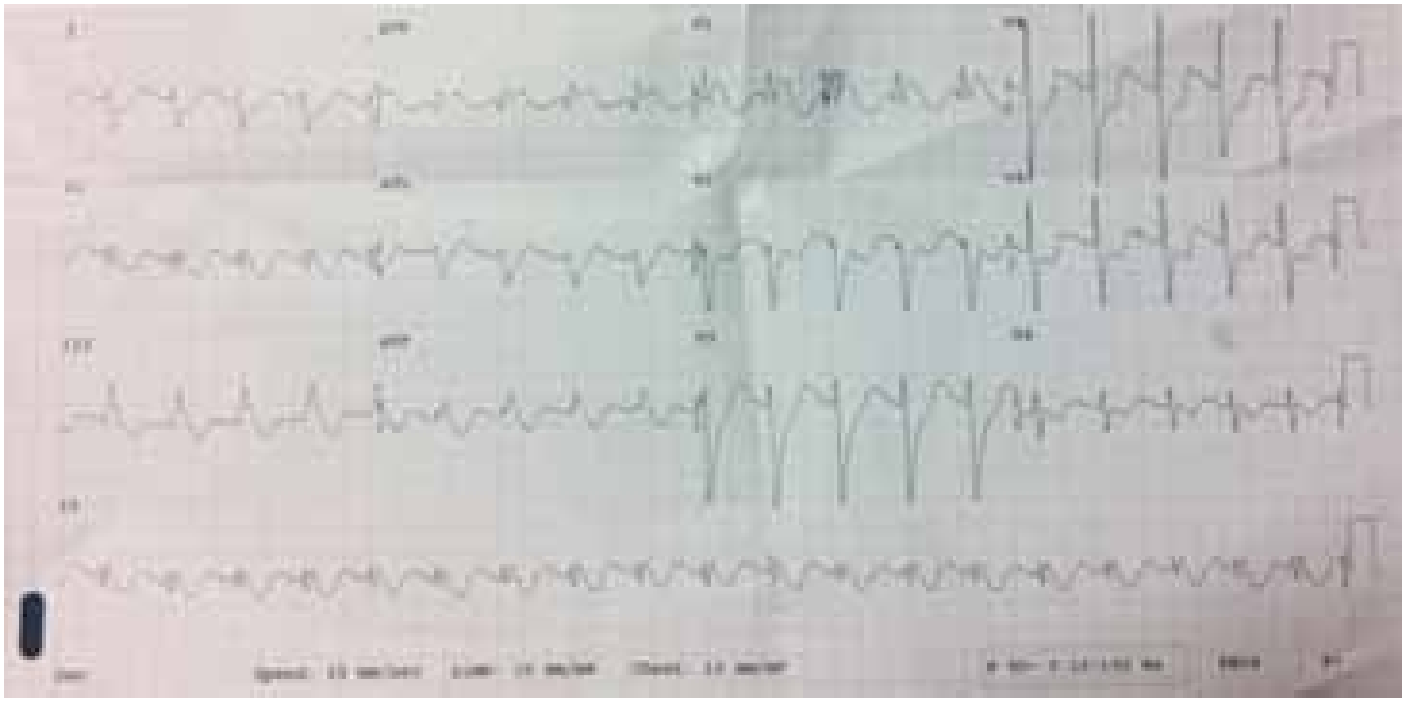

Figure 2 : ECG with Brugada pattern in a 4 year old child with recurrent near-syncope

repolarization syndrome are clubbed together into J wave syndrome[10,11]. This paper reviews mechanisms, prevalence and morphological variations of the J wave syndrome and discusses the clinical implications in terms of diagnosis, prevention and treatment of the J wave syndrome.

Prevalence

The prevalence of early repolarization in the general population has been observed to vary from less than 1% to 13%, with the prevalence of ER in these studies being significantly influenced by age, gender and ethnic race[12-20]. Surawicz et al showed that the male pattern prevalence increased at puberty, reached 91% in the age group of 17 to 24 years and declined gradually with advancing age to 14% in the oldest males[13]. This has been attributed to the effect of androgens with the prevalence of ER appearing to parallel the rise of testosterone blood level in males during puberty and the decline of testosterone level in elderly males[13]. The early repolarization pattern is seen in 50–80% of trained athletes[16,21].

ER was observed in 31% of idiopathic VF cases vs 5% (21/412) of well-matched healthy subjects (P<0.001). The prevalence of the ER pattern with J-wave elevation ≥0.2 mV in patients with idiopathic VF was found to be 16%[7].

Based on data from implantable cardioverter-defibrillators (ICD), 64 idiopathic VF survivors with ER experienced higher VF recurrence than 142 VF survivors without ER (41% vs 23%, P=0.008). Rosso et al [22] found that early repolarization was more common among the patients with VF than among the control subjects (42% vs 13%, P=0.001). This was particularly true for J-point elevation in the inferior leads (27% vs 8%, P= 0.006) and was true for J-point elevation in leads I to aVL (13% vs 1%, P=0.009). Nam et al. found that early repolarization was observed in (57.9%) of baseline ECGs in patients with VF compared to 3.3% of 1,395 controls [23].

Mechanisms of J wave

Antzelevitch et al in 1991 proposed that the transmural differences in the phases 1 and 2 components of the cardiac action potential may be responsible for the J wave in 12-lead ECG [24]. J wave has been attributed to an overlap between the end of depolarization and beginning of repolarization or a modification of the time course of repolarization across the ventricular wall caused by selective depression, delayed activation (global or regional), excitation failure or current to load mismatch of the epicardial action potential[25]. The presence of Ito-mediated action potential notch in ventricular epicardium but not in endocardium produces a transmural voltage gradient seen as a J-wave in 12-lead ECG[26]. Boineau has attributed ER to the deep invagination of Purkinje fibres into the sub-epicardial tissue resulting in early transmural activation followed by early repolarization[27].

Early repolarization-associated events of idiopathic ventricular fibrillation are known to occur more likely in vagal contexts such as sleeping with J-point amplitude increases at night[26]. Conversely, adrenergic stimulation is known to suppress early repolarization and associated arrhythmic events. It has been suggested that time-dependent recovery of Ito from inactivation

could explain the decreased J-wave amplitude with increased heart rate[4]. Similarly, temperature can also modulate J waves and ST-segment elevation with prominent J waves, classically known as the Osborn waves typically seen in hypothermia[10]. An outward shift in repolarizing current caused by a decrease in sodium or calcium channel currents or an increase in Ito, IK-ATP, IK-ACh, or other outward currents generates an early gradient in the repolarization currents within the ventricles and results in the J-wave syndromes[4,26]. Typically phase 2 reentrant beat causes the R on T phenomenon leading to pause and short coupled extrasystole like bradycardia resulting in polymorphic ventricular fibrillation in the J wave syndrome [11,26]. Isoproterenol and the class Ia antiarrhythmic drug like Quinidine can decrease the J waves in idiopathic VF patients and suppress recurrences of arrhythmic events[25].

Brugada Syndrome

The Brugada syndrome has a prevalence of prevalence 1: 2000 and has a male predominance[28]. ECG findings Brugada syndrome (Figure 2) includes coved type R'-ST segments, > 0.2 mV ST elevation and negative T waves in the right precordial leads V1–V3 and a shorter QT interval, particularly when associated with a loss-of-function mutation in the cardiac channels. These ECG findings can be elicited by sodium channel blockers such as ajmaline[28]. Sodium channel dysfunction is of critical importance in the Brugada syndrome and provocation of the Brugada ECG pattern by sodium channel blockers has been incorporated in the diagnostic criteria of the Brugada syndrome[25]. Diagnostic criteria for Brugada syndrome include a history of ventricular tachycardia, ventricular fibrillation, a family history of sudden death at < 45 years old, coved type ECGs in family members, inducibility of ventricular fibrillation, unexplained syncope or nocturnal agonal respiration and the absence of gross structural abnormalities[28].

Difference between Brugada syndrome and early repolarization syndrome

Gussak and Antzelevitch have proposed that early repolarization syndrome occurs because of a greater transmural gradient of repolarization which is akin to that in the Brugada syndrome [2]. However, short–long–short sequence and extrasystoles with short coupling intervals were observed more frequently in patients with early repolarization syndrome than in those with Brugada syndrome[10]. However, povocation by sodium channel blockers and positive signal averaged ECG are not observed in early repolarization syndrome[26].

Other conditions where J wave plays an important role

Myocardial infarction (MI)

Prominent J waves on the ECG have been reported to occur in association with acute myocardial ischemia. Excitation failure is a known cause of J point and ST segment elevation in the setting of cardiac inexcitability by local myocardial ischemia[25]. The incidence of primary ventricular fibrillation is higher in patients with acute inferior myocardial infarction who have right ventricular involvement (8.4%) than in those without (2.7%) or in those with an anterior myocardial infarction (5.0%). Clinical observations suggest an association between Ito density and risk of primary VF during acute myocardial infarction. This may be due to the fact that Ito channel is more prominent in right than left ventricular epicardium[10]. The presence of large number of Ito channels may also explain the higher prevalence of ventricular fibrillation post-MI in males than in females. Acidosis and hyperkalemia are thought to be critical elements for development of J waves and subsequent arrhythmias in MI patients[28].

Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy is seen in 1:10,000 individuals and marked by presence of an epsilon wave in conjunction with negative T waves in the right precordial leads [28]. These patients may also show the coved ST segments typically observed in Brugada syndrome and may show similar response to ajmaline challenge. [28]

Controversies in evaluation of ER pattern with increased risk of VF

The identification of ER as a potential harbinger of ventricular arrhythmias through these studies represents a major advancement in the search for risk stratification tools for sudden cardiac death. However, the definition of what truly constitutes the pathological or malignant form of early repolarization is yet to be refined and has been the source of much controversy [29]. Case control studies have provided some insight into the characteristics that could distinguish between benign and malignant forms of J waves. In a study by Haissaguerre et al, a history of familial sudden death was significantly more frequent among idiopathic VF patients with J waves than among patients without J waves[7]. Merchant et al found that QRS notching is found more commonly with malignant ER than with the benign variant [30]. Tikkanen et al and Rosso et al found that patients with J wave and horizontal/descending ST segment have higher risk of arrhythmias than those with ascending ST segment [8,31]. Haissaguerre et al reported that the J-wave amplitude in idiopathic VF patients was significantly larger than that in the controls suggesting that higher the amplitude of J wave more the risk of ventricular arrhythmias [7].

Another source of controversy is that whether J wave represents a repolarization abnormality or a depolarization abnormality. Few authors suggest that the slurring or notching of the terminal part of QRS complex may be due to a depolarization abnormality[28]. Abe at al found that J waves were significantly associated with all Late Potential parameters and therefore J waves are more strongly associated with a depolarization abnormality than with a repolarization abnormality [32]. However, others believe that an outward shift in repolarizing current caused by a decrease

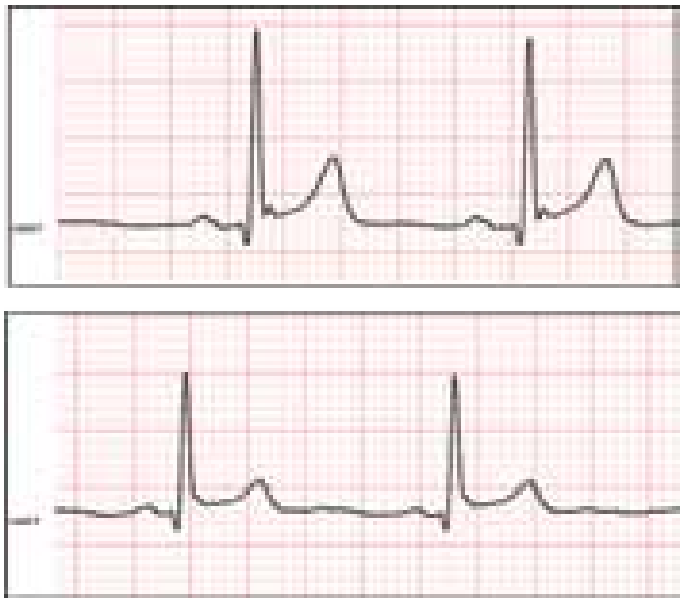

Figure 3 : Electrocardiograms from different healthy individuals showing notched and slurred patterns of the terminal portion of the QRS complex

in sodium or calcium channel currents or an increase in Ito, IK-ATP, IK-ACh, or other outward currents that generates an early gradient in the repolarization currents within the ventricles results in the J-waves and therefore J wave syndrome is a repolarization abnormality [4,26].

J wave syndrome and the risk of ventricular fibrillation and sudden cardiac death

Sudden cardiac death is defined as an unexpected death from a cardiac cause within a short time period of ≤1 hour from the onset of symptoms, in an individual without any prior cardiovascular abnormalities [33]. Idiopathic ventricular fibrillation is a disorder presenting as syncope or cardiac arrest caused by polymorphic ventricular tachycardia (VT) that invariably is triggered by ventricular extrasystoles with a very short coupling interval, falling on the peak or on the descending limb of the preceding T wave[11]. The mean age of patients with idiopathic VF was around 35 ± 10 years in a population based study[7]. The association of J waves with increased risk for VF was first noted for hypothermia and incidentally quinidine was effective in preventing hypothermia-induced VF[11].

Several case reports published in the last two decades, highlighted the fact that patients with idiopathic ventricular fibrillation had J waves and demonstrates augmentation of J-wave amplitude immediately prior to the onset of malignant arrhythmias. Subsequent experimental studies also demonstrated that the J wave is a marker of increased dispersion of repolarization, which is a substrate for reentrant arrhythmias. J-point elevation > 0.1 mV in inferior leads was associated with an increased risk for death from cardiac causes (adjusted relative risk 1.28, P < 0.03). J-point elevation > 0.2 mV in inferior leads increased the adjusted relative risk of death from cardiac causes to 2.98 (P < 0.001)[8]. The subjects with J waves had a threefold higher risk for cardiac death in two population studies[8].

Subsets of ER pattern with increased risk of VF/cardiac death

The type 1 ER pattern, which manifests in the lateral precordial leads, is prevalent among healthy male athletes and is associated with a relatively low risk of arrhythmic events. Early repolarization pattern in the inferior or inferolateral leads, type 2 pattern, is associated with a moderate level of risk. Finally, type 3 ER pattern appearing globally in the inferior, lateral, and right pre-cordial leads is associated with the highest level of risk[26]. ER in the inferior leads was found to be associated with an increased risk of cardiac death (adjusted relative risk, 1.28; 95% confidence interval (CI), 1.04 to 1.59, P=0.03) in their general population[8]. Tikkanen et al.[34] found that ER patterns showing horizontal or descending ST segments after the J point are associated with an increased risk for arrhythmic death (hazard ratio of 1.43, 95% confidence interval 1.05-1.94), while ER with rapidly upsloping ST segment after the J point is not associated with such risk. The highest risk is observed with the combination of ER pattern in the inferior leads, J point amplitude > 0.2 mV and horizontal or descending ST segment.

Rosso et al. found that the J waves with horizontal/descending ST segment improved the ability to distinguish patients with idiopathic VF from age and gender matched controls[31]. They also found that subjects showing a J wave on the baseline ECG had an odds ratio of 4 for developing VF and while in those subjects showing J waves with horizontal ST segment, the odds ratio for developing VF increased to 13.8. To put the things in perspective, the estimated odds for developing idiopathic VF for an individual are 3.4 in 100,000. With presence of J waves in the ECGs, the risk increases 3 fold to 11 in 100,000 individuals and when the J-waves is associated with horizontal/ descending ST segment the risk increases 9 fold to 30.4 in 100,000 individuals[31].

Diagnosis and Treatment of J wave syndrome

Patients having idiopathic VF due to early repolarization syndrome can be diagnosed when they have no identifiable structural heart disease demonstrated by echocardiographic evaluation, no detectable coronary artery disease on coronary angiography or exercise testing, and no known repolarization abnormalities including short QT syndrome, long QT syndromes or Brugada syndrome. Additionally, patients with catecholaminergic arrhythmias should be excluded[4]. Patients with transient J wave augmentation, presence of ER pattern (≥0.2 mV) in inferio-lateral leads or global ER pattern in ECGs portend a high risk for VF in patients with ER[4]. Patients with the Brugada syndrome can be diagnosed with ECG pattern of right bundle branch block and ST-segment elevation (≥0.2 mV) in ≥2 precordial leads V1–3, without intervention or following

infusion of a sodium-channel blocker. Presence of mutations in the cardiac sodium channel gene SCN5A also aids in diagnosis of Brugada syndrome.

Given that at least 6% of healthy individuals will have inducible VF during aggressive EP studies, usually an asymptomatic subject with early repolarization pattern does not warrant an electrophysiologic study [11]. A low rate of VF inducibility (34%) in the patients with history of VF makes the electrophysiologic study less sensitive for risk stratification even in symptomatic patients [4]. Acute control of arrhythmias due to J wave syndrome can be achieved by deep sedation and/or isoproterenol infusion whereas Quinidine can be used for management of recurrent VF in chronic phase [4]. Catheter ablation of the ectopy initiating the VF can be used in VF patients with ER who fail to respond to drugs. Implantation of ICD is indicated for those with aborted sudden death. However, there are no data available for long-term follow-up and further studies are warranted to address this issue.

Conclusions

In conclusion, current body of evidence suggests that J wave syndromes characterized by ER pattern in infero-lateral lead with J point elevation of > 0.2 mV along with horizontal/descending ST segment increases the risk of ventricular fibrillation and sudden cardiac death. Stringent evaluation is warranted when these findings are observed especially in patients with prior history of VF or unexplained syncope. Quinidine can be used for the prevention of recurrence of arrhythmias in such patients. Long term follow-up studies are required to further streamline the risk mitigation strategies in patients with malignant ER pattern.

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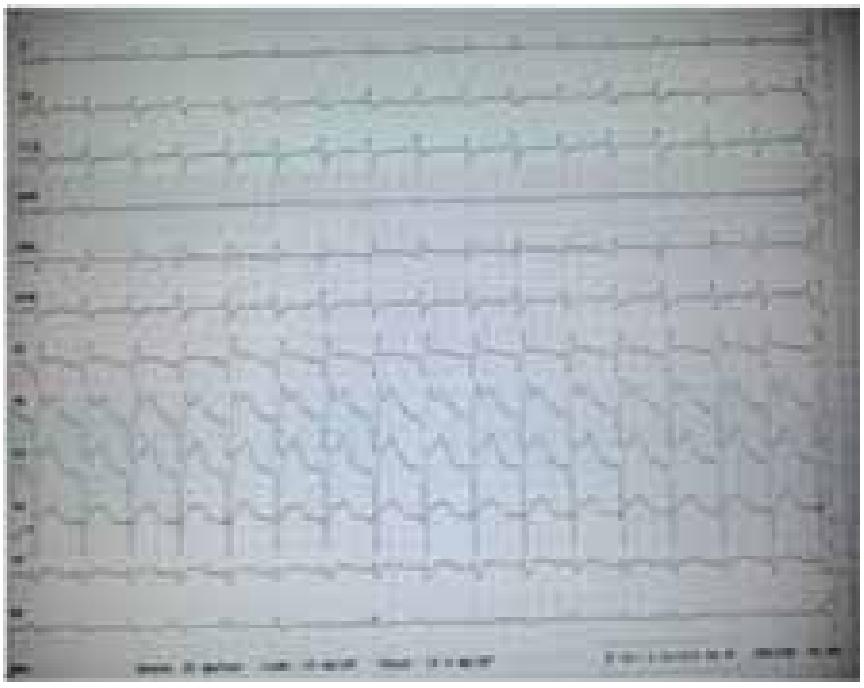
ECG Quiz

Yash Lokhandwala[*], Gopi Krishna Panicker[#]
[*]Arrhythmia Associates, [#]Quintiles Cardiac Safety Services

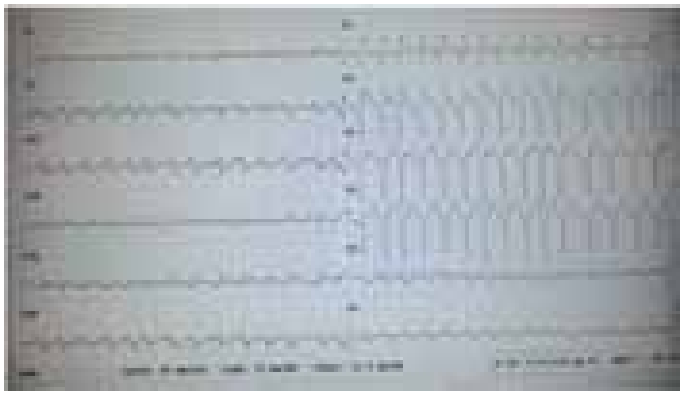
The answers and explanations are on the reverse side of the page.

ECG - 1

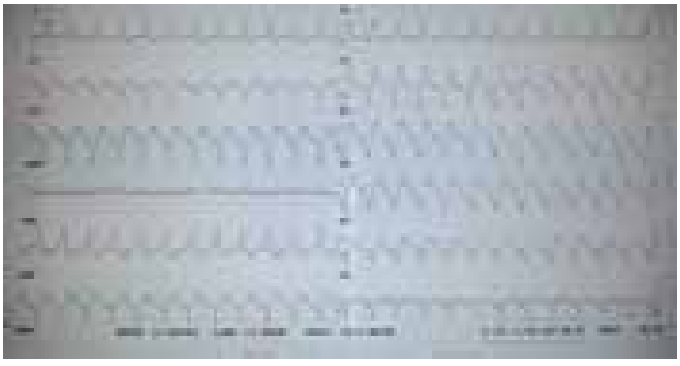
A 65 yr old man with rest angina 24 hrs ago. Cardiogenic shock on presentation. Underwent LAD stenting, on intra-aortic balloon pump and ventilator…...



The next day…



A few hours later…What is this arrhythmia?



a. Sinus Tachycardia

b. Atrial Fibrillation

c. Atrial Flutter

d. Ventricular tachycardia

For correct answer see overleaf

ECG - 1

The correct answer is 'd'

The ECG shows a wide QRS tachycardia with a QS morphology in leads V3 to V5 and a QR pattern in leads V1 and V2. There is also left axis deviation. This clearly does not conform to any bundle branch block pattern. Normally, it would be straightforward to diagnose it as VT. However, one could be confounded by the second ECG which shows AF with rapid ventricular rate and a ***seemingly*** similar QRS morphology.

Teaching point from Figure 1: RBBB in MI is seen with proximal LAD occlusion. This is a poor long-term prognostic indicator if permanent. The risk of progression to CHB is twice that of LBBB especially if associated with fascicular block.

Teaching point from Figure 2: Atrial fibrillation has an incidence of 6-20% with increased morbidity and mortality (both In-hospital and long-term). In recent years, there has been a notable decline with widespread use of PCI. When AF occurs within 48 hrs and with inferior MI, it is often transient, due to electrical instability/ atrial ischemia. In such cases, the prognosis is fair. When AF occurs with anterior MI, especially after 48 hrs, the prognosis is poor. It is often associated with significant MR/severe LV dysfunction.

Teaching point from Figure 3: Sustained monomorphic VT is rare in acute MI with an incidence of 1%. It is indicative of large MI and is associated with heart failure. It has a poor prognosis.

This patient was in cardiogenic shock. He underwent rescue PTCA and recovered gradually. He was readmitted within 2 weeks, with heart failure.

ECG - 2

60 year old man, recurrent unexplained near-syncope since 3 months

Figure 2

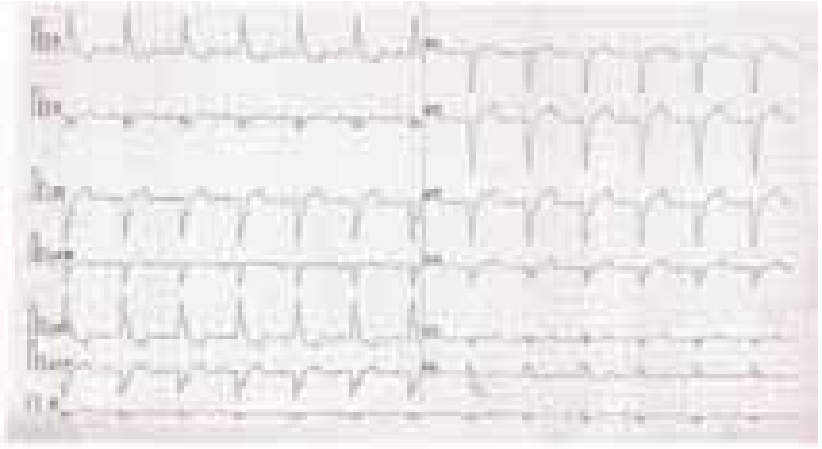
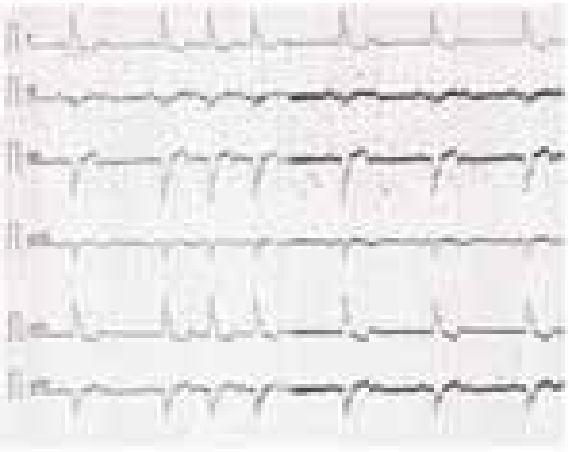
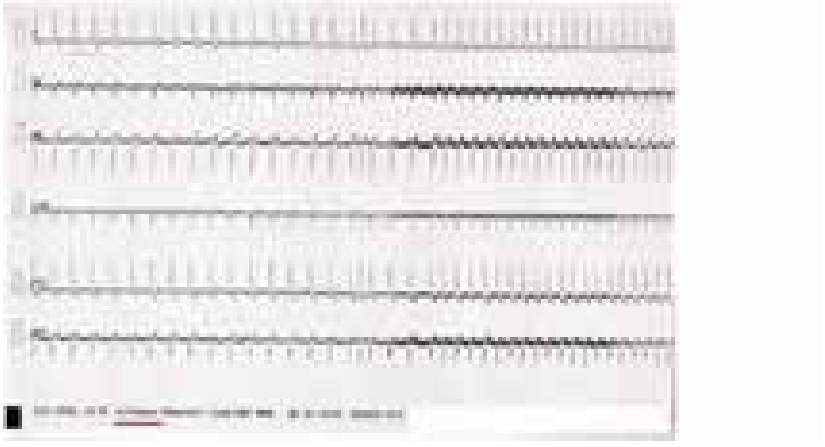


Figure 2a



On Carotid sinus massage- the ventricular rate increases!



a. Sympathetic stimulation

b. Response to hypotension

c. Enhanced AV nodal conduction

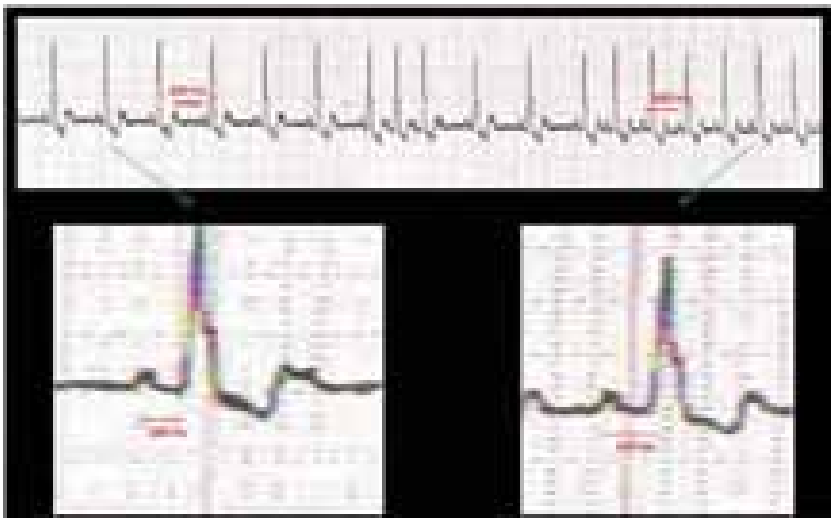
d. Enhanced infra-nodal conduction

For correct answer see overleaf

ECG - 2

The correct answer is 'd'

There is sinus and AV nodal slowing during carotid sinus massage..



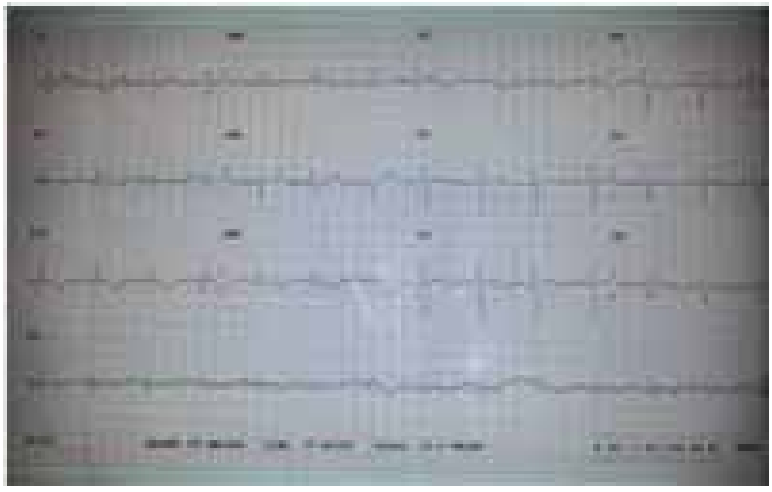
The noninvasive interventions to determine site of AV block includes interventions like use of atropine, exercise or catecholamines and carotid sinus massage.

AV nodal conduction improves with all these interventions except carotid sinus massage.

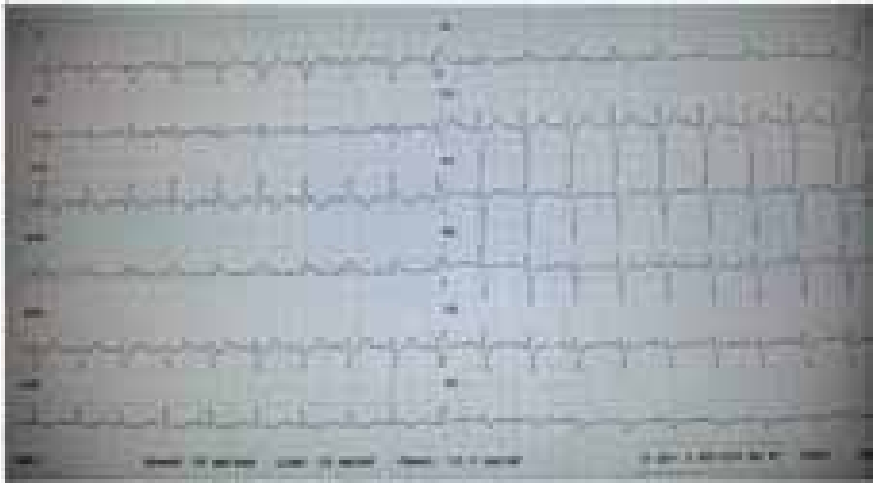
In comparison, subnodal conduction improves with carotid sinus massage and worsens with use of atropine, exercise or catecholamines.

ECG - 3

A 64 year old man with recent onset of recurrent chest heaviness with dyspnea. LVEF and Coronary angiogram was normal. Patient while being mobilised prior to discharge, collapsed, pulseless, shocked and put on ventilator



Soon after resuscitation…



What to suspect?

a. Pulmonary embolism

b. Aortic dissection

c. Iatrogenic coronary ostial dissection

d. Brugada syndrome

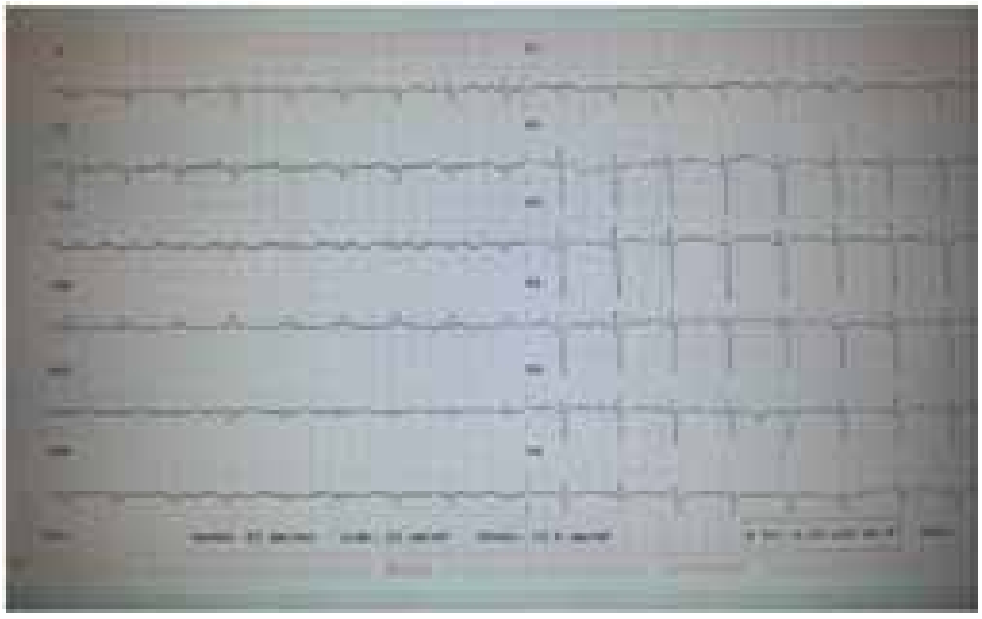
For correct answer see overleaf

ECG - 3

The correct answer is 'a'

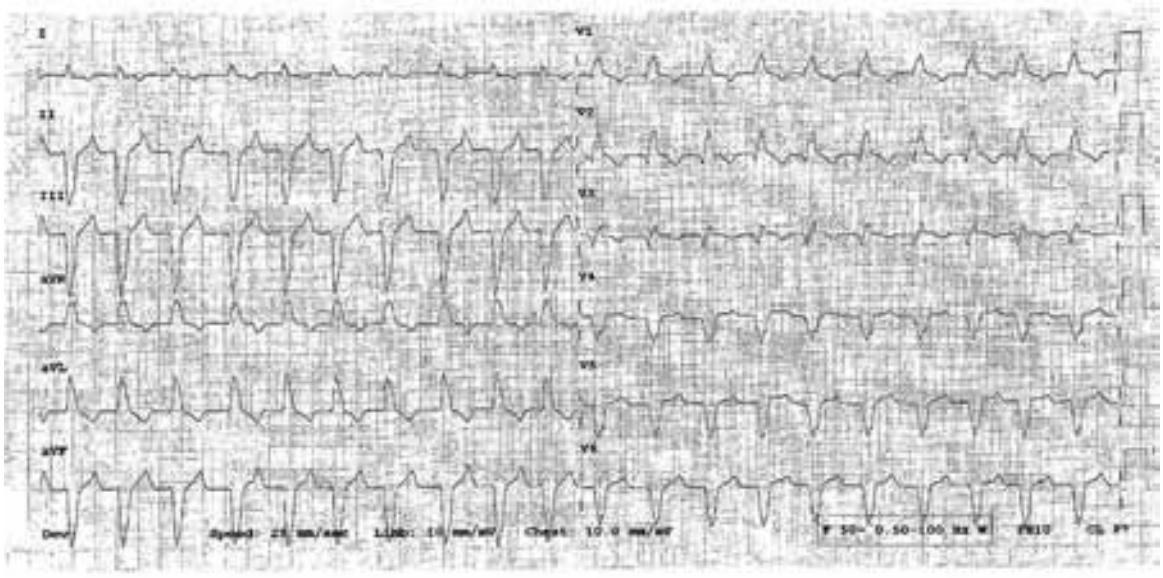
ECG is of great importance in patients with acute PE and normal BP. In these patients, an RV strain pattern with RBBB, S1Q3T3 and negative T waves in V1-V4 is seen in 34% of patients. This strain pattern was associated with adverse outcome which were independent of echo findings. In patients without RV strain, the echo adds little prognostic information.

I.V. streptokinase infusion was given for 36 hours, followed by heparin and warfarin. There was marked clinical improvement, along with the ECG.



ECG - 4

65 year old man with a “heart attack” several years ago



The rhythm is:

a. Sinus tachycardia

b. Atrial tachycardia

c. VT with AV dissociation

d. VT with retrograde conduction

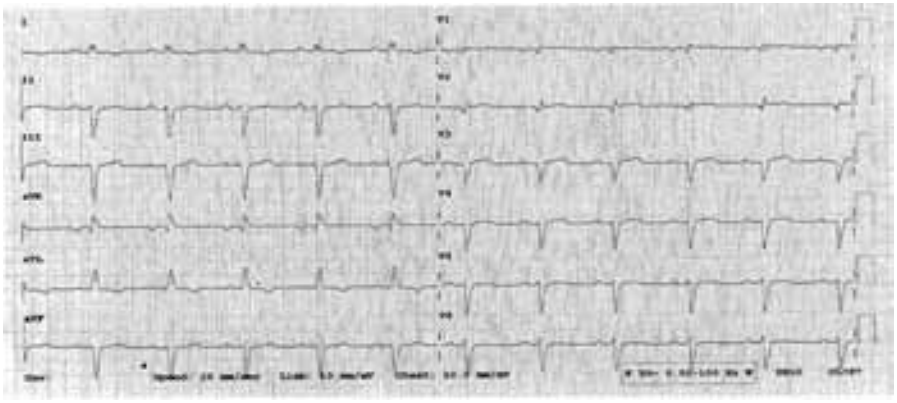
For correct answer see overleaf

ECG - 4

The correct answer is 'a'

The T waves in lead II are peaked indicative of positive P waves, suggesting sinus tachycardia.

The ECG at normal heart rate recorded later was as follows:



This showed that this was a regular LBBB-type wide QRS tachycardia.

In wide QRS tachycardia in a patient with an old MI, the diagnosis is VT, until proved otherwise. This case was an exception to the rule.

INDIAN SOCIETY OF ELECTROCARDIOLOGY

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Yours sincerely,

Signature of the Applicant
Proposed by (the Member of the Society)

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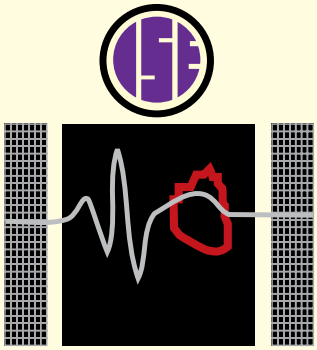

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