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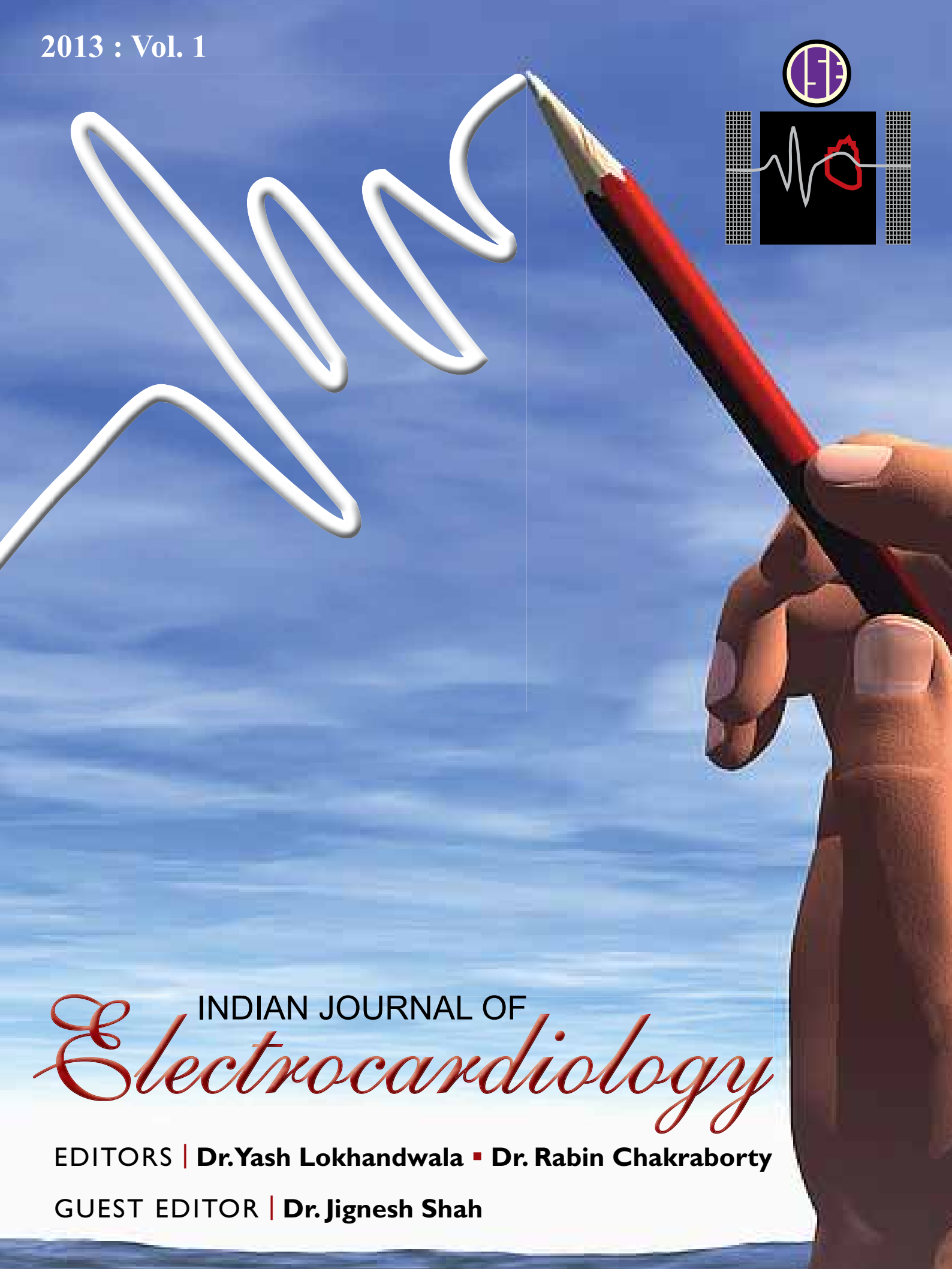

INDIAN JOURNAL OF Electrocardiology

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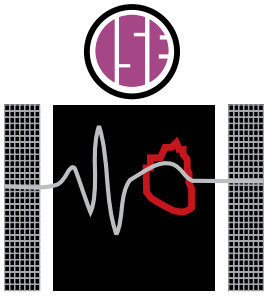
11TH ANNUAL CONFERENCE



ISECON 2013

6th - 7th April 2013

Hotel Intercontinental Eros, Nehru Place, New Delhi

C O N T E N T S

SECRETARIAT
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PRESIDENT ELECT

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Editorial

Dear Friends,

As we release this issue of the IJE, we are at the threshold of ISE New Delhi. 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 clinical relevant review articles.

We have been fortunate to have a number of clinicians contributing excellent review articles to this issue of IJE. Dr. Maheswari's group continues to support the IJE and have presented an interesting case of RV infarct, followed by a comprehensive review of literature related to the same. This review will assist us in better management of patients with Inferior ST elevation MI by making us lookout for RV infarct.

Dr. Nathani's group has summarized the known data regarding Coronary artery spasm, another important clinical entity which all of us will face in our clinical practice. A good summary of the data is the highlight of this article.

A comprehensive review of pacemaker follow-up in clinical practice has been presented by Drs. Nadig and Kapoor. They describe the requirements and essentials to establish a good pacemaker clinic. Their experienced insights will help us understand various issues encountered during pacemaker follow-up. As always, IPEJ has graciously allowed us to borrow their article, "Cardiac pacing and Defibrillators in Children and Young adults" by Dr. Singh et al. With the improving survival of pediatric patients with surgically repaired congenital heart disease, all of us will encounter young patients with implantable devices and this review article will help us approach such patients with greater understanding and less anguish.

Dr. Pandurangi's group has presented several examples of QRS axis alternans as well as QRS alternans. They have selected excellent ECGs of this phenomenon and have provided ladder diagrams to explain the mechanism of QRS axis alternans. Dr. Vora has presented an excellent clinical vignette of "the missing beat". The vignette helps us review an oft neglected physiological cause of 'missing beats'.

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 Yash Lokhandwala S.B. Gupta

From President Elect's Desk

Dear Members,

It is our great pleasure in bringing out the 1st issue of Indian Journal of Electrocardiology of the year 2013 on the eve of Annual Conference of Indian Society of Electrocardiology at New Delhi.

ISECON 2012 – The Annual Conference of Indian Society of Electrocardiology was organized by Dr Jitendra Makkar and the team at Jaipur from 17th to 19th February 2012. It was a great scientific feast! Our heartiest congratulations to Dr Jitendra Makkar and his team.

Mid-Term Conference of Indian Society of Electrocardiology was organized by Dr Ajit R Bhagwat at Aurangabad on 15th – 16th September 2012 and a very well attended meeting with excellent scientific material. Dr Ajit Bhagwat and his team needs worthy praise.

Indian Society of Electrocardiology also organized many programs during the year :

a. "ECG Learning Courses" for postgraduate students were organized at Bangalore on 7th and 8th April 2012, at Sri Lanka on 21st and 22nd April 2012, 9th and 10th June 2012 at Chennai, 16th and 17th June 2012 at Pune, 18th and 19th August 2012 at Rohtak, 13th – 14th October 2012 at Dibrugarh, 15th – 16th December at Jodhpur, 19th – 20th January 2013 at Mangalore, 9th March at Kathmandu and 16th and 17th March at Mumbai. About 60-100 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading

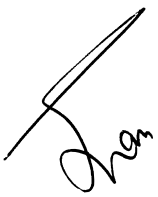
b. ISE Satellite Symposium at Mumbai on 6th July 2012 and 19th August 2012 and at Kathmandu on 8th March 2013.

c. For the first time, a successful ECG Technician Course was organized at Mumbai on 26th – 27th January 2013. 50 candidates participated.

d. ISE has initiated Pacemaker/CRT/ICD Survey. We received approx. data for more than 1000 persons. A lot more to be done.

My sincere thanks to Dr Yash Lokhandwala, Dr Jignesh Shah and the Editorial Team for bringing out the ISE Journal – 2013, 1st Volume.

Long Live Indian Society of Electrocardiology.



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

Review Article

Coronary Artery Spasm

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In 1959, Dr. Myron Prinzmetal described a syndrome of nonexertional chest pain with ST-segment elevation on electrocardiography (ECG) as a "variant form of angina pectoris". It is caused by coronary artery spasm (CAS), a sudden, intense vasoconstriction of an epicardial coronary artery that causes complete vessel occlusion or near occlusion. This vasospasm is due to the smooth muscle constriction of the coronary artery (as opposed to atherosclerotic process) and, is an important cause of chest pain syndromes that can lead to myocardial infarction (MI), ventricular arrhythmias, and sudden death. This review article will discusses the natural history, physical exam findings, pathophysiology, natural history and management options with this variant form of angina.

History and Physical Examination

The age of onset of Prinzmetal angina is highly variable, but most patients are in their 50s at symptom onset. Variant angina is believed to be more common in female patients. Patients often have 3- to 6-month clusters of recurrent attacks, separated by relatively asymptomatic periods, with a gradual reduction of symptoms in the long term. Patients with coronary artery vasospasm typically describe anginal symptoms, including retrosternal pain or pressure with radiation to the neck, jaw, left shoulder, or arm. This may be particularly true if there is significant coexistent atherosclerosis. Notably, symptoms associated with vasospastic angina often occur at rest and may exhibit a circadian pattern, with most episodes occurring in the early hours of the morning. In severe cases, associated arrhythmias may be present and lead to lightheadedness and dizziness due to either heart block or ventricular tachycardia.

Although CAS is more likely to occur in the presence of atherosclerotic lesions, the absence of traditional risk factors for atherosclerotic CAD may evoke suspicion of vasospastic angina. Cigarette smoking, however, is a common risk factor for both atherosclerosis related typical angina as well as CAS related variant angina. CAS is found more often in patients with symptoms that occur at rest (55.5%) than in those with exertional angina. A minority of patients with variant angina may have a more systemic abnormality of vasomotor tone; this may include symptoms of migraine headache and Raynaud phenomenon.

No features on physical examination are specific for vasospastic angina. Signs may be absent between symptomatic episodes. During periods of angina, physical findings relating to ischemia and ventricular dysfunction may be present, including rales, jugular venous distention, peripheral edema, extra heart sounds, ectopy or other arrhythmia (eg, tachycardia or bradycardia), and murmurs (such as occur with ischemic mitral regurgitation).

Diagnosis

Severe chest pain, usually without physical effort and with a concurrent ECG showing transient ST elevation, is the key for the diagnosis of CAS. Diagnosis of the silent variety of CAS is possible if the vasospastic attack occurs under medical observation or during ambulatory ECG monitoring, but long-term surveillance may be needed to establish the diagnosis. Exercise testing may also be helpful, although approximately equal numbers of patients show ST depression, ST elevation, or no change whatsoever during the exercise. Further pharmacological testing, such as provocation with intravenous ergonovine, should be used only under special conditions and with extreme care.[1] Definitive diagnosis requires coronary angiography demonstrating focal spasm (defined as a 75% reduction in artery diameter) on the administration of ergonovine.

Distinguishing unstable angina pectoris related to coronary atherosclerosis from variant angina may be difficult and the distinction may be an arbitrary one because CAS and atherosclerosis co-exist in many patients. Moreover, CAS may be the cause as well as a consequence of plaque rupture and thrombosis in patients with unstable angina pectoris.

Complications

Myocardial infarction (MI) is a potential complication of variant angina, especially in the myocardial territory involved in the previous anginal attacks. The incidence of MI is varied but has been reported to be as high as 30% in some series. The prognosis of MI in patients with CAS depends on the extent and severity of any underlying atherosclerosis. Adverse outcomes are more likely and survival poorer in patients with multivessel atherosclerotic disease.. Most often, a relatively small myocardial territory is affected since quick reperfusion by relief of vasospasm is common. Transient sympathovagal imbalance, detected during Holter monitoring by a marked decrease in heart rate variability in the period immediately preceding the onset of the ST shift, was suggested as the trigger for sudden death during CAS related ischemia. Ventricular fibrillation, tachycardia, and complete atrioventricular block were repeatedly observed

during ischemic episodes caused by CAS, even if the attack was painless. The risk of sudden death is approximately 2% and is most common in patients with multivessel spasm and prior serious arrhythmia during anginal attacks. The natural history of patients undergoing medical therapy for coronary vasospasm may involve significant morbidity, but mortality is low in most cases, even on long-term follow-up**.**

Coronary Spasm Under Special Circumstances

In teenagers and young adults, the use of illicit substances, primarily cocaine, is an important cause of drug-induced CAS and resultant MI, with important therapeutic and prognostic implications. CAS is also associated with use of marijuana, alcohol, butane, amphetamine, chemotherapy, antimigraine, and antibiotic medications. Perioperative CAS is prevalent in elderly male patients with coronary risk factors; instability of the autonomic nervous system and vascular hyperactivity in these patients are presumed to be the underlying mechanism of CAs.

Catheter-induced spasm of the right coronary artery is a common phenomenon (however, left main coronary artery (LMCA) spasm during routine diagnostic catheterization is a relatively rare occurrence (Figure 1 and 2). Due to the wide disparity between treatment strategies for fixed obstruction versus spasm of the left main coronary artery, it becomes imperative that the operators differentiate spasm from fixed obstruction during coronary angiography. However, the inability to distinguish vasospasm from obstructive disease of the LMCA can lead to inappropriate referral for coronary artery bypass graft (CABG) surgery.[2] Factors predictive of LMCA spasm were increased catheter-to-LM diameter ratio, catheter-to-LM wall contact, vessel bulging, and acute catheter-to-LM angle. Attempts to ameliorate possible spasm by using intracoronary nitroglycerin seems like a reasonable routine in hemodynamically stable patients with significant LMCA stenosis.CAS is reported to occur in 1% to 5% of percutaneous coronary interventions and may be precipitated by guide wire insertion. Cardiogenic shock caused by severe coronary artery spasm immediately after stenting is frequently but not always resolved by local injection of nitroglycerin.[3]

Pathophysiology of CAS

Vagal withdrawal is most often the mechanism leading to spontaneous spasm, but a change in sympathetic activity may also have a role in CAS. Endothelial dysfunction through abnormalities of nitric oxide (NO) synthase and its reduced bioavailability and hypercontractility of vascular smooth muscle in spastic arteries are major factors in the development of CAS. However, Egashira and coworkers[3] demonstrated that NO was not decreased at the spastic sites of the coronary arteries; they pointed to additional mechanisms, such as enhanced phospholipase C enzyme activity inducing focal smooth muscle cell hypersensitivity in variant angina patients.[4]

Management

Initial acute medical management should include sublingual, topical, or intravenous (IV) nitrate therapy. Nitroglycerin administered by any route (intracoronary, IV, topical, or

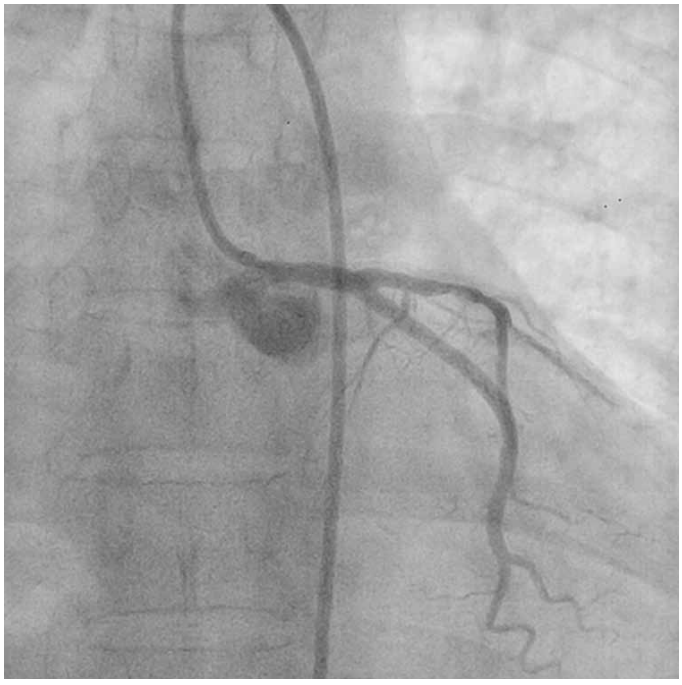

Figure 1: Spasm of LMCA after placing the guiding catheter for angioplasty

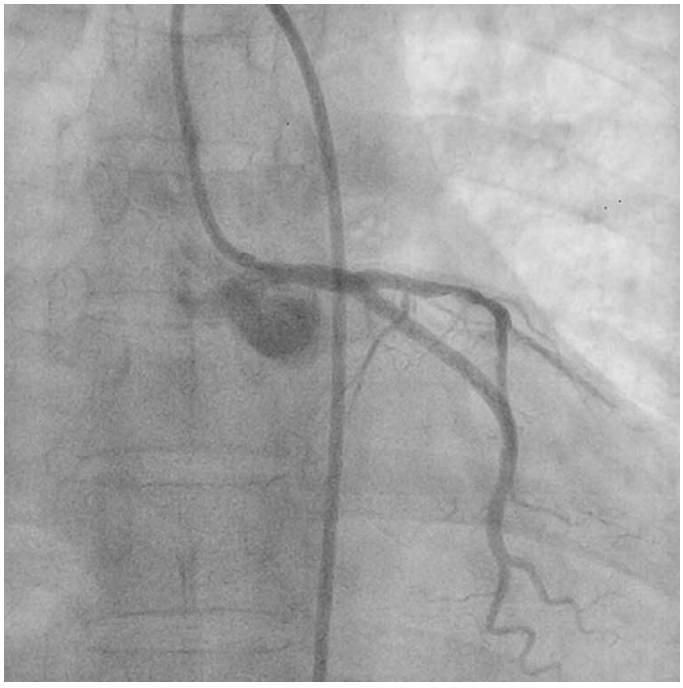

Figure 2: Spasm of LMCA relieved with NTG

sublingual) effectively treats episodes of angina and myocardial ischemia within minutes, and long-acting nitrate preparations reduce the frequency of recurrent events. The calcium channel blockers nifedipine, amlodipine, verapamil, and diltiazem effectively prevent coronary vasospasm and variant angina, and they should be administered in preference to beta blockers. Amlodipine may be preferable because of its long half-life.

Beta blockers are beneficial in most patients with atherosclerotic coronary stenoses and exertional angina pectoris and are sometimes helpful in combination with the above drugs to achieve control of symptoms in patients of CAS with co-existing atherosclerosis. However, nonselective beta blockers may be detrimental in some patients because blockade of the beta receptors, which mediate vasodilation, allows unopposed alpha receptor–mediated coronary vasoconstriction to occur and may worsen vasospastic angina in selected cases.

Until atherosclerotic coronary disease is excluded, standard therapies, including antiplatelet or antithrombotic agents, statins, and beta blockers, may be administered. Once the diagnosis of isolated CAS, calcium channel blockade and long-acting nitrates may be used for long-term prophylaxis. Magnesium deficiency is a possible factor contributing to CAS, and Teragawa and coworkers suggested that its long-term supplementation might also have a preventive effect[5].After an early report on the beneficial effect of cholesterol-lowering therapy on endothelial function and, consequently, a reduced coronary vasoconstrictor response to acetylcholine, suppression of acetylcholine-induced CAS through the addition of a statin (fluvastatin) to conventional calcium-channel blocker therapy was reported; the purported mechanism is inhibition of the RhoA-associated kinase pathway.[6] For patients who continue to have significant symptoms or signs of coronary vasospasm despite maximally tolerated medical therapy, in whom the culprit segment can be identified, coronary stenting may be considered. Coronary angioplasty performed in CAS patients produced results similar to those with typical angina.[7] However, bypass grafting of arteries without baseline obstruction should be reserved for patients with life-threatening ischemia that is refractory to maximal medical therapy. In these patients, adding complete plexectomy to the procedure may provide additional benefit. The indication for implantable cardioverter defibrillator (ICD) implantation in a patient with CAS is still not clearly established.

Prognosis

Spontaneous remission may occur, and some patients may be able to wean off or reduce their drug dosage. Long-term survival is believed to be good, especially in patients avoid smoking and are compliant with medical management including calcium channel blockers. Predictors of poorer prognosis include the presence of concurrent coronary atherosclerosis, ongoing smoking, intolerance of calcium antagonists, and spasm of multiple coronary arteries. The natural history of patients undergoing medical therapy for coronary vasospasm may involve significant morbidity, but mortality is low in most cases, even on long-term follow-up.

Conclusions

The key for diagnosis of vasospastic angina is episodic ST elevation concomitant with the rest angina. Ambulatory ECG monitoring, exercise testing, or both may provide a clue for the diagnosis, and coronary angiography with ergonovine challenge may provide definitive diagnosis. There is significant co-existing atherosclerosis in patients with CAS. Calcium antagonists are extremely effective in treating and preventing coronary spasm, with or without the additive vasodilatory effect of nitroglycerin, and may provide long-lasting relief for the patient. For patients with drug refractory angina and significant morbidity due to CAS, therapies include coronary stenting or bypass surgery may be considered if fixed coronary occlusions are demonstrated.

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Review Article

Pacemaker Follow Up

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Introduction

In the last few years, there has been an exponential rise in the number of pacemaker implants and the continuing evolution of device technology has resulted in increasingly complex devices capable of extensive programmability. However it is important to realize that the real challenge lies not only in the implantation of these devices but also in a comprehensive follow-up program, which should be an integral part of the long term patient management. The standards of pacemaker follow up vary widely across implanting centers and it is not uncommon to encounter patients with implanted pacemakers who have had a sporadic follow up, and then present only towards the end of pacemaker battery life. An organized and targeted follow-up is key to optimal utilization of the pacing system and therefore for the safety of the patient and continuity of medical care. Considering the evolution of pacemaker technology it is advisable that device follow up is best done under the domain of specialized pacemaker clinics under supervision of a Cardiologist or medical personnel sufficiently trained in pacemaker care. The article shall detail the scope, infrastructure and protocols to be followed in pacemaker clinics with a brief description about commonly encountered situations leading to pacemaker malfunction.

The aims of a comprehensive pacemaker follow up should be

a. To optimize the pacing system to the patient's needs and maximize the device capabilities and the generator life.

b. Identify abnormalities in the pacemaker system and any complications so as to be able to apply appropriate therapeutic algorithms

c. To reliably predict end-of-life of the pulse generator in order to permit elective and non-urgent change of the pulse generator.

d. To monitor the device implant site and manage any risk of infection

e. Accumulation of a patient database that offers information on patient demographics, present and past pacing systems including generators and leads. This can also expedite and initiate appropriate follow-up in the event of recall or advisory.

f. Provision of training opportunities for medical and para medical personnel.

g. To be able to address any patient queries related to pacing and provision of patient support and education.

Most pacemaker follow-up in our country occurs in the setting of a hospital based clinic, and the responsibility for programming is usually with a supervising physician. However in many cases pacemaker follow up is performed by a cardiac technician with/without physician supervision. Trans-telephone or remote home monitoring of patients involves additional expense and remains largely unapplied in our country, despite its technical feasibility.

The requisite personnel and infrastructure of a pacemaker follow up program include:

a. A physician who is well versed with pacing should be the primary person in-charge of the program. The lead consultant should not only be regularly performing pacemaker implants, but should also be closely involved in the pacemaker follow up program to remain familiar with the evolving pacing technology.

b. Although at many centers, pacemaker follow up is performed by paramedical personnel including nurses and technicians, they must always function under direct physician supervision.

c. The role of the Industry Professionals should be limited to

- Providing technical support about the pacing system
- Updating the physicians/paramedical staff on device related advisories and recalls.
- It is important for the pacemaker program in-charge to ensure that the program is not run under the sole supervision of Industry Professionals

The following basic equipment should be available in the pacemaker clinic or in its immediate vicinity: (which need not necessarily be on site).

a. 12-Lead electrocardiograph (ECG) machine

b. An appropriate range of manufacturer programmers (with appropriate documentation for use of each specific model) and contact details of all device manufacturers.

c. Emergency 'crash' trolley, defibrillator with integrated

pacing function and emergency drugs. Although these may rarely be needed, it is imperative for them to be on site.

d. Magnet

e. Dressing pack to address infection related issues.

f. Data management system to enter/access patient rated information

Presence of X-Ray, fluoroscopy, EP/cath lab, ambulatory ECG Recording, Echocardiography, stress testing and head-up tit testing etc. though not mandatory on site, should be available in close vicinity, if the need arises to order these investigations. **A system for rapid referral of any patient needing urgent admission should also be available.**

Pacemaker Clinic Procedures:

All follow up clinics need to operate under standard procedures/ protocols. Although these may vary according to local needs/ logistics, few minimum requirements should be met in all cases and should include the following at each visit, where possible:

a. Identification of the device and make of leads from the patient records

b. Documentation of symptoms like syncope, presyncope, dizziness, dyspnea or fatigue and concomitant drug history. While syncope and presyncope may result from failure to capture or pace, worsening dyspnoea or fatigue may be due to pacemaker syndrome in patients with VVI pacemakers or inappropriately programmed AV delays in those with DDD pacemakers.

c. Recording of an ECG to verify pacemaker function

d. Interrogation of the device and recording of any relevant information (any telemetry recorded events, appropriate counters/histograms etc.)

e. Assessment of device battery and lead status and comparison with previous records

f. Safe testing of device including thresholds for sensing and capture and impedance measurements

g. Appropriate troubleshooting for complications/problems and reprogramming to ensure that optimal settings for clinical outcomes are provided for the patient

h. Documenting all the above in patient records/pacemaker logbooks

i. Scheduling of the next appointment or referral

j. **Accurate record keeping is of utmost importance** and includes documentation of patient related demographics (including name, age, address and contact details), diagnosis and indication of implant, details of other co-morbid conditions, pacemaker operative record and manufacturer, model number and serial number of all implanted hardware. Patients who fail to attend for routine follow-up must be persuaded and encouraged to do so.

The frequency of follow up visits varies widely and is governed by existing co-morbid conditions as well as the geographic accessibility of the patient to medical care. The usual schedule of visits includes:

a. An early assessment should be performed, preferably within 72 hours of the implant, to assess the site of implant, rule out any acute infection and confirm adequacy of pacing. At hospital discharge, all patients should be issued an identification card with details of the pacing system and the hospital/physicians contact details. At this early visit, if there is a superficial infection often only aseptic cleaning and dressing with/without oral antibiotics is enough. Small boggy and soft hematomas usually self reabsorb within 4-6 weeks and should be managed conservatively. Deeper dermal pus collection needs explanation and subsequent re-implantation of the device. Needle aspiration should be avoided, as it more often tends to introduce infection.

b. A second follow up within 6-12 weeks with the aim to adjust for acute threshold changes and requisite output programming of the pacemaker settings to optimize safety margin. Since the capture threshold initially rises within the first 2-6 weeks post-implant, and then plateaus again, a visit between 2-4 months allows reprogramming of pacemaker settings based on the chronic values to enhance longevity of the pacemaker. Any patient questions/concerns about the pacing system need to be addressed.

c. Usually patients with single chamber pacemakers are reviewed annually while those with dual chamber pacemakers need twice a year follow up. A yearly follow up for pacemakers implanted for less than 7-10 years depending on the manufacturers recommendation and expected battery longevity is sufficient. Troubleshooting of any pacemaker/ lead malfunction is part of each pacemaker clinic visit. Intensified follow up (~6 monthly) should be instituted once the end of life (EOL) period approaches or 75% of expected battery longevity has been reached or when unexpected changes in lead impedance or threshold occur.

d. Pacemaker replacement should always be an elective procedure and is usually reliably diagnosed during regular pacemaker clinic follow up. Pacemaker upgrade or mode change should also be actively considered at the time of pulse generator change.

Common Clinical Situations

a. Failure to capture: Capture failure occurs whenever there is an absence of ventricular depolarization despite the pacemaker having delivered an output. Pacing artifacts are demonstrable on the surface ECG without accompanying

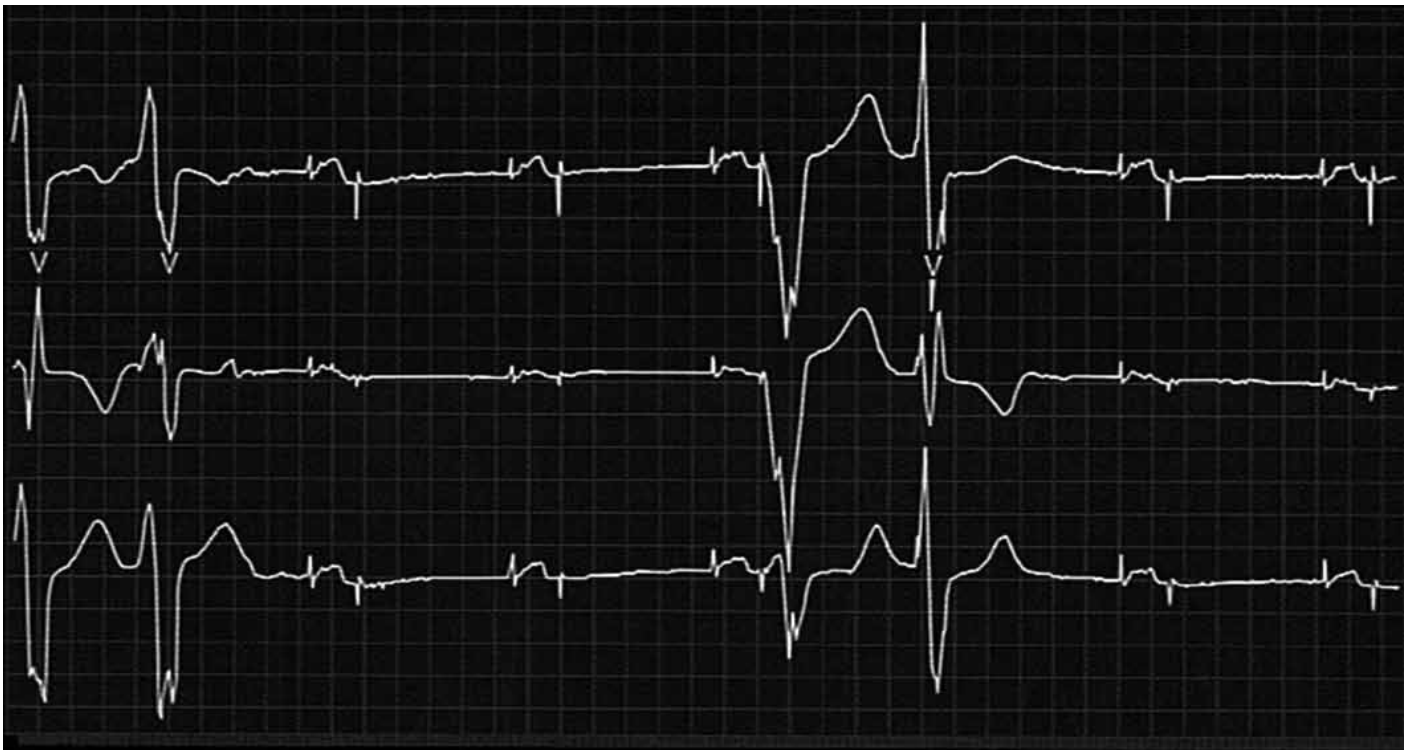

Figure 1 : Failure to capture – A Dual chamber pacemaker with normal atrial pacing and Intermittent ventricular pacing spikes not followed by ventricular paced complexes

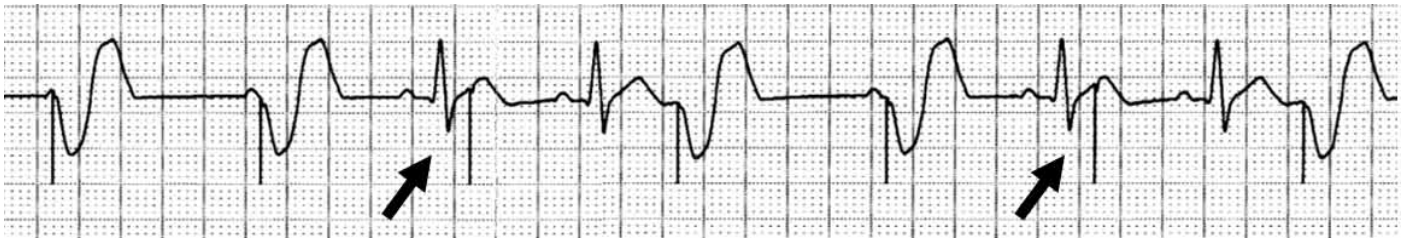

Figure 2 : Undersensing – The QRS complexes marked by the arrows are not sensed by the pacemaker, leading to inappropriate delivery of pacing.

QRS complexes. (Figure 1) Failure to capture can lead to presyncope, syncope, precipitation of heart failure and less commonly bradycardia dependant ventricular arrhythmias. While early lead dislodgment is the commonest cause of failure to capture (within first few weeks), in the time period beyond this, development of electrode tip fibrosis with an exit block is an important cause. Reprogramming to a higher output or urgent correction of metabolic parameters are often needed.

b. **Battery depletion**: Once battery depletion occurs beyond a particular limit, most pacemakers automatically reprogram to slower rates, and either single-chamber pacing or fixed rate pacing modes. When the ECG Is recorded with a magnet placed on the generator pocket site, most pacemakers pace at the magnet rate (ie. a fixed rate of 100 bpm). Although usually battery depletion initially causes the magnet rate to drop below 100 bpm, occasionally there may be a total lack of pacemaker output which can be disastrous for the pacemaker-dependent patient. These changes signify the need for elective pacemaker replacement; however even once these appear, there is usually a lag period of several months before the battery reaches a critically low voltage and pacing fails.

c. **Undersensing / Oversensing:** *Undersensing* is the failure of the pacemaker to sense and results in "pacing in excess of what is required or desired." (Figure 2). Causes include break in lead insulation, lead tip fibrosis, intrinsic myocardial disease or inadequate pacemaker programming. Undersensing "P" or "QRS" complexes may result in atrial/ventricular arrhythmias due to pacing occurring too soon after spontaneous cardiac activity. Programming to a numerically lower value makes the pacemaker "more sensitive" and may correct the abnormality. *Oversensing* is the unexpected or unwanted sensing of an intracardiac or

extracardiac signal, leading to inappropriate pauses in the paced rhythm. Electrical signals that may cause oversensing include myopotentials, T waves, and P waves. In dual-chamber pacemakers, pacemaker stimulus in one chamber may sometimes be sensed in the other chamber leading to chamber cross-talk (oversensing by the ventricular channel may lead to complete inhibition of pacing output causing asystole).

d. Pacemaker lead malfunction including lead or conductor fracture, insulation defect etc. can lead to pacemaker malfunction. *Lead fracture* is characterized by an increase in lead impedance and leads to capture failure. It may be visible on digital X –ray imaging or fluoroscopy and often need lead revision. *Insulation break* is characterized by reduced lead impedance (due to excessive current drainage) and may either lead to capture failure or loss of pacing output due to oversensing.

Conclusion

The increasing number of pacemaker implants as well as the evolving pacemaker and device technology, have made the management of patients with implanted pacemakers a clinical challenge. Appropriate pacemaker follow up should preferably be carried out by cardiologists or physicians trained in the basics of pacemaker management. Careful attention to basic tenets of pacing and ECG interpretation, and seeking expert opinion, whenever needed form an integral part of a comprehensive pacemaker follow up program.

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Cardiac Pacing and Defibrillation in Children and Young Adults

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Abstract

The population of children and young adults requiring a cardiac pacing device has been consistently increasing. The current generation of devices are small with a longer battery life, programming capabilities that can cater to the demands of the young patients and ability to treat brady and tachyarrhythmias as well as heart failure. This has increased the scope and clinical indications of using these devices. As patients with congenital heart disease (CHD) comprise majority of these patients requiring devices, the knowledge of indications, pacing leads and devices, anatomical variations and the technical skills required are different than that required in the adult population. In this review we attempt to discuss these specific points in detail to improve the understanding of cardiac pacing in children and young adults.

Keywords: Pacemakers, pacing, ICDs, pediatrics, congenital heart defects

Introduction

Pediatric pacing has progressed substantially since the first implant in a 14 yr old with myocarditis in 1962.[1] Current pacemakers have a much smaller size, longer battery life, multiple pacing and sensing modalities, and therapeutic capabilities in the form of detecting and treating tachyarrhythmias as well improving the contractility of a failing heart. Hence there is an increasing demand for pediatric pacing devices due to increase in clinical indications, technological advances and innovative techniques. However based on the 2010 Health Care Cost and Utilization Project (HCUP) database, only 0.6% of all the implanted cardiac devices have been in the pediatric population. The number of pediatric patients receiving pacemaker implantation has been stable over the past decade; however there has been a 4-fold rise in the number of patients receiving defibrillators and biventricular devices.[2]

Indications

a. Permanent Pacemakers

The most common indications for permanent pacemaker implantation in children, adolescents, and patients with congenital heart disease are:

1. Symptomatic sinus bradycardia related to sinus node dysfunction, associated with poor cardiac output or to prevent episodes of recurrent atrial tachycardias.

2. Advanced second- or third-degree AV block, either congenital or postsurgical, when associated with low cardiac output, ventricular dysfunction, complex ventricular ectopy, syncope or potential of recovery is minimal, especially after cardiac surgery.[3]

Important considerations in children and young adults are 1) an increasing number of young patients are long-term survivors of complex surgical procedures for congenital heart defects that result in palliation rather than correction of circulatory physiology. The residua of impaired ventricular function and abnormal physiology may result in symptoms due to sinus bradycardia or loss of AV synchrony at heart rates that do not produce symptoms in individuals with normal cardiovascular physiology. Hence, the indications for pacemaker implantation in these patients need to be based on the correlation of symptoms with relative bradycardia rather than absolute heart rate criteria. 2) The clinical significance of bradycardia is age dependent; e.g. a heart rate of 45 bpm may be a normal finding in an adolescent, the same rate in a newborn or infant indicates profound bradycardia. 3) Significant technical challenges may complicate device and transvenous lead implantation in very small patients or those with abnormalities of venous or intracardiac anatomy. 4) As there are no randomized clinical trials of cardiac pacing in pediatric or congenital heart disease patients, the level of evidence for most recommendations is consensus based.

b. Implantable Cardioverter-Defibrillators (ICDs)

ICDs are recommended for patients who have survived an episode of cardiac arrest, patients with poor cardiac function with evidence of moderate to severe heart failure, patients with inducible ventricular dysrhythmia in a setting of symptomatic CHD and in patients with genetic cardiomyopathy. Sudden cardiac death (SCD) in childhood and adolescence is associated with congenital heart disease, cardiomyopathies, and genetic arrhythmia syndromes. There is paucity of clinical experience and data regarding ICD implantation for primary prevention of SCD in young patients and therefore recommendations are based on extrapolation

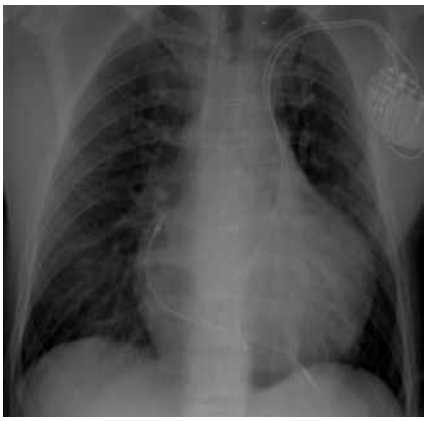

Figure 1: Dual chamber pacemaker lead implantation in a patient with left SVC without a bridging vein.

of data from adult studies. Unexpected sudden death is reported in 1.2% to 3.0% of patients per decade after surgical treatment of tetralogy of Fallot, with risk factors including ventricular dysfunction, QRS duration, and atrial and ventricular arrhythmias.[4] A significantly greater risk of SCD has been identified for patients with transposition of the great arteries or aortic stenosis, with most cases presumed to be due to a malignant ventricular arrhythmia associated with ischemia, ventricular dysfunction, or a rapid ventricular response to atrial flutter or fibrillation.[5] The lack of prospective and randomized clinical trials precludes exact recommendations regarding risk stratification and indications for ICD implantation for primary prevention of SCD in patients with postoperative congenital heart disease and ventricular dysfunction. ICDs may also be considered as a bridge to orthotopic heart transplantation in pediatric patients, particularly given the longer times to donor procurement in younger patients.[6]

c. Biventricular pacing (Cardiac Resynchronization Therapy, CRT)

There are no randomized multicenter studies regarding use of CRT in pediatrics and young adults. The limited worldwide pediatric experiences has shown that CRT is useful in select younger patients with clinical improvements comparable to adult patients and, in some instances, can delay or remove the need for heart transplant.[7] The current guidelines for adults suggests biventricular pacing for patients with wide QRS rhythm of left bundle branch block morphology with ejection fraction $\leq$ 35% and in functional heart failure class 2-3 despite medical management.[8] These are not easily extrapolated to the pediatric population. The incidence of ischemic heart disease is very low in pediatric patients. Younger patients typically require pacing therapy for bradycardia associated with congenital heart block (often with normal ventricular contractility) or progressive damage to the atrioventricular conduction system following surgical repair of various structural congenital heart defects.

Technical aspects of device implantation

The implantation of devices in children and young adults can be challenging especially in view of anatomical variations due to congenital defects and surgical procedures to repair the heart defects.

Anatomical considerations

It is important to understand the anatomy and have a thorough knowledge of any underlying heart defects, presence of intracardiac shunts and type(s) of surgical procedure(s) if any performed in the past. Venography may help define presence or absence of left superior vena cava, any obstruction, or anomalies as well as patency of the vasculature if previous leads are present. Patients with d-transposition of the great arteries (d-TGA) with atrial switch operation have surgical baffles connecting the superior vena cava (SVC) and inferior vena cava to the pulmonary (left) ventricle. If placement of a lead is anticipated and there is presence of narrowing across the superior baffle, it is useful to consider stent angioplasty of the SVC baffle prior to lead implantation. Patients with previous cardiac surgery may have their right atrial appendage amputated at the time of canulation. In patients with Fontan palliation for single ventricular physiology, an atrial lead may be implanted transvenously in patients with atrio-pulmonary connection or lateral tunnel palliation (but not the extracardiac conduit), keeping in mind that they have a passive venous flow circulation. The presence of left SVC without a bridging vein can make the implantation technically challenging albeit possible (Figure 1). Future growth of the patient must be taken into account during lead implantation.

Route of lead implantation

The pacing and defibrillator leads can be implanted via the transvenous (endocardial) or surgical (epicardial) route. The choice of route is dependent upon the size of the patient, anatomy and surgical procedures performed. The primary risk factor for obstruction after pacemaker lead implantation in children was found to be related to the size of the lead as compared to the body surface area at implantation. A ratio > 6.6 mm2/m2 was found to best predict venous obstruction, with a sensitivity of 90% and specificity of 84%. This data can be used to aid the physician in selection of a single or dual chamber lead system appropriate for the patient's size, thus decreasing the risk of venous obstruction, and hence preserving venous access.[9] Patient age, body size and lead characteristics at implant do not appear to predict occlusion in patients aged over 3 years.[10] For patients less than 10-15 kilograms, intracardiac shunt lesions, prosthetic tricuspid valve and circumstances where the anatomy or surgical palliation precludes access via the transvenous route, epicardial implantation is the route of choice. Epicardial lead

implantation requires sternotomy or thoracotomy or subxiphoid approach, and is associated with higher chronic stimulation threshold, higher lead failures and fractures and early depletion of battery life.[11-13] However it preserves the venous access for future use. There have been case reports and small series of patients less than 10 kg who have successfully undergone transvenous lead implantation.[14-16]

Endocardial lead placement offers the advantages of avoidance of thoracotomy, lower pacing thresholds, and a lower incidence of lead fractures. However its disadvantages include a greater risk of lead dislodgment, venous occlusion, embolic vascular events, and endocarditis.[17]

Programming of the device

Children have faster resting heart rates than adults and higher peak heart rates - it is not unusual for infants to have resting heart rates between 120 and 150 beats/min, and it is easy for children of all ages to attain sinus rates in excess of 200 beats/min during active play. Many pacemakers cannot pace at or track sinus rates beyond 180 beats/min and rates with defibrillators are even lower. These limits to the maximum tracking rate can result in a substantial decrease in exercise performance, peak oxygen consumption and anaerobic threshold.[18] In addition, higher heart rates result in increased battery utilization that can significantly impact the longevity of the pulse generators.

Use of single chamber vs. dual chamber ICD

Younger patients have higher sinus rates during exertion and increased frequency of supraventricular tachycardias especially in patients with congenital heart defects. It has been reported that 30% of patients with ICDs and congenital heart disease will develop supraventricular tachyarrhythmias during follow-up.[19,20] Meta-analysis of data from patients with ICDs reveals evidence that in those with dual-chamber ICD's, arrhythmia discrimination shows improved detection specificity without jeopardizing sensitivity; however the proportion of patients with inappropriate therapy was still approximately 20%, despite sophisticated arrhythmia discrimination.[21] There is no evidence that empirical use of any dual-chamber pacing approach improves mortality, quality of life, or reduces heart failure, ventricular tachyarrhythmia, or atrial arrhythmia. Moreover, the pulse generator longevity is about one-third less in dual- versus single-chamber ICDs.[22] In the case of obligatory pacing for symptomatic sinus node dysfunction, a dual-chamber strategy for minimal pacing at all chamber levels is recommended.[23]

Technique of device implantation

The handedness of the patient is determined as the device is preferably implanted on the nondominant side. The procedure is usually performed under general anesthesia. Antibiotic coverage is provided during and immediately after the procedure.[24] Based on the size of the patient and the device as well as cosmetics, the site of implantation is chosen. Subcutaneous pocket or submuscular pocket is created and rinsed with antibiotic solution. There is no difference in the pacing, sensing thresholds or defibrillation thresholds for ICDs in either the subcutaneous or submuscular implantation.[25] Some prefer the submuscular implantation in extremely thin individuals with minimal fat tissue to prevent device erosion and in patients with or at risk of Twiddler's syndrome. The vein is accessed with modified Seldinger technique or a venous cutdown. The number of leads decides the number of access sites in the veins. We attempt to access the axillary vein to avoid the complication of subclavian crush at the site of the ligament, reduce the risk of pneumothorax or hemothorax and less cumbersome extraction if necessary. The axillary vein is accessed by creating a roadmap by either placing a pacing catheter or by performing a venogram in the innominate vein or the cubital vein from a peripheral venous line. The ventricular leads are usually implanted first. The RV septum is usually targeted with manually shaping the stylets and the positioning confirmed on a biplane fluoroscope. The RV low septum is targeted in most of the patients with a routine curve to the stylet that lets it across the tricuspid valve followed by a posterior smaller curve near the tip of the stylet to obtain a septal position. If implanting a LV lead, the coronary sinus is accessed with special sheaths, an angiogram performed to delineate the anatomy and choose the target vein. Once the target vein is identified, the LV lead is implanted over a guide wire. If a dual chamber pacemaker is planned, an atrial lead is implanted next. The site of the Bachmann's bundle is preferred as it is associated with lower far field R wave sensing, atrial synchronization and prevention of atrial arrhythmias.[26-28] The Bachmann's bundle is located in the posterior high right atrial septum near the superior vena cava. The site is easily accessible using long sheaths and manually shaped stylets that have a smaller curve than the routine J-shaped stylet for positioning the lead in the right atrial appendage.[29] Pacing and sensing thresholds are determined. Pacing from each lead at 10V is performed to determine any potential phrenic nerve stimulation. The leads are secured with stay sutures in the musculature and around the pacing lead sleeves and attached to the generator. If placing an ICD, the defibrillation threshold (DFT) is obtained by the upper limit of vulnerability (ULV) testing or the binary search method. The upper limit of vulnerability (ULV) is the weakest shock strength at or above which VF is not induced when the shock is delivered at any time during the vulnerable period, which is the portion of the cardiac cycle during which shocks induce VF. ULV testing can be applied at ICD implant to confirm a clinically adequate defibrillation safety margin without inducing VF in 75%–95% of ICD recipients.[30] The binary search algorithm uses step-wise successive shock energies depending on the success of the previous shock. The lowest energy that successfully terminates the ventricular tachycardia is termed as the DFT.[31] The generator is secured and the incision is closed in multiple layers. The ipsilateral arm is immobilized in a sling for a period of 1-2 weeks to prevent lead dislodgement. The incision is kept dry for 7-10 days.

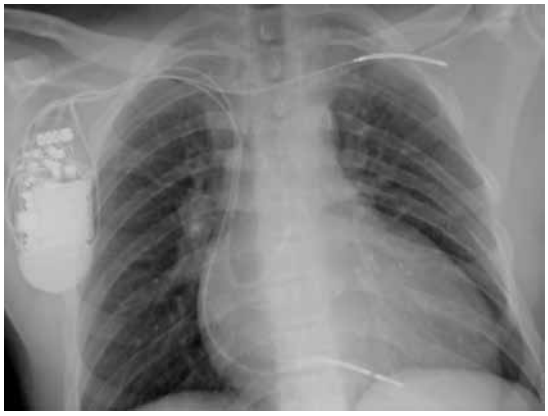

Figure 2: Use of an ICD coil in the left axillary vein in a patient with right sided generator implant to lower the DFTs. The atrial lead is in the posterior high right atrial septum near the Bachmann's bundle.

Based on variations in anatomy, the lead implantation technique may have to be revised. In patients with high DFTs at the time of ICD implantation, additional coils or subcutaneous array are implanted.[32] The placement of additional coils can be in the coronary sinus, azygous vein, or the left innominate or axillary vein (Figure 2). Implanting the device in the left axillary region has also been postulated to reduce the DFT.[33] Use of medications like Sotalol has been reported to lower the DFT.[34,35] In some group of patients a 'hybrid' approach to lead implantation is performed. If biventricular pacing is contemplated in patients with d-TGA with atrial switch palliation, a mini-sternotomy or thoracotomy is used to implant the systemic (RV) ventricular epicardial lead that is tunneled to the pocket where the generator with the transvenous leads is placed. In very small patients, ICD is implanted using a pericardial patch or a coil in the pericardial space with a bipolar sensing lead on the ventricle and implantation of the device in the abdomen.[36]

Follow-up

The patient and the device are assessed prior to discharge, in 1- 2 weeks for incision check, at 2- 3 months to assess chronic pacing thresholds and cardiac function (because of the risk of pacing-induced cardiac dysfunction), and then 6 months to yearly. Patients are advised to transmit data using the remote monitoring services on a 3 monthly basis or if any change in clinical status occurs. The remote monitoring is intensified to a monthly basis in pacemaker dependent patients and in patients nearing end of battery life. Any patient experiencing an ICD discharge is recommended to follow up in the closest emergency room to evaluate the appropriateness of the discharge, need for in-patient admission, or pharmacological intervention. Chest radiographs have been advised on a yearly basis in small children to recognize any growth related lead damage. Echocardiograms are performed on an annual basis to evaluate valvular and cardiac function. Stress test is recommended to assess exercise tolerance, maximal heart rates achievable, assess the rate response settings, and exercise related arrhythmias for fine tuning of the device for allowing maximal functionality in children and young adults.

Complications

Device implantation data using the Kids' Inpatient database from 1997-2006 revealed specific complication rates for all device types were pneumothorax 2.2%, hematoma 3.3%, endocarditis/ pericarditis 1.1%, surgical infection 2.4% and death 1.7%. Biventricular pacemakers have the highest percentage of acute complications (42.3%) whereas pacemakers (17.3%) and defibrillators (16.8%) were lower. Pacemakers had higher patient-related complications (11.2%) in comparison to ICDs and biventricular pacemakers and ICDs had higher device-related complications (11.5%) in comparison to the pacemakers and biventricular pacemakers.[2]

Techniques of implantation to aid extraction

With the technological advances making more devices compatible for younger patients and the increasing population of adult congenital heart defect patients requiring device implantation, the need for revision or extraction will continue to increase. Considering lead implantation techniques and hardware that lend them to easier extractions would be helpful. Older lead age, a lead in the ventricular position, and polyurethane lead insulation were found to be independent predictors of the decreased likelihood of a simple extraction.[37] Long implantation time, lack of operator experience, ICD lead type and female gender are possible risk factors for life-threatening complications.[38] Implantation durations of less than 3 years had a success rate of 100% whereas it was only 65.5% in those that were older than 3 years, most probably due to robust fibrosis in the young patient population.[39] Medial subclavian vein approaches are discouraged due to the risk of crush requiring subsequent and likely difficult extraction. In the dual coil leads, the SVC coil stimulates more aggressive fibrosis with high risk for vascular tear at time of extraction.[40] Use of leads that are appropriately sized for the patient will reduce the amount of extra lead left in the pocket that may need to be dissected. Leads that are constructed well so as not to fall apart easily, and leads that are isodiametric with active fixation, are likely to be more easily and completely removed.[41] If passive fixation leads are to be used, shorter tine length will make extraction easier. As the IS-4 standard becomes widely available for ICD leads, this will eliminate the "yoke" on these leads, making dissection easier as well. The use of ICD leads that use coils backfilled with medical adhesive, or that are covered with Gortex™ markedly reduces the tissue in-growth and facilitates easier and safer extraction.[41,42]

Conclusion

The utility of cardiac devices in the pediatric population is increasing due to technological advances as well as improved survival of patients with congenital heart defects. Symptomatic

bradyarrhythmias, risk of sudden death, heart failure are the broad indications for implantation of a cardiac device. The selection of device and leads as well as the technique of implantation are based on the patient size and anatomy. Careful selection of the device, leads and technique can help reduce complications associated with the implantation as well as aid in extraction of the devices in the long term.

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Case Report

Isolated Right Ventricular Infarction - A Case Report and Review of Literature

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Abstract

A rare case of isolated right ventricular infarction is described herewith along with the review of literature.

Key Words

Right ventricular infarction, Angiography, Right coronary artery stenosis.

Introduction

Right ventricular myocardial infarction (RVMI) usually accompanies inferior-posterior wall myocardial infarction.[1] Isolated RVMI is rare, accounting for 3% of all infarctions[2] and reported in literature secondary to occlusion of the right ventricular branch as a complication during percutaneus intervention.[3,4] In the present report, we describe a 45 year old man presenting with isolated right ventricular myocardial infarction in the absence of any percutaneous intervention.

Case Report

A 45 year old man presented in emergency department with severe retrosternal chest pain, associated with sweating since 2 hours. He was a smoker and had a positive family history of cardiac disease. On examination his pulse was 74/min, blood pressure 100/70 mmHg, respiratory rate 20/min and cold extremities. There was pallor, but no cyanosis, clubbing, icterus, pedal odema or lymphadenopathy. Jugular venous pressure was raised (10cm of H_2O) with prominent inspiratory rise. Cardiovascular examination was unremarkable. Lungs were clear. Chest skiagram showed mild cardiomegaly. The electrocardiogram (ECG) showed selective ST- segment elevation in right precordial leads from RV_6 to RV_3 (Figure-1). Serum cardiac markers were elevated. Transthoracic echocardiogram in apical 4 chamber view showed hypokinetic free wall of right ventricle (RV) (Figure-2). His coronary angiogram revealed patent left coronary arteries (LAD & LCx) (Figure-3) with isolated stenosis (99%) in right coronary artery (Figure-4). He was thrombolysed with fibrinolytic and prescribed enoxaparin, aspirin, clopidogril, ramipril and atorvastatin. The in-hospital course was uneventful, and he was discharged after 5 days.

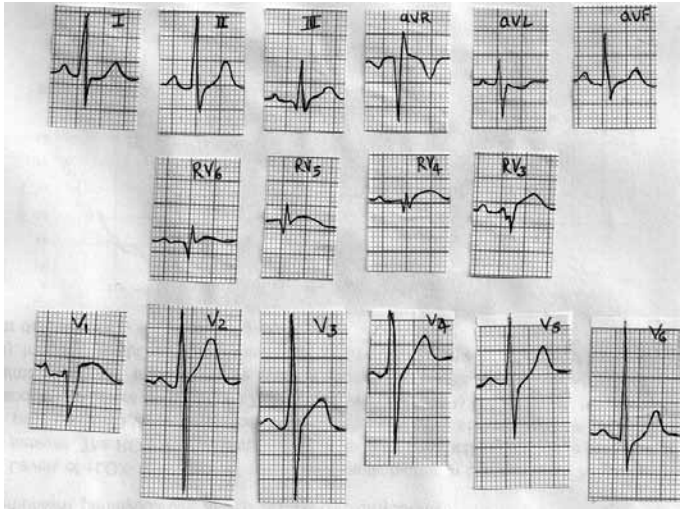

Figure 1: Electrocardiogram showing 'ST' segment elevation in right precordial leads from RV_6 to RV_3

Discussion

Early recognition of the RVMI is important, as it defines a significant clinical entity, associated with considerable immediate morbidity and mortality.[5] Inferior myocardial infarction (IWMI) with RVMI has a high mortality rate of 25% to 30% as compared with only 6% for IWMI not involving the

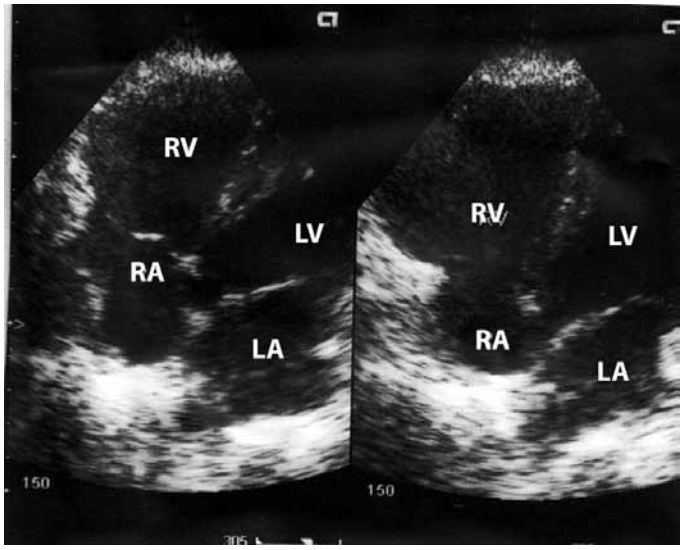

Figure 2: Transthoracic echocardiogram (4 chamber view) showing hypokinetic free wall of right ventricle.

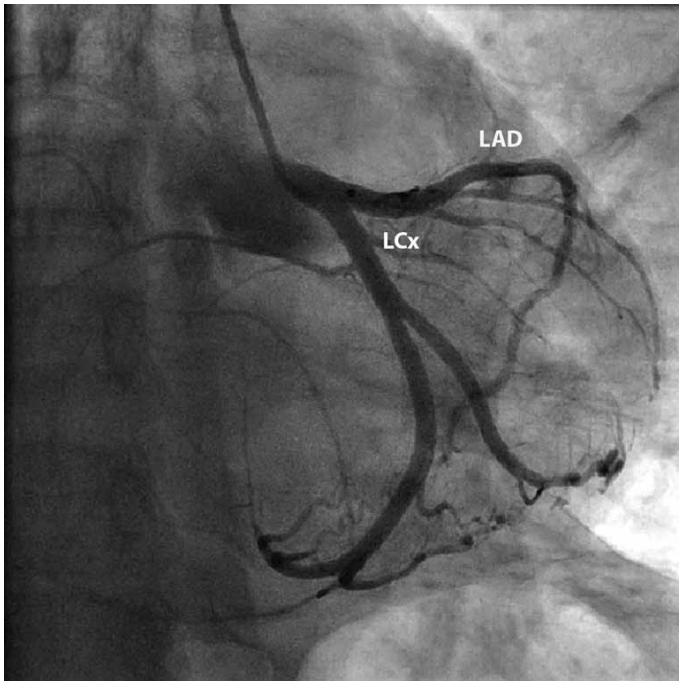

Figure 3: Left coronary angiogram showing patent left coronary arteries (LAD & LCx).

right ventricle.[6] Most often, an RVMI which occurs in concert with an IWMI is caused by occlusion of the proximal right coronary artery. If occlusion occurs distal to acute marginal and right ventricular branches, RVMI does not occur.[7] The triad of hypotension, elevated jugular venous pressure and clear lung fields has been recognized as clinical markers of RVMI in acute IWMI. Pulsus paradoxus and Kussmaul's sign in the setting of an IWMI indicate a hemodynamically significant RVMI (sensitivity - 88% and specificity - 100%).[8] The right ventricular involvement can be diagnosed with a predictive accuracy well above 80% by the presence of ST-segment elevation of ≥1 mm in the right-sided precordial lead, V_4R in the presence of an acute IWMI (sensitivity - 70% and specificity - 100%).[9] The ST-segment elevation in V_4R is a strong independent predictor of major complications and in-hospital mortality.[10] It is important to recognize that ST-segment elevation in right precordial leads may be transient[11] and absent in one half of patients with RVMI after 12 hours of onset of chest pain.[12] Thus it is imperative to record right-sided precordial leads in all patients with IWMI as soon as possible for an accurate diagnosis and prompt initiation of treatment.

After RVMI, mechanical or electrical complications may develop. Papillary muscle dysfunction or rupture may result in marked worsening of mitral or tricuspid regurgitation. As right atrial pressure increases, right-to-left shunt may occur through a patent foramen ovale. Ventricular septal defects frequently complicate RVMI. So in patients with RVMI continuous monitoring and echocardiography is warranted for early detection of these developing complications.[13]

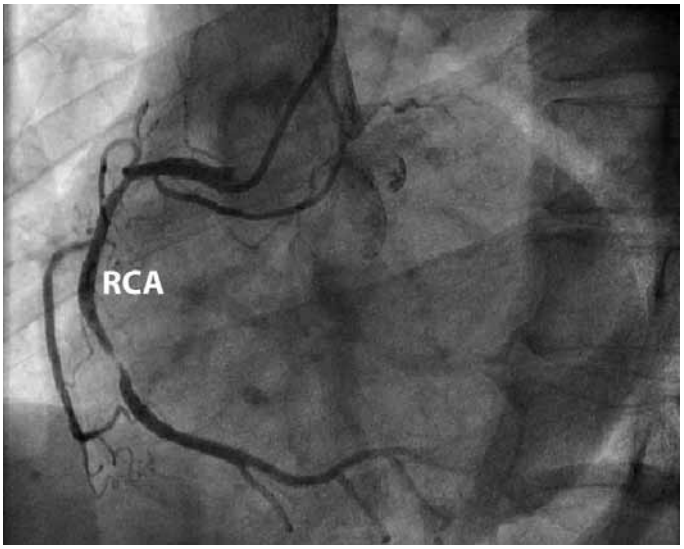

Figure 4: Right coronary angiogram showing 99% stenosis of right coronary artery.

Patients with RVMI are prone to develop Bezold-Jarisch reflex mediated bradycardia.[14] This is secondary to stimulation of receptors heavily concentrated in the inferior and posterior walls of the heart, which through afferent and efferent limbs of the vagus nerves, leads to enhanced parasympathetic tone. Bradyarrhythmias result in atrioventricular dyssynchrony and loss of right atrial contribution leading to severe hemodynamic compromise.[15] The development of high-degree atrioventricular block has been reported to occur in as many as 48% of patients with RVI.[16] Although atropine may restore physiologic rhythm, in some patients atrioventricular sequential pacing may be necessary.[17] Atrial fibrillation can be managed by using immediate cardioversion to reverse signs and symptoms of cardiogenic shock.[13] Hence the presence of RVMI should raise a clinical alert for its potential immediate life threatening consequences and has a well-delineated set of priorities for its management.[1]

The therapeutic options for RVMI differ from those in a patient with isolated or predominant left ventricular infarction. Vasodilators (Nitroglycerin), diuretics, and morphine are not well tolerated by patients with RVMI and may lead to severe hypotension by reduction in preload by decreasing filling pressures and subsequently decreasing cardiac output. Therefore, volume loading with an isotonic solution is recommended as the initial therapy in RVMI. Progressive volume loading can produce an incremental increase in right-sided filling pressures, systolic blood pressure, and cardiac output.[18] If the hemodynamic parameters do not change markedly after volume loading with intravenous fluids, addition of an inotropic medication, such as dobutamine , may be beneficial to enhance ventricular contractility and cardiac output. In patients with acute RVMI, treatment with aspirin, clopidogrel, intravenous heparin, and glycoprotein IIa/IIIb inhibitors should be considered. Urgent

reperfusion with fibrinolytic agents or percutaneous coronary intervention is warranted because the earlier reperfusion occurs, the better is the chance of decreasing the size of the RV infarct and more preservation of ventricular function with rapid hemodynamic improvement.[19] When substantial left ventricular dysfunction occurs in conjunction with an RVMI, and cardiac output continues to decrease and shock is eminent, an intra-aortic balloon pump can be used to reduce afterload and provide the added benefit of augmenting coronary perfusion.[13] Coronary angiography with percutaneous coronary intervention should be performed in this situation.

Conclusion

It is important to record right sided precordial leads in patients presenting with chest pain in emergency department. This will assist in diagnosing patients with isolated RVMI cases. Early recognition and prompt reperfusion with thrombolytic therapy or coronary angioplasty, supported with intravenous fluids (maintaining preload), inotropic support, AV synchrony, and rate and rhythm control is warranted. It is heartening to know that, patients who survive the acute phase by timely diagnosis and intervention, often recovery occurs over a period of weeks to months.

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Case Report

QRS Axis Alternans

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The Madras Medical Mission

A 55 year old diabetic and hypertensive male presented with acute onset typical angina. His ECG at the time of admission (Figure 1) showed alternating QRS axis with sinus tachycardia of 155 bpm. With the relief of pain his heart rate gradually slowed. However the alternating QRS axis pattern persisted (Figure 2). His coronary angiogram revealed triple vessel disease. Post bypass surgery the ECG showed normal axis with no significant ST-T changes.

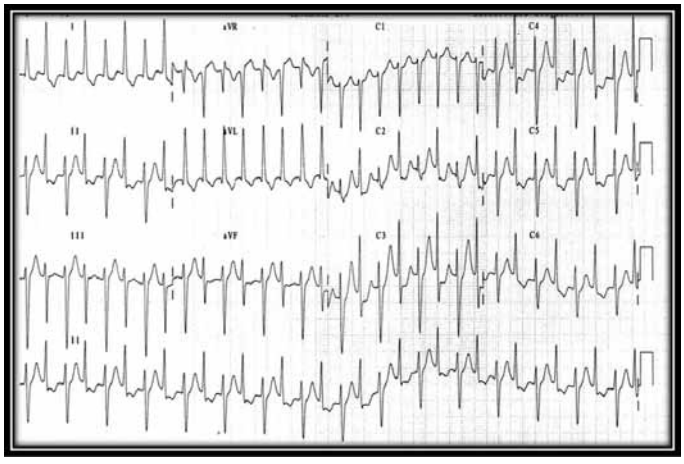

Figure 1 : Sinus tachycardia with QRS axis alternans during chest pain. The rate gradually slowed with relief of pain (Figure 2). The ST segment depression is pronounced during incomplete LAFB.

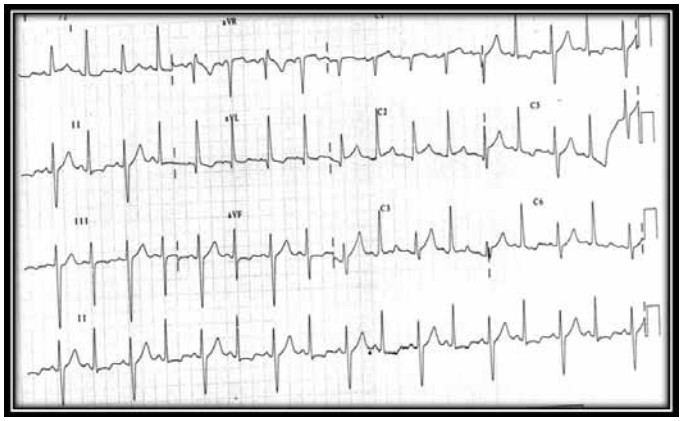

Figure 2 : The ECG after relief of pain. The alternating QRS axis is due to complete and incomplete LAFB. The first QRS complex is wider (100 ms) and has more leftward axis (-45^{o}) than the second QRS complex (80 ms and -10^{o}).

Discussion

The wider QRS complex has the following characteristics.

1. Duration of 100 ms
2. Axis of -45^{o}
3. q in aVL
4. r S in inferior leads

The above features are typical of complete LAFB. The larger than usual 'R wave' in the inferior leads during both complete and incomplete LAFB is probably due to significant underlying left ventricular hypertrophy.

The Narrow QRS complex also has q in aVL with r S in Lead III and aVF. These features are suggestive of incomplete LAFB.

The ladder diagram depicts the mechanism of alternating QRS axis. The right bundle (A) and the left posterior fascicle (B) are shown conducting normally. The left anterior fascicle is shown failing to conduct completely (C) and sluggishly (D) during the alternate beats. The alternating complete and incomplete LAFB is a manifestation of 2:1 Wenckebach phenomenon in the fascicle. The conduction velocity and refractory period determine the degree of block. The reversal of LAFB post revascularization indicates the underlying mechanism of QRS axis alternans is ischemia.

Case examples of QRS axis alternans:

1. QRS axis alternans during Sinus Rhythm:

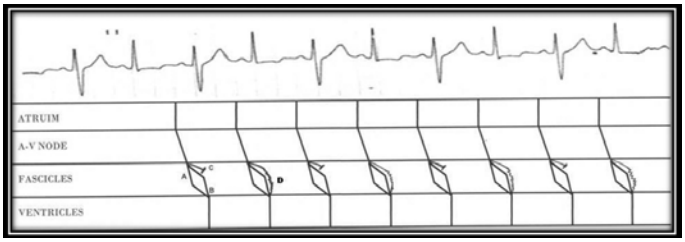

Figure 3 : The ladder diagram depicting the mechanism behind QRS axis alternans. The first wider QRS complex is due to complete LAFB and the narrower QRS complex is due to incomplete LAFB. (LAFB- Left Anterior Fascicular Block, A- Right Bundle Branch, B- Left Posterior Fascicle, C- Complete LAFB, D- Incomplete LAFB).

a. Alternating complete and incomplete left anterior fascicular block as discussed above (Figure 2).

b. Right Bundle Branch Block with alternating left anterior fascicle and left posterior fascicular block (Figure 4).

c. Rate dependent LAFB (Figure 5).

d. Intermittent left anterior fascicular block. (Figure 6).

e. Alternating RBBB and LBBB (Figure 7).

2. QRS axis alternans during Ventricular Tachycardia:

The prototype (Figure 8) ventricular tachycardia with QRS axis alternans is commonly described as "Bidirectional VT". The causes of bidirectional VT are

a. Digoxin toxicity

b. Familial catecholaminergic polymorphic ventricular tachycardia

c. Aconite poisoning

d. Acute myocardial infarction

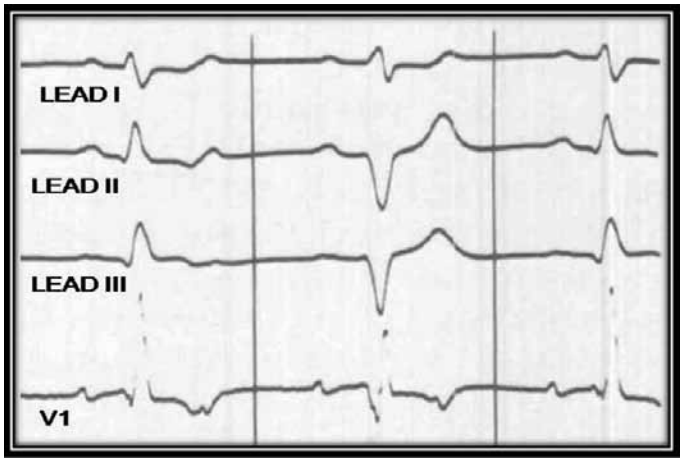

Figure 4 : Right Bundle Branch Block with alternating left anterior fascicle and left posterior fascicular block.

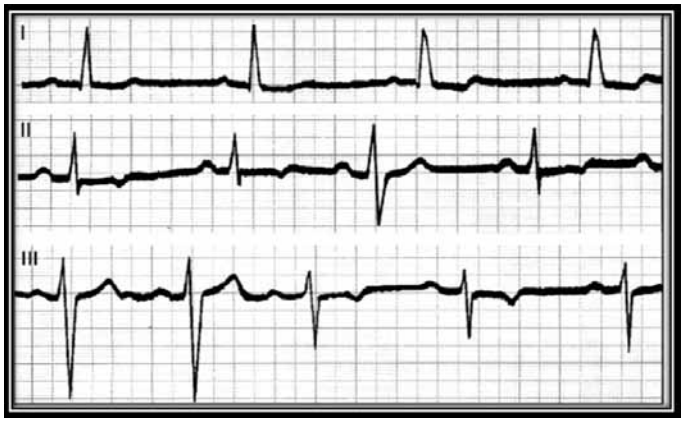

Figure 5 : Rate dependent LAFB.

QRS Alternans

The phenomenon of the beat to beat QRS voltage changes is called QRS alternans. Typically QRS alternans is not associated with QRS axis alternans. The mechanism of QRS alternans is not clear. However beat to beat change in the cardiac position and cardiac volume has been implicated in the mechanism.

1. QRS Alternans during Sinus Rhythm:

Massive pericardial effusion, bronchial asthma and emphysema may occasionally produce QRS alternans during sinus rhythm (Figure 9).

2. QRS alternans during Tachycardia:

Any rapid tachycardia, especially in the young, may produce QRS alternans. More often for no known specific mechanism orthodromic tachycardia using an accessory pathway in young is associated with QRS alternans. However QRS alternans alone is not specific for any tachycardia mechanism (Figure 10).

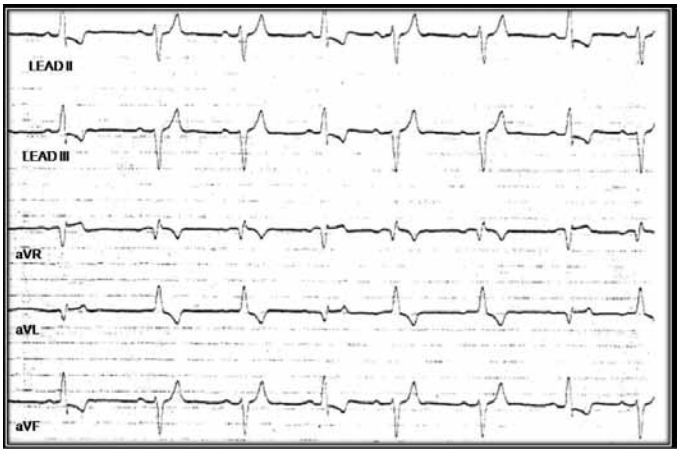

Figure 6 : Intermittent left anterior fascicular block.

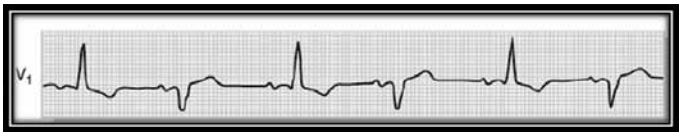

Figure 7 : Alternating RBBB and LBBB.

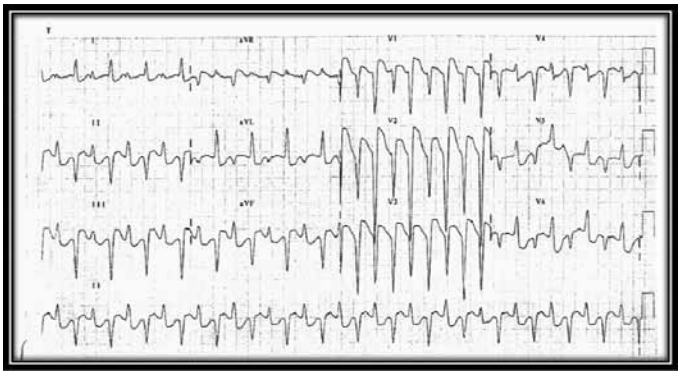

Figure 8 : Bidirectional Ventricular Tachycardia.

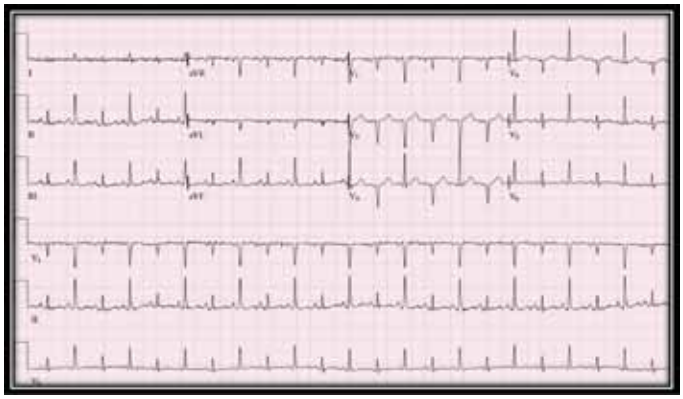

Figure 9 : QRS alternans in a case of pericardial effusion

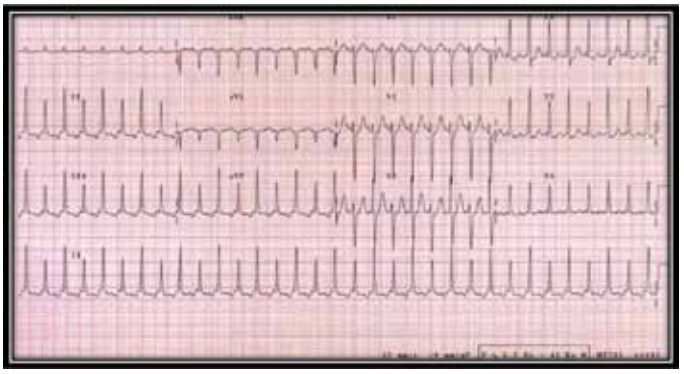

Figure 10 : QRS alternans during Orthodromic tachycardia

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Case Report

Mystery of the missing QRS complex!

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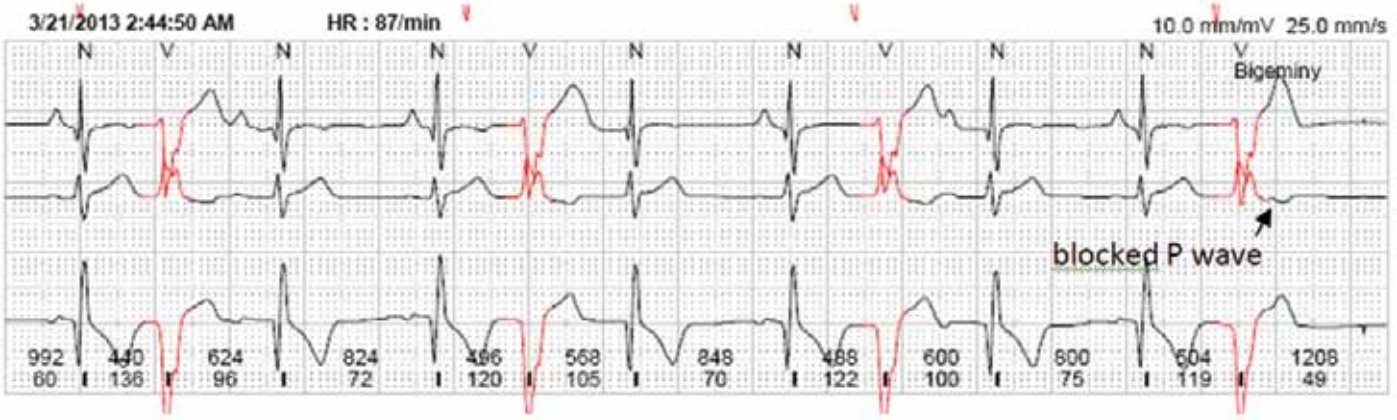
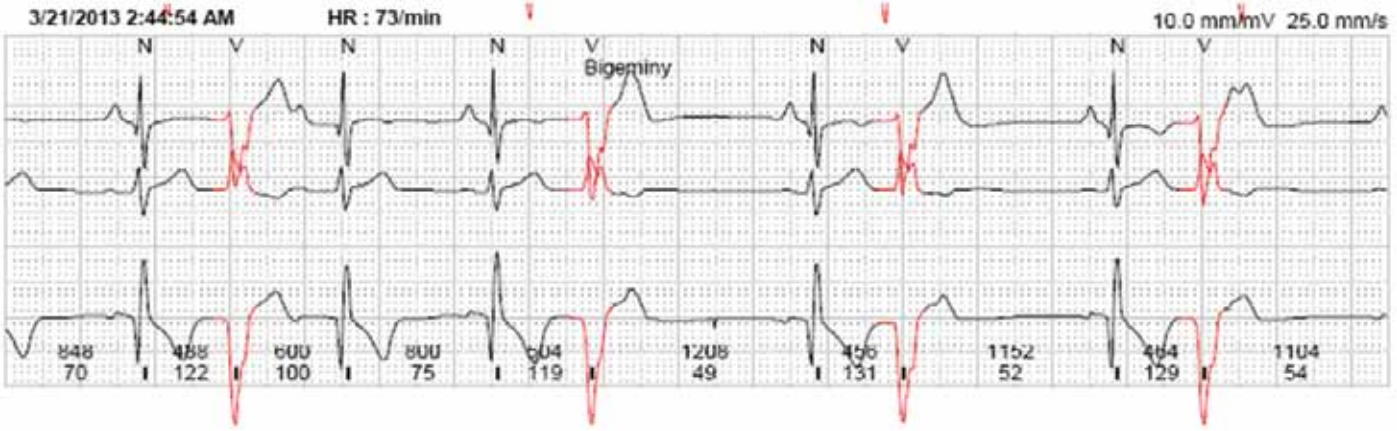
This is a routine Holter monitor performed on a patient with frequent bigeminy and occasional dizziness. What is the diagnosis?



Ventricular premature complexes (VPC) can penetrate the AV conduction system retrogradely and affect the antegrade conduction of the subsequent P wave.

This Holter shows variable degree of the AV conduction following the VPCs, depending on its coupling interval. The VPC with coupling interval of 440 ms in the first strip results in PR prolongation and with progressive delay in the PVC, there is further prolongation of the PR interval and eventually a 504 ms coupling interval results in blocked P wave (last complex).

How then can you the following strip in the same patient, wherein a PVC with a coupling interval of 488 ms prolongs the PR, 504 ms coupling interval blocks the P wave but the following 456 ms coupled PVC continues to result in subsequent P wave being blocked?



This is because the preceding longer RR interval prolongs the AV nodal refractory period and therefore even a relatively shorter coupled PVC blocks the following P wave.

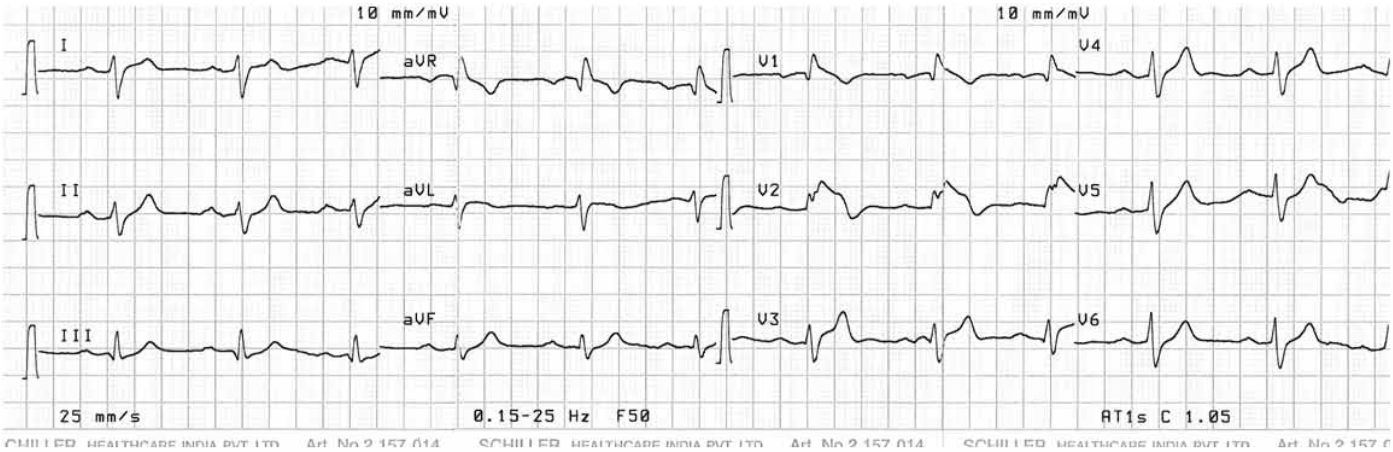
ECG Quiz

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

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

ECG - 1

45 yr old man; unexplained syncope. Brother had died suddenly



Examination, echocardiogram normal. What next?

a. Holter

b. Coronary angiography

c. EP study

d. ICD

For correct answer see overleaf

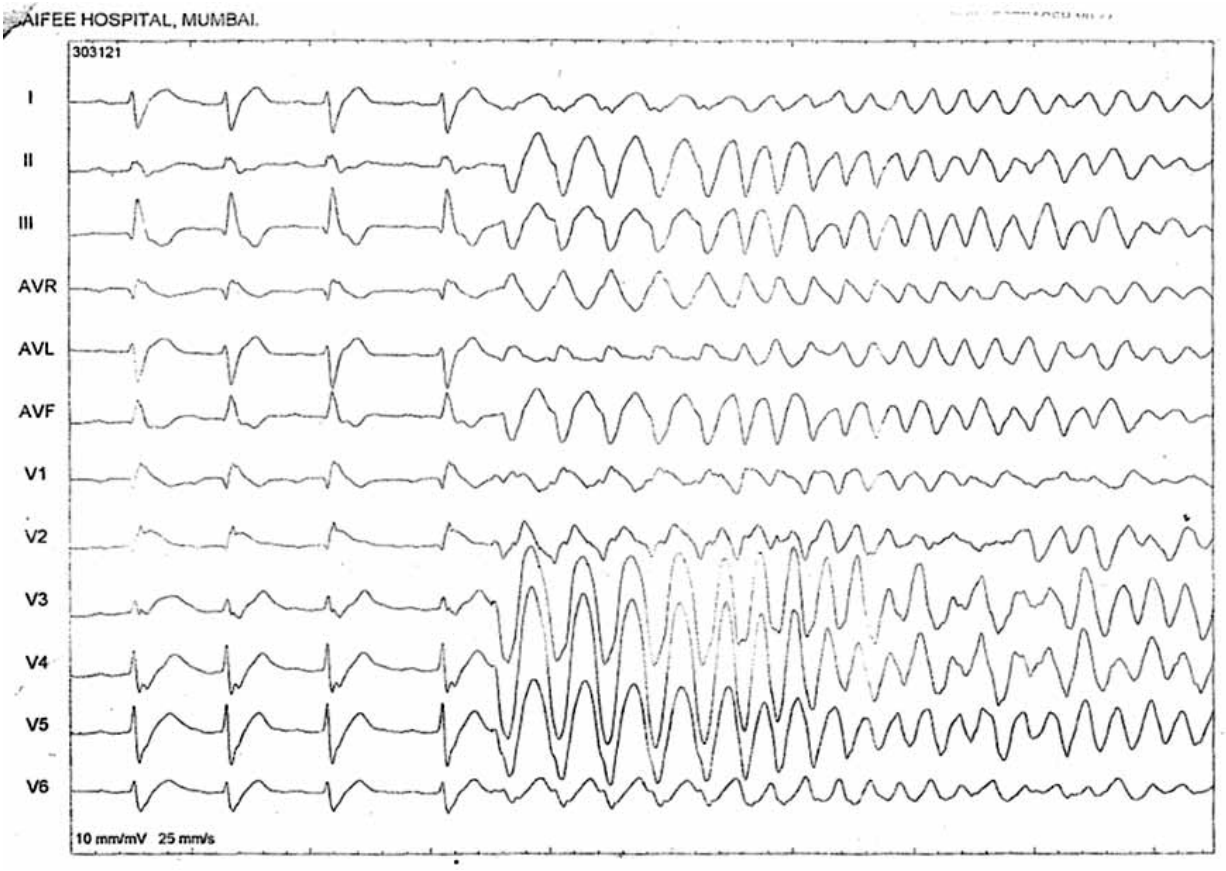
ECG - 1

The correct answer is 'd' – ICD.

The limb leads show an indeterminate QRS axis and PR prolongation (240 ms). Lead V1 and V2 suggest an incomplete RBBB pattern along with marked convex ST elevation. This is a classical Type I Brugada pattern. In view of the 'malignant' history, this patient has Brugada syndrome and is at a high risk of SCD due to VT/VF. Unfortunately, there is no reliable test to prognosticate and there is no effective drug therapy. Therefore, an ICD (preferably dual chamber in view of AV delay) becomes mandatory.

However, the patient and his family requested if any further studies could be done to be certain about the diagnosis. Therefore, provocative testing with IV flecainide was performed along with an EP study. Such maneuvers do have a reasonable positive predictive value. Figure 1a shows the gross QRS widening with flecainide, alongwith easy inducibility of VF. Subsequently, the patient underwent a dual-chamber ICD implantation.

Figure 1 a



ECG - 2

48 yr old man. Asymptomatic. “Routine” stress test….

Figure 2

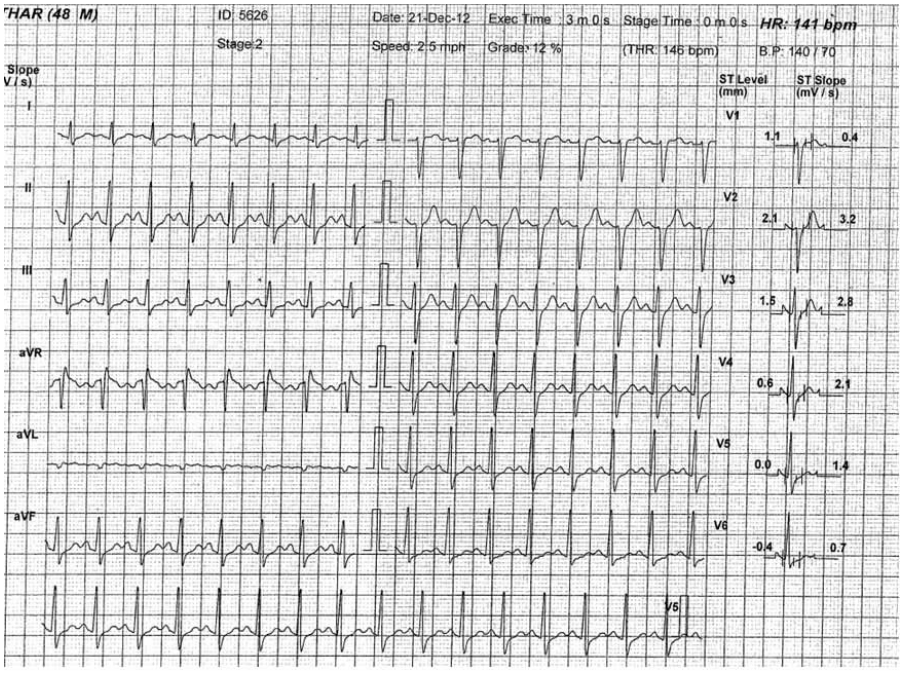
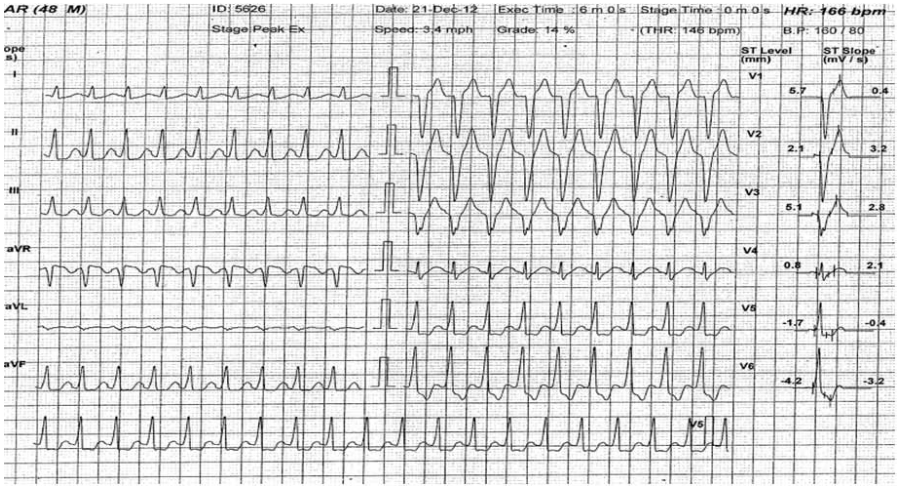


Figure 2a



a. Artifactual trace

b. Sinus tachycardia with LBBB

c. SVT

d. VT

For correct answer see overleaf

ECG - 2

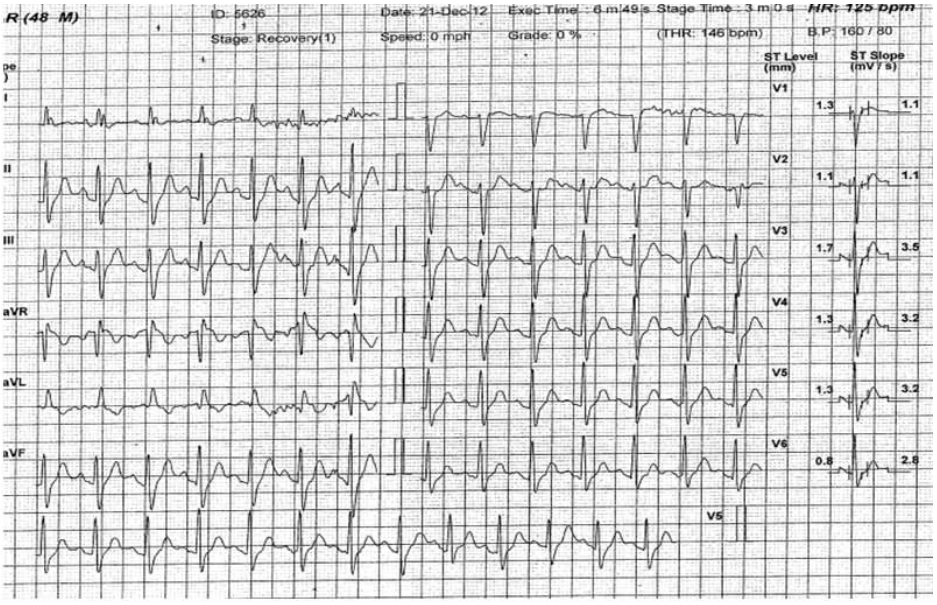
The correct answer is 'b'

There is an increase in the ventricular rate to 166 bpm with exercise alongwith widening of the QRS complex, The QRS resembles the LBBB pattern and has a sharp initial downstroke in lead V1. While P waves are not clearly seen, careful observation in lead II suggests that the P wave merges with the downslope of the T wave.

Artifactual waveforms are not uncommon with 'Linked Median' software manipulation as seen in many stress test machines. These are done to make the ECG look free of noise but in reality are a distortion and should be avoided. The typical exercise-induced VF has a RVOT origin with LBBB-like pattern ***(but with a slow downstroke in lead V1)*** and right axis deviation.

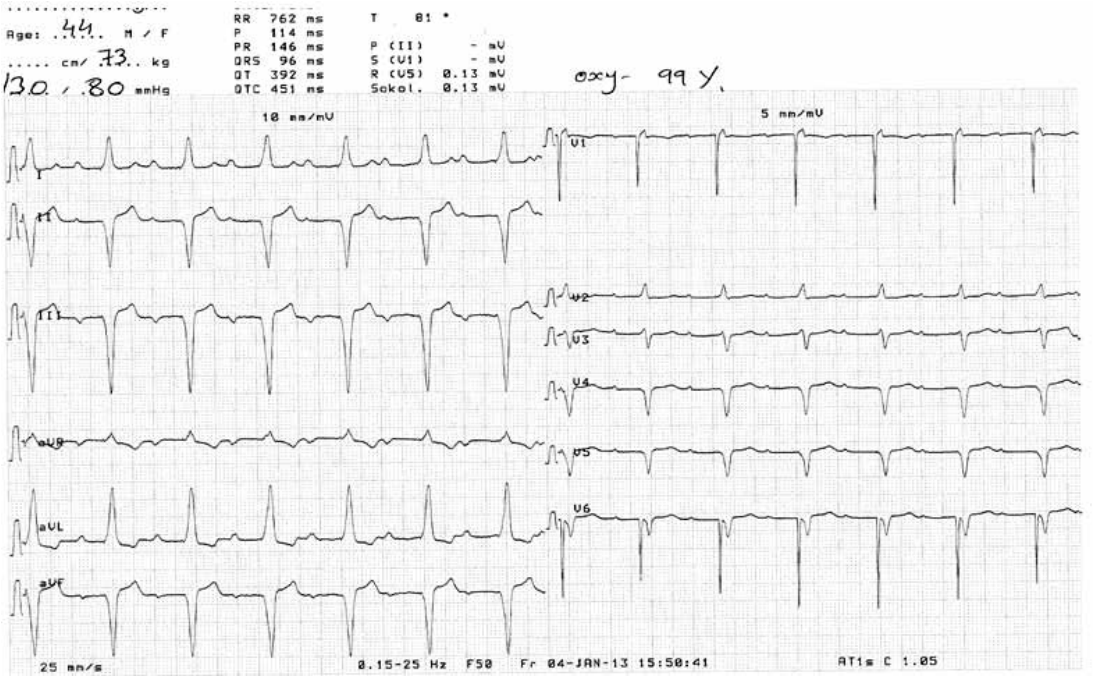
The simplest way to confirm the diagnosis of rate-related LBBB would be to observe that the onset and termination of LBBB occurs in conjunction with a change in heart rate. Figure 2b shows that as the heart rate settles, the LBBB disappears.

Figure 2b 30 seconds later…



ECG - 3

A pacemaker was implanted several years ago…



a. Single chamber (VVI) pacemaker, retrograde P waves

b. Dual chamber pacemaker (P sensed, QRS paced)

c. None of the above

For correct answer see overleaf

ECG - 3

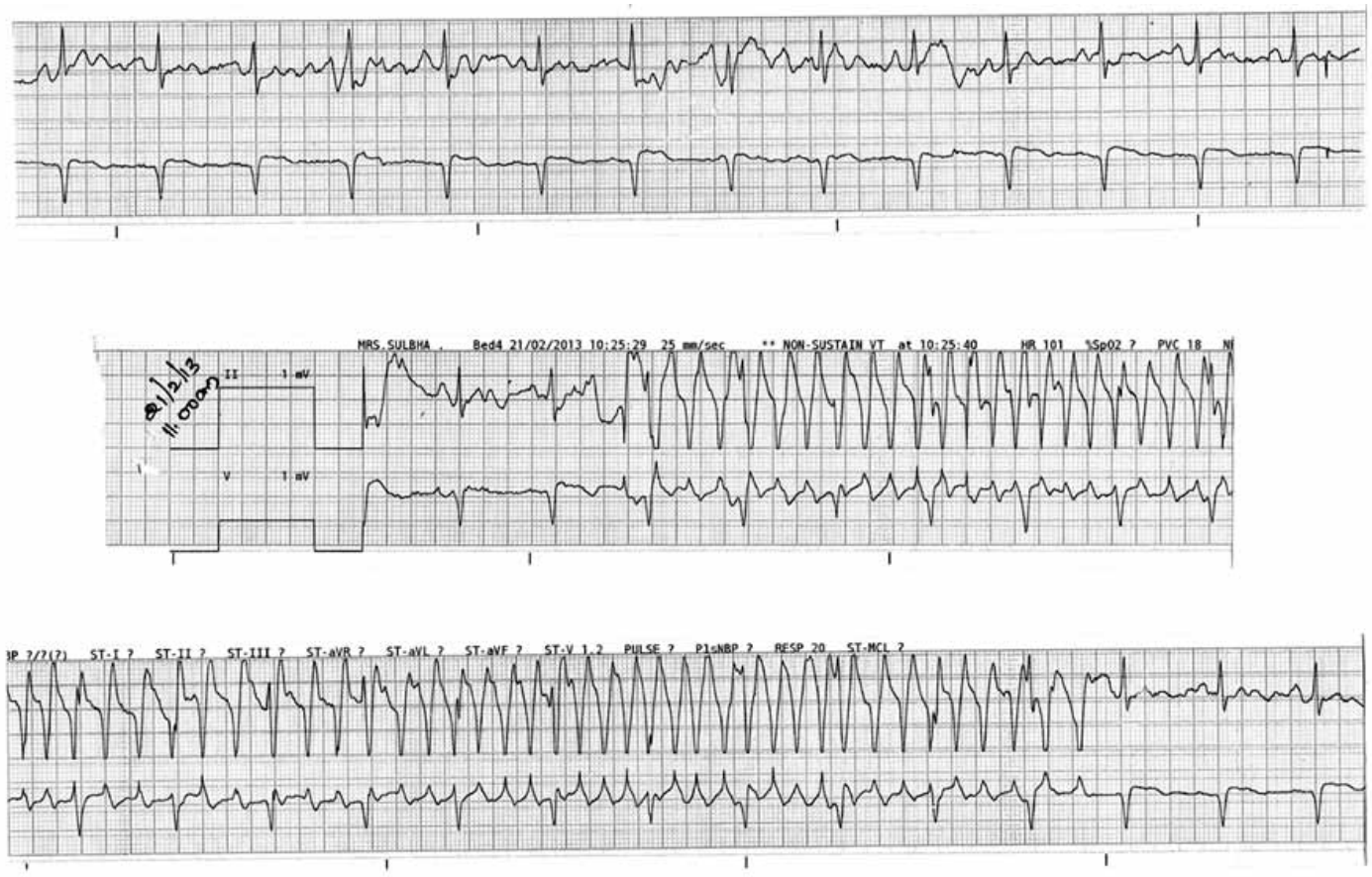
The correct answer is 'c'

The ECG shows constant ventricular pacing. The pacemaker artifacts are best seen in leads V1 and V6. There is one P wave seen before each QRS complex. These P waves are positive in leads I, II, V4, V5 and V6. Hence, these are normal P waves. Retrograde P waves would be inverted in lead V2. The PR interval is constantly changing which cannot happen with a dual-chamber pacemaker (except in the rare situation with marked sinus tachycardia which exceeds the programmed upper rate).

Hence, this is a single chamber VVI pacemaker which is functioning normally. Coincidentally, the sinus rate is similar to the pacing rate. Since, no P wave is conducted, the indication for pacing must have been AV block.

ECG - 4

65 yr old lady. Recent PAMI for anterior MI. Needed ventilator and IABP. 1st day of mobilsation



a. Artifact

b. Monomorphic VT

c. Polymorphic VT

d. SVT with aberrancy

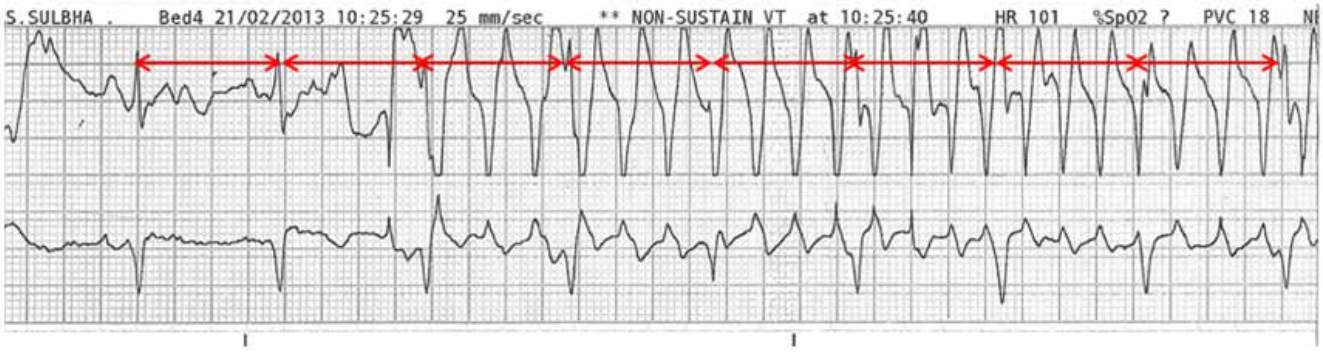
For correct answer see overleaf

ECG - 4

The correct answer is 'a'

The difference in pattern between the two leads helps in discerning out the artifact which was mimicking a tachycardia. The QRS complex can be detected in the leads which are synchronously manifested as compared to the artifactual complexes.

Figure 4a



INDIAN SOCIETY OF ELECTROCARDIOLOGY

APPLICATION FORM FOR LIFE MEMBERSHIP/FELLOWSHIP

SECRETARIAT
S. B. GUPTA
Indian Society of Electrocardiology
Head, Department of Medicine and Cardiology, C. Rly, Head Quarters Hospital, Byculla, Mumbai - 400 027.
Phone : 2371 7246 (Ext. 425), 2372 4032 (ICCU), 2373 2911 (Chamber) • Resi: 2262 4556 • Fax : 2265 1044
Mobile : 0 98213 64565 / 0 99876 45403 • E-mail : sbgupta@vsnl.net • www.iseindia.org

Dear Sir,

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

My particulars are as follows :

Name in full (Surname first) _____

Qualifications _____

University (Post-Graduation obtained) _____

Year of obtaining first Post-Graduation _____

Mailing Address _____

_____

Tel. No. Hospital _____ Clinic _____ Residence _____

Fax _____ E-Mail _____

Enclosed a cheque/draft of Rs. 2000/- (for outstation cheques add Rs.100/- more) towards Membership of the Society

No. _____ Dated _____ of _____

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

Thanking you,

Yours sincerely,

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

Name _____

Address _____

Signature _____

FOR OFFICE USE ONLY

Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No.

_____

Hon. Secretary, ISE

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

***Life Members :**

1. Person should be a Post-Graduate in Medicine/ Pediatrics/Anaesthesia/ Physiology or other allied subjects from an University recognised by Medical Council of India, with interest in Cardiology / Electrocardiology.
2. Candidates are requested to submit **Xerox** copies of the PG Certificate and Medical Council of India Registration Certificate alongwith Application Form.

****Fellowship:**

1. Person should be a Member of the Society.
2. He/She should be of atleast 7 years of standing after Post-Graduation.
3. He/She should have minimum 3 publications In Cardiology In Indexed Journals (Not Abstracts)
4. List of Publications to be submitted for the Fellowship.
5. Fellowship Fees: Rs.2,000/- (+Rs.100/- for outstation cheque) only. Incase, fellowship not approved by the Credential Committee, the cheque / draft will be returned.

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

www.iseindia.org

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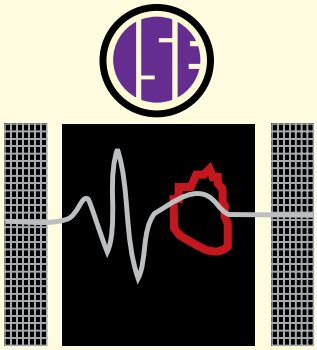
latest updates



SECRETARIAT

S. B. GUPTA

PRESIDENT ELECT

Indian Society of Electrocardiology

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