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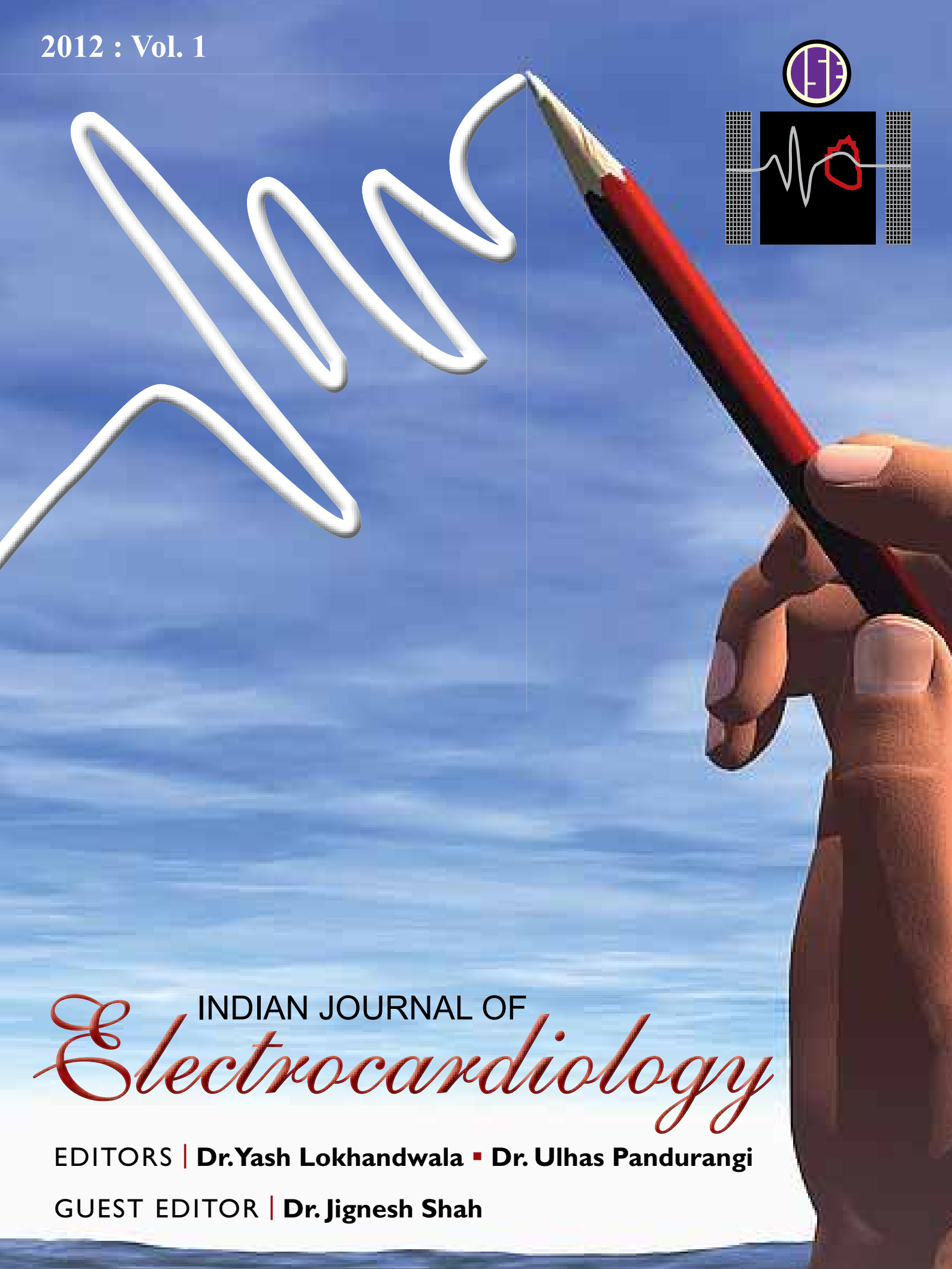

INDIAN JOURNAL OF Electrocardiology

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Editorial

Dear Friends,

As we release this issue of the IJE, we are at the threshold of ISECON Jaipur. The scientific committee has prepared an excellent arrhythmia course encompassing interesting topics relevant in day to day clinical practice. ECG based learning is well incorporated in the scientific session. Discussions regarding devices have been given considerable amount of time considering the increasing number of patients with these devices. Arrhythmias encountered under various settings need individualized treatment plan and will be well covered during the meeting. 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 and device related review articles as well as the ever popular ECG quiz.

Dr. Nathani and colleague have presented an excellent review of Accelerated Idioventricular rhythm, an oft encountered clinical arrhythmias with perplexing clinical significance. Dr. Darrat has written an excellent review article explaining the interpretation of ECG to identify the origin of PVC. The same criteria will help us interpret the origin of VT and guide us to plan treatment strategy. Dr. Bohra presents the various myths among clinicians regarding cardiac devices. He has presented a good discussion to help us in caring for patients with cardiac devices. In our country, reuse of cardiac devices is a reality due to socioeconomic status of a large proportion of our patients. There is an increasing body of literature documenting the safety of these re-used devices. Dr. Bindra has reviewed the literature and presented us with up to date data on the safety and efficacy of these devices. Dr. Bohra has presented an excellent review of ICD related complications. His comprehensive review of literature will further help us in our discussions with the patients regarding these devices. This article has been re-printed with permission from the web-based Indian Pacing and Electrophysiology journal. As always, the ECG Quiz presented by Dr. Lokhandwala and Panicker is one of the highlights of the IJE. It includes ECG from some common and some rare clinical scenarios and test our ability to apply ECG reading skills picked up during the meeting.

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

Jignesh Shah
Guest Editor

Yash Lokhandwala
Editor

Ulhas Pandurangi
Editor

From Vice President's Desk

Dear Members,

It is our great pleasure in bringing out the 1st issue of Indian Journal of Electrocardiology of the year 2012 on the eve of ISECON 2012 – The Annual Conference of Indian Society of Electrocardiology at Jaipur.

Goa Arrhythmia Course was organized by Dr Ramdas Nayak and the team at Goa from 16th to 18th September 2011. The attendance was mammoth and scientific program was a treat. Dr Ramdas Nayak and his team need kudos !

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

a. "ECG Learning Course" for postgraduate students at Sri Lanka on 26th and 27th November 2011 and at Mumbai on 11th and 12th February 2012. 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 9th October 2011 - "Battle Hymn of the Argumentative Cardiologist" – one of the most appreciated program.

c. Indian Society of Electrocardiology is accredited by Maharashtra Medical Council and our programs are now getting the credit hours (if done in Maharashtra).

My sincere thanks to Dr Yash Lokhandwala, Dr Ulhas Pandurangi, Dr Jignesh Shah and the Editorial Team for bringing out the ISE Journal – 2012, 1st Volume.

Long Live Indian Society of Electrocardiology

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

Reuse of Pacemakers: A Primer

Sanjay Bindra
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Why do we Need to Reuse Pacemakers?

Many patients with indications for permanent pacing are unable to afford the implant due to inadequate financial resources. International aid organizations like Heart beat international, estimate that more than 1 million people die annually from lack of a pacemaker. It is estimated that the cost of the devices is more than the per capita income in a large number of countries. For eg. in India, the cost of devices ($2200 to 6600) surpasses what many families can afford. Hence, great disparity in health care is evident across the globe. Many developing countries like India and Bangladesh average < 8 new implants per million as compared with 738 new implants in France.[2]

As, Complete Heart Block is the primary indication for device implantation in underserved countries, the risks of pacemaker reuse must be weighed against the obvious benefit to patients with no other options of receiving the device.

Is It Safe?

Multiple studies performed in various countries over a long period of time and with a significant follow up have demonstrated the safety of these devices in terms of their infection rates as well as device failure rates. (Table 1) A recent meta-analysis included pooled analysis of 18 studies and 2270 patients implanted between 1970 to 2010. About 1.97% (1.15% to 3.00%) patients developed infection after pacemaker reuse. There was no difference in infection rate between pacemaker reuse and new device implantation. (P=0.580). Less than 1 % patients in the reuse group developed device malfunction , this was significantly increased compared to new device implant (odds ratio, 5.80 [1.93 to 17.47], P=0.002). This difference was mainly driven by abnormalities in set screws, which possibly occurred during device extraction, as well as nonspecific device "technical errors." Safety of reuse of ICDs has also been documented.[4]

Small studies in various developed countries have demonstrated the safety of the reuse of devices. Holland, Norway, Sweden, France, Italy and Australia are among the few nations where reuse of devices has been documented. In Sweden, this appears to be a nationwide and long standing tradition with about 5% (in 1996) of pacemakers were re-used devices. Patients receiving resterilized devices demonstrated complication rates similar to those receiving new units in a Swedish study.

Is it Practiced in the Developing World?

Multiple developing countries have annual per capita income substantially below the cost of these devices. Use of resterilized devices has been documented in India, Brazil, Phillipines, Hungary and Romania (Table 1). At Holy Family Hospital in Mumbai, explanted pacemakers donated by funeral homes from the US have saved and improved quality of life of needy poor patients.[1] STIMUBANK an organization in France provides refurbished pacemakers to JIPMER, Pondicherry, a process which has continued for over 20 years, with safe and reliable performance. Feasibility of this practice has been demonstrated in numerous settings worldwide.

What is the Source for these Reused Pacemakers?

Post-mortem: The largest source of used pulse generators is obtained post-mortem since over 30% of pacemaker implants occur in patients over 80 years of age. These pulse generators, often with adequate battery life of greater than 3 years, can be recovered by embalmers prior to burial and could be removed prior to cremation. In a study of funeral directors in Michigan, 84% of the devices collected were discarded as medical waste or were collected with no intended use.[5] By one estimate, about 300000 devices are implanted in the US every year. Though the survival of patients with initial pacemaker implant is long (8.5 years) those with generator change live for shorter duration. Furthermore, patients in the SCD HeFT trial had a mortality of 7% per year and that of COMPANION trial was 12% per year. Patients who receive an appropriate shock have an median expected survival of 168 days. Thus, a significant number of patients underutilize the 7 – 10 year device life guarantee from the manufacturers and hence are available for reuse.

Upgrades : Some patients receiving right ventricular pacing will require device upgrades to an ICD and/or cardiac resynchronization therapy device potentially making the old pulse generators available for reuse.

Infections : Since the devices are adequately resterilized, devices explanted secondary to infection could be reimplanted.

Expiration of Shelf life : Device companies donate the devices that are close to or beyond the "use before" date. These devices may be utilized in the developing countries where the same regulatory hurdles do not exist.

Which Devices can be Reused and how should they be Sterilized?

Battery life and performance testing specification should be the initial criteria to determine whether a device is sent to underserved nations. Adequate pre-defined battery threshold of

Table 1: Safety of reuse of devices demonstrated in multiple studies.

Study	Duration	Country	Pacemakers Reused, n	Follow-Up	Infection	Other Complications	Comments
Balachander et al[27]	1983–1999	India	453	17 y	…	…	
Balachander[28]	6 y	India	140	6 y	2	0	
Pescariu et al[40]	1993–2001	Romania	365	35±21 mo	6	0	No statistical difference in infection or device failure rates between device reuse and new implantation
Linde et al[21]	1998–1993	Sweden	100	32±11 mo	2	1	No statistical difference in infection or device failure rates between device reuse and new implantation
Panja et al[39]	1979–1992	India	120	7.5±5.6 y	6	0	Morbidity and mortality were similar to control population
Kruse[36]	1969–1985	Sweden	487	…	1	2	Of the 487 patients, 118 had already received a refurbished device
Kovacs et al[35]	1975–1980	Hungary	28	…	0	0	
Copperman et al[30]	5 y	Israel	78	…	0	0	
Mond et al[37]	1975–1978	Australia	83	…	1	0	
Amikam et al[25]	1976–1982	Israel	132	5 y	3	0	
Havia and Schuller[34]	1968–1974	Sweden/ Finland	50	22 mo	1	0	
Grendahi[33]	1974–1993	Norway	310	…	14	4	
Costa et al[31]	…	Brazil	22	16 mo	1	2	
Rosengarten et al[41]	1981–1987	Canada	18	29 mo	1	3	No statistical difference in infection or device failure rates between device reuse and new implantation
Mugica et al[38]	1971–1981	France	151	10 y	…	…	No statistical difference in actuarial survival between device reuse and new implantation
Namboodiri et al[24]	2000–2001	India	5	19 mo	0	0	
Sedney et al[42]	1978–1983	Holland	214	31.5 mo	1	1	
Arén and Larsson[26]	1977–1979	Sweden	19	26 mo	0	0	
Ferugilo and Pagani[32]	1971–1978	Italy	87	14 mo	1	0	
Baman et al[29]	2008	Philippines	12	4 mo	0	0	
Is it Practiced in the Developed World?							

Figure 1 : Proposed initiative for delivery of electrophysiological devices to LIMCs

4 years for pacemakers may be utilized to guarantee minimal duration of device function. Adequate sterilization of pulse generators requires removal of all protein material, and a standardized cleansing and sterilization. Multiple studies have demonstrated no increase in rate of infection compared to new devices.

What are the Barriers and Solutions?

Significant logistical, legal and ethical barriers exist.

Logistical barriers : Reuse is hindered by the fact that most devices are unexplanted and buried with the patient. A significant number of explanted devices are discarded as medical waste. In various studies, the willingness of the funeral home directors, pacemaker patient and general population to reuse these devices after patient's death provides an optimistic picture for reuse of these devices. However, the logistics of family consent, transportation and a central agency to send the device to, are the major barriers to reuse of these devices.

Solution: A "pacemaker living will" filled out by patients at the time of device implantation could be used to authorize pulse generator recovery for reuse after upgrade or death. Education and involvement of mortuary directors and personnel may assist in retrieval of suitable devices. Creating a validated network with central office which can assist with consent, transport and oversight of resterilizing process may help in overcoming the logistical barriers.

Legal Barriers : The FDA compliance manual labels "pacemaker reuse as an objectionable practice". The FDA considers the resterilizing and transporting of the devices to another country as a regulated activity. Laws concerning reuse of "single use devices (SUDs)" and product and handling standards do not exist in most developing countries.

Solution: One potential solution is that the device is explanted in the developed country and transported to developing country where it can be resterilized. Another solution could be to collect these devices under an organization which can apply for a FDA export license. Further, patients in developing nations should be fully informed that the device they are reusing is not being deployed according to manufacturer's recommendations, and there may be unknown risks associated with the reused devices.

Ethical Barriers : Psychological benefits of device donation for family members and benefits to patients in the developing countries must be balanced by concerns over the societal benefits of testing the returned devices by the manufacturers to improve the quality of the devices and hence improving the lives of future patients. The incremental quantitative benefit derived from extensive testing of the returned devices vis a vis the benefit from just the device interrogation print outs is difficult to quantify. In a survey, 84% of device patients and 71% of general population expressed their desire to donate these devices for re-use. This was significantly compared to the number of patients (44%) and the general population (53%) in favor of returning the device to the manufacturers for testing.[5] There is also a potential concern for the inappropriate distribution of these devices. These donated devices may end up in the black market or be donated to someone with more clout and less deserving. Determining the hierarchy of deservedness may be a problem.

Solution: One can reasonably argue that devices with minimal battery life may be returned to the device manufacturers for extensive testing and those with significant battery life be reused in developing countries. Exploitation can be minimized with meticulous chain of custody, proper documentation of handlers from explant to reimplant. For programs using donated devices, careful policies regarding patient screening, need of implant, establishing financial hardship and follow-up documentation needs to be in place.

What Organization can Help this Process?

Project My Heart-Your Heart:[6] Baman and colleagues at University of Michigan, have collected more than 4000 pacemakers from funeral homes in the past three years. Working with a Detroit based charity called World Medical Relief, which specializes in delivering used medical equipment to underdeveloped countries, with a plan on getting pacemakers to the developing world on a wider scale. This project lays out the groundwork for logistics of acquiring post-mortem devices from funeral homes and crematories. The project has documented high success rate by distribution of flyers, presence of a website (*myheartyourheart.org*) which provides consent forms for family members, instructions and tools for the funeral home directors to enable them to mail the devices appropriately.

Heartbeat International: This charitable organization, has been responsible for implantation and follow-up in over 9000 indigent recipients in developing countries. The organization which

works through Pacemaker Banks established by local Rotary International chapters in 24 countries over four continents. It is dependent on the generosity of device manufacturers who supply devices close to the expiration of shelf life.[7]

What are the Benefits

It is estimated that about 1 million patients die per year for lack of pacemakers. Approximately 21% of devices have adequate battery life for reimplant. Reutilizing these devices can turn potentially wasted resource into new life opportunities for many citizens of the world.

Conclusion

The reuse of pacemakers for the poor seems feasible and safe when guided by proper procedures, including patient education, informed consent, transport to established networks, meticulous chain of custody and established, ethical programs using these devices.

References

1. Kantharia BK, Patel SS, Kulkarni G. Reuse of Explanted Permanent Pacemakers Donated by Funeral Homes. *Am J Cardiol* 2012;109:238-40.

2. Mond HG, Irwin M, Ector H, Proclemer A. The world survey of cardiac pacing and cardioverter-defibrillators: calendar year 2005 an International Cardiac Pacing and Electrophysiology Society (ICPES) project. *Pacing Clin Electrophysiol* 2008;31:1202-1212.

3. Baman TS, Kirkpatrick J, Romero J. Safety of Pacemaker Reuse, *Circ Arrhythm Electrophysiol* 2011;4:318-323.

4. Pavri BB, Lokhandwala Y, Kantharia BK, Mascarenhas D. Preliminary experience regarding reuse of explanted resterilized defibrillators. *Circulation* 2010;122:A18350

5. Gakenheimer L. Lange DC. Romero J. Kirkpatrick JN. Sovitch P. Oral H. Eagle KA. Baman TS. Societal views of pacemaker reutilization for those with untreated symptomatic bradycardia in underserved nations. *Journal of Interventional Cardiac Electrophysiology* 2011;30:261-6.

6. Baman TS, Kirkpatrick JN, Romero J Pacemaker reuse: an initiative to alleviate the burden of symptomatic bradyarrhythmia in impoverished nations around the world. *Circulation* 2010;122:1649–1656

7. Mond HG , Mick W , Maniscalco BS . Heartbeat International: making “poor” hearts beat better. *Heart Rhythm* 2009;6:1538–1540

Predicting the Origin of PVC Based on EKG Characteristics

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Introduction

Premature ventricular complex (PVC) is a frequently encountered finding on electrocardiography in clinical practice. It is considered to be the most common arrhythmia that physicians often see during patient visit and can provide clues to the physical or electrocardiographic diagnosis.[1] It is caused by an ectopic cardiac electrical focus located in the ventricle. PVCs (Figure 1) are usually wide (>120msec) and abnormally shaped QRS complexes that are not preceded by a P wave. The ensuing T wave is generally wide and deep and is in the direction opposite to the major deflection of the QRS of the PVC. Prevalence ranges from less than 3% for young white women without heart disease to almost 20% for older African American individuals with hypertension.[2] PVCs can arise from various locations in either ventricles. Determining the origin of PVCs from a 12 lead electrocardiogram (EKG) is not only of significance to the cardiac electrophysiologist prior to ablation but could also be of clinical and prognostic value.

Clinical and prognostic significance of PVCs

The clinical implication of PVCs depends primarily on their frequency, complexity and hemodynamic impact. Similarly the origin of PVCs carries a clinical and prognostic value. Isolated PVCs are the most common arrhythmias observed in patients without structural heart disease and they most commonly arise from the right ventricular outflow tract (RVOT) beneath the pulmonic valve. RVOT PVCs are generally considered to carry a favorable outcome but occasionally can result in malignant arrhythmias.[3] The classical differential diagnosis of right ventricular arrhythmia is either idiopathic right ventricular outflow tract tachycardia or arrhythmogenic right ventricular dysplasia.[4] The later is characterized pathologically by fibro-fatty replacement of the right ventricular myocardium and clinically by risk of sudden cardiac death.[5,6] Meanwhile PVCs associated with coronary artery disease (CAD) commonly arise from the left ventricle[7] and are associated with poor prognosis.[8,9] Therefore, predicting the PVC origin can assist the clinician in appropriately counseling patients and in deciding when to refer to a cardiac electrophysiologist for specialized care. We will discuss a diagnostic approach using 12 lead EKG to predict the probable anatomical origin of PVCs.

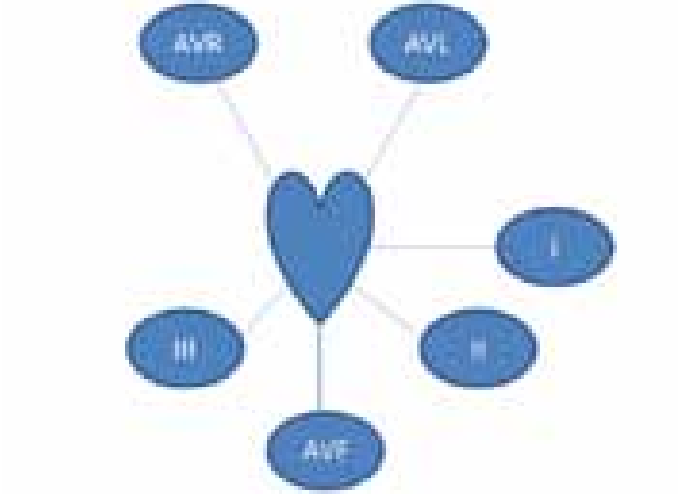

Figure 1 : Orientation of limbs leads in relation to the heart in the vertical axis.

Predicting the ventricular origin of PVCs

The 12-lead EKG provides a powerful tool for localizing the focal origin of PVCs and ventricular tachycardia.[10] There are several electrocardiographic features that can assist in predicting the region the PVCs arise from. It is instrumental to understand the orientation of the 12 leads to interpret PVCs and ventricular tachycardia on EKG. Limbs leads look at the heart in a vertical plane (Figure 1) while precordial leads in a horizontal plane (Figure 2) from the anterior aspect of the chest wall to the left side. It is advised to refer to figures 1 and 2 while reading the text to comprehend the QRS polarity caused by spread of the electrical activity form the PVC focus. The accuracy to predict the origin of PVCs on an EKG can be affected by several limiting factors.[10] However, it is still considered as a valuable tool particularly in the structurally normal heart.[10]

1. Right side versus left side origin

   Bundle branch block pattern of the PVC helps to determine if it is originating from the left or right ventricle. PVC is said to have a right bundle branch block (RBBB) pattern if the terminal portion of the QRS complex in V1 is positive, and as having a left bundle branch (LBBB) pattern if this is

Figure 2 : Orientation of precordial leads in relation to the heart in the horizontal plane.

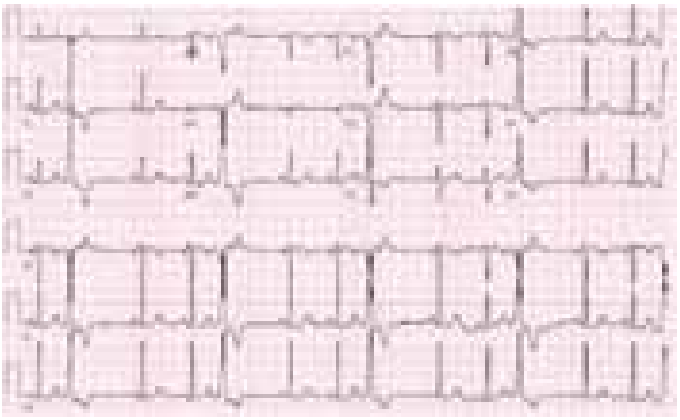

Figure 3 : LBBB with inferior axis and narrow QRS complex indicating a septal RVOT origin of the PVCs.

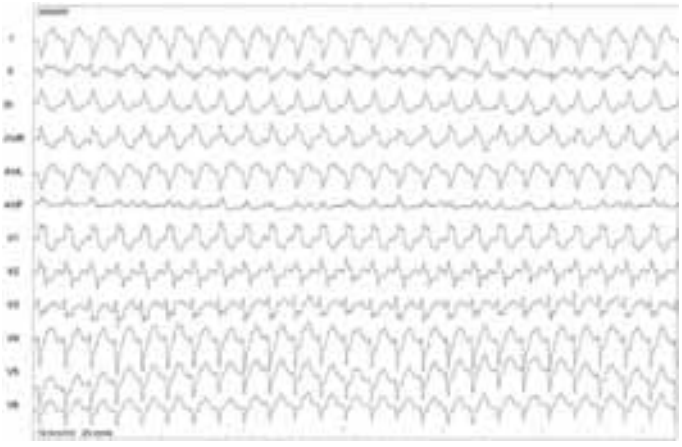

Figure 4 : Ventricular tachycardia with RBBB pattern indicating left side origin

negative. The majority of right ventricular tachycardias exhibit a LBBB pattern (Figure 3).[11] However, PVCs from the left ventricular side of the septum also exhibit a LBBB pattern. Ventricular arrhythmia originating from the left ventricle predominantly demonstrate a RBBB pattern (Figure 4) but rarely LBBB pattern with early precordial transition in lead V1 (Figure 5). The R/S transition in the precordial leads of the 12-lead surface EKG has been useful to differentiate between a left or right sided origin. Precordial transition in lead V4 to V6 indicates an RVOT origin, as shown in (figure 3), with 92.3% sensitivity and 100% positive prediction value.[12] In addition, an R wave duration index of less than 0.5 and the R/S wave amplitude index of less than 0.3 predicts RVOT PVCs with 95% sensitivity and 100% positive prediction value.[12]

Another method to assist in differentiating between left side and right side originating PVCs is to look at QS wave amplitude in leads AVR and AVL.[13] If the QS wave depth in lead AVL was larger than that in lead AVR, the origin was likely to be on the left side because AVL monitors the ex-

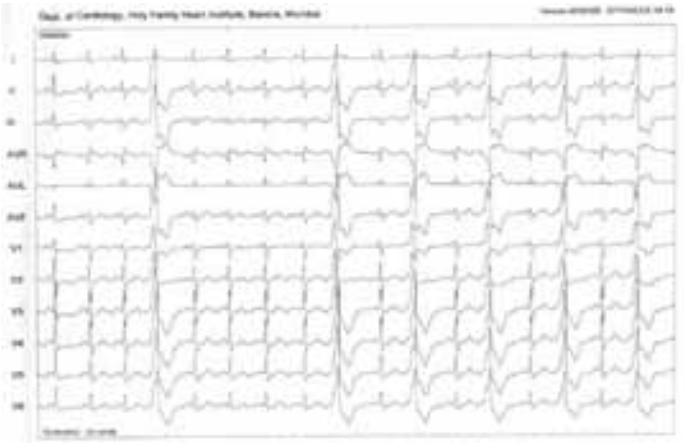

Figure 5 : LBBB with inferior axis with early precordial transition in lead V1 indicating left ventricular outflow tract origin of PVCs

citation front away from the left upper region of the heart. If the QS amplitude in lead AVR was equal to or larger than that in lead AVL, the origin was likely to be on the right side.[13] In simple terms, AVR being a right-sided lead is negative with right-sided origin; AVL being a left-sided lead is negative with left-sided origin.

The QRS wave polarity of lead I is another useful tool in differentiating between left and right side focus. If lead I showed negative polarity (QS, Qr, or rS wave pattern), the origin was likely to be on the left side If lead I showed positive polarity (R-wave pattern), the origin was likely to be on the right side (diagnostic accuracy: 83%).[13]

2. Septal versus free wall origin

Septal origin PVCs are generally narrower than those originating from the free wall. The QRS duration and the QRS wave morphology in leads II and III are informative. QRS duration of less than 140ms predicted septal origin (Figure 3) while duration greater than 140ms predicted free wall origin with 80% diagnostic accuracy.[13] The shorter QRS duration in the PVC arising from the septum can be explained by the spread of excitation downward over the right and left ventricles more rapidly through the septal Purkinje network than the excitation from the free wall. If the RR' or Rr' wave (notching) pattern was observed in leads II and III, the origin was on the free-wall side. If the R-wave pattern was seen in leads II and III, the origin was likely to be on the septal side (diagnostic accuracy: 86%).[13]

3. Basal versus apical origin

Inferior axis is a positive deflection of QRS complex of the PVC in the inferior limb leads (leads II, III and aVF) and it suggests a very superior origin (basal) either on the anterior wall or the outflow tract (Figure 3 and 5). In contrast, superior axis PVCs originate from the apical region. Concor-

dance of QRS can be also used to determine origin from the basal versus the apical region and it indicates that precordial leads have similar morphology, either all positive or all negative. Positive concordance is seen with PVCs arising from the base of the heart (Figure 5) since the spread of electrical excitation is towards the precordial leads. Similarly, negative concordance is seen when the origin is from the apical septum because spread of electrical excitation is away from the precordial leads.[10]

4. Epicardial versus endocardial origin

Ventricular arrhythmias can arise from different locations in the epicardium as well as the endocardium. Variation in the QRS morphology can be detected with epicardial origin of PVCs in the form of pseudo delta wave or slurring of the intial forces of QRS,[10,14] resulting in widening of the QRS complex. The differences between endocardially and epicardially originated QRS complexes are probably due to fast depolarization of the ventricles along the specialized conducting system when the stimulus is delivered in the endocardium, resulting in a narrow QRS and rapid initial forces on the 12 lead EKG.[14] In contrast the intramyocardial delay of conduction in case of epicardial PVCs produces a slurred initial portion of QRS and thus a pseudo delta wave.[14] The recognition of epicardial ventricular arrhythmia is significant since it dictates that approach to ablation should be epicardial rather than the traditional endocardial approach.

Conclusion

PVCs can arise from different anatomical locations in the ventricles depending on the underlying etiology and on the substrate. Recognizing the origin of PVCs can be of clinical and prognostic significance. The 12 lead EKG is a feasible way to predict the origin via analyzing the bundle branch pattern, QRS morphology, axis and precordial R/S transition.

References

1. K Wang, M Hodges. The premature ventricular complex as a diagnostic aid. *Ann Intern Med* 1992;117: 766-770.

2. Simpson RJ Jr, Cascio WE, Schreiner PJ, Crow RS, Rautaharju PM, Heiss G. Prevalence of premature ventricular contractions in a population of African American and white men and women: the Atherosclerosis Risk in Communities (ARIC) study. *Am Heart J* 2002;143:535-40.

3. Shimizu W. Arrhythmias originating from the right ventricular outflow tract: how to distinguish “malignant” from “benign”? *Heart Rhythm* 2009;6:1507-11.

4. Hans Kottkamp, Gerhard Hindricks . Right ventricular tachycardia—arrhythmogenic right ventricular cardiomyopathy or idiopathic? *Eur Heart J* 2003;24:787-788.

5. McKennaWJ, Thiene G, Nava A et al. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. *Br Heart J* 1994;71:215–218.

6. GemayelC, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular cardiomyopathy. *J Am Coll Cardiol* 2001;38:1773–1781.

7. Josephson ME, Horowitz LN, Farshidi A, Spear JF, Kastor JA,Moore EN: Recurrent sustained ventricular tachycardia. 2. Endocardial mapping. *Circulation* 1978;57:440.

8. Ruberman W, Weinblatt E, Goldberg JD, Frank CW, Shapiro S. Ventricular premature beats and mortality after myocardial infarction. *N Eng J Med* 1977;297:750-7.

9. Bigger JT Jr, Fleiss JL, Kleiger R, Miller JP, Rolnitzky LM, and The Multicenter Post-Infarction Research Group. The relationships among ventricular arrhythmias, left ventricular dysfunction, and mortality in the 2 years after myocardial infarction. *Circulation* 1984;69:250-8.

10. Josephson ME, Callans DJ: Using the twelve-lead electrocardiogram to localize the site of origin of ventricular tachycardia. *Heart Rhythm* 2005;2:443–446.

11. Buxton AE, Waxman HL, Marchlinski FE, Simson MD, Cassidy D, Josephson ME. Right ventricular tachycardia: clinical and electrophysiologic characteristics. *Circulation* 1983;68:917-927.

12. Zhang F, Chen M, Yang B, Ju W, Chen H, Yu J, Lau CP, Cao K, Tse HF. Electrocardiographic algorithm to identify the optimal target ablation site for idiopathic right ventricular outflow tract ventricular premature contraction. *Europace* 2009;11:1214-20.

13. Kamakura S, Shimizu W, Matsuo K, Taguchi A, Suyama K, Kurita T, Aihara N, Ohe T, Shimomura K. Localization of optimal ablation site of idiopathic ventricular tachycardia from right and left ventricular outflow tract by body surface EKG. *Circulation* 1998;98:1525-33.

14. Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation* 2004;109:1842–1847.

Common Myths Regarding Cardiac Devices

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Increasing number of patients are getting cardiac devices implants including pacemakers, Implantable cardiac defibrillators (ICD) and cardiac resynchronization therapy (CRT) devices. Every clinician is encountering these devices in day to day practice. There are a lot of myths surrounding these devices. This review attempts to discuss these myths and clarify some misunderstandings regarding these devices.

Myth 1. Single chamber pacemaker is appropriate for most patients with bradycardia.

Pacemakers are needed for multiple indications such as symptomatic sinus bradycardia, symptomatic pause, complete heart block, AF with slow ventricular response, tachy-brady syndrome etc. Patients with intact sinus rhythm requiring pacemakers for eg. Those with complete heart block should receive a VDD/DDD pacemaker to maintain AV synchrony and avoid pacemaker syndrome. In fact by implanting a single chamber pacemaker in patient with complete heart block, we are converting a complete heart block with slow ventricular escape to a complete heart block with increased ventricular rate with partial relief of symptoms. There is ample data to show that these patients have poor long term outcome as measured by increased incidence of AF, increased CHF etc. Always prefer dual chamber pacemakers in patients with intact sinus rhythm with complete heart block.

However, a patient with symptomatic sick sinus syndrome may benefit from single chamber pacemaker in the form of AAI (R). However, the incidence of CHB in patients with sick sinus may be 2-20% on follow up which may warrant a further ventricular pacing and need to upgrade the pacemaker to a DDDR pacemaker. During implantation of a AAIR pacemaker if there is presence of a wide QRS with/without a bundle branch block, PR prolongation and failure to conduct 1:1 at heart rates < 130 beats/min on atrial pacing, then a DDDR pacemaker should be preferred at the onset. Patients who are given VVIR pacemaker for sick sinus syndrome often experience pacemaker syndrome and are similarly at higher risk to develop AF and CHF over the long run. With the availability of active fixation leads the chances of lead dislodgement from the atrium are minimal and should not be a reason to avoid doing atrial based pacing.

Ideally, single chamber pacemaker in VVI mode is preferred only in patients with chronic atrial fibrillation with slow ventricular rate or with symptomatic long pauses.

Myth 2. Device implantation is critical and once well implanted, all is well.

Adaptive pacemakers presently automatically optimize output depending on the threshold and hence conserve battery automatically. Latest pacemakers may give alerts in case of device or clinical problems and may alert the patient to contact the clinician immediately. Home monitoring keeps the patient under constant surveillance and hence patients may not come for regular follow up.

However, devices need regular planned follow up to optimize device function and battery life. Checking the local site, assessing diagnostic data like heart rates and Optivol fluid monitoring and appropriately changing coexisting therapy may help optimize treatment and patient outcome. Stable lead measurements such as impedance, sensing and threshold confirms lead integrity and ensures optimal device functioning. Early recognition of the possibility of inappropriate ICD shocks due to high baseline heart rate in active young individuals or development of asymptomatic AF with RVR can help modify therapy as well as programming so as to prevent inappropriate shocks. Asymptomatic VT and its treatment with painless ATP therapy should prompt clinicians to initiate antiarrhythmic treatment to prevent further arrhythmias. Asymptomatic AF documented via device can prompt early thromboprophylaxis and stroke prevention.

CRT implantation with LV lead in an appropriate target vein is very important for clinical response. However, 30% of the patients are non-responders and a small percent of patients deteriorate after CRT. Programming of the device to optimize AV and VV intervals based on device algorithms and/or echocardiography techniques can significantly improve clinical benefit. With progression of the disease process appropriate modifications may be needed during follow up.

Overall, all device patients need to undergo programming according to their clinical scenario and their lifestyle. It is important to ensure that the device is programmed appropriately to detect and treat appropriate arrhythmias, avoid inappropriate shocks, optimize heart failure therapy and detect asymptomatic AF to optimize patient outcomes

Myth 3. Procedure of device implantation is as risky as an open heart surgery.

Generally, all device implantations are done under local anesthesia with mild sedation and do not carry major risk in

presence of expertise and current hardware. Among 2010 patients undergoing pacemaker implant in the MOST study, there were no deaths in the first 24 hours after surgery, 1.7% had atrial lead dislodgement, 0.7% had ventricular lead dislodgement and 0.7% had pneumothorax. Pocket hematoma was noted in a small percentage of patients. Infection of the device occurs in 1-2% of the patients over the ensuing 1 year. Complication rate of ICD is considered similar to that of pacemakers, especially with incremental data showing no clinical benefit of DFT testing. CRT devices carry additional risk of coronary venous dissection (0.3-0.5%), coronary vein perforation (0.8–1.1%) coronary vein tamponade (0.3–0.5%). There is a higher risk of LV lead dislodgement in CRT implant to the tune of 2-4%. However, none of these risks are comparable to that of open heart surgery. Most implant patients are discharged within 1-2 days and return to routine activity within a week.

Myth 4. Patients with devices should not operate any electronic items or vehicles.

In the modern era, electronic gadgets are ubiquitous and cell-phones, computers, television etc are part of the daily life of an individual. Modern day cardiac devices are not affected by the day to day electronic gadgets. However, it is advisable to use the cell-phone from the ear on the opposite side of the implanted device. It is important to avoid high voltage equipments like transformers and heavy industrial electrical equipments. Driving commercial vehicles is restricted for ICD patients due to possibility of appropriate or inappropriate shock during driving and causing loss of control of the vehicle. However pacemaker patients can drive without any restrictions after the surgical recovery. Professions requiring vigorous hand movements on the side of implantation (eg. drilling) should be avoided as it may damage the lead.

Myth 5. Device patients may undergo all diagnostic and therapeutic procedures without any additional precautions.

Patients with devices need peri-procedural preparation for various diagnostic and therapeutic procedures. MRI should be avoided in patients with devices as it has the potential to cause reversible or irreversible damage to the device. There is a concern of heat injury to the myocardium in patients with devices undergo MRI. A small percentage of pacemakers are MRI safe but majority of the pacemakers and all ICD and CRT devices are not. Hence, MRI is contraindicated in these device patients.

During surgical procedures, operators should be aware that the use of cautery close to the device may interfere with its normal functioning. Cautery close to the pacemaker may inhibit pacing function and may lead to catastrophe in a pacemaker dependent patient. Hence, pacemaker dependent patients need to be programmed to non-sensing mode prior to such surgery. Cautery used in the vicinity of ICD may lead to oversensing and inappropriate shock. ICD therapies should be switched off prior to the surgery and post surgery the device should be interrogated and parameters restored.

In case the device programmer is not available during the pre-operative period and in case of emergencies a magnet placed over the pacemaker will lead to pacemaker pacing at non-sensed mode and in ICD will lead to disabling the therapies and hence can be an acceptable alternative.

Therapeutic radiation over the site of the device may damage the device and hence post radiation device check is recommended. With increasing number of patients with these devices, clinicians need to be aware of the possibility of various diagnostic and therapeutic procedures interacting with the device.

Myth 6. Properly implanted CRT device in cardiomyopathy patients with wide QRS and heart failure will lead to clinical benefit.

Currently CRT devices are indicated in patients with LVEF of less than 35% with no reversible cause, QRS duration of >120 msec, Class III-IV heart failure despite optimal medical management. Even in clinical trials where all the above criteria were met, the response rate was 70%.

There has been no single test to accurately predict responders. Evidence of dys-synchrony on echocardiogram has not been demonstrated to improve the predictability of responder. Patients with LBBB with QRS width greater than 150ms, non-ischemic cardiomyopathy and those who demonstrate narrow QRS post procedure appear to benefit the most from their CRT. Wide QRS with RBBB or IVCD may not have similar clinical benefit as compared to those with LBBB. Large anterior wall scar, infarct in the LV postero-lateral region also decreases the clinical benefit. Hence though indicated in all patients with wide QRS, CRT should be implanted only after reasonable evaluation and expected clinical benefit should be discussed in detail with the patient based on such evaluation prior to the implantation.

Myth 7. Implantable Cardiac Defibrillator (ICD) should be given to patients with untreated recurrent Ventricular Tachyarrhythmias

An ICD treats ventricular tachyarrhythmias by either overdrive pacing or by cardioverting with an internal shock. In cases of untreated recurrent ventricular tachyarrhythmias, there is a potential for multiple shocks. Multiple ICD shocks are known to cause increased emotional trauma, increase in the long term mortality and early battery depletion. In fact, incessant VT is an absolute contraindication for implantation of ICD. In such cases, initial treatment involves prevention of VT with the help of antiarrhythmic drugs or radiofrequency ablation so as to decrease the burden of ventricular tachyarrhythmias prior to implantation of device. Thus, ICDs are useful in preventing sudden cardiac death due to ventricular tachyarrhythmia.

Myth 8. Pacemakers are not required in patients with CHB or sick sinus syndrome with no syncope.

Many patients with CHB or sick sinus have no symptoms at rest and have fairly good heart rates at rest. However, they have chronotropic incompetence and experience symptoms on exertion because the heart rates fail to rise according to the metabolic needs. Many patients notice an increase in fatigue levels which is not commensurate with their age. In patients with tachy-brady syndrome, rate control cannot be achieved during the tachycardia phase due to the fear of excessive bradycardia during the bradycardia phase. All these patients may benefit from pacemaker implant and notice a significant improvement in their quality of life.

Myth 9. The costlier devices have all the functions.

Device functions are mixed and matched within and between the different product lines of various manufacturers. A certain function eg. MRI safety may be available in a costlier device but it may not have other function such as rate drop feature, which may be necessary in some patients. There is no substitution for a thorough knowledge of the patient's clinical condition and matching it with the specific device function available.

Myth 10. A person standing next to ICD patient gets shocked when the ICD patient receives a shock.

ICD lead in the RV detects ventricular arrhythmia and the pulse generator provides high voltage to the tune of 600V between the RV lead and the active pulse generator. Thus, this vector is contained within the patient and the current does not leak outside of the patient's body. However, the mechanical jolt from this high voltage electricity experienced by the patient may be transmitted to the person in contact with him. It is not the electric current but this mechanical jolt that is felt by the bystander.

Accelerated Idioventricular Rhythm- An Overview

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Introduction

Accelerated idioventricular Rhythm (AIVR) is a ventricular rhythm consisting of three or more consecutive beats, with gradual onset and gradual termination. Clinically, accelerated idioventricular rhythm can occur in any form of structural heart disease and occasionally in adults or children without structural heart disease. It is most often seen in patients with coronary artery disease. In the past, it was believed to be a sign of successful reperfusion, but it usually signifies an underlying myocardial necrosis and hence not a favorable prognostic marker. The present overview discusses etiology, possible mechanisms, and the clinical significance of accelerated idioventricular rhythms.

Definition

Accelerated idioventricular rhythm (AIVR) was first described by Thomas Lewis in 1910.[1] AIVR is currently defined as an enhanced ectopic ventricular rhythm with at least 3 consecutive ventricular beats, which is faster than normal ventricular escape rhythm (≤40 bpm), but slower than ventricular tachycardia (100 bpm).[1]

The ventricular beats are usually monomorphic and demonstrate gradual onset. Less commonly, AIVR is polymorphic.

ECG characteristics

Typical AIVR demonstrates regular rhythm at a rate of 40 to 100 BPM with wide and abnormal QRS complexes. The QRS complexes are usually dissociated from the P waves (Fig. 1). Furthermore, capture and fusion beats are common.

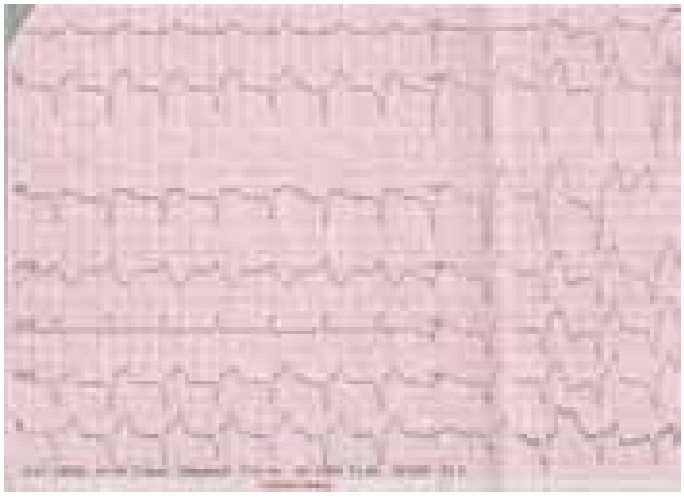

Figure 1 : ECG showing AIVR at a rate of 80/ min. with evidence of AV dissociation in a case of acute anterior wall MI in a 45 years old female.

Terminology

Different terminology was used to describe AIVR: Non paroxysmal ventricular tachycardia (VT), isorhythmic slow VT, and the curious benevolent tachycardia. Non paroxysmal VT is no longer an accepted terminology since the rate is less than the tachycardia definition of 100bpm.

Differential Diagnosis

Accurate diagnosis of AIVR is critical because this rhythm by itself is hemodynamically stable but misdiagnosis of AIVR can lead to inappropriate therapies with potential complications

1. This rhythm may resemble sinus rhythm due to similar rate of 40 to 100 bpm. Differentiation may be made due to numerous fusion beats in case of AIVR unlikely to be seen in sinus rhythm.
2. AIVR must be distinguished from junctional tachycardia with preexisting intra ventricular conduction defects. Once again, such rhythm is not associated with fusion or capture beats as noted in AIVR.
3. AIVR differs from ventricular tachycardia by rate of ventricular rhythm and by additional features such as the onset with a long coupling interval, the termination by a gradual decrease of the ventricular rate or increase of the sinus rate.

Causes

AIVR often occurs during acute myocardial infarction with reperfusion. It has been also described with several drug intoxications such as digitalis, halothane, aconitine, desflurane, and cocaine. Electrolyte imbalance has also been associated with AIVR. There are also association of AIVR with the post-resuscitation period (as an initial resolving rhythm), in chronic ischemic and non ischemic dilated cardiomyopathy, and less frequently in cardiomyopathies such as hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia (ARVD), and in newborn infants with different congenital heart diseases. Rarely, AIVR can be detected in the youth and it is usually benign.

Pathophysiology

In most cases, the mechanism of AIVR appears to be related to the enhanced automaticity in His-Purkinje fibers and/or

myocardium[2] sometimes accompanied with excessive vagal activity and / or decreased sympathetic activity [(3)]. Ischemia, reperfusion, hypoxia, drugs, and electrolyte abnormalities can all accelerate the phase 4 action potential depolarization rates in His-Purkinje fiber and myocardium, leading to faster spontaneous cell depolarization (enhanced automaticity).[4] When the enhanced automaticity in His-Purkinje fiber or myocardium surpasses that of sinus node, AIVR manifests as the dominant rhythm of the heart. Sinus bradycardia may facilitate the appearance of AIVR. When AIVR is associated with digitalis intoxication, the main arrhythmogenic mechanism involved is trigger activity.

AIVR in acute myocardial infarction

In acute ST elevation myocardial infarction, AIVR observed in 9%- 46% of patients without thrombolysis and in 23%- 82% of patients with thrombolysis. AIVR is frequently observed during the reperfusion phase that follows an acute myocardial infarction. It remains controversial whether or not, AIVR implies complete reperfusion of the culprit lesion or only some degree of reperfusion. It was previously reported as AIVR was commonly occurs in inferior than anterior wall MI but recent studies have demonstrated similar incidence of AIVR in inferior and anterior wall MI.

AIVR generally initiates after a long coupling interval to the underlying sinus rhythm and is regular. Configuration of AIVR depends on the reperfused infarct related vessel. Reperfusion of the left anterior descending branch showed various different configurations of AIVR and with the least QRS width. Reperfusion of the circumflex branch never had a left bundle branch block-like configuration. AIVR from reperfusion of the right coronary artery never has an inferior axis. AIVR occurring during persistent ischemic chest pain is a marker for both myocardial necrosis and reperfusion of the infarct vessel.[5]

AIVR during Primary PCI

AIVR is the commonest arrhythmia observed during Primary PCI (42%) followed by sinus bradycardia (28%), and nonsustained ventricular tachycardia (26%). Patients with AIVR during PCI correlated with decreased incidence of spontaneous ST resolution before PCI, decreased incidence of Thrombolysis In Myocardial Infarction(TIMI) flow 3 on admission, had a larger area at risk, had a longer time to complete ST resolution, had a larger final infarct size, but had similar mortality compared to patients without AIVR.[6]

Patients with ST elevation MI patients with reperfusion-induced AIVR after Primary PCI showed more pronounced diastolic LV dysfunction before and after AIVR than patients without AIVR, which suggests that diastolic LV dysfunction contributes to the occurrence of AIVR and that AIVR is a sign of diastolic LV dysfunction.[7]

Occasionally, AIVR following reperfusion is associated with marked reduction in both systolic and diastolic BP, irrespective of infarct-related artery. These hemodynamic effects are accompanied by only a very modest increase in heart rate during AIVR. Patients with a culprit lesion in the proximal left coronary artery showed a smaller reduction in systolic blood pressure than distal left coronary artery lesions probably due to preexisting more compromised hemodynamic condition. [(8)] Such differences were not noted if the culprit lesion was in the right coronary artery.

Management

Treatment for AIVR does not change the prognosis. **AIVR is generally a transient rhythm, rarely causing hemodynamic instability and rarely requiring treatment.** The most important therapy for patients with AIVR is to treat the underlying etiology eg. digoxin toxicity, myocardial ischemia etc.

Occasionally, patients may not tolerate AIVR due to (1) loss of atrial-ventricular synchrony, (2) relative rapid ventricular rate, or (3) ventricular tachycardia or ventricular fibrillation degenerated from AIVR (extremely rare). Under these situations, atropine can be used to increase the underlying sinus rate to inhibit AIVR. Other treatments for AIVR, which include isoproterenol, verapamil, antiarrhythmic drugs such as lidocaine and amiodarone, and atrial overdriving pacing are only occasionally used today.

Prognosis

It is a mostly self-limiting rhythm and has a benign prognosis. The prognosis of patients with AIVR largely depends on their underlying conditions. AIVR is the most frequent arrhythmia occurring during primary PCI in patients with ST-elevation myocardial infarction. However, it is not a marker of successful reperfusion but is associated with extensive myocardial damage and delayed microvascular reperfusion.[6]

AIVR on postresuscitation ECG offers a prognostic factor related to a higher repeated CPR rate within 1h after return of spontaneous circulation and a lower 7-day survival rates in successfully resuscitated victims.[9]

Conclusion

AIVR is a common reperfusion arrhythmia noted after thrombolysis as well as after primary PCI, usually a benign self limiting rhythm. It is a marker of extensive myocardial damage and poor diastolic dysfunction. It is a self limiting rhythm and rarely causes hemodynamic instability. Treatment of the underlying etiology is key to optimal outcome.

Reference

1. Grimm W, Marchlinski FE. Accelerated Idioventricular Rhythm and Bi-directional Ventricular Tachycardia. In: *Cardiac Electrophysiology: From Cell to Bedside*. 4th ed. 2004:700-704.

2. Castellanos A Jr, Lemberg L, Arcebal AG. Mechanisms of slow ventricular

tachycardias in acute myocardial infarction. *Dis Chest* Dec 1969;56:470-6.

3. Bonnemeier H, Ortak J, Wiegand UK, Eberhardt F, Bode F, Schunkert H, et al. Accelerated idioventricular rhythm in the post-thrombolytic era: incidence, prognostic implications, and modulating mechanisms after direct percutaneous coronary intervention. *Ann Noninvasive Electrocardiol*. Apr 2005;10:179-87.

4. Hasin Y, Rogel S. Ventricular rhythms in acute myocardial infarction. *Cardiology* 1976;61:195-207.

5. Anton P.M. Gorgels, Marc A. Vos, Ingrid S. Letsch, Erik A. Verschuuren, Frits W.H.M. Bär, Johan H.A. Janssen, Hein J.J. Wellens Usefulness of the accelerated idioventricular rhythm as a marker for myocardial necrosis and reperfusion during thrombolytic therapy in acute myocardial infarction. *The American Journal of Cardiology* 1988;61:231-235.

6. Terkelsen CJ, Sørensen JT, Kaltoft AK, Nielsen SS, Thuesen L, Bøtker HE, Lassen JF. Prevalence and significance of accelerated idioventricular rhythm in patients with ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. *Am J Cardiol* 2009;104:1641-6.

7. Remmelink M, Delewi R, Yong ZY, Piek JJ, Baan J. More pronounced diastolic left ventricular dysfunction in patients with accelerated idioventricular rhythm after reperfusion by primary percutaneous coronary intervention. *J Invasive Cardiol* 2010;22:574-8.

8. Delewi R, Remmelink M, Meuwissen M, van Royen N, Vis MM, Koch KT, Henriques JP, de Winter RJ, Tijssen JG, Baan J, Piek JJ. Acute haemodynamic effects of accelerated idioventricular rhythm in primary percutaneous coronary intervention. *EuroIntervention* 2011;7:467-71.

9. Tsai MS, Huang CH, Chen HR, Hsieh CC, Chang WT, Hsu CY, Ma MH, Chen SC, Chen WJ Postresuscitation accelerated idioventricular rhythm: a potential prognostic factor for out-of-hospital cardiac arrest survivors. *Intensive Care Med* 2007;1628-32.

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Implantable Cardiac Pacing Devices Related Complications: Keeping Pace with Time

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Keywords: Cardiac Pacing Devices; Complications

The physician should look upon the patient as a besieged city and try to rescue him with every means that art and science place at his command. ~ Alexander of Tralles.

Science of medicine (and less of art) has progressed tremendously over the last few decades and engineering innovations have constantly changed the way we used to treat a patient. The field of cardiology is the best example of how technology has invaded into a human body making interventions a lot easier and safer. Cardiac pacing devices which initially were designed to treat bradycardia, have now found place for treatment for ventricular arrhythmia, heart failure and for prevention of sudden cardiac arrest. With increasing number of patients having cardiac disease and with the ever expanding indications of device therapy in clinical practice, the numbers of patients who are undergoing device therapy is expanding exponentially.

Based on knowledge of inadequacies from prior implant data, cardiac pacing devices and related hardware are constantly undergoing refinement and are continuously being upgraded to being better and safer each time. Lead is placed in the right atrial appendage for atrial pacing and right ventricular apex or septum for right ventricular pacing for most of the patients through the axillary/subclavian/cephalic venous access. The pulse generator is kept most commonly in the subcutaneous or submuscular pocket in the pectoral region. Active fixation lead (screw in lead) or passive fixation (tined) leads are selected based on patient's disease, need to do special site pacing or purely on operator preference. Pacing in the right atrial free wall, interatrial septum, right ventricular outflow, His bundle and left ventricle requires greater understanding of anatomy and/or training in using appropriate hardware and techniques for appropriate lead placement. Occasionally surgeons help needs to be taken for epicardial lead implantations. [1-3

Though implantation of cardiac pacing device is now a safe time tested procedure, complications related to implantation, when enumerated are many, but occur in about 5.7% of patients and can be grouped as either procedural, component or biophysical interface related problems.[1-3] Intuitively and scientifically complications are more likely to occur with increased procedure time, more difficult procedure or implantation technique (like upgrade of existing devices or left ventricular lead implantation), with implantations in higher risk patients and with lesser operator experience.[2] Though, once in a while, complications do occur in hands of even the most experienced operator in the most simple device implantation procedure and in an absolutely normal risk patient. Precautions hence need to be taken appropriately and hardware selection always needs to be individualized.

Commonly occurring complications of percutaneous venous access and blind subclavian puncture are subclavian artery puncture, pnuemothorax, hemothorax and hemo-pneumothorax. Lacerations of subclavian artery, nerve injury, thoracic duct injury, chylothorax and lymphatic fistula have occasionally been described. Contrast venography-guided venipuncture, ultrasound-guided puncture and an extra-thoracic subclavian puncture may decrease such complications and fluoroscopy time.[4] Though cephalic venous cut-down has decreased such vascular and pleural complications, multiple lead insertions cannot be achieved with cephalic venous cut-down alone and hence venous access related complications occasionally do continue to occur.[5,6]

Acute complications of lead placement include thromboembolism (air/clot), arrhythmia, tricuspid regurgitation due to damage to the valvular apparatus and chamber perforation associated with or without cardiac tamponade. Left ventricular lead placement presents special challenges and complications due to need for coronary sinus cannulation and placement of a lead in a desired vein. Lead displacement with rise in thresholds, loss of pacing, diaphragmatic pacing and chamber perforation, pericarditis with or without cardiac tamponade can occur either immediately or at a later date. Misplacement of a lead is very uncommon, though described (lead placed in a left ventricle through the interatrial septum instead of desired right ventricle pacing). [2,4,7]

Device related complications include battery failure and pulse generator circuit failure, lead failure, conductor coil fracture and insulation failure. [3] Manufacturing deficiencies in software or hardware have rarely led to device/lead recalls. Under-sensing, over-sensing and programming related issues leading to inappropriate therapy tend to crop up every now and then and most of the time can be appropriately rectified non-invasively. Electromagnetic interference may occasionally cause device malfunction.

Axillary vein thrombosis is rare occurring in 0.5-1% of cases.

Partial venous obstruction in the great veins is almost a rule and occurs to some degree in up to 100% of cases. Clinically, pulmonary embolism however is extremely rare. Partial or silent inconsequential thrombosis is considered extremely common but generally of no clinical significance. [1,4] Pain at the local site and shoulder pain can sometimes be annoying.

Pocket related complications like pocket hematoma, wound dehiscence, migration, erosion, pain and infection are well known and almost all who perform the procedure routinely have come across varying severity of such complications. [3] Twiddler's syndrome is very uncommon. [8] Device related infections possibly present the greatest challenges in clinical practice. Infections may present acutely with septicemia with or without endocarditis with vegetation on the lead, valve or the cardiac tissue or a pocket abscess. Chronic infection most often presents as a chronically discharging sinus, device erosion or a granulomatous mass. [3] Explantation of the whole system and reimplantation at a different site or reimplantation after adequate debridement and antibiotic therapy at the same site are almost always required in either acute or chronic settings and requires patience and persistence of both the patient and the treating physician. Extraction of chronically implanted leads can be challenging and can be associated with significant complications. [3]

If not vigilant, recognition of uncommon complications like a pneumothorax due to a right atrial micro-perforation by an atrial screw in lead described by Syamkumar et al in the issue of this journal sometimes can get delayed.[9] Delayed perforation leading to migration of the lead to the pericardium or the pleural cavity with or without pericarditis, cardiac tamponade or hemothorax have been described.[10-14] Such complications though very uncommon are potentially life threatening and should be recognized and treated immediately.

Follow up of patients needs to be done regularly with cardiac devices and should be emphasized in all patients who receive them. It is not only necessary for optimizing battery life, but also to detect complications early related to the device or the biophysical interface so that correction can be done before significant symptoms develop. The case report by Garg et al presents an interesting patient, who after initial implantation never came for follow up, and presented with end of life of pulse generator and a chronic granulomatous mass over the incision site described due to hypersensitivity to non-absorbable suture material. [15] Ideally chronic infection should be considered in all such patients and further treatment should be done accordingly. [3]

Atypical complications can occur. Complications though unavoidable, its manifestations can be minimized by early recognition and appropriate treatment. The absolute number of complications that we may see in the near future is going to increase, though the percentage of complications decrease with increase in the operator experience and better hardware, as absolute number of implantations occurring are increasing. Till the day, when advances in biotechnology would change the way we implant cardiac pacing devices for treatment of bradycardia, heart failure, ventricular tachycardia and for prevention of sudden cardiac arrest, we may still continue to face complications related to the interventions we do.

References

1. Barrold SS, Mugica J: Recent advances in Cardiac Pacing: Goals for the 21st Century, Vol 19. Mt Kisco, NY, Futura, 1998, pp 213-231.
2. Parsonnet V, Bernstein AD, Lindsay B. Pacemaker-implantation complication rates: an analysis of some contributing factors. J Am Coll Cardiol. 1989 Mar 15;13:917-21.
3. Byrd C: Clinical Cardiac Pacing, Defibrillation, and Resynchronization therapy, 3rd edition. Saunders, Elsevier, 2007, pp 855-930.
4. Belott P, Reynolds D: Clinical Cardiac Pacing, Defibrillation, and Resynchronization therapy, 3rd edition. Saunders, Elsevier, 2007, pp 561-651.
5. Furman S. Venous cutdown for pacemaker implantation. Ann Thorac Surg. 1986;41:438-9.
6. Furman S. Subclavian puncture for pacemaker lead placement. Pacing Clin Electrophysiol. 1986;9:467.
7. McManus DD, Mattei ML, Rose K, Rashkin J, Rosenthal LS. Inadvertent lead placement in the left ventricle: a case report and brief review. Indian Pacing Electrophysiol J. 2009;9:224-8.
8. Veltri EP, Mower MM, Reid PR. Twiddler's syndrome: a new twist. Pacing Clin Electrophysiol. 1984;7:1004-9.
9. Syamkumar M Divakara Menon, Glen L Sumner, Carlos S Ribas, Jeff.S.Healey, Girish.M.Nair, Stuart.J.Connolly, Carlos. A Morillo. Contralateral Pneumothorax in Left Sided CRT Device Implantation. Indian Pacing Electrophysiol J; 2011; 11:16-19.
10. Refaat MM, Hashash JG, Shalaby AA. Late perforation by cardiac implantable electronic device leads: clinical presentation, diagnostic clues, and management. Clin Cardiol. 2010;33:466-75.
11. Bohora S, Unnikrishnan M, Ajit Kumar VK, Nayyar S, Tharakan J. Left hemothorax: a presentation of a late ventricular perforation caused by an active fixation pacing lead. Int J Cardiol. 2010;141:e43-6.
12. Ellenbogen KA, Wood MA, Shepard RK. Delayed complications following pacemaker implantation. Pacing Clin Electrophysiol 2002; 25:1155–1158.
13. Sivakumaran S, Irwin ME, Gulamhusein SS, Senaraine PJM. Post pacemaker implant pericarditis: Incidence and outcomes with active fixation leads. Pacing Clin Electrophysiol 2002; 25:833–837.
14. Khan MH, George J, Kahykin Y, Ziada KM, Wilkoff B. Delayed lead perforation, a disturbing trend. Pacing Clin Electrophysiol 2005; 28:251–253.
15. Naveen Garg, Nagaraja Moorthy. A Mysterious Pacemaker Suture: An Uncommon Foreign Body Reaction. Indian Pacing and Electrophysiology Journal; 2011; 11:27-30.

ECG Quiz

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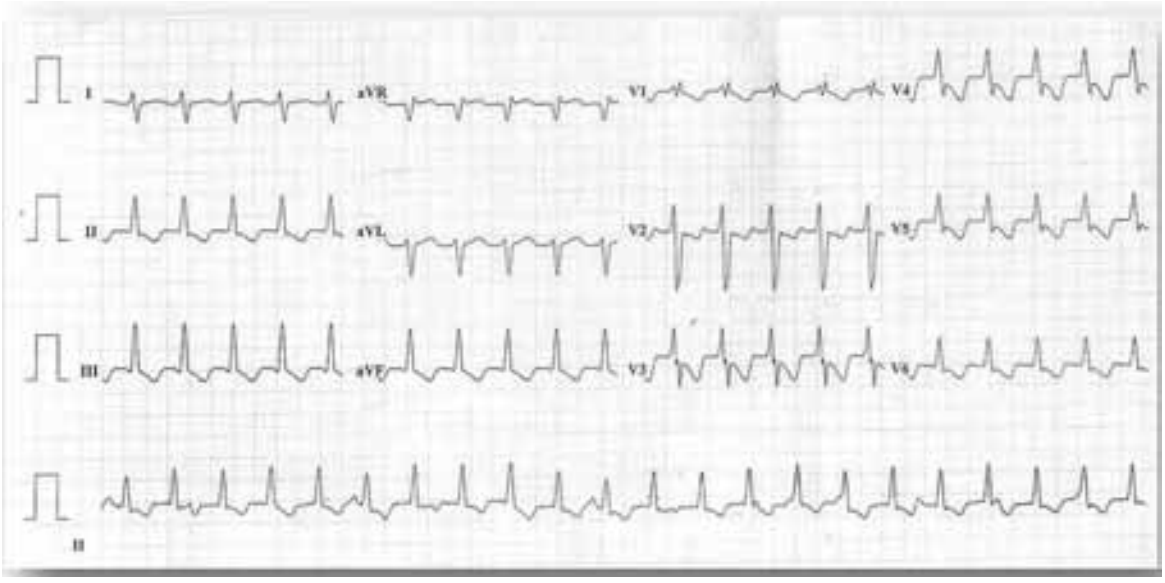
The answers and explanations are on the reverse side of the page.

ECG - 1

Young man. Recurrent episodes of tachycardia. Normal echo.



The diagnosis is:

a. Supraventricular tachycardia

b. Junctional ectopic tachycardia

c. Ventricular tachycardia

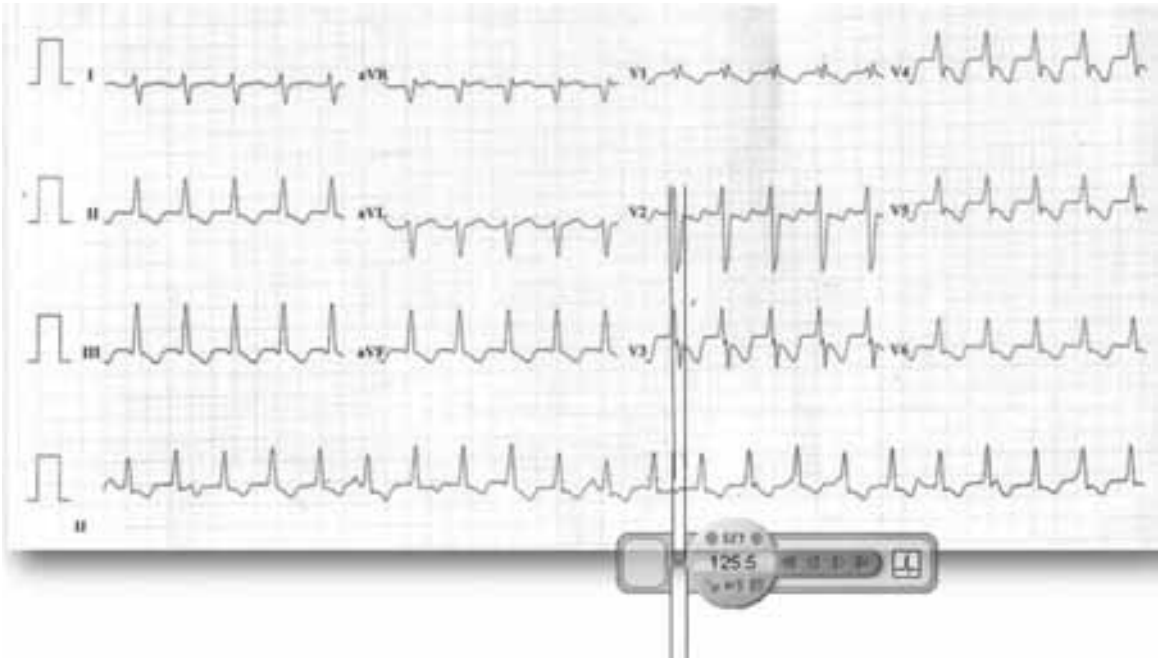
For correct answer see overleaf

ECG - 1

The correct answer is 'c' – Ventricular tachycardia (VT)

There is regular tachycardia at the rate of 136 bpm. The QRS complexes in the limb lead appear narrow. However, if one looks at lead V2, the QRS measures 126 ms. Hence, this qualifies as a broad QRS complex. The QRS morphology does not conform to any typical BBB pattern.

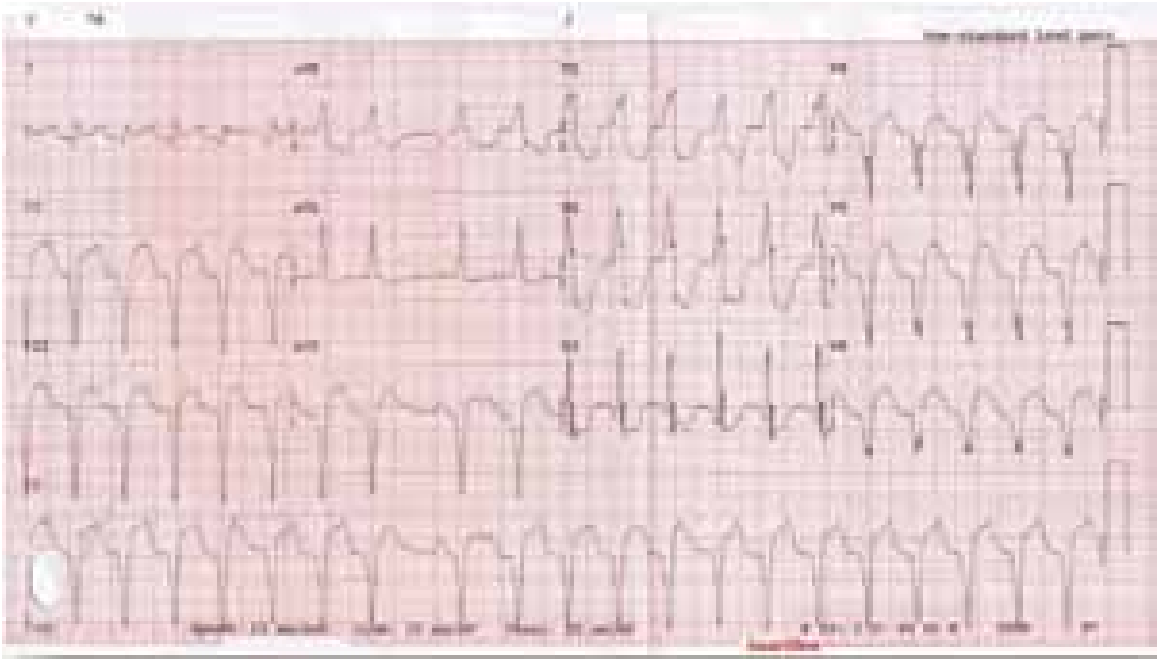
All these features favour the diagnosis of VT. The clinching evidence is seen in long lead II, in the form of AV dissociation.



In view of a structurally normal heart, the monomorphic VT will be labeled as 'idiopathic'. It does not conform to the classical idiopathic morphologies like fascicular VT and RVOT VT. The site of origin of this VT was mapped during EP study and was found to be in left anterior fascicle.

ECG - 2

Elderly man with pulmonary edema



This ECG shows:

a. Atrial tachycardia

b. Scar VT

c. Fascicular VT

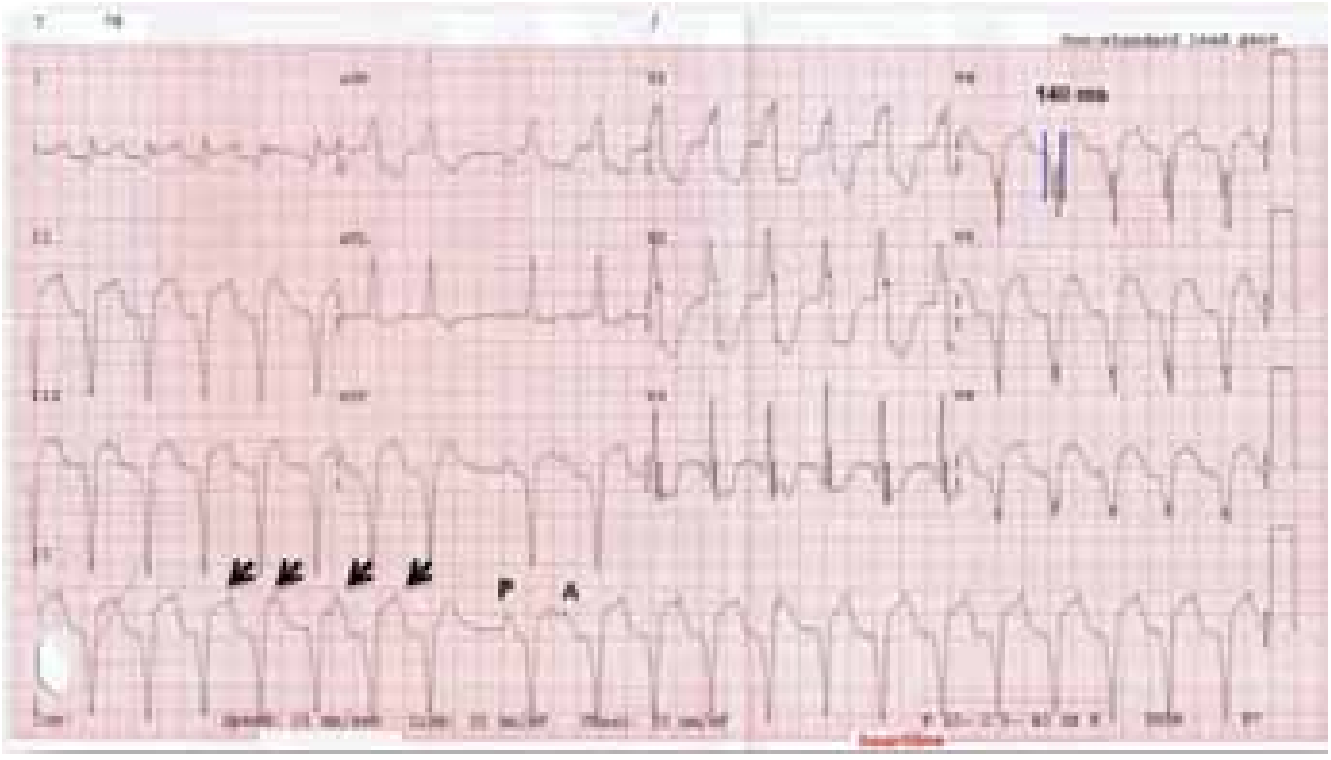
d. None of the above

For correct answer see overleaf

ECG - 2

The correct answer is 'a' – Atrial tachycardia

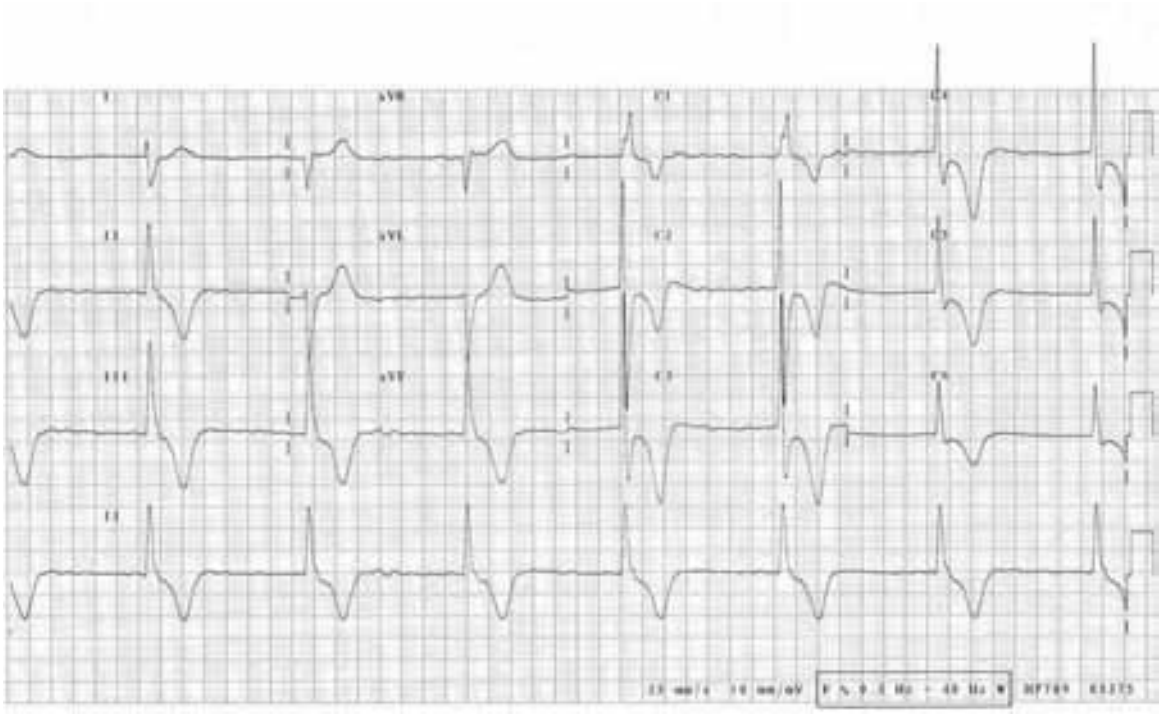
The ECG shows a regular wide QRS tachycardia except for a transient pause. The QRS width (lead V4) is around 140 ms. There are QS complexes in lead V4 to V6. The QRS axis is -60o. The P waves are clearly discernible. These P waves are positive in lead II and cannot be retrograde P waves of VT.



Importantly, the tachycardia terminates after the last P waves conduct to the ventricle.

A sinus complex (P) is seen after that. Immediately after that a premature atrial complex (A) sets of the atrial tachycardia. The QS complexes results from a large inferolateral MI. There is also a RBBB+LAFB. Hence, although there is a large underlying scar, the arrhythmias is atrial tachycardia.

ECG - 3



This ECG shows:

a. Atrial fibrillation

b. CHB

c. Idioventricular rhythm

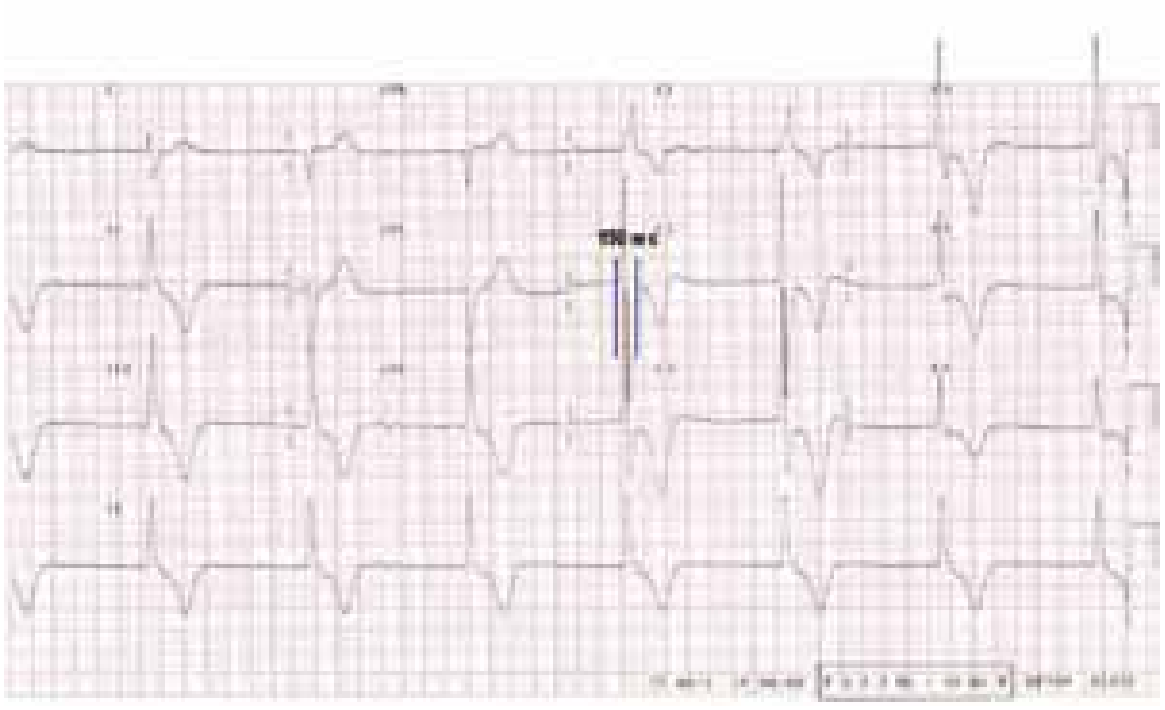
d. All of the above

For correct answer see overleaf

ECG - 3

The correct answer is 'd' – All of the above

There is a slow ventricular rate (35 bpm). The RR intervals are regular but the QRS complex is wide (150 ms- see annotations in lead V2).

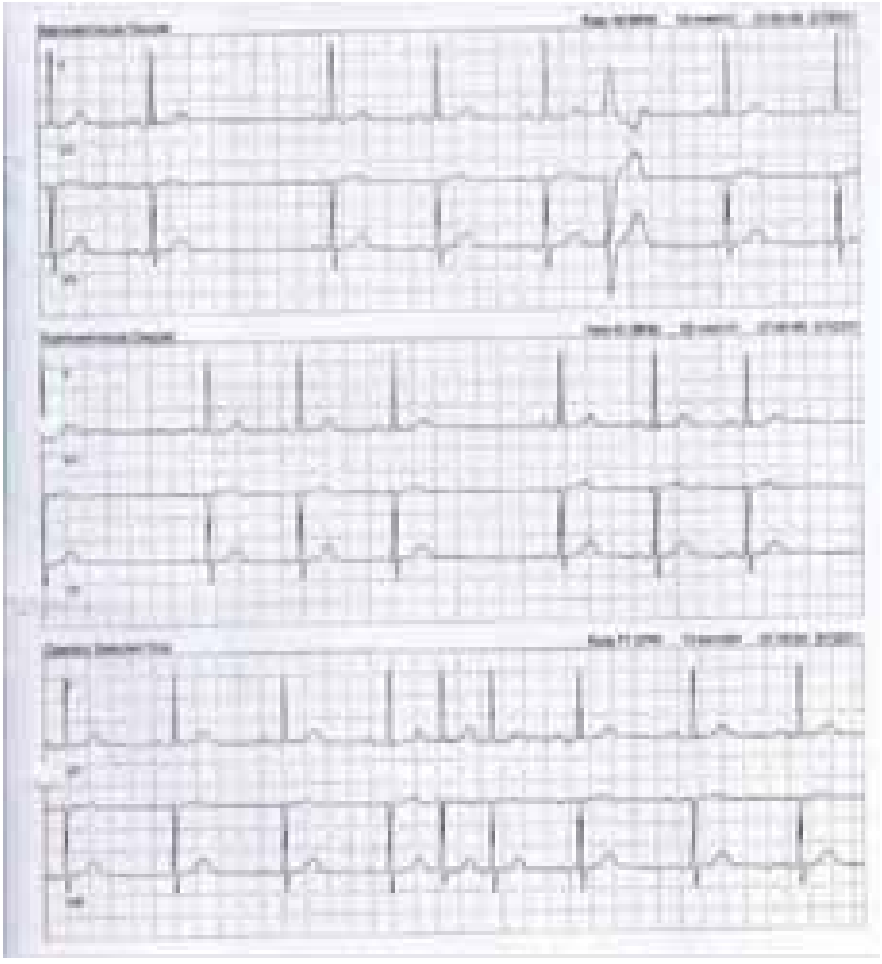


There are deep symmetrical T wave inversions in inferior and precordial leads. No normal P waves are seen. There is a clear atrial fibrillation.

In atrial fibrillation, a slow regular ventricular rate can only be the result of complete heart block.The wide QRS complexes originate in the ventricular myocardium.

ECG - 4

55 yr old man; palpitations, “missed beats



This ECG shows:

a. Tachy-brady syndrome

b. Atrial arrhythmias

c. Sick sinus syndrome

d. AV nodal disease

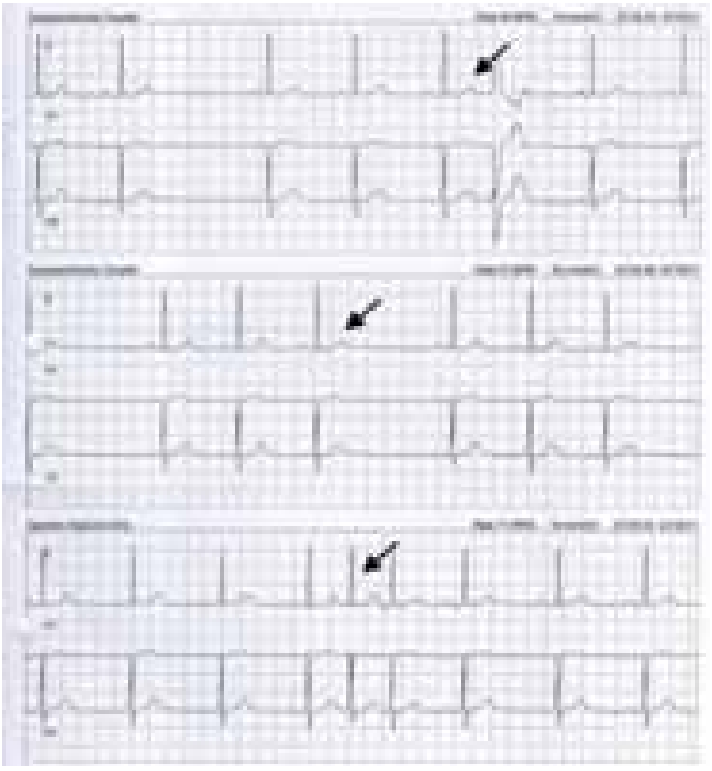
e. Both C and D

For correct answer see overleaf

ECG - 4

The correct answer is 'b' – Atrial arrhythmias

There are several pauses in the two Holter strips. A PAC is seen at the peak of T wave in top strip (arrow).



Similar notches in the T waves are seen in the middle strip.

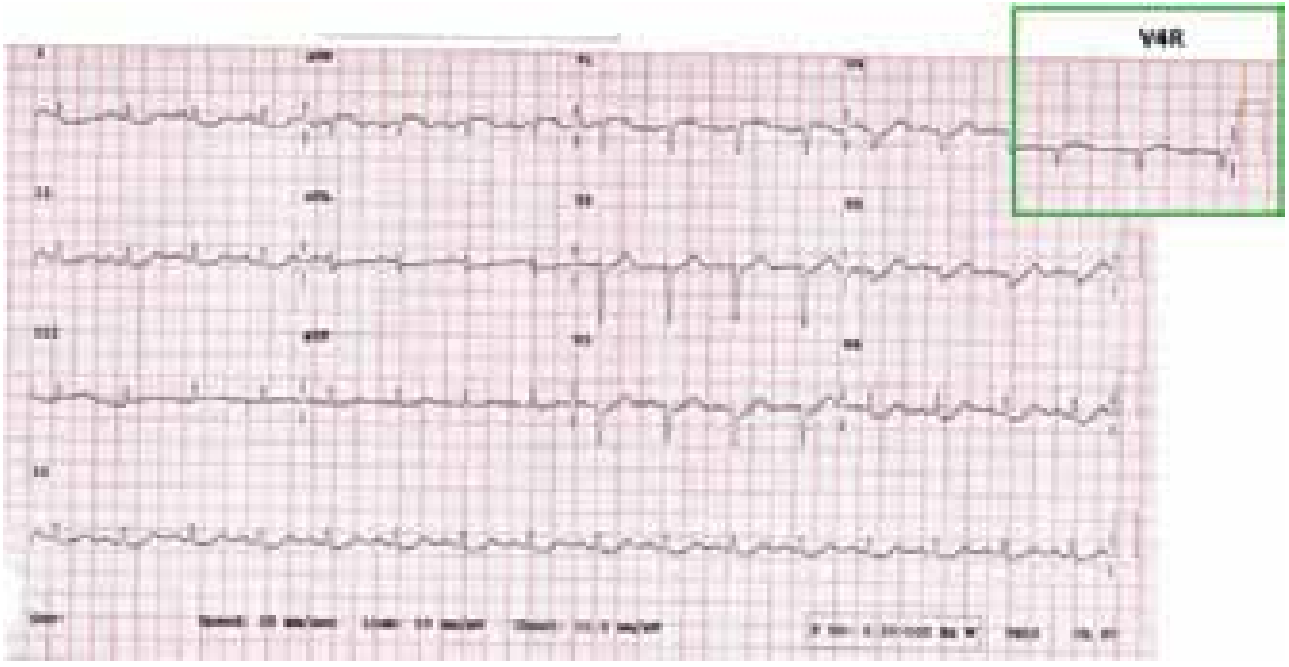
These PACs come so early that they are 'physiologically' blocked.

When the PACs come slightly later (as seen in the bottom strip), they are conducted to the ventricles. This shows that the AV conduction system is normal.

One PAC conducts with a long PR interval and associated BBB.

ECG - 5

70 yr old lady. Chest heaviness with transient collapse 8 hrs ago, after a long train journey. Basal crepitations, O2 saturation at 90%



This ECG shows:

a. ACS

b. Pulmonary thromboembolism

c. Myocarditis

For correct answer see overleaf

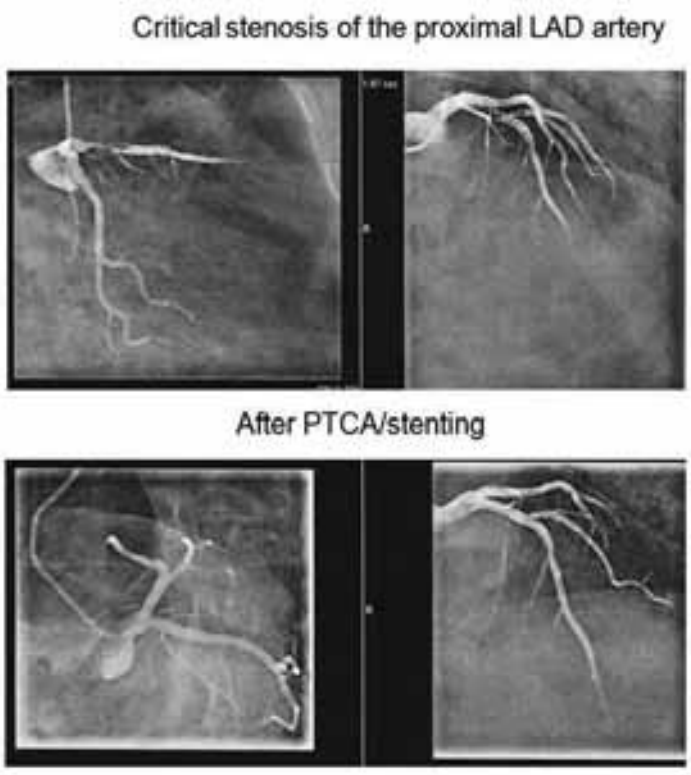
ECG - 5

The correct answer is 'a' – ACS

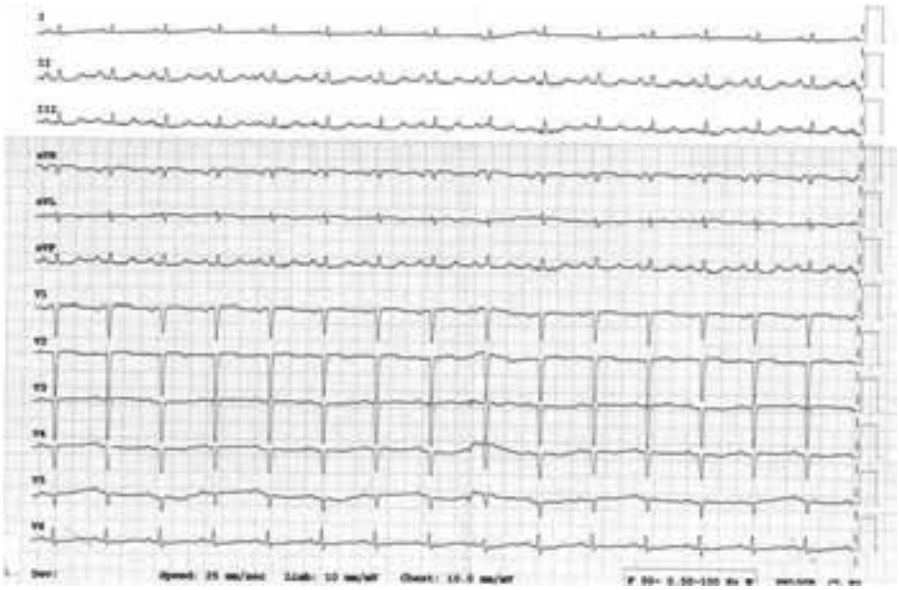
The ECG shows ST coving in lead V1 and aVR. There is also mild horizontal depression in the lateral leads. There is a poor progression of R waves till lead V5. The basal crepitations suggest early pulmonary edema.

The entire combination suggests a large area of ischaemia and severe LV dysfunction.

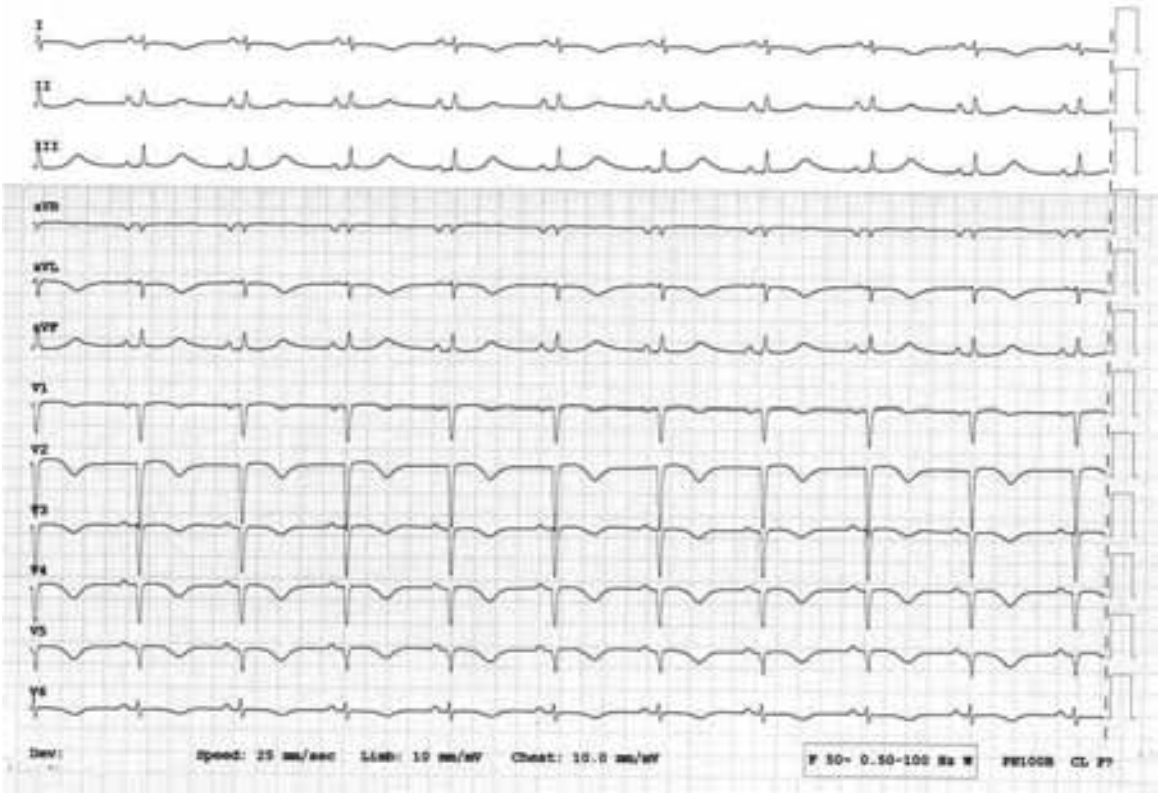
The coronary angiogram done the next day after stabilization, showed a flow-limiting stenosis of the proximal left anterior descending (LAD) artery. This was stented with a good result.



2 hrs after the intervention: The ST segments have normalized. QS complexes V1-V5.



The next day…. Evolutionary T inversion with mild QT prolongation V2-V6. This is actually a favourable sign, indicating stunned myocardium, likely to improve over the next few weeks (Hirota Y, Kita Y, Tsuji R, Handa H, Ishii K, Yoneda Y, et al. Prominent negative T waves with QT prolongation indicate reperfusion injury and myocardial stunning. J Cardiol 1992; 22: 325 –340).



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.
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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

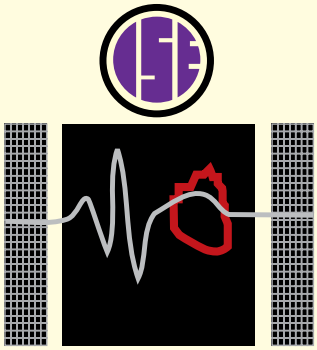
***Life Members :**

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.



SECRETARIAT
S. B. GUPTA
VICE PRESIDENT

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