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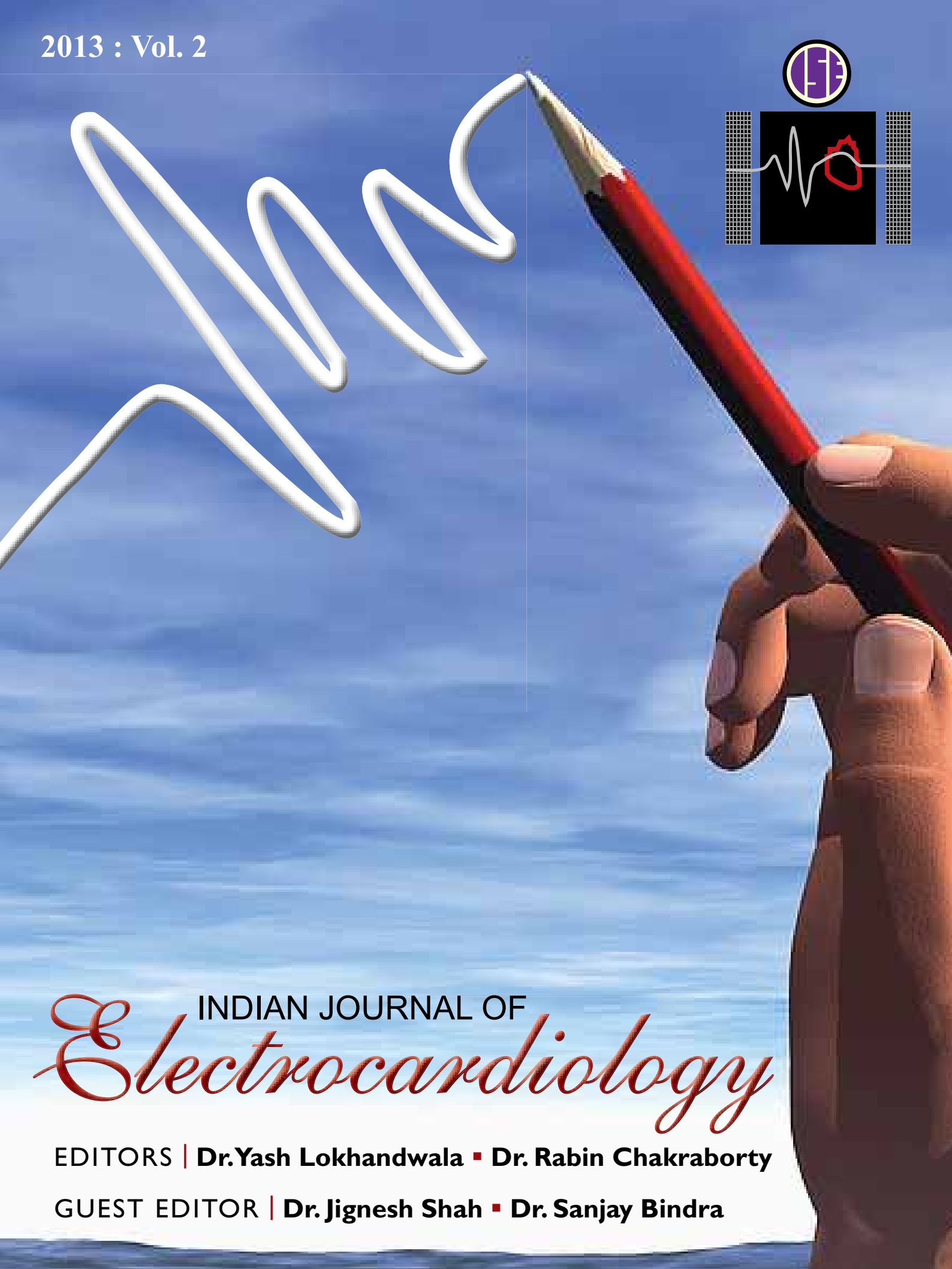

INDIAN JOURNAL OF Electrocardiology

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Editorial

Dear Friends,

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

We have been fortunate to have a number of clinicians contributing excellent review articles to this issue of IJE. Drs. Devidutta and Lokandwala present a case of accessory pathway in a patient with hypertrophic cardiomyopathy, two rare diseases occurring together. Coincidence? May be not. Read and find out. Dr. Soni presents a case of reversible cardiomyopathy and explores the etiology based on the time course of the disease. Dr. Darrat continues his support of the IJE and presents a case report and review of literature regarding twiddler's syndrome, a rare but curious device related complication. Dr. Arbune presents a typical case of digitalis toxicity, a disease entity rich in its ECG manifestations. He further discusses various presentation and the management of this frequent toxidrome.

A comprehensive review of the utility of internal loop recorder in syncope of unknown etiology is presented by Drs. Puri and Srivastava. This review article appeared in IPEJ and as always, IPEJ has graciously allowed us to borrow their article. Amit Desai and team has put together a comprehensive review of electromagnetic interference of cardiac devices from gizmos and gadgets of daily use. This review will help us answer some of our patients daily concerns regarding cardiac devices. 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's Desk

Dear Members,

It is our great pleasure in bringing out the 2nd issue of Indian Journal of Electrocardiology of the year 2013 on the eve of Mid-Term Annual Conference of Indian Society of Electrocardiology at Mangalore.

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

Mid-Term Conference of Indian Society of Electrocardiology was organized by Dr 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 :

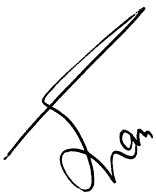
a. "ECG Learning Courses" for postgraduate students were organized at Thiruvanthapuram 0n 20th -21st April 2013, Bhubaneswar on 22nd – 23rd June 2013, Mumbai on 21st July 2013, Bhopal on 3rd and 4th August 2013 and Ahmedabad on 18th August 2013. Successful candidates were awarded the Certificate of Competence for ECG reading

b. ISE Program at Mumbai on 23rd June 2013.

c. 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 Rabin Chakraborty, Dr Jignesh Shah, Dr Sanjay Bindra and the Editorial Team for bringing out the ISE Journal – 2013, 2nd Volume.

Long Live Indian Society of Electrocardiology.



Dr. S.B. Gupta
President
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Review Article

Electromagnetic Interference with Pacemakers and Defibrillators in "Day to Day" Life

Amit Desai[*], Bianca Honnekeri[**], Disha Lokhandwala[***], CK Ponde[****]

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Introduction

With the ever-expanding indications for device therapies for management of cardiac rhythm disorders, the number of patients receiving pacemakers and implantable cardioverter-defibrillators (ICDs) are increasing every year. These devices rely on complex micro-circuitry and use electromagnetic waves for their communication with the programmers. Therefore, these devices are susceptible to interference from surrounding electromagnetic radiation. Electromagnetic interference (EMI) can be defined as any signal, either biologic or non-biologic, that falls within a frequency spectrum that is detected by the sensing circuitry of the pacemaker. This may interfere with the optimal function of the pacemaker and is always a concern.

Table 1

Electromagnetic fields
Daily life:
1. Cellular telephone
2. Electronic article surveillance devices
3. Metal detectors
4. Some home appliances (e.g., electric razor, toy remote controls)
5. Music and Media devices (e.g. MP3 Player, Ipods, Ipads)
Work and industrial environment:
1. High voltage power lines
2. Transformers
3. Welders
4. Electric motors
Medical Environment:
1. Magnetic resonance image scanners
2. Electro surgery
3. External Defibrillation
4. Neurostimulators
5. TENS units
6. Therapeutic diathermy
7. Ionizing radiation
8. Radiotherapy
9. Lithotripsy

EMI may potentially affect pacemakers and ICDs in one of the following ways:

1. Inhibiting the pacemaker from delivering the stimulating pulses that regulate the heart's rhythm
2. Causing the pacemaker to deliver the impulse irregularly
3. Causing the pacemaker to ignore the patient's own rhythm and deliver impulses at a fixed rate
4. Inappropriate shocks due oversensing of EMI perceived as ventricular fibrillation by the ICD

Over the last 2 decades there has been an explosion of high-tech gadgets and gizmos. On parallel grounds, there has also been increased complexity introduced into pacemaker and ICDs. Despite the fact that newer generation cardiac devices have better band pass filters, it is imperative to be concerned about EMI. It is important to note that the newer generation devices with remote monitoring system are not immune to EMI. This article, focuses on the effect of various environmental EMIs on cardiac devices, their impact on daily activities of the patients and the methods to prevent negative consequences of these EMI.

Sources of EMI

The common home and workplace items that can generate EMI are described in Table 1

EMI in daily life

Portable headphones

Portable headphones such as those used with portable digital music players (MP3 players) like iPods, generate powerful magnetic fields that have the potential to cause clinically relevant EMI. Keeping portable headphones in the shirt pocket may temporarily deactivate the pacemaker. Because magnetic field strength decreases exponentially with distance, keeping the portable headphones even a

short distance (3cm) from the pacemaker can effectively eliminate the potential for EMI.

iPods

iPods may cause interference if they are brought very close to an implanted device monitored through the telemetry wand. Thaker et al. (2008) tested four types of iPods, and found that 19% of the patients studied showed interference associated with atrial and/or ventricular high rates on rate histograms, while 32% showed interference that did not affect pacemaker rate counters. The iPods demonstrated maximum interference when they were turned 'on' with headphones attached, implying that active iPods placed within 2 inches of implanted pacemakers can cause interference. However Shah et al. (2010) found that while iPods can interfere with the establishment and maintenance of the telemetry link, they do not corrupt the vital stored downlink data. It is recommended that iPods be kept at least 2 inches away from the patient's chest, and not be used at all during device interrogation.

iPads

Under standard functionality, ICD Therapy suspension and Asynchronous Pacing of IPG can be triggered by exposure to a magnet with a static magnetic field greater than 10 gauss. Chien et al. reported that iPad2 can trigger similar magnet mode in ICDs and therefore suspend anti-tachycardia therapy. Other devices with embedded magnets like iPad2 can cause similar interference. Furthermore, since the covers of iPads often use magnets to secure them to the tablet, one needs to be vigilant with these accessories. By maintaining 15cm distance with iPads from the implanted devices, EMI can be avoided.

Cellphones / Cellular Towers / R.F. Transmissions

Radio Frequency Transmission Chart

The following guidelines are suggestions for safe use of radio equipment: Power in Watts	Minimum Distance of device from Antenna	Example(s)
3 watts or less	6 inches (15 cm)	Cellular Telephone, Cordless Telephone, Cordless Microphone, Home wireless electronics, Smart key/Remote car starter
>3 – 15	12 inches (30 cm)	Citizens Band, Long Range Cordless Telephone, Invisible Fences, Walkie-Talkies
>15 – 30	24 inches (60 cm)	Marine band radios, GPS survey equipment, some jobsite radios
>30 – 50	3 feet (1 meter)	Commercial and government dispatch, e.g. taxis, emergency vehicles
>50 – 125	6 feet (2 meters)	
>125 – 250	9 feet (3 meters)	Commercial broadcasting towers, Ham Radio
>250 – 500	12 feet (4 meters)	
>500 – 1000	20 feet (6 meters)	
>1000 – 2000	30 feet (9 meters)	
>2000	No exposures >100V/meter	High power broadcast towers

These EMI occur without user initiated action and hence caution is needed. In terms of use of cell phones, standard recommendations include: Talk into the hand-held phone with the phone against the ear opposite the side of an upper thorax implant and do not carry the cellular phone in pockets or on a belt adjacent to or over the implant site.

Hearing aids

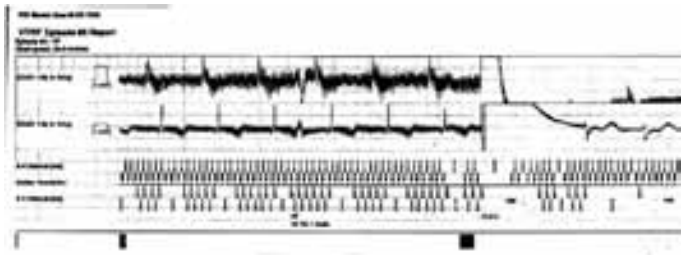
Cochlear implants have a low risk of EMI with the pacemakers. Hearing Aids with transmitting necklace loops coupled into the ear piece Telcoil (T-coil) of the hearing aid emit magnetic fields. The transmitting antenna associated with this type of hearing aid system is incorporated into the necklace loop. This antenna radiates a magnetic field that is coupled into the T-coil in the earpiece of the hearing aid. Maintaining a 6" (15cm) distance between the pacemaker and the portion of the hearing aid necklace radiating the magnetic field is recommended.

Work/environment

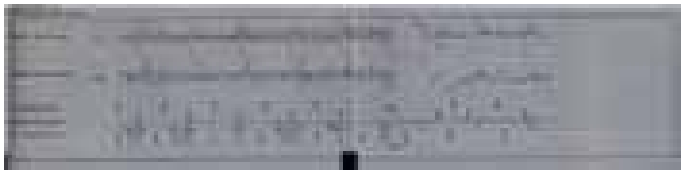
Electric shock

EMI can occur if patients come in proximity to high voltage overhead power lines (accidentally or by occupation) or it may be caused by electrical appliances held close to the chest. A momentary shock from an electrical outlet (110 / 220 volts) or higher voltages, if in a commercial or industrial setting, will cause pacemaker inhibition or inhibition of the pacing function of the ICD. Permanent damage to the pacemaker or ICD is unlikely to occur unless the shock is of large magnitude. EMI from household appliances is more likely with improper grounding.

Example 1 : A 62 year old man received an inappropriate shock while handling a washing machine which had leakage of 50Hz AC current because of improper grounding. This current which passed through the patient's body got inappropriately sensed by defibrillator device as ventricular fibrillation and gave an inappropriate shock.



Example 2 : A 25-year-old lady with an ICD for idiopathic ventricular fibrillation. The ICD tracing below was recorded when she got an inappropriate shock while opening the bathroom tap. One can clearly see electrical noise being picked up by the ICD and misinterpreted as ventricular fibrillation.



Note how the disturbance is noted in both the atrial and ventricular channels with EMI, an important point to differentiate it from a local hardware or lead issue

Anti-theft systems (also called electronic article surveillance or EAS):

Interactions with EAS systems are unlikely to cause clinically significant symptoms in most patients. However, the American Heart Association recommends that the patient:

- Is aware that EAS systems may be hidden or camouflaged in entrances and exits in many businesses.
- Don't stay near the EAS system longer than is necessary.
- Don't lean against the system.

Metal detectors for security:

Interactions with metal detectors are unlikely to cause clinically significant symptoms in most patients. However, the American Heart Association recommends that the patients:

- Don›t stay near the metal detector longer than is necessary.
- Don›t lean against the system.
- If scanning with a hand-held metal detector is necessary, tell the security personnel that you have a pacemaker. Ask them not to hold the metal detector near the device any longer than is absolutely necessary. Or ask for an alternative form of personal search.

Devices / Activities	Low Risk/ Safe to Use	Pacemaker Inhibition	Possible Defibrillator Shock	Remark
Television Sets	✓			
Radios	✓			
Microwave Ovens	✓			
Washing Machines	✓			
Laptops/Desktop Computers	✓			
Cordless Phones	✓			
TV Remotes	✓			
Electric Hair Dryers	✓			
Electric Shavers	✓			
Electric Toasters	✓			
Electric Vacuum Cleaners	✓			
Cellular Phones	✓	✓	✓	Maintain 15cm Distance
Mobile Phone Towers	✓			Maintain 9 meter Distance
Metal Detectors	✓			Don't stay near detector frames
TV Transmitters (Cordless Headphones)		✓		
MP3 Players Headphones		✓		Not to keep headphones near chest area
Electric Razors		✓	✓	
Play Stations		✓		
Antitheft Systems	✓	✓		Don't stay near detector frames
Slot Machines		✓		

Interference: Quick Look-up Tables for Pacemakers/ Defibrillators

If patient experiences dizziness or a feeling of rapid or irregular heartbeats (palpitations), or an extra shock caused by interference from equipment, simply move further away from the item. The pacemaker/ICD will immediately return to working normally.

It is always worth spending some time discussing about EMI with every patient. If EMI is suspected, get the device thoroughly evaluated as soon as possible.

Acknowledgement: Dr. Mehul Patel, Baylor College of Medicine, Texas Heart Institute

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Review Article

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Use of Implantable Loop Recorders to Unravel the Cause of Unexplained Syncope

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Abstract

Syncope is a symptom of many underlying disease states, which range from the relatively benign to the life threatening. There are numerous investigations done for patients with recurrent unexplained syncope which may have very low yield when it comes to making a definitive diagnosis. Recently, the implantable loop recorder (ILR) for continuous monitoring of the cardiac rhythm has been launched in India. This review will briefly discuss these current available strategies and focus on the usefulness of an ILR in the definitive diagnosis and treatment of patients with a recurrent unexplained syncope.

Key words: Syncope, implantable loop recorder

Introduction

Syncope is an abrupt and transient loss of consciousness (TLOC) associated with loss of postural tone that follows a sudden fall in cerebral perfusion. Syncope is a symptom of many underlying disease states, which range from the relatively benign to the life threatening.[1] At assessment, patients are often asymptomatic and the diagnosis is unclear, culminating in frequent hospitalization and resulting in expensive and often repeated investigations, most of which are inconclusive.[2]

Syncope accounts for approximately 3% of emergency room visits and 1-6 % of hospital admissions. The prevalence of syncope increases with age from 0.7% in men aged 35-44 to 5.6% in men over the age of 75. In long- term care institutions, the annual incidence is approximately 6 %.[3] The elderly represent the population at greater risk for most causes of syncope.

Once it has been established that the patient has true syncope, it is useful to further classify into 4 categories: 1) reflex neurally mediated; 2) cardiac; 3) orthostatic hypotension; or 4) unexplained. The major causes of syncope are enumerated in **Table 1**.

Investigation of patients with recurrent unexplained syncope may include electrocardiography, ambulatory Holter monitoring, treadmill exercise testing, neurologic testing, tilt table testing and electrophysiological testing. Recently, a new device, an implantable loop recorder (ILR) has been developed for continuous monitoring of the cardiac rhythm to unravel the cause of unexplained syncope. This review will briefly discuss these current available strategies and focus on the usefulness of an ILR in the definitive diagnosis and treatment of patients with a recurrent unexplained syncope.

More commonly, a focused initial evaluation of syncope leads to a suspected diagnosis, which needs to be confirmed by directed testing. If a diagnosis is confirmed by specific testing, treatment may be initiated. On the other hand, if the diagnosis is not confirmed, then patients are considered to have unexplained syncope. The strategy of evaluation varies according to the severity and frequency of the episodes. The majority of patients with single or rare episodes probably have neurally mediated syncope and tests for confirmation are usually not necessary.

If it is not clear that it was syncope, the term 'transient loss of consciousness (TLOC) is preferable and reappraisal is warranted.

Approach to assess unexplained syncope

History, physical examination, and electrocardiography are the core of the investigations of syncope with a combined diagnostic yield of 50%. Neurological testing is rarely helpful unless additional neurological signs or symptoms are present and the diagnostic yield for electroencephalography, computed tomography, and Doppler ultrasound is only 6%.[4]

Clinical history and physical examination

History should focus on postural symptom, palpitations, family history and should include the use of medication particularly in the elderly patients. Physical examination should focus on orthostatic blood pressure, cardiac murmurs and specific cardiac disorders e.g. aortic or mitral stenosis. The clinical features

Table 1 : Different causes of syncope

Neurally-mediated (reflex)

i. Vasovagal syncope (common faint)
ii. Carotid sinus syncope
iii. Situational syncope
    - Cough, sneeze
    - Gastrointestinal stimulation (swallow, defecation, visceral pain)
    - Micturition (post-micturition)
    - Post-exercise
    - Post-prandial
iv. Glossopharyngeal neuralgia

Orthostatic hypotension

i. Autonomic failure
    - Primary autonomic failure syndromes (e.g. pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure)
    - Secondary autonomic failure syndromes (e.g., diabetic neuropathy, amyloid neuropathy)
    - Post-exercise
    - Post-prandial
ii. Drug (and alcohol) – induced orthostatic syncope
iii. Volume depletion

Cardiac Arrhythmias as primary cause

i. Sinus node dysfunction (including bradycardia / tachycardia syndrome)
ii. Atrioventricular conduction system disease
iii. Paroxysmal supraventricular and ventricular tachycardias
iv. Inherited syndromes (e.g. long QT syndrome, Brugada syndrome)
v. Implanted device (pacemaker ICD) malfunction
vi. Drug-induced proarrhythmias

Structural cardiac or cardiopulmonary disease

i. Obstructive cardiac valvular disease
ii. Acute myocardial infarction / ischemia
iii. Obstructive cardiomyopathy
iv. Atrial myxoma
v. Acute aortic dissection
vi. Pericardial disease / tamponade
vii. Pulmonary embolus / pulmonary hypertension

Cerebrovascular

Vascular steal syndromes

suggestive of a particular cause of syncope are enumerated in **Table 2**.

Table 2 : Clinical features suggestive of specific causes of syncope

Neurally – mediated syncope

- Absence of cardiac disease
- Long history of syncope
- After unpleasant sight, sound, smell or pain
- Prolonged standing or crowded, hot places
- Nausea, vomiting associated with syncope
- During or in the absorptive state after a meal
- With head rotation, pressure on carotid sinus (as in tumours, shaving, tight collars)
- After exertion

Syncope due to orthostatic hypotension

- After standing up
- Temporal relationship with start of medication leading to hypotension or changes of dosage
- Prolonged standing especially in crowded, hot places
- Presence of autonomic neuropathy or Parkinsonism
- After exertion

Cardiac syncope

- Presence of electrical or structural heart disease
- During exertion, or supine
- Predicted by palpitation or accompanied by chest pain
- Family history of sudden death

Cerebrovascular syncope

- With arm exercise
- Differences in blood pressure or pulse in the two arms

Cardiac diagnostic approach

Electrocardiography

Electrocardiography is essential in the work-up of patients with unexplained syncope but may reveal a direct cause in only 5% of patients. Pre-excitation patterns, a long QT- interval, the recently reported Brugada syndrome and characteristic features in patients with arrhythmogenic right ventricular dysplasia should all be considered.[5-7] **Table 3.**

Echocardiography

No studies have been specifically designed to assess the usefulness of echocardiography in syncope. However, in patients known to have or suspected of having heart disease, patients suspected of having arrhythmias, echocardiography is an important initial step in diagnostic testing. Unsuspected findings on echocardiography are reported in only 5% to 10% of un-selected patients.[8] This yield is similar to that of 12-lead electrocardiography. The cost-effectiveness of echocardiography in diagnosing the cause of syncope has yet to be determined. In

Table 3 : ECG abnormalities suggesting an arrhythmic syncope

- Bifascicular block (defined as either left bundle branch block or right bundle branch block combined with left anterior or left posterior fascicular block)
- Other intraventricular conduction abnormalities (QRS duration >0.12 s)
- Mobitz I second degree atroventricular block
- Asymptomatic sinus bradycardia (<50 bpm), sinoatrial block or sinus pause in the absence of negatively chronotropic medications
- Pre-excited QRS complexes
- Prolonged QT interval
- Right bundle branch block pattern with ST – elevation in leads V1-V3 (Brugada syndrome)
- Negative T waves in right precordial leads, epsilon waves and ventricular late potentials suggestive of arrhythmogenic right ventricular dysplasia
- Q waves suggesting myocardial infarction

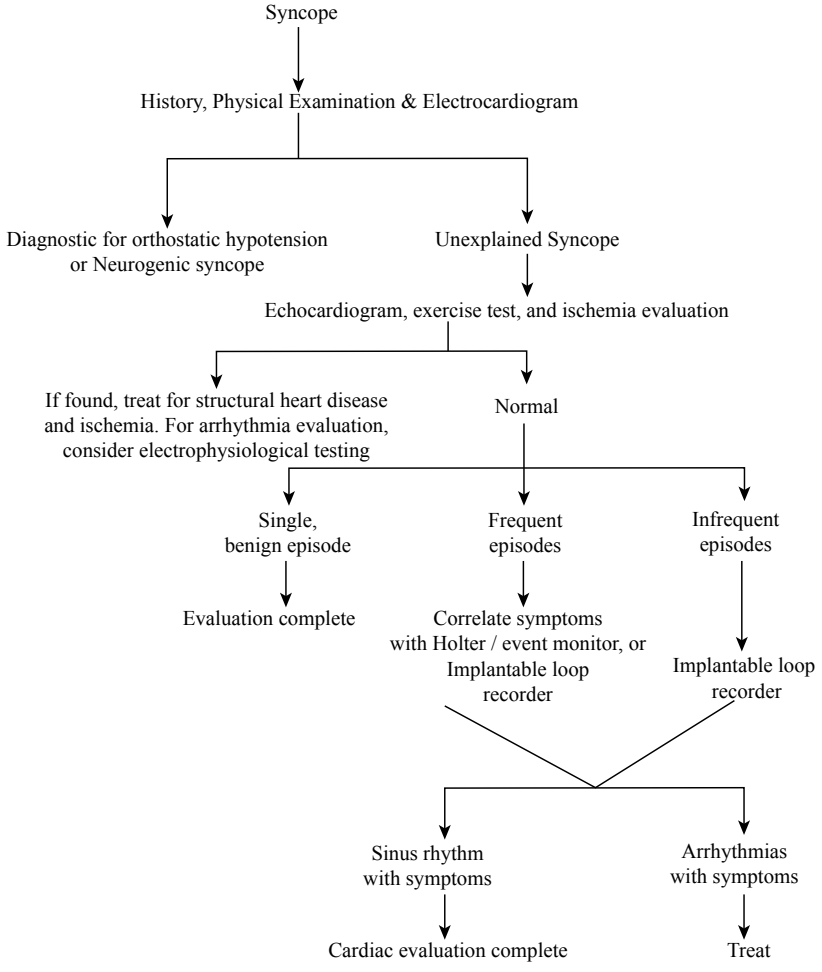

Figure 1 : Flow chart for the diagnostic approach to the patient with syncope using an implantable loop recorder

patients with exertional syncope, echocardiography should be done first to exclude hypertrophic cardiomyopathy.

Exercise Testing

Exercise stress testing can be used for the evaluation of exertional syncope to diagnose ischemia or exercise-induced tachyarrhythmia's or to reproduce post exertional syncope. In one population study of patients with syncope, the yield of the exercise stress test was less than 1% .[9] Data is scarce to determine the yield for ischemia or exercise-induced tachyarrhythmia's or to define the test's usefulness in diagnosing exercise-associated syncope. Exercise stress testing is recommended if patients have exercise-associated syncope and if the results of clinical evaluation suggest ischemic heart disease.

Tilt table testing

Tilt table testing has emerged as a safe and effective method of identifying individuals with a susceptibility to neurally mediated syncope.[10] Tilt table testing may be performed alone or with pharmacological provocation using isoprenaline or nitrate preparations. The tilt table test is best considered for patients with suspected neurally mediated syncope but in whom the cause is not obvious or in patients with syncope of otherwise unknown origin with no evidence of heart disease. The major problem with tilt table testing is quantifying the test's sensitivity. The sensitivity of the test has been calculated at between 20% and 75%.[11] In those known to have structural heart disease in which electrophysiological studies have not given a diagnostic clue, tilt table testing may prove cost-effective by avoiding expensive and unrewarding neurological investigations such as computed tomography or electroencephalography which might otherwise be requested. Interestingly, the frequency of syncope decreases following a positive test regardless of therapeutic intervention. The test may educate the patient to recognize the warning signs of syncope and to make appropriate changes in posture.[12,13]

Holter monitoring

A 24-hour Holter monitor or inpatient telemetry is recommended when symptoms suggest arrhythmic syncope with brief loss of consciousness, no prodrome, palpitations with syncope and in patients who have syncope of unexplained cause, heart disease, or an abnormal electrocardiogram.

Neurological diagnostic approach

Neurologic tests used for patients with syncope include electroencephalography, brain imaging (computed tomography or magnetic resonance imaging), and neurovascular studies (carotid and

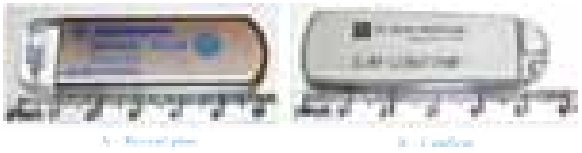

Figure 2 : Two type of ILR, the REVEAL PLUS device (MEDTRONIC, Minneapolis, USA) is about 62 × 19 × 8 mm in size and the CONFIRM device (ST JUDE, St Paul, USA) , it is about 563 × 18.5 × 8 mm in size

transcranial doppler ultrasonographic studies). To determine which patients may benefit from neurologic testing, physicians should take a particularly careful neurologic history for example, patients should be asked about a history of seizure activity, prolonged loss of consciousness, diplopia, headache, and post ictal symptoms and perform a thorough, focused physical examination including a search for bruits or focal neurologic signs. A diagnostic algorithm and approach to a patient with syncope is suggested in **Figure 1**.

New method of monitoring to diagnose syncope: "Implantable loop recorder" (ILR)

In spite of a detailed screening of patients and often multidisciplinary investigation, more than one third of the patients may remain undiagnosed.[14] Recently, an implantable loop recorder (ILR) has been developed for continuous monitoring of the cardiac rhythm to unravel the cause of unexplained syncope. The ILR is an implantable device that has a solid state loop memory capable of storing electrocardiographic events up to 40 minutes before and 1 to 2 minutes after activation. The ILR has built in electrodes on the back of the device to detect patients' cardiac rhythm and does not require any intra cardiac leads. This device is very small and is typically implanted subcutaneously in the left pectoral region as an outpatient procedure.[15] Currently, there are 2 FDA-approved ILRs available for clinical use as shown in **Figure 2**. The REVEAL PLUS device (MEDTRONIC, Minneapolis, USA) was the first approved device, it is about 62 × 19 × 8 mm in size and the battery of the device generally lasts 18 months. The CONFIRM device (ST JUDE, St Paul, USA), it is about 56.3 × 18.5 × 8 mm in size and its battery is expected to last up to 3 years. Data are retrieved and analyzed with a compatible programmer but this device also has a remote real-time monitoring capability that allows patients to send data directly to their health care providers.[16]

Method of Implantation

After identification of the most appropriate pectoral position to record an electrocardiographic lead with a prominent QRS wave, the loop recorder is subcutaneously implanted snugly in a pocket similar to that of a pacemaker implantation, the size of a little finger. After suture of the skin, the quality of electrocardiographic recording is tested in supine and standing positions and during movements of both the arms. The device is explanted after a diagnosis is obtained or if syncope did not recur. The first experiences with the ILR have shown that the device was safe and was able to detect cardiac arrhythmias in about 23-42% of patients. The population in these studies was, however, not systematically evaluated prior to the ILR implantation to reach a diagnosis.[17]

Clinical evaluation of the ILR

In an initial study of 24 patients, 52% presented with an arrhythmic cause with the vast majority of patients having bradycardia. Treatment was directed at the underlying cause in the 18 patients who received a specific diagnosis. During follow up, syncope did not occur in 16 of the18 treated patients. In 2 patients who underwent explantation of the device after the end of battery life, no recurrence was seen with a final analysis extending the follow-up to 40 ± 10 months.[18] A larger series of 85 patients with recurrent undiagnosed syncope despite extensive evaluation had patients who were eligible if they had at least two syncopal episodes within the previous 2 months or if they had a single syncope with a history of presyncope. In all patients ILR was implanted which resulted in detection of abnormal rhythms in 21 patients, the vast majority having a bradycardia. Interestingly, in 29 patients no arrhythmia was detected despite symptoms. Patients with syncope were more likely to record an arrhythmia during symptoms compared to patients with a history of presyncope (70 % vs 24 % p = 0.005).[19] Recently another large and prospective study was carried out to collect information on the use of the REVEAL (MEDTRONIC, Minneapolis, USA) ILR in the patient care pathway and to investigate its effectiveness in the diagnosis of unexplained recurrent syncope in everyday clinical practice. Eligible patients had recurrent unexplained syncope or pre-syncope. Follow up was until the first recurrence of a syncopal event leading to a diagnosis or till the end of 1 year. In the course of the study, patients were evaluated by an average of three different specialists for management of their syncope and underwent a median of 13 tests. Average follow-up time after ILR implant was 10±6months. The percentages of patients with recurrence of syncope were 19%, 26% and 36% after 3, 6 and 12 months respectively. Of 218 events within the study, ILR-guided diagnosis was obtained in 170 cases (78%), of which 128 (75%) were of cardiac origin.[20] The findings support the recommendation in current guidelines that an ILR should be implanted early rather than late in the evaluation of unexplained syncope.

Recently we conducted a similar work and enrolled 20 patients of unexplained syncope, out of which 11 patients completed a 12 month, follow up. The diagnostic yield was 100% in the 11 patients among which 7 patients got permanent pacemaker implantation and 4 patients having no arrhythmic event noted with symptoms and 9 patients continue in follow up.[21]

Table 4 : Summary of Recommendations

Implantable loop recorders (ILRs) can be used

1. Patients with recurrent syncope of uncertain origin after the initial evaluation who have the following:
   - Clinical or electrocardiographic features suggesting an arrhythmic syncope
   - High probability of recurrence (i.e. ≥ 3 episodes of syncope during last 2 years)
   - Absence of high-risk criteria that require immediate hospitalization and intensive evaluation
2. To assess the contribution of bradycardia before embarking on cardiac pacing in patients with suspected or certain neutrally mediated syncope presenting with frequent or traumatic syncopal episodes.
3. Patients in whom the cause of syncope remains unexplained despite full evaluation and who have clinical or electrocardiographic features suggesting an arrhythmic syncope.

Diagnosis:

ILR findings are diagnostic when either of the following is true:

1. A correlation between presyncope and an arrhythmia (bradyarrhythmia or tachyarrhythmia) is detected.
2. In the absence of such correlation, ventricular pauses >3 sec when awake or periods of periods of Mobitz II or III atrio-ventricular block when awake, or rapid paroxysmal ventricular tachycardia are detected.
3. ILR findings exclude an arrhythmic cause when there is a correlation between syncope and no rhythm variation. Presyncope may not be an accurate surrogate for syncope in such circumstance, and therefore monitoring should be continued until syncope is documented.

Therapy Guided by ILR

Little is known about the outcome after ILR-guided specific therapy. In the East Bourne Syncope Assessment Study,[22] performed in a typical unselected population, there was an increased diagnostic rate in the group of patients randomized to ILR management and ECG-directed treatments than in conventional investigation group. Despite that a specific ILR directed therapy could be applied to only a minority of patients, the long-term follow-up demonstrated a significant reduction in syncopal events with improved quality of life with ILR based treatment. Since prolonged asystole was the most frequent finding at the time of syncope, pacing was the specific therapy mostly used in the ILR population. In a pooled data of 720 ILR patients from 4 studies, a pacemaker was implanted in 17% of patients, with 45% of those having an ILR-documented event. In the ISSUE 2 study, the 1-year burden of syncope decreased from 0.83±1.57 episodes per patient per year in the control group of patients without any ILR-guided specific therapy to 0.05±0.15 episodes per patient per year in the patients treated with a ILR-guided specific therapy of pacemaker (87% relative risk reduction; P=.001).[23] In another study, after the insertion of a cardiac pacemaker, syncope burden decreased from 2.17 per year to 0.45 per year in 1A or 1B group syncope patients (P=.02) and from 4.57 per year to 0 per year in the type 1C syncope patients (P=.001). Nevertheless, syncope still recurred in 12% (range, 3%–18%) of the patients during the long-term follow up (2.0–3.6 years), especially in those patients more likely to be affected by neurally mediated syncope, accounting for the coexistence of some vasodepressor reflex that cannot be overcome by pacing. Implantable cardioverter-defibrillator and radiofrequency catheter ablation were also consistently used in a few selected patients with ILR documented ventricular and atrial tachyarrhythmia's in 1.5% and 3.0% of the patients, respectively.[23]

Limitation of the device

Limitations of the device include the inability to monitor blood pressure changes, the necessity for surgical implant, cost of the device and a small risk of infection. Most patients in whom an arrhythmia is not documented during syncope have either hypotensive syndromes or psychogenic syncope. Hypotensive syndromes include vasovagal syncope, orthostatic hypotension, postprandial hypotension, and vasodepressor carotid carotid sinus hypersensitivity. A facility to track blood pressure behavior in addition to heart rate during symptoms would undoubtedly advance real-time hemodynamic monitoring with implantable devices. The inability of the present device to detect hypotension is a limitation and in the near future the devices capable of recording other physiological parameters may further increase the diagnostic yield.[24] With increasing healthcare costs, a proper selection of patients for implantation of an ILR is mandatory. Total costs of the investigation of patients with recurrent syncope are high because of extensive diagnostic testing.[25] However in a cost analysis study it was demonstrated that ILR implantation could reduce costs as was seen in a pilot study of 24 patients referred for recurrent syncope.[26] There may also be a small risk of interference from electronic article surveillance devices and found that interference may occur causing malfunction of this device.[27]

Conclusion

The prognosis of syncope ranges from benign to life threatening situations and risk stratification should be based on the result of history, physical examination, electrocardiography, and selected noninvasive test. ILR is a novel and useful diagnostic tool in patients with unexplained syncope. ILR is easy to implant and explant and is based on electrocardiography which is useful to establish if there is an arrhythmic cause of syncope, especially when symptoms are recurrent but too infrequent for conventional monitoring techniques.

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Case Report

Twiddler's Syndrome

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Introduction

Twiddler's syndrome is a rare presentation of pacemaker malfunction. We present a case of twiddler's syndrome along with review of literature.

Case

A 57 year old white male, with history of non ischemic cardiomyoapthy post implantable cardiovertor defibrillator (ICD) for primary prevention, presented to device clinic 6 weeks after implantation for interrogation. He was complaining of muscle twitching in his left upper abdomen that resolves once he raises his left arm above his head. Right ventricular lead was assessed and despite various maneuvers diaphragmatic stimulation could not be reproduced. Atrial lead showed failure to capture and sense. A 12 lead electrocardiogram (Figure 1) showed sinus rhythm with intact conduction above lower rate interval. A chest X-ray was obtained to examine the pacing leads position and integrity. The X-ray demonstrated atrial lead dislodgement and it's coiling around the pulse generator (Figure 2) while the right ventricular lead was intact. Therefore, atrial lead sensing and pacing function was turned off and patient was referred to the implanting physician for lead revision.

Discussion

Twiddler's syndrome, a rare etiology of device malfunction, was first described in 1968 by Bayliss et al.[1] It occurs due to deliberate or subconscious manipulation of the pulse generator in a capacious pocket by the patient, resulting in torsion, dislodgement, and often fracture of the pacing lead.[2] It is generally diagnosed within the first year of implantation. Twiddler's syndrome can be a life threatening condition in pacemaker dependent and ICD patients. It can lead to under or over sensing, loss of capture, and inappropriate ICD shocks. It is more common in the elderly and obese patients since the presence of loose subcutaneous tissue allows for rotation of the pulse generator in its pocket.[3] Such a rotation results in lead dislodgement, reeling of the leads around the generator and possibly ipsilateral phrenic nerve stimulation. As the leads are further wrapped around the generator, rhythmic arm twitching occurs when the brachial plexus is paced.[3,4] It can also lead to increasing lead impedance, without any of these symptoms.[5] Typically rotation of pulse generator occurs on its long axis. A different form of the syndrome, termed "Reel syndrome", was described in which the generator is rotated on its transverse axis.[6] Recently, reverse Twiddler's syndrome was described as pulse generator manipulation resulting in lead advancement rather than retraction.[7] Variations of the syndrome have been reported in medical literature with the manipulation of other devices, including deep brain stimulator implants[8] and chemotherapy infusion pumps.[9]

Twiddler's syndrome can easily be diagnosed with a chest X-ray. In addition, inappropriate sensing and capture seen on a 12 lead EKG can alert the physician to potential lead dislodgment. Timely pocket and lead revision is the mainstay of management. Creation of a small surgical pocket and suturing of the device to the fascia can help prevent pulse generator manipulation. Alternatively, Twiddler's syndrome can be possibly prevented

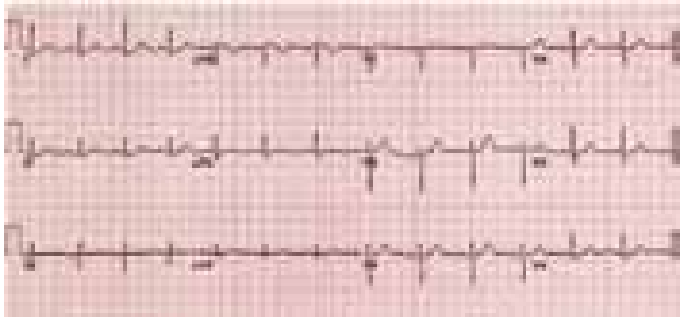

Figure 1 : 12 lead EKG demonstrating sinus rhythm and no significant findings.

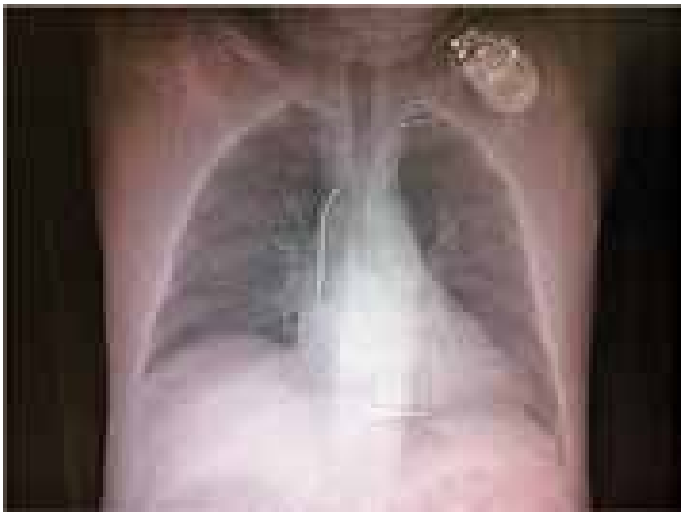

Figure 2 : Chest X-ray demonstrating atrial lead dislodgement and coiling of the lead around the longitudinal axis of the pulse generator.

by submuscular implantation and use of active fixation leads. Although, a case has been reported in which the syndrome occurred in a child despite use of the former mentioned approach.[4] We therefore recommend educating patients about the adverse consequences of pulse generator manipulation.

Conclusion

Twiddler's syndrome is a potentially life threatening condition occurring within the first year of device implantation. Patients can be asymptomatic or present with diaphragmatic or brachial plexus stimulation. Diagnosis can be readily achieved using chest x-ray. Expedient pocket and lead revision constitutes the mainstay of therapy. Prevention of the syndrome can be achieved through suturing of the pulse generator to fascia, submuscular implant and use of active fixation leads. Furthermore, patient education about the risks of pulse generator manipulation is of utmost importance.

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Case Report

Reversible Cardiomyopathy- Peripartum, Tachycardia-Induced or Myocarditis?

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Introduction

Tachycardia, extreme emotional stress, alcoholism, hypoparathyroidism, peri-partum state and end-stage renal disease have been shown to result in reversible impaired left ventricular function. We present a case of cardiomyopathy where more than one of these factors were at play.

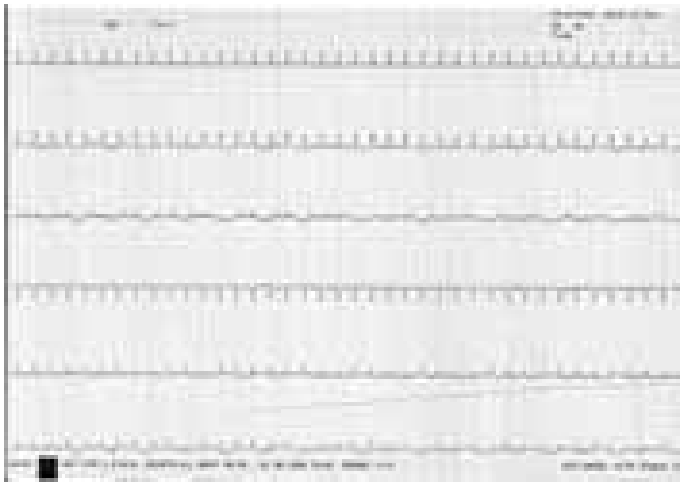

Figure 1 : Narrow complex tachycardia, cycle length of 180 ms, P waves could not be deciphered.

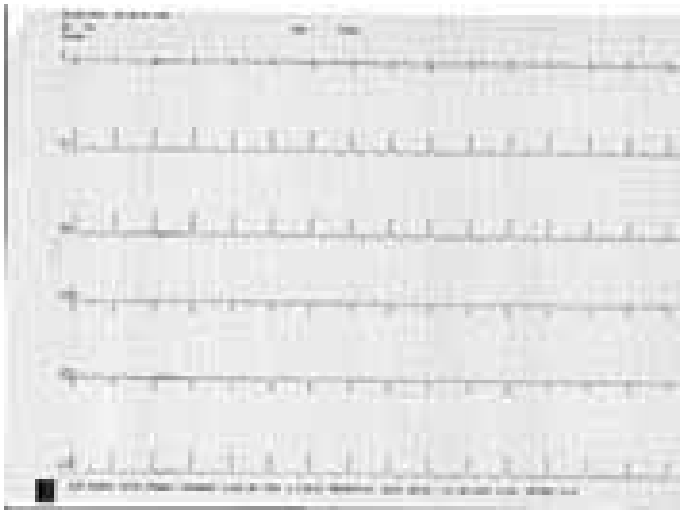

Figure 2 : Transient slowing & intermittent 2:1 AV conduction with upright P wave in inferior leads during carotid massage.

Case

A 23 year lady, post-partum day 25, presented with continuous fast regular palpitation since 3 days associated with progressive dyspnea over the past 3 days. On admission, the examination revealed heart rate-190/minute, regular, BP 106/70 mm Hg, basal crepitations and SPO_2 of 90%. The patient was in NYHA Class IV heart failure. The ECG showed narrow QRS tachycardia, cycle length of 180 ms, indistinct P waves (Figure 1); during carotid massage, transient slowing with intermittent 2:1 AV conduction with upright P wave in inferior were seen (Figure 2). 2D echocardiogram showed severe global LV hypokinesia (EF.15), mild mitral regurgitation and mild pulmonary hypertension. The routine biochemistry, thyroid profile and cardiac enzymes were normal.

The patient was stabilized with IV diuretics, digoxin and non-invasive ventilation. Intravenous amiodarone was started with a diagnosis of incessant atrial tachycardia. After 3 days DC cardioversion was tried but was ineffective.

The differential diagnoses at this point of time were:

1. Post-partum cardiomyopathy with superimposed atrial tachycardia

Figure 3 : RA appendage angiogram showing position of RF catheter

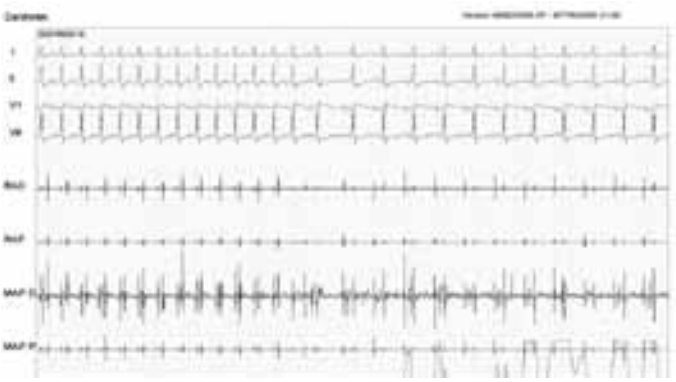

Figure 4 : Successful ablation of atrial tachycardia in the right atrial appendage.

2. Incessant atrial tachycardia causing cardiomyopathy
3. Viral myocarditis with atrial tachycardia

In view of drug refractory atrial tachycardia, it was decided to perform EP study with possible RF ablation. The focus of the atrial tachycardia was found to be just within the right atrial appendage; this was successfully ablated (Figures 3 & 4).

There were a few self-terminating episodes of tachycardia over the next week. The patient improved symptomatically over next week, ***but LVEF showed only marginal improvement***. After 2 weeks her LVEF started improving rapidly and normalised at 8 weeks.

Discussion

Tachycardia-induced cardiomyopathy (TIC) is caused by persistent supraventricular or ventricular tachyarrhythmias. It is characterized by ventricular systolic dysfunction and dilatation and by clinical manifestations of heart failure that are reversible with normalization of heart rate. Tachycardia-induced cardiomyopathy should be considered in all patients with a dilated cardiomyopathy of uncertain origin, who have concomitant tachycardia. TIC occurs in association with supraventricular arrhythmias such as atrial tachycardia, atrial flutter, atrial fibrillation, Coumel tachycardia/PJRT. Rarely, TIC may follow incessant right ventricular outflow tachycardia or idiopathic left ventricular "fascicular" tachycardia.

Abolishing tachycardia with drugs or catheter ablation often results in clinical improvement and recovery of ventricular function. However, the time course of such recovery is variable. It depends on the rate and duration of the responsible arrhythmia. Both experimental models and some reports in humans showed the greatest LVEF recovery 1 month after arrhythmia cessation and optimal heart rate control.[1] In animal models, complete resolution of cardiomyopathy was observed 4 weeks after cessation of pacing which is similar to recovery in humans.[2] In one study in which sinus rhythm was maintained in all patients after successful cardioversion, there were no notable differences in LVEF before and at 24 hours or 1 week after cardioversion. However, at 1 month, the LVEF was nearly normal and remained so for 6 months.[3]

Peripartum cardiomyopathy is one of the known etiologies for reversible LV dysfunction. Reported risk factors for peri-partum cardiomyopathy include age, pregnancy-induced hypertension or preeclampsia, multiparity, multiple gestations, obesity, chronic hypertension, and the prolonged use of tocolytics.[4,5] ECG abnormalities frequently noted on presentation include sinus tachycardia, nonspecific ST-T segment changes, LV hypertrophy, premature ventricular contractions, and bundle branch block. Recovery of LV function in patients with peripartum cardiomyopathy takes weeks to months.[6]

Based on the above studies, it can be postulated that our patient, whose LV function recovered in 8 weeks, probably had a combination of the above mentioned etiologies of reversible cardiomyopathy.

Conclusion

Tachycardia-induced cardiomyopathy is a reversible form of dilated cardiomyopathy and heart failure caused by supraventricular and ventricular tachyarrhythmias. Its diagnosis requires a high index of suspicion, and the clinician should consider the diagnosis in patients with unexplained systolic dysfunction and any form of tachyarrhythmia. Heart rate normalization, by either rate or rhythm control, is the cornerstone of therapy, which may result in improvement or normalization of systolic function in about 4 weeks.

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Case Report

Radiofrequency Ablation for WPW Syndrome in a Child with Hypertrophic Cardiomyopathy

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Introduction

Wolff-Parkinson-White syndrome (WPW) syndrome, defined as ventricular prexcitaiton pattern on EKG along with symptoms of syncope or tachycardia, has an incidence of 0.15-3 per 1,000.[1] Hypertrophic cardiomyopathy is the most common familial genetic disease affecting the heart with autosomal dominant inheritance and an estimated prevalence of 0.2%.[2] It is characterized by unexplained ventricular hypertrophy with myocyte and myofibrillar disarray. Reports suggest that about 5% of HCM patients have associated ventricular preexcitation.[3] As early as 1960 Braunwald et al.[4] had suggested that localized ventricular hypertrophy might disrupt normal electrical insulation at the atrioventricular ring. Studies through genetic linkage analyses have demonstrated that defect in chromosome 7q3 can give rise to both conditions.[5]

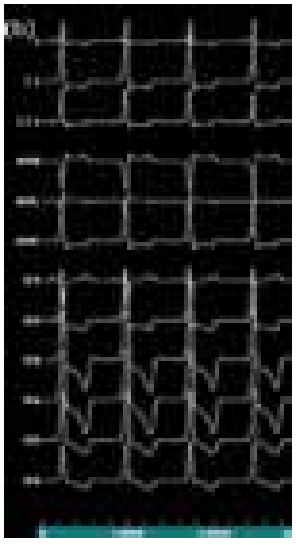

Figure 1 : ECG during sinus rhythm.

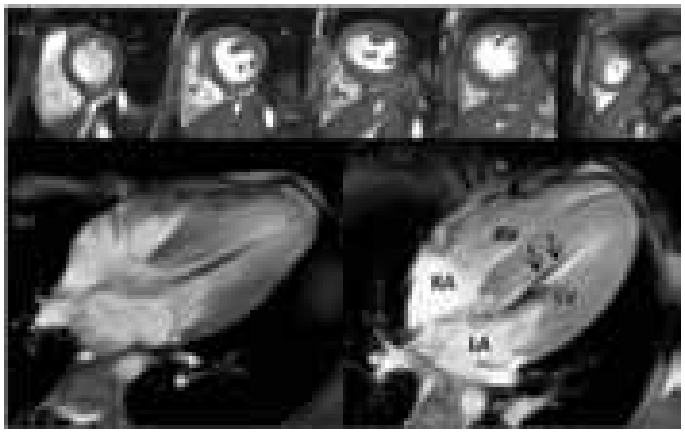

Figure 2 : MRI images in (a) short axis view during diastole from base towards apex demonstrating focal asymmetric hypertrophy of the inferior LV septal myocardium (arrow). (b) 4 chamber view in systole shows mid-cavitatory jet (black arrows) seen as black linear area in the left ventricle (LV). Right atrium – RA , Right ventricle – RV , Left atrium – LA , Left ventricle – LV.

We report a young girl with HCM with associated WPW syndrome with a left sided accessory pathway and orthodromic atrioventricular re-entrant tachycardia (AVRT). We successfully performed radiofrequency ablation to cure the WPW syndrome.

Case

A nine year old girl with WPW syndrome was referred to us for ablation. She had two episodes of sudden rapid palpitations with listlessness during which her mother had noted rapid precordial activity. The episodes terminated spontaneously. She was

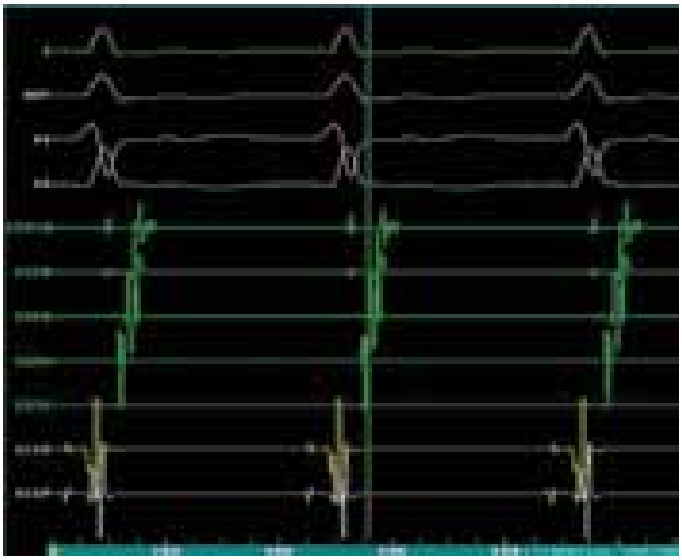

Figure 3 : Intracardiac electrogram showing orthodromic AVRT using left lateral accessory pathway. (AVRT- atrioventricular re-entrant tachycardia).

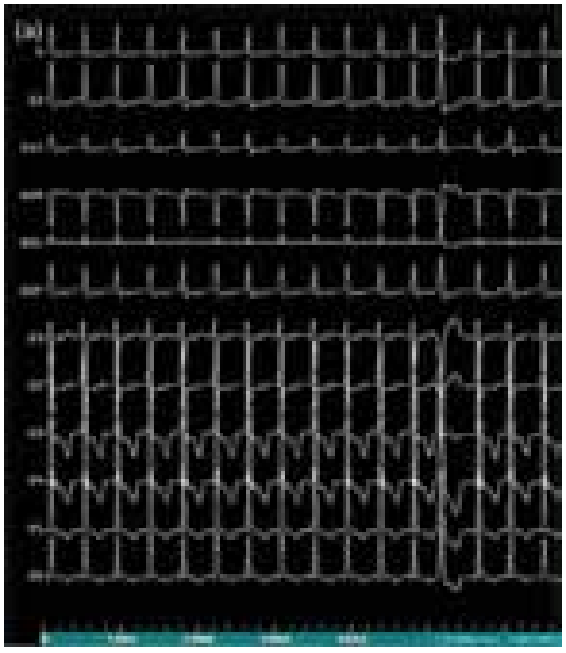

Figure 4 : ECG during AVRT

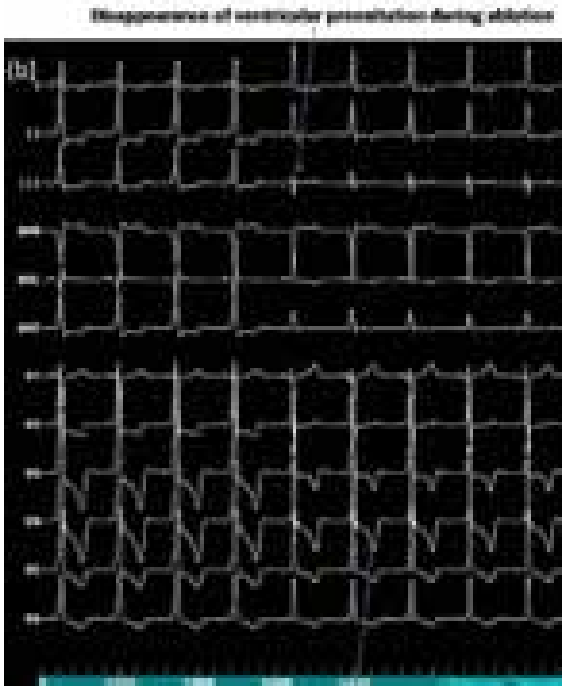

Figure 5 : During radiofrequency energy delivery, the preecxitation disappears, while the high voltages and deep T wave inversions persist.

otherwise active and had achieved the expected developmental milestones. Her family history was non-contributory and the physical examination was unremarkable. Her electrocardiogram (ECG) revealed sinus rhythm with preexcitation and very high QRS voltages suggestive of associated ventricular hypertrophy (Figure 1). Echocardiogram revealed concentric left ventricular hypertrophy with a subvalvular left ventricular outflow tract gradient of 20 mm Hg. Cardiac MRI demonstrated symmetric concentric hypertrophy of both the ventricles. (Figure 2). Electrophysiology study revealed a left lateral accessory pathway and an orthodromic re-entrant tachycardia could be induced repeatedly (Figures 3, 4) which reproduced her symptoms. Radiofrequency ablation was performed and the accessory pathway was successfully ablated (Figure 5).

This association between accessory pathways and HCM may be more than just coincidental. In some patients with HCM small muscle bundles in the hypertrophied myocardium within the atrio-ventricular junction may acquire conduction properties like an accessory pathway and lead to preexcitaion and hence, re-entrant AVRT.

Acknowledgement: Dr Alpa Bharti, M.D., consultant cardiac imaging, NM medical Center, Mumbai - interpreted the MRI pictures.

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Case Report

Digitalis Toxicity : Case Report and Review of Literature

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Introduction

Digitalis is commonly used drug for heart failure and atrial fibrillation. Though it has been used for over 200 years, its use has declined in recent times due to poor outcomes associated with its use.[1,2,3,10] Due to narrow therapeutic window, digitalis toxicity is not uncommon. It remains an important clinical toxidrome, which is diagnosed, based on the clinical presentation, EKG findings of cardiac dysrhythmias and serum digoxin level.[5-7] The incidence of digitalis toxicity varies from 0.4% in hospitalized patients to 1.1% in outpatients.[4, 8] We present a typical case of digitalis toxicity with its myriad of findings.

Case

A 73 year old Caucasian women with past medical history of diabetes mellitus, hypertension, hyperlipidemia, chronic atrial fibrillation, congestive heart failure, diastolic heart failure and COPD, presented to the hospital with nausea, vomiting, diarrhea, lethargy, decreased appetite and confusion. The patient denied any other symptoms including chest pain, syncope, headache, palpitations, dizziness, lightheadedness or any visual changes. On examination, the patient was found to be afebrile, with BP of 111/49 mm Hg, pulse of 32 bpm with oxygen saturation of 98%. The physical exam was unremarkable other than low pulse rate. The EKG on presentation (Figure 1) demonstrated regular narrow complex rhythm at 32 bpm with left axis deviation and QT interval of 544 msec.

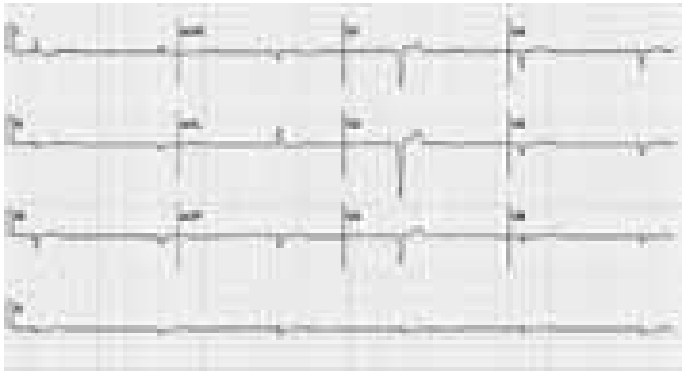

Figure 1 : EKG on presentation

Patient's home medications included furosemide, diltiazem, spironolactone, lisinopril, digoxin, carvedilol, rosuvastatin, dexlansoprazole, insulin and ipratropium-albuterol inhaler.

Basic laboratory investigation including comprehensive metabolic panel, complete blood count were all within normal limit except for patient's serum creatinine was increased to 2.2 mg/dL from the baseline of 0.8 mg/dL (2 months ago). The patient previous EKG from a month ago is shown in Fig. 2, which shows rate of 138, atrial fibrillation with rapid ventricular response, leftward axis, non-specific T wave changes and low QRS voltages in precordial leads.

Based on the clinical presentation, laboratory data and EKG findings, digitalis toxicity was suspected. It was confirmed by measured digoxin level of 6.4 ng/ml (normal range 0.5 to 0.8 ng/ml). In the meanwhile, due to persistent bradycardia down to the 20s, temporary pacemaker wire was placed (Figure 3). The patient was hydrated and was treated for digitalis toxicity with digoxin-specific fragment antigen binding (Fab) antibody fragment (known as Digibind or Digifab in US). Over next 24 hours the patient remained hemodynamically stable, serum

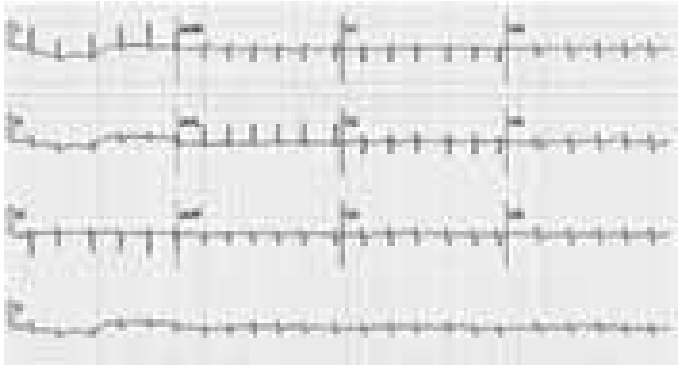

Figure 2 : Patients baseline EKG, 1-month prior

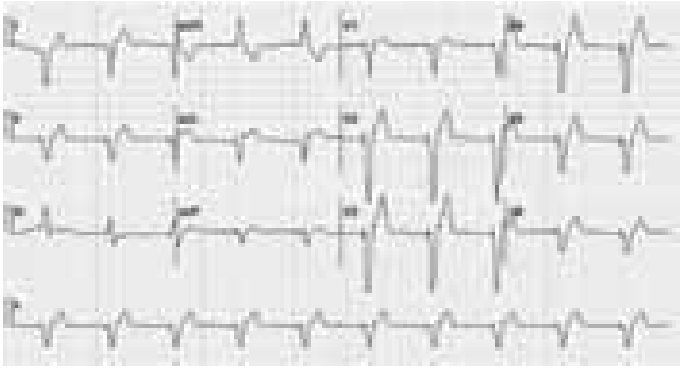

Figure 3 : Patients EKG with the temporary pacing

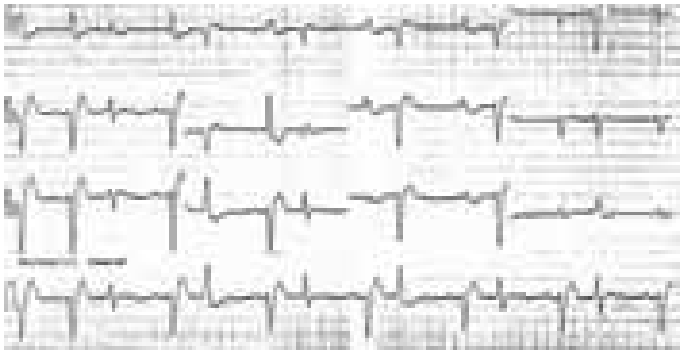

Figure 4 : "Paroxysmal" Atrial Tachycardia with block and frequent PVC's

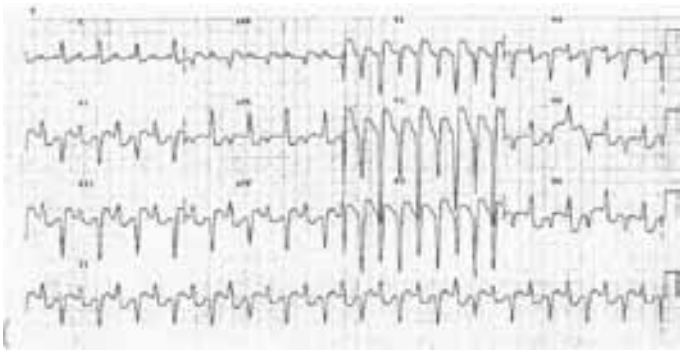

Figure 5 : Bidirectional VT

creatinine level improved and so did the native heart rate. The temporary pacemaker was removed the next day. The patient's rest of the hospital stay was uneventful and was subsequently discharged in a few days.

Discussion

Digoxin has been used commonly in heart failure patients due to its positive ionotropic effect in heart failure and in atrial fibrillation with rapid ventricular response for AV nodal blocking action by increased vagotonia.[1-3,6,10] Its use has decreased over the past decade because of potential for toxicity acutely and association with increased mortality in the long term.[4,6,7] Digitalis toxicity can vary from acute toxicity due to acute high dose ingestion or drug interactions and chronic toxicity can result from declining renal or hepatic function or even drug interactions.[6,7] In our patient the toxicity related to the renal failure, which resulted in decreased clearance of digoxin, which is primarily cleared by the kidneys.

Typical symptoms of digitalis toxicity vary from neurological symptoms of lethargy, confusion and vision changes; gastrointestinal symptoms including nausea, vomiting, poor appetite; cardiac manifestation including but not limited to various dysrhythmias, with the cardiac manifestations being the primary concern due to associated mortality.[5,6] Various types of arrhythmias associated with digitalis toxicity include premature atrial contractions, premature ventricular contraction, sinus brady or tachy arrhythmias, ectopic atrial tachycardia with block, also known as "paroxysmal" atrial tachycardia (Figure 4),[12] AV nodal blocks, junctional arrhythmias, ventricular arrhythmias including ventricular fibrillation, ventricular tachycardia and bidirectional ventricular tachycardia (Figure 5).[12,5,7] Of note is that digitalis toxicity can rarely cause atrial fibrillation, but is most commonly noted in the patient with underlying atrial fibrillation treated with digoxin for rate control. AV blocks are more commonly seen in patients with atrial fibrillation or atrial flutter.[6]

Serum Digoxin Concentration can be used to evaluating and confirming prevent digitalis toxicity. Life threatening arrhythmias occur usually above the digoxin level of 2.0 ng/lm (2.6 mmol/L).[7] It is critical to remember that life-threatening arrhythmias can even occur in patients with lower concentration especially with co-existent electrolyte abnormalities such as hyperkalemia, hypomagnesaemia and hyperkalemia. It is to be noted that hyperkalemia is related to higher mortality.[5,6,9]

Treatment of digitalis toxicity include continuous cardiac monitoring, maintaining electrolyte balance, GI decontamination with activated charcoal (in case of acute ingestion), use of digibind, supportive management and pacing if need be.[4,6,9] Use of Digibind has shown great results in treatment of acute as well as chronic toxicity.[4,5,9] Indications for use of digibind include severe CNS side effects like altered mental status, renal failure, life threatening arrhythmias, end organ damage and significant hyperkalemia.[6,9] The does of digibind can be calculated based on serum digoxin level or patients dose and body weight.[5,6,7] Temporary pacing should be considered in patients who are hemodynamically unstable with the dysrhythmia or who have very high serum concentration along with symptoms and life threatening arrhythmias like complete heart block, as in our case.[5]

Conclusion

With the above case we want to reemphasize the importance of detecting digitalis toxicity in a patient who is on digoxin based on the clinical presentation and EKG findings in addition to serum digoxin level. Serum digoxin level does not correlate to toxicity since older patient and those with other co-morbidities may show signs and symptoms of toxicity while in "therapeutic range" while others may not demonstrate toxicity at higher digoxin levels.[6,8,10] Regardless, we want to emphasize the narrow therapeutic index for digoxin should be kept in mind, with regular drug level monitoring and lower dose should be used in older patient, especially with other cardiac comorbidities.[4,5,10,11]

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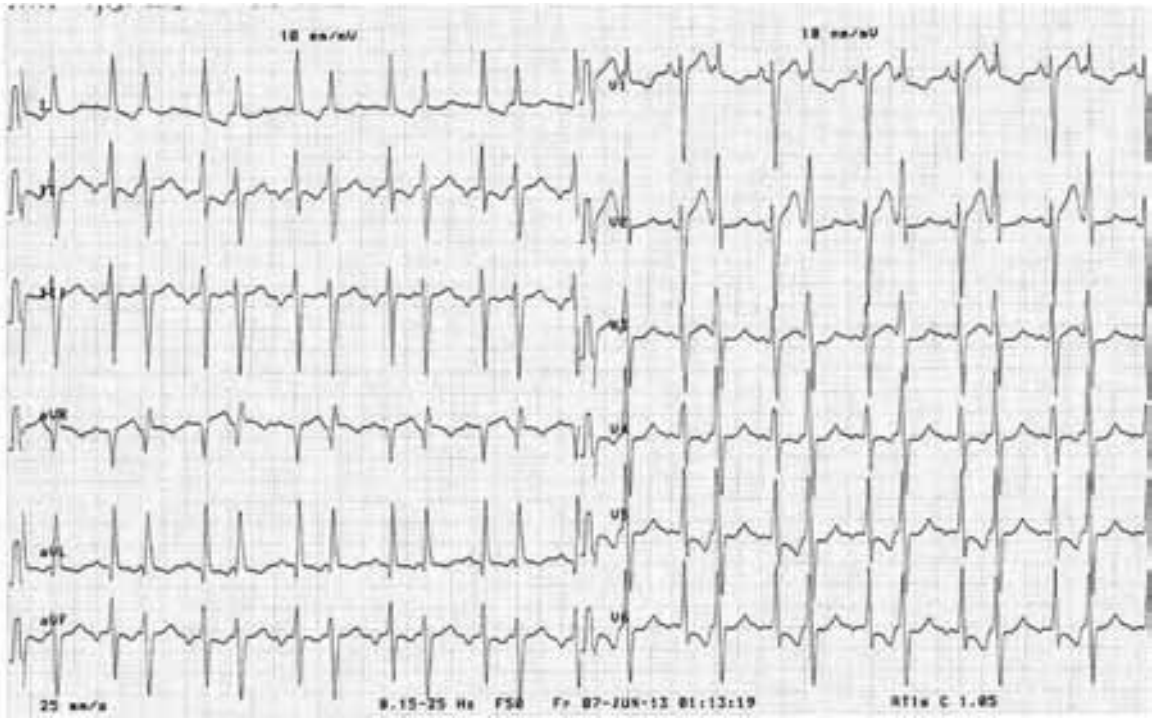
ECG Quiz

Yash Lokhandwala[*]
[*]Arrhythmia Associates

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

ECG - 1

48 yr old man. 10 yr history of increasing palpitations/SOB/occasional edema feet. Fair RV and LV EF on echo



The ECG shows

a. Atrial bigeminy

b. Ventricular bigeminy

c. Ectopic atrial rhythm

d. A and C

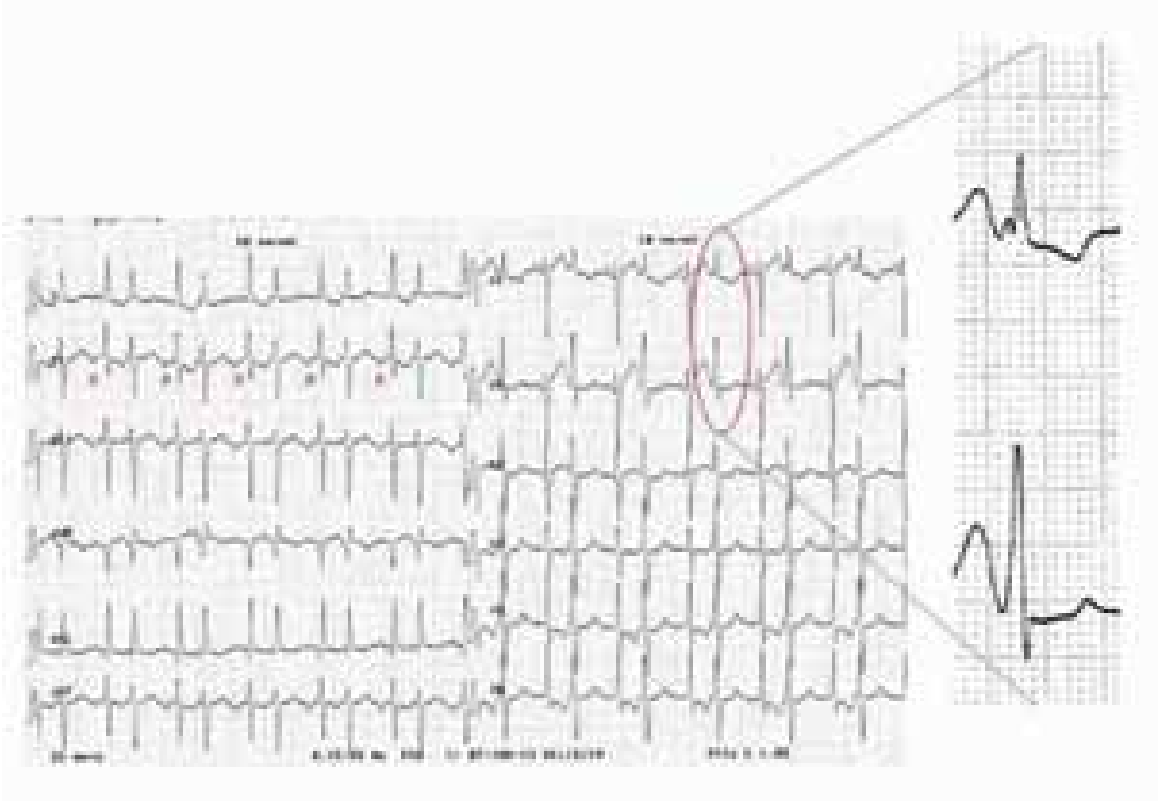
e. B and C

f. None of the above

For correct answer see overleaf

ECG - 1

The correct answer is 'e' – Ventricular bigeminy and ectopic atrial rhythm.

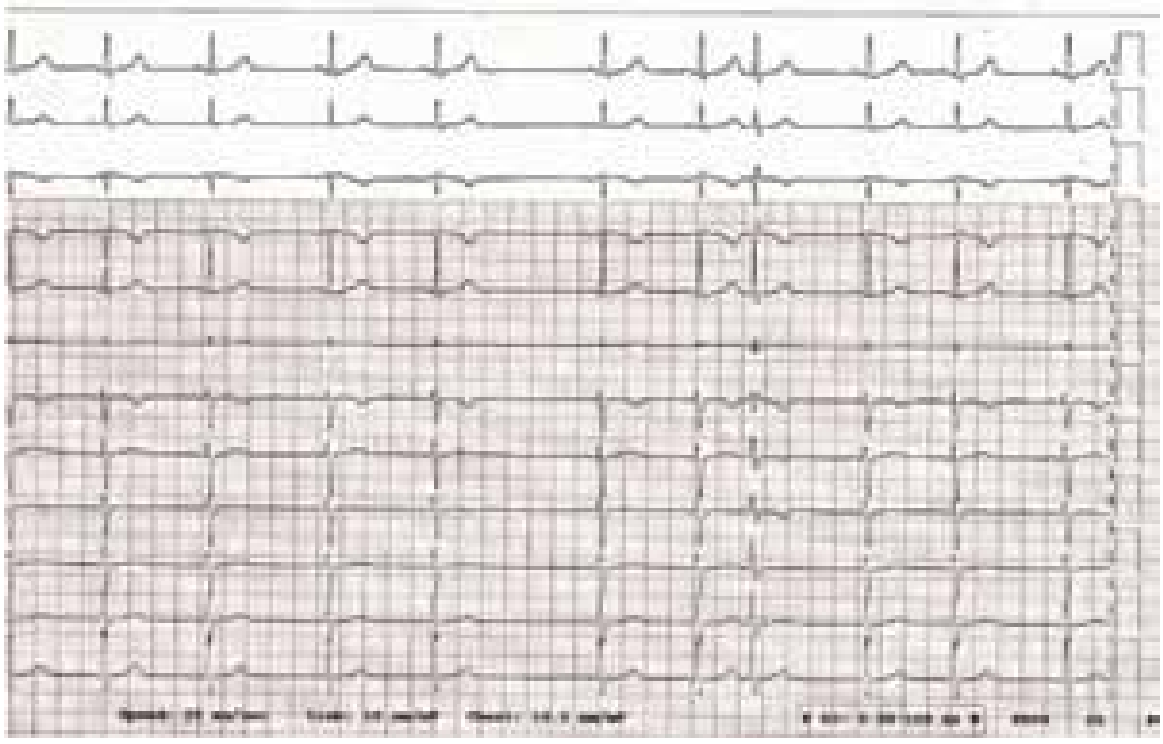


There is a bigeminal rhythm. There is an inverted P wave in lead II which precedes every alternate QRS complex (P). Thus, there is an underlying ectopic low atrial rhythm. The QRS complex during this rhythm is normal with the QRS axis being near horizontal.

There is downsloping ST depression in the inferior and lateral leads.

The premature QRS complexes show a RBBB morphology with QRS of 120 ms. There is a prominent S wave in V6 with R/S ratio of 1. The secondary ST changes eliminate the ST depression in inferior and lateral leads. Thus, these are clearly PVCs. The QRS not being very wide indicates that the origin is close to the conduction system in the interventricular septum.

ECG - 2



The cause of pause is:

a. Sinus pause

b. SA block

c. Blocked PAC

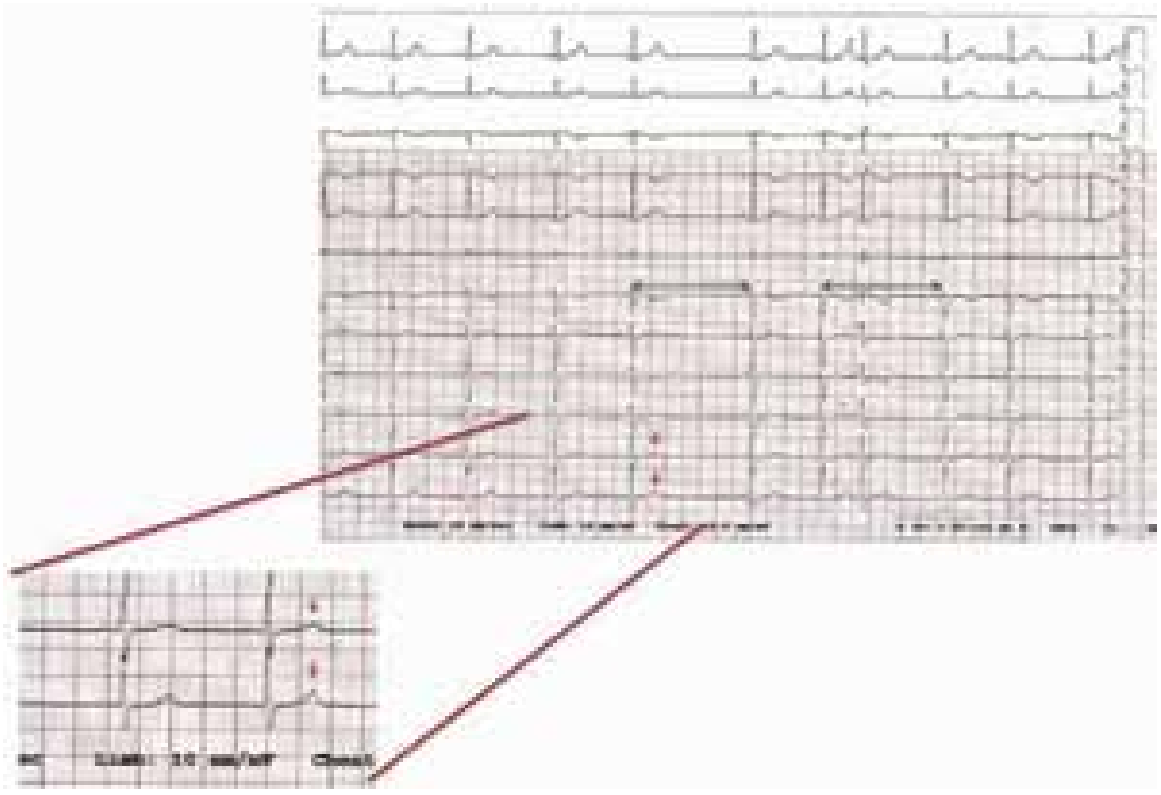
d. Sinus arrhythmia

For correct answer see overleaf

ECG - 2

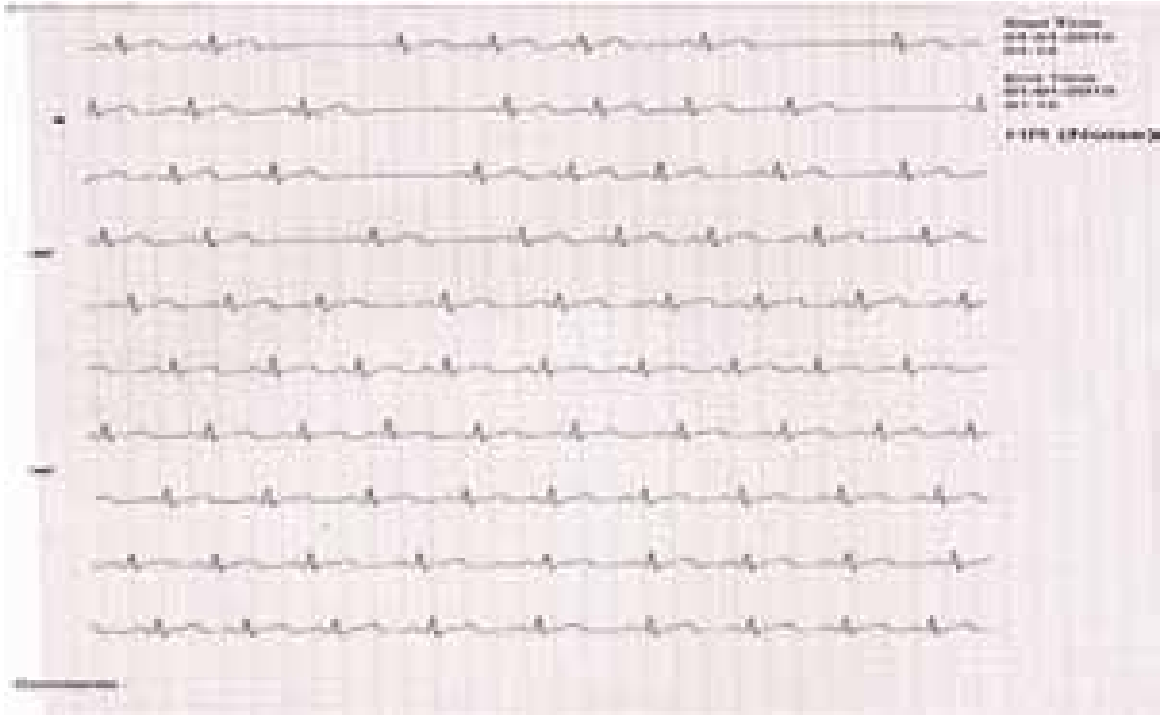
The correct answer is 'c' – Blocked PAC

One obvious premature complex is seen preceded by a 'peaked' T wave in lead I (*) and a notched T wave in V4. These are clearly indicative of a PAC. The pause is exactly equal to the compensatory pause encompassing the PAC. Close scrutiny of the T wave in V5-V6 preceding this pause shows a peaking which is not seen in the T waves before this. This confirms a blocked PAC which comes very early and hence finds the AV conduction refractory. Such pauses are benign and need no treatment.



ECG - 3

14 yr old girl. History of syncope



The pauses are due to:

a. Sinus arrhythmia

b. SA block

c. Sinus pauses

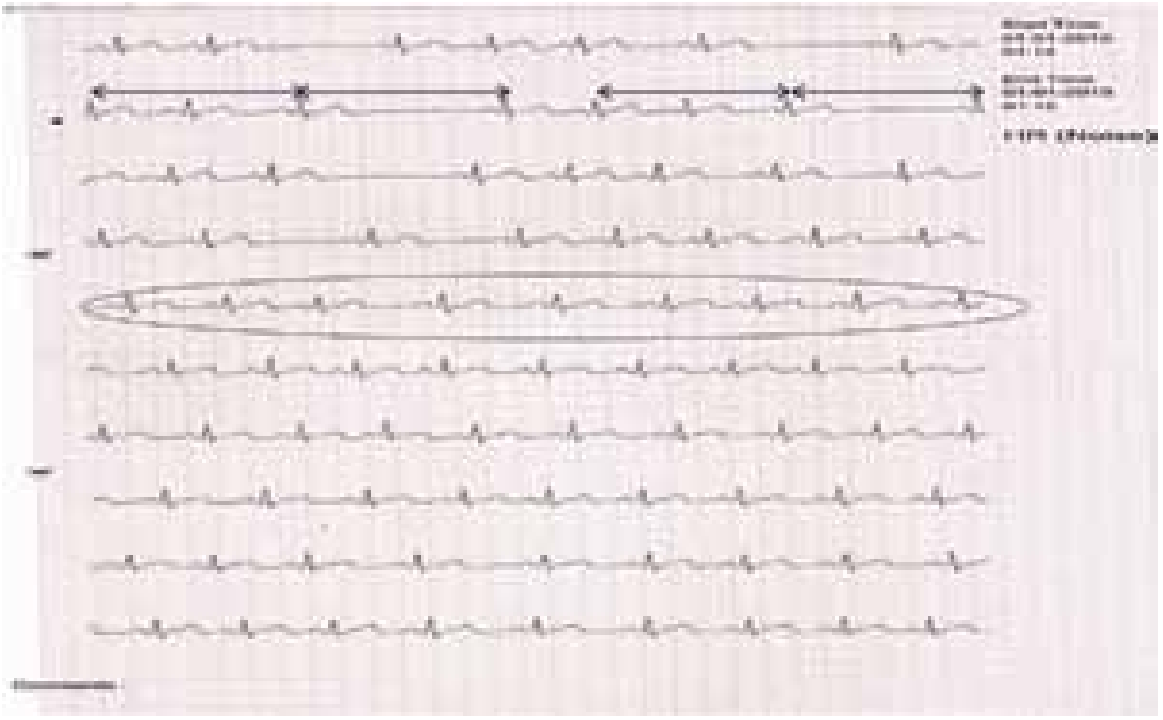
d. All of the above

For correct answer see overleaf

ECG - 3

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

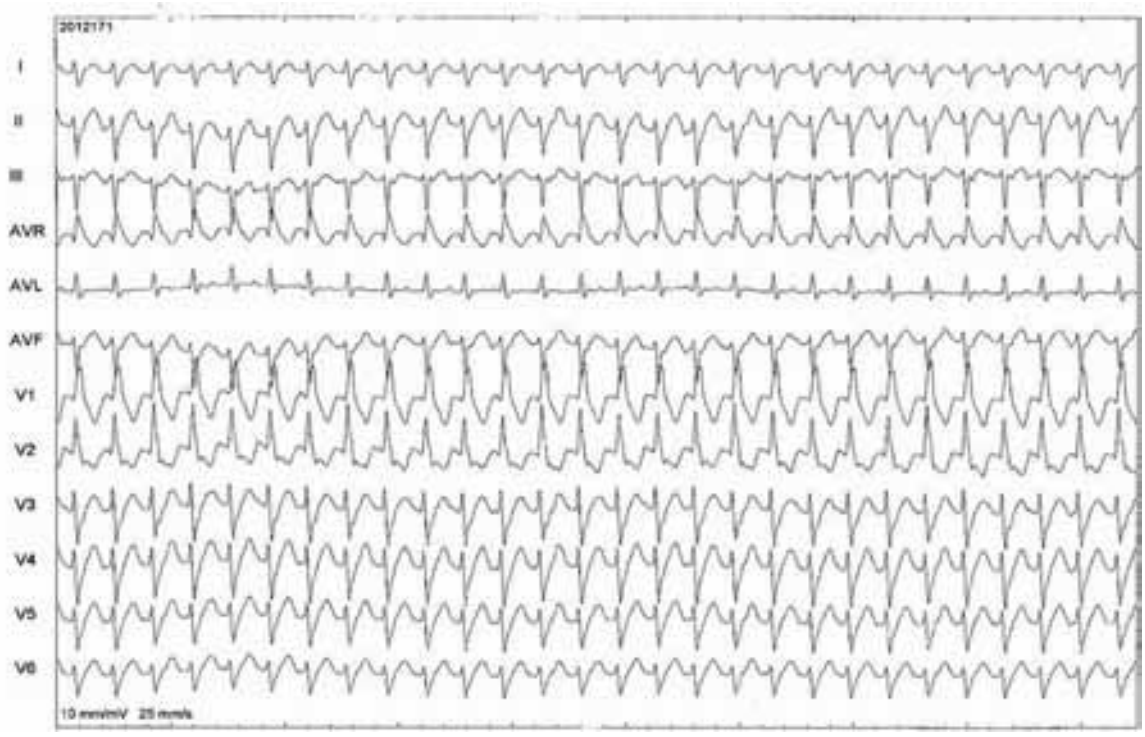
This is a continuous single monitor strip from an event recorder. Multiple pauses are seen which occurs abruptly.



Only two such pauses are an exact multiple of the underlying cycle length indicating 2:1 SA block. The others are sinus pauses. There is also a gradual variation in the PP intervals indicative of sinus arrhythmia (⬭).

ECG - 4

17 yr old girl. Structurally normal heart



The ECG shows:

a. SVT with RBBB

b. VT with VA dissociation

c. VT without VA dissociation

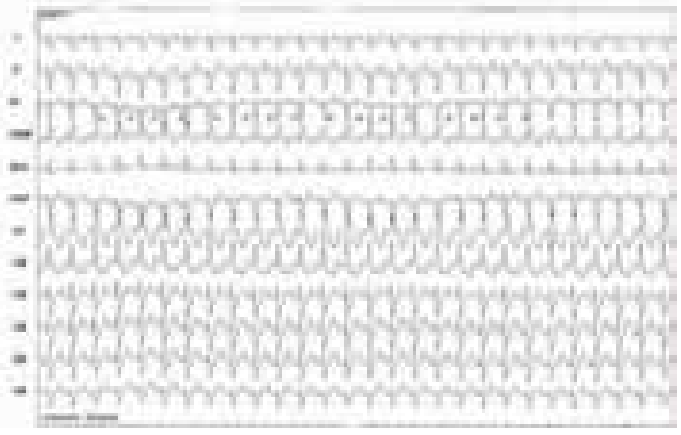
For correct answer see overleaf

ECG - 4

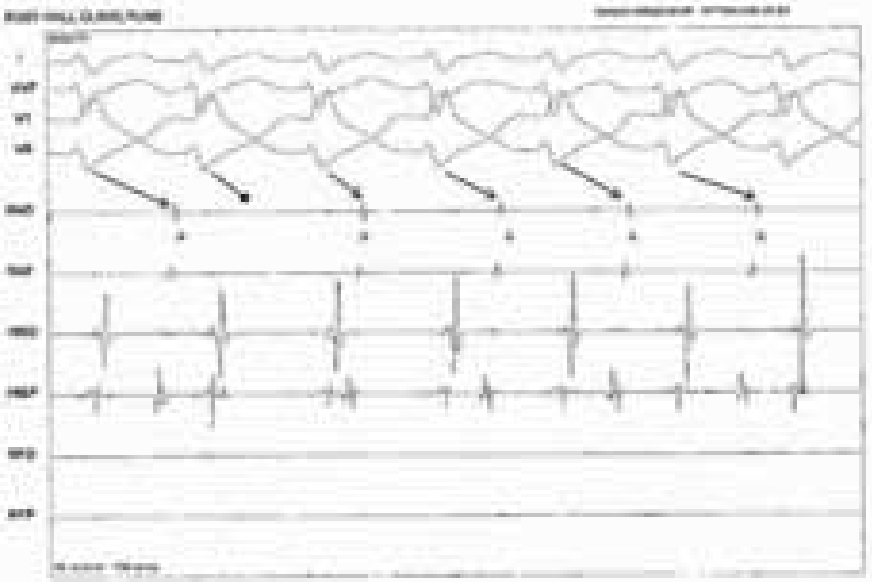
The correct answer is 'c' – VT without VA dissociation.

There is a regular wide QRS tachycardia with an RBBB-like pattern. There are deep S waves in V6 with R/S ratio less than 1, which is highly unusual for RBBB which favours the diagnosis of VT without VA dissociation. Also, the QRS axis is -90° (extreme LAD) which is discordant for RBBB and again favours VT. This is idiopathic "fascicular" VT.

On careful inspection, inverted P waves are seen in lead III suggesting that these are retrograde P waves. The RP interval keeps increasing until the P wave is intermittently absent. This indicates a VA Wenckebach.



The figure below shows the intracradiac ECG during this period confirming the VA Wenckebach. 'A' waves correspond to 'P' waves, as recorded in right atrium. The term VA dissociation is used when the atria and ventricle are activated by separate pacemakers.



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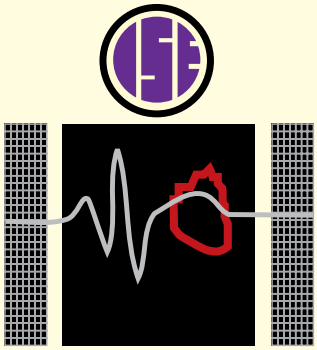

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