C O N T E N T S

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Editorial

Dear Colleagues,

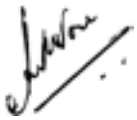
Wishing you a joyful and academically enriching 2006. The ISECON 2006 is around the corner, and a wide-ranging scientific program has been prepared by the scientific committee. We wish them all the best and are sure that the conference will be a resounding success.

After last year's ISECON at Bangalore, the mid-term conference was held at Varanasi. This year, the mid-term conference will be held at Aligarh in September. The bi-annual meetings of ISECON are able to provide valuable updates and training in ECG and related subjects in different parts of the country.

The ISE has also continued to conduct ECG courses in different medical institutes. Such courses were held in the Central Railway Hospital, Byculla, Mumbai and at Mysore. The first examination evaluating competence in ECG reading was conducted by the ISE in 2005 in Mumbai. This evaluation was conducted in accordance with the pattern followed by other international bodies which conduct similar examinations. Similar examinations are planned on a regular bi-annual basis in the future.

The current issue has 2 articles taken from the web-based Indian Journal of Pacing and Electrophysiology. These are followed by an ECG Quiz and a couple of interesting ECG vignettes. Happy reading!

Yash Lokhandwala
Editor



Amit Vora
Editor

From Hon. Secretary's Desk

Dear Members,

I am proud to mention that the Indian Society of Electrocardiology is growing in numbers as far as members are concerned and in the academic programs too.

Dr. Ravi Kishore and their team organized ISECON-2005 at Bangalore from 2nd and 3rd April 2005. It was a grand success.

Indian Society of Electrocardiology organized the dream - "ECG Learning Course" for postgraduate students on 27th August, 2005, which was attended by approximately 100 students. Successful candidates were awarded the Certificate of Competence for ECG reading.

Then, Mid-Term CME Course (ECG CON 2005) was conducted on 29th and 30th October 2005 at Varanasi by Dr. B.V. Agrawal, Dr. Vineet Agrawal and their team successfully.

Indian Society of Electrocardiology organized Euro-India Satellite Symposium at Mumbai on 5th January, 2006, which was very well attended.

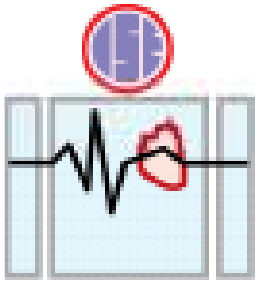
ISECON 2006 has arrived and Dr. K.P. Misra and Dr. Ulhas Pandurangi are very enthusiastic in organizing ISECON 2006 and planning to make it a huge success.

I look forward to see you all at the above meet, which will be a real treat.

My sincere thanks to Dr. Yash Lokhandwala, Dr. Amit Vora and the Editorial Team for bringing out the ISE Journal - 2006.

Long live Indian Society of Electrocardiology

Dr S B Gupta
Hon. Secretary
Indian Society of Electrodardiology



Long live

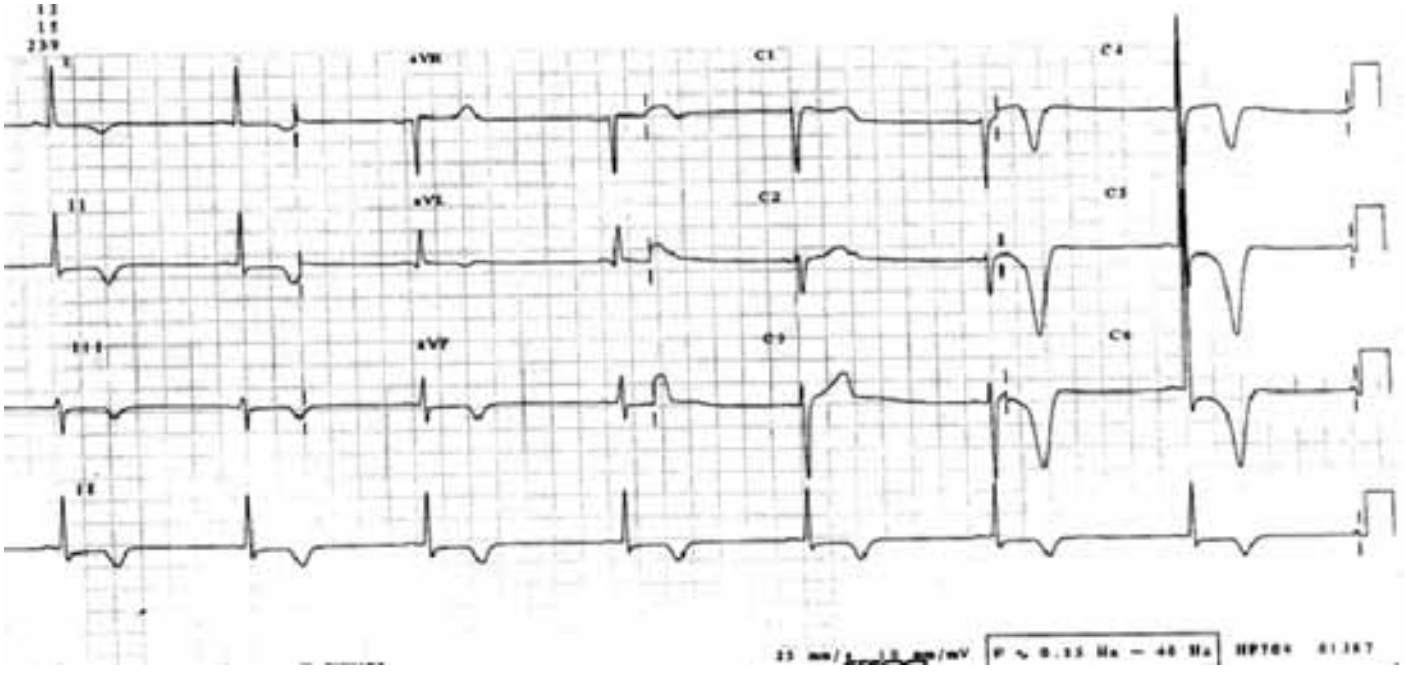
Indian Society of Electrocardiology

ECG Quiz

ISECON 2005, Bangalore

ECG - 1

49 yr old man. Recurrent chest discomfort, only on exertion.
ECG at rest.



1. This ECG is suggestive of:

a. Coronary ischemia
b. Hypertrophic cardiomyopathy
c. ↑ ICT
d. Don't know

For correct answer see overleaf

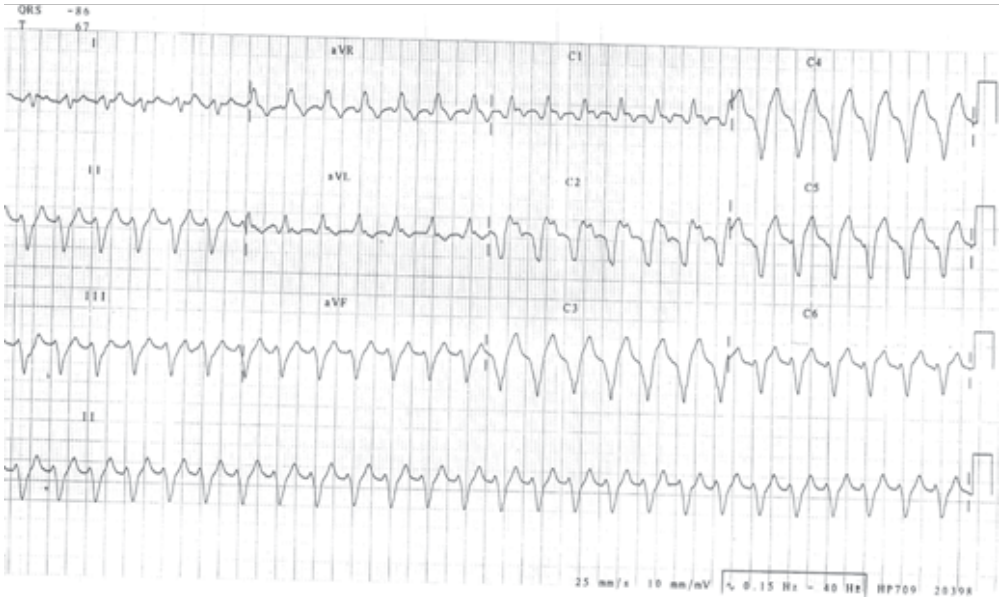
ECG - 1

The correct answer is "b" - Hypertrophic cardiomyopathy

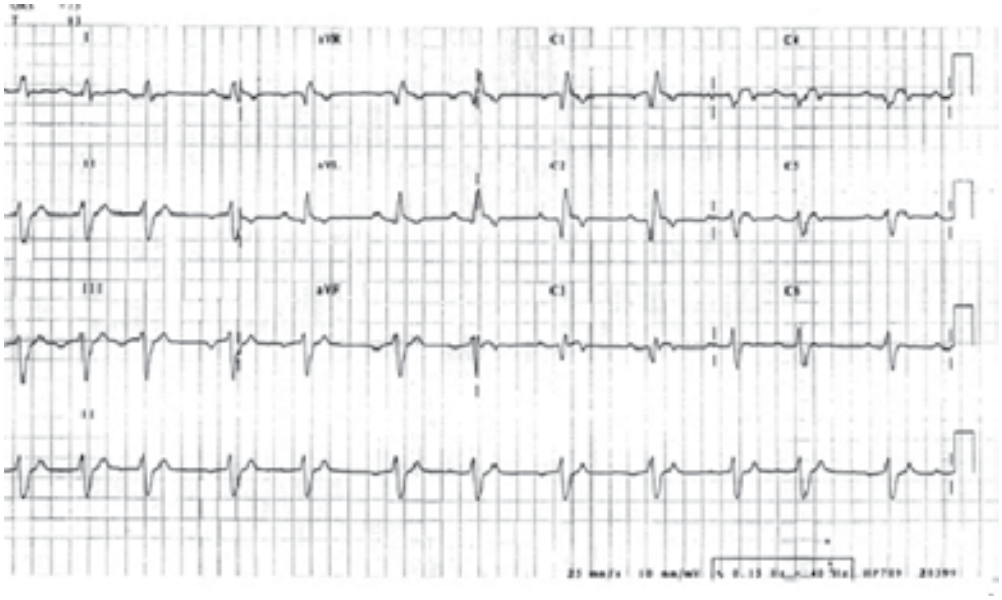
The 12-lead ECG shows deep T wave inversions in leads V4- V6, I, aVL, II, III and aVF. Coronary ischemia is unlikely in view of the T wave inversions present even at rest. Also, there is evidence of left ventricular hypertrophy by voltage criteria. The history of exertional chest pain does not support raised ICT. LVH with global T wave inversions at rest suggest hypertrophic cardiomyopathy and these patients often complain of exertional chest pain in view of increased myocardial oxygen demand during exercise.

ECG - 2

2 65 yr-old, past MI. Admitted with palpitations
Post conversion..



2a



2. The tachycardia ECG is:

a. SVT with RBBB
b. Sinus tachycardia
c. Atrial flutter
d. VT

For correct answer see overleaf

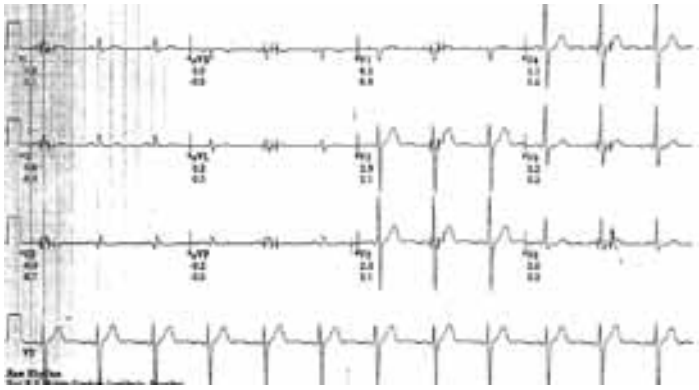
ECG - 2

The correct answer is "d" - VT

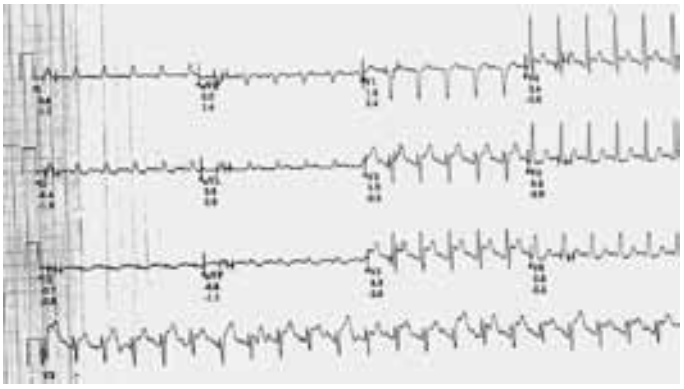
This ECG (figure 2) shows a wide QRS tachycardia. Figure 2a, the post conversion ECG shows healed anterior wall myocardial infarction (AWMI) with RBBB. The tachycardia ECG appears similar to the sinus rhythm complexes at an initial glance, however a detailed analysis reveal i) qR pattern in V1-V2; ii) QS pattern from V3- V6; iii) QRS-12 lead morphology not a perfect 12-12 lead match between the tachycardia & sinus rhythm. All of the above suggest the tachycardia to be a VT as opposed to SVT with RBBB. A wide QRS tachycardia in the setting of old myocardial infarction should be presumed to be VT unless proved otherwise.

ECG - 3

3a. Stress Test. Baseline



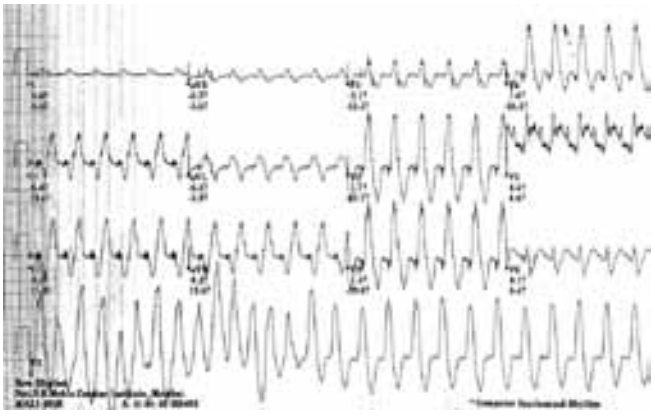
3b. Beginning of stage 2, Bruce protocol



Would you stop the stress test?

a. Yes b. No

3c. Early recovery



3. What critical lesion is likely?

a. Left main/proximal LAD
b. Proximal RCA
c. Circumflex
d. None of the above

For correct answer see overleaf

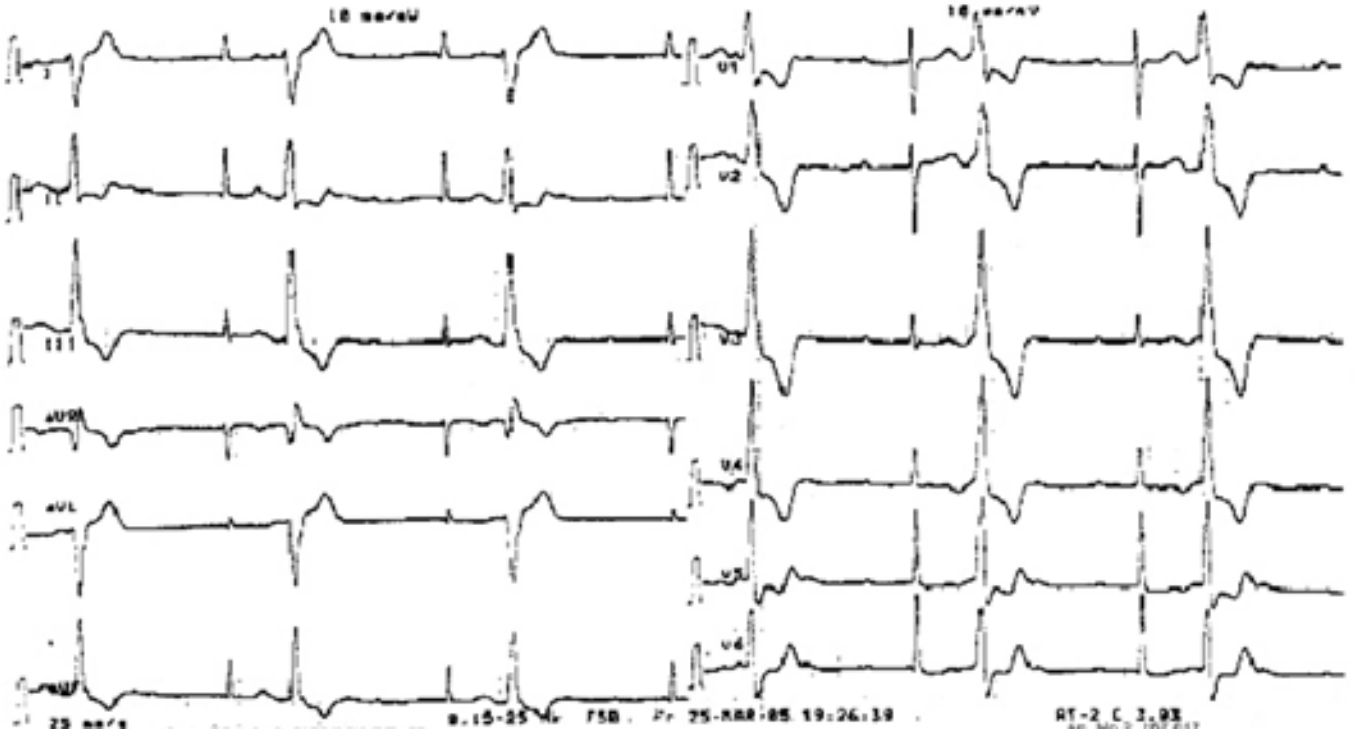
ECG - 3

The correct answer is "a" - Left main/proximal LAD

The baseline ECG before starting the stress test is normal. After initiating the stress test, the anterior leads V2- V3 show mild ST elevation and some widening of the QRS complexes. The stress test should be stopped as these changes signify severe coronary ischemia. During recovery phase there is a wide QRS polymorphic ventricular tachycardia suggestive of ischemia induced VT. Later in recovery RBBB with left axis deviation is seen; ischemic RBBB is only seen when the proximal LAD is involved. ST depression, a common finding during a positive stress test is not localizing, however, ST elevation is localizing. ST elevation in anterior leads suggests LAD ischemia. Thus, in this case, proximal LAD or left main lesion is most likely.

ECG - 4

18 year old girl, asymptomatic



4. This ECG shows:

a. First degree AV block
b. Second degree AV block
c. Complete AV block
d. Don't know

For correct answer see overleaf

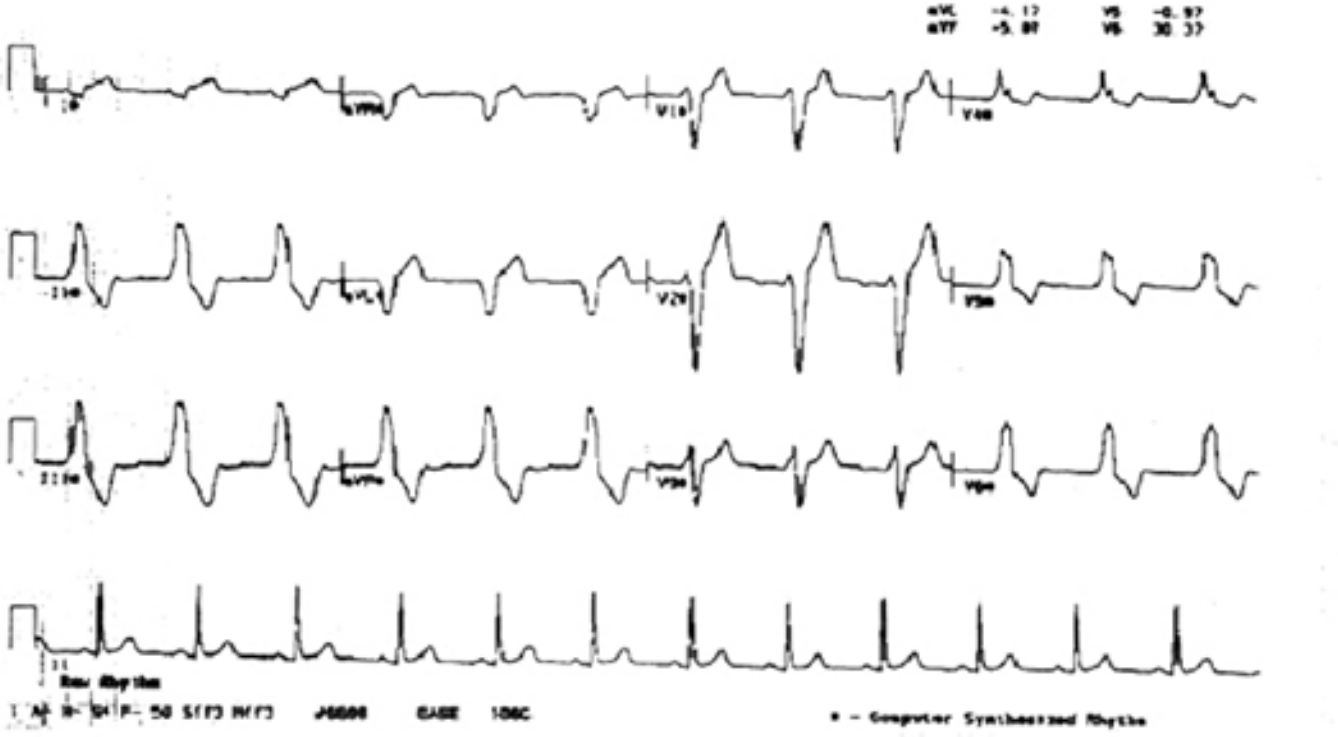
ECG - 4

The correct answer is "c" - Complete AV block

The 12 lead ECG shows a bradycardia with narrow QRS complexes and ventricular bigeminy. However, there is no definite relationship between P waves and the narrow QRS complexes. This is therefore complete AV block. The ventricular ectopics have a constant coupling interval, are monomorphic and also do not seem to have any relation with the P waves. Ventricular ectopy often gets a chance to manifest during bradycardia.

ECG - 5

About to start Stress test…



5. This ECG is:

a. Slow VT
b. Rate related BBB
c. WPW pattern
d. Normal

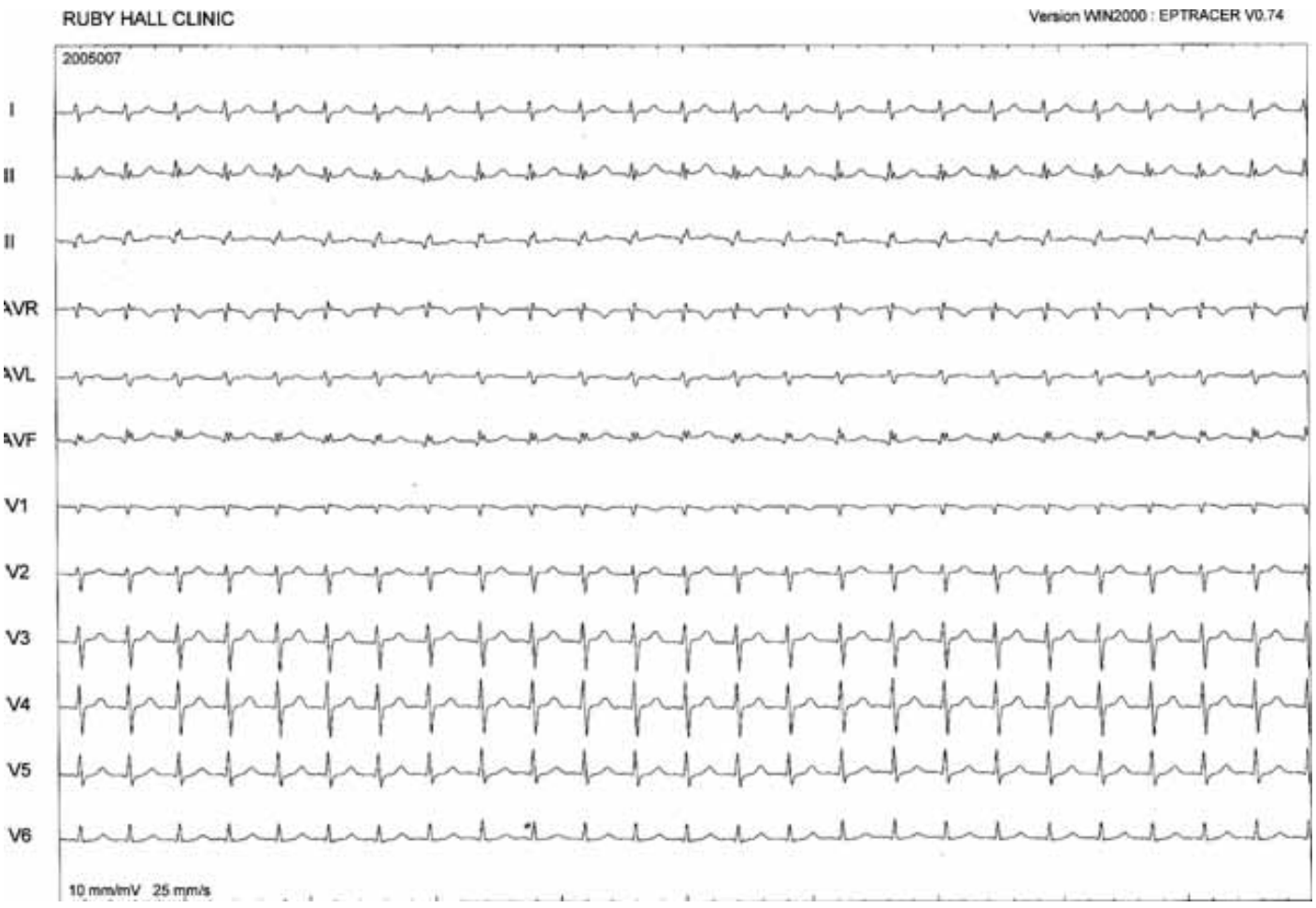
For correct answer see overleaf

ECG - 5

The correct answer is "d" - Normal

The 12-lead ECG is taken during a stress test and computer synthesized i.e. averaged by the computer except for the raw rhythm of long lead II at the bottom of the ECG. The wide QRS computer generated rhythm is therefore deceptive. The raw ECG of lead II shows a normal sinus rhythm with narrow complexes. The computer has likely picked up preceeding ventricular ectopics and averaged them to display a 12 lead ECG with wide QRS complexes giving a false impression, although the patient's true ECG is normal. Thus, it is very important to evaluate and observe the raw ECG and not to rely on the computer averaged ECG, which can often be misleading.

ECG - 6



6. This tachycardia is AVNRT because:

a. Its most common SVT
b. P waves not discretely identifiable
c. Pseudo Q waves
d. All of the above

For correct answer see overleaf

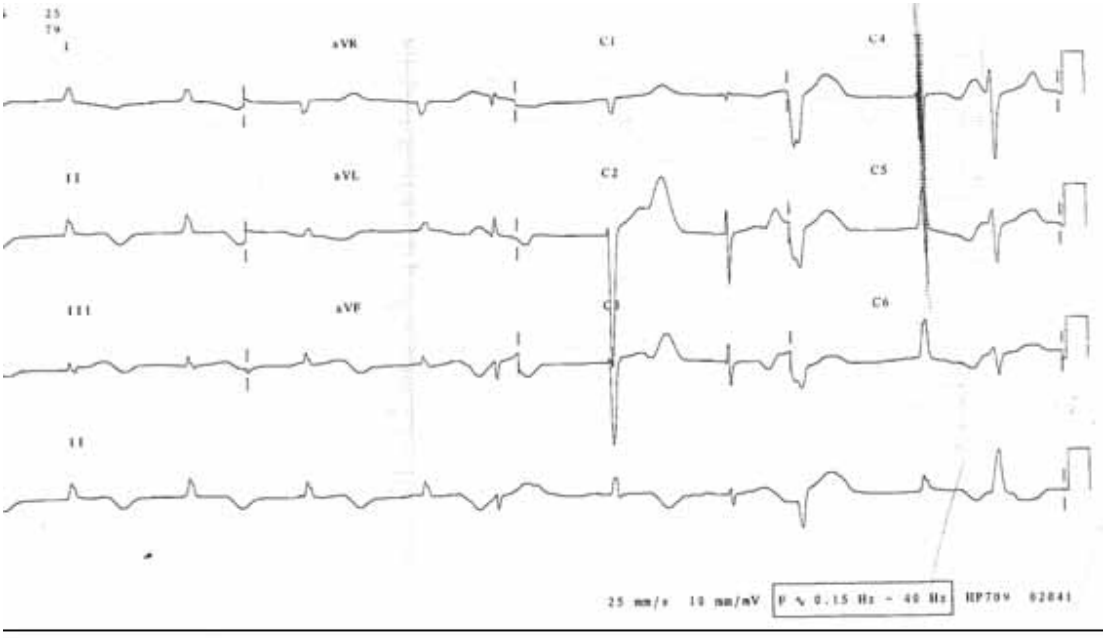
ECG - 6

The correct answer is "d" - All of the above

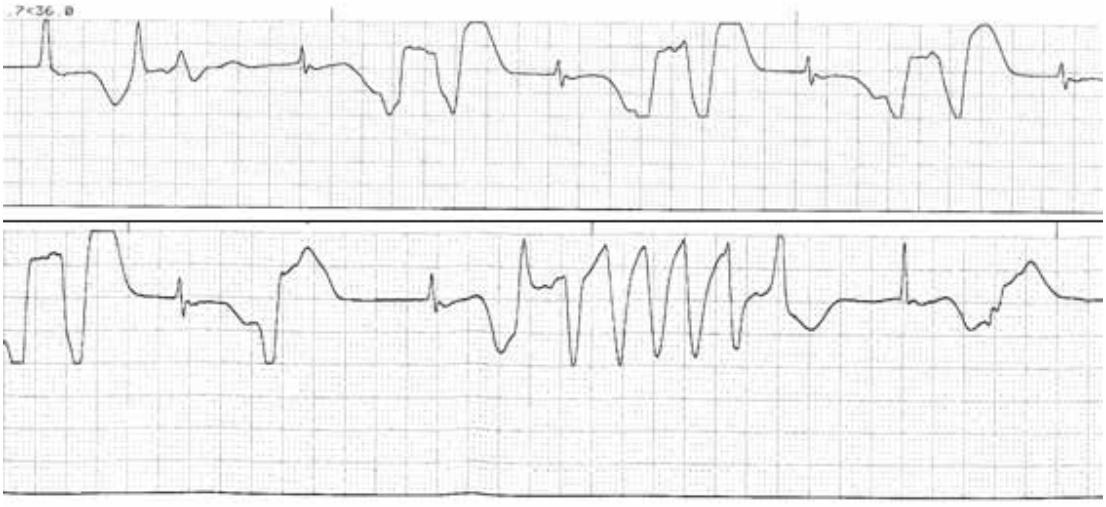
The 12 lead ECG shows a narrow QRS, regular tachycardia with no obvious P waves. This is likely to be AVNRT which is also the most common SVT. It is common for P waves to appear within the terminal portion of the QRS giving rise to a pseudo S wave during AVNRT. However, sometimes the P waves appear within the initial part of the QRS complexes thereby giving rise to pseudo Q waves, as evident in the leads II, III and aVF in the above ECG.

ECG - 7

50 yr old lady. Re-redo MVR. Post-VF



After IV amiodarone bolus…



7. Best Rx option is:

a. Lignocaine
b. ↑ dose of amiodarone
c. Beta-blockers
d. Pacing / isoprenaline

For correct answer see overleaf

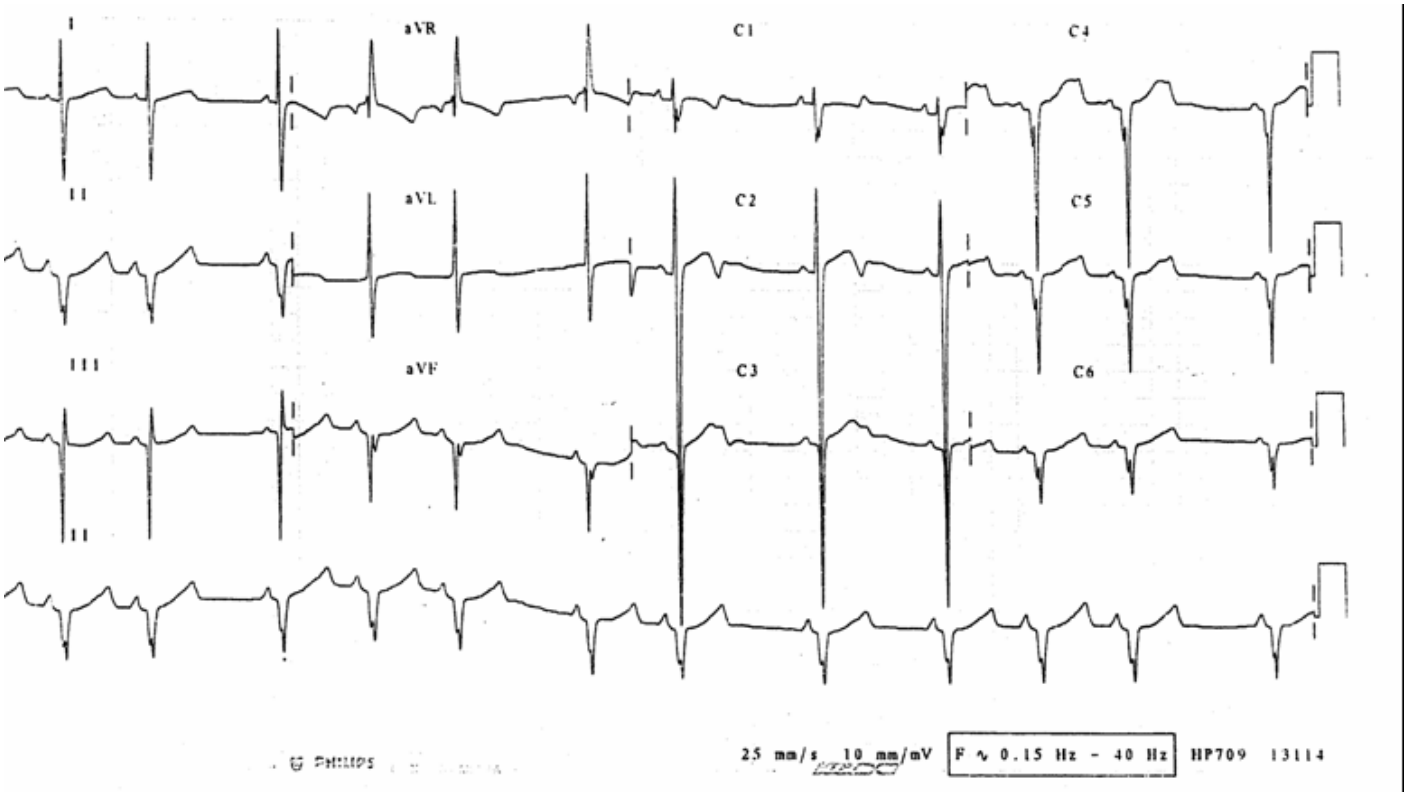
ECG - 7

The correct answer is "d" - Pacing/ Isoprenaline

The top rhythm strip shows bradycardia with a prolonged QT interval and PVCs. The bottom strip shows ill-sustained polymorphic VT, after a late coupled PVC in the latter part of the T wave (with long QT interval). Amiodarone is contra-indicated in view of long QT and polymorphic VT. The long QT is likely acquired and bradycardia dependant and therefore betablockers may worsen the VT. The efficacy of lignocaine is doubtful. Pacing and /or iosprenaline infusion, known to shorten the QT interval, is the most logical step to prevent further polymorphic VT. Till such time the underlying cause for long QT is corrected, maintaining adequate K+ / Mg++ would be essential.

ECG - 8

4 yr-old-girl, HCM with small VSD….



8. This ECG shows:

a. AV Wenckebach
b. Sinus arrhythmia
c. Sinus arrests
d. Both a & b

For correct answer see overleaf

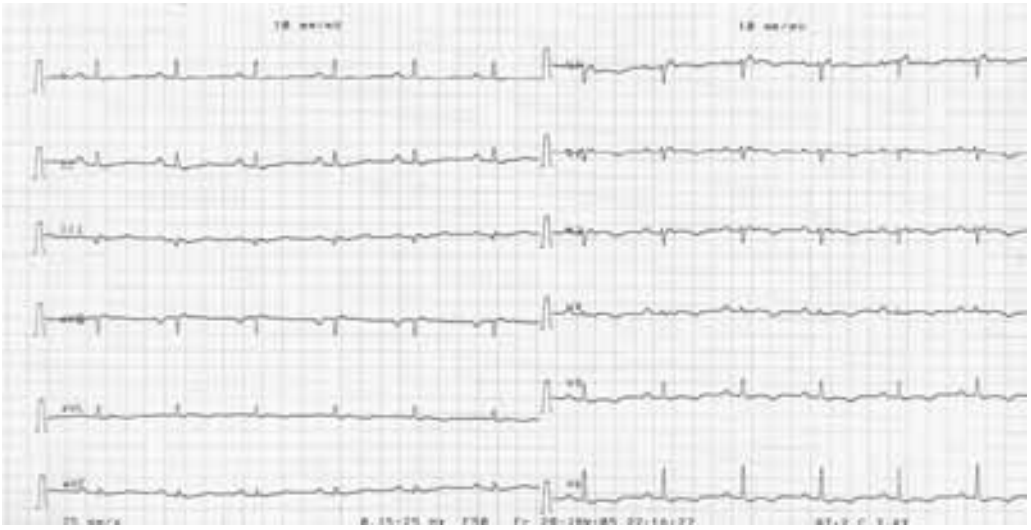
ECG - 8

The correct answer is "c" - Sinus arrhythmia

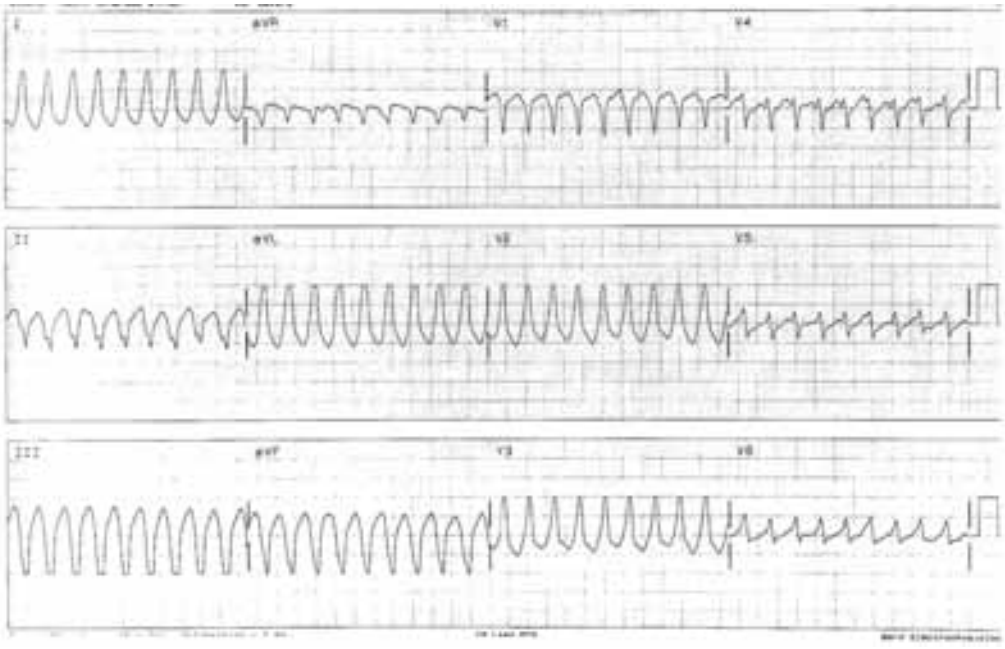
This 12 lead ECG shows irregular R-R interval with a P wave preceding every QRS complex. Sinus arrest is unlikely as the pauses are not long. The PR interval in all the complexes is constant and normal suggesting a normal AV conduction, thereby ruling out AV Wenckebach. The irregular rhythm is more likely to be sinus arrhythmia which is physiological at this young age.

ECG - 9

9 37 year old lady. Recurrent paroxysmal palpitations



9a



9. The likely cause of palpitations is:

a. Anxiety neurosis (Sinus tachycardia)
b. SVT
c. VT
d. Don't know

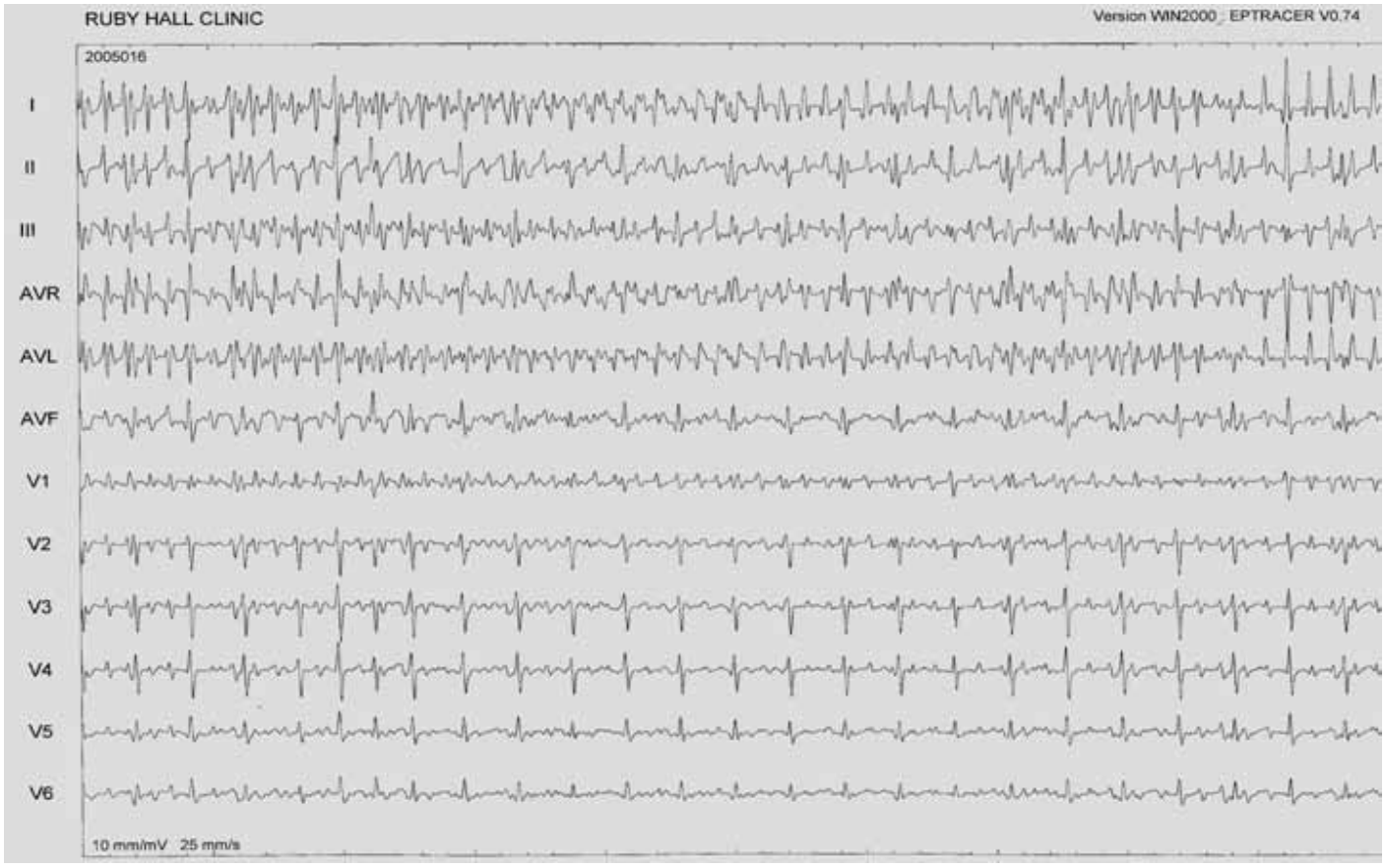
For correct answer see overleaf

ECG - 9

The correct answer is "c" - VT

The 12 lead ECG shows 'epsilon' like wave in lead V1 just at the end of QRS complex. Also, there is poor progression of R wave with T wave inversions in the chest leads. These findings suggest a strong possibility of arrhythmogenic right ventricular cardiomyopathy (ARVC). And so the cause of recurrent palpitations in this patient could be VT (fig. 9a) arising from the right ventricle.

ECG - 10



10. The ECG shows:

a. Atrial flutter
b. Atrial fibrillation
c. VT
d. None of the above

For correct answer see overleaf

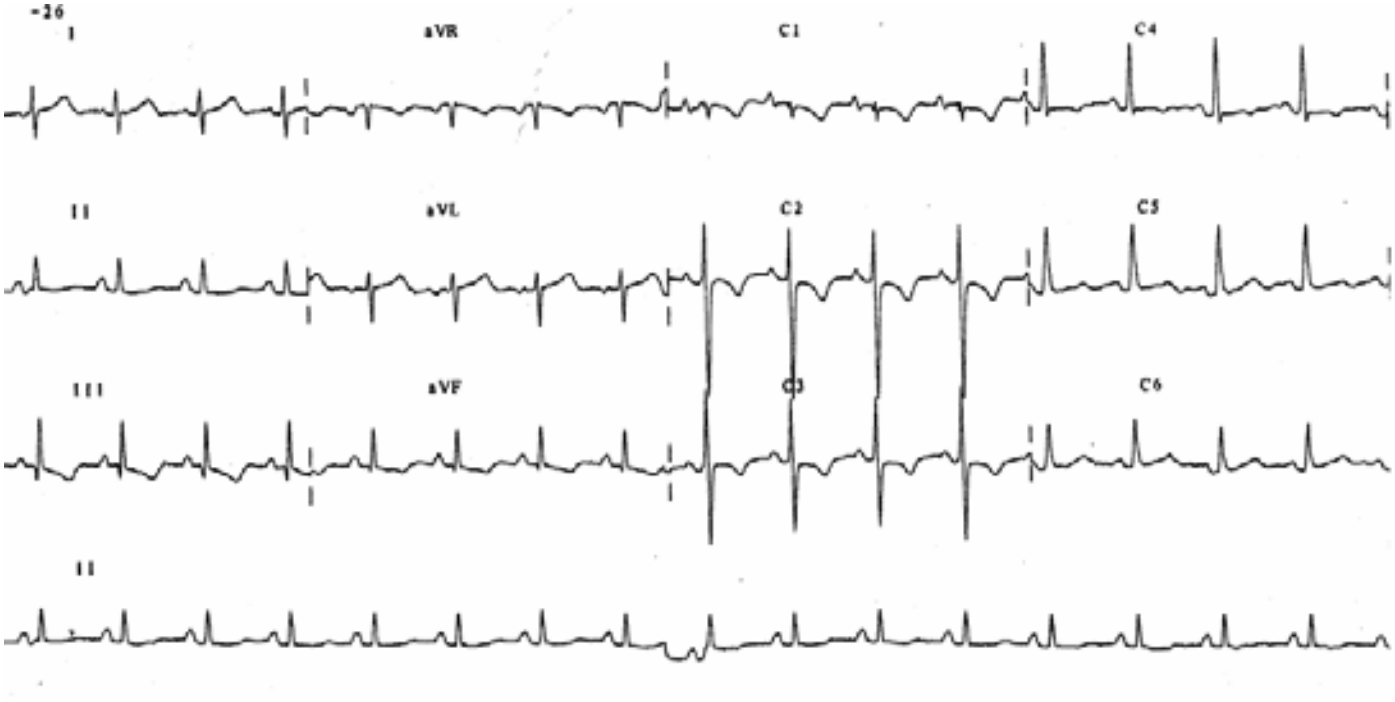
ECG - 10

The correct answer is "d" - None of the above

This 12 lead ECG shows normal QRS complexes with mostly a regular RR interval. Although there are multiple irregular waves intervening the QRS complexes resembling some form of atrial arrhythmia, they are too bizarre and not classic of any arrhythmia. This is basically sinus tachycardia with artifacts due to rigors manifest more in the limb leads.

ECG - 11

45 yr-old, atypical chest pain & fever..



11. The likely diagnosis is:

a. Myocarditis
b. Pericarditis
c. Coronary ischemia
d. Pulmonary embolism

For correct answer see overleaf

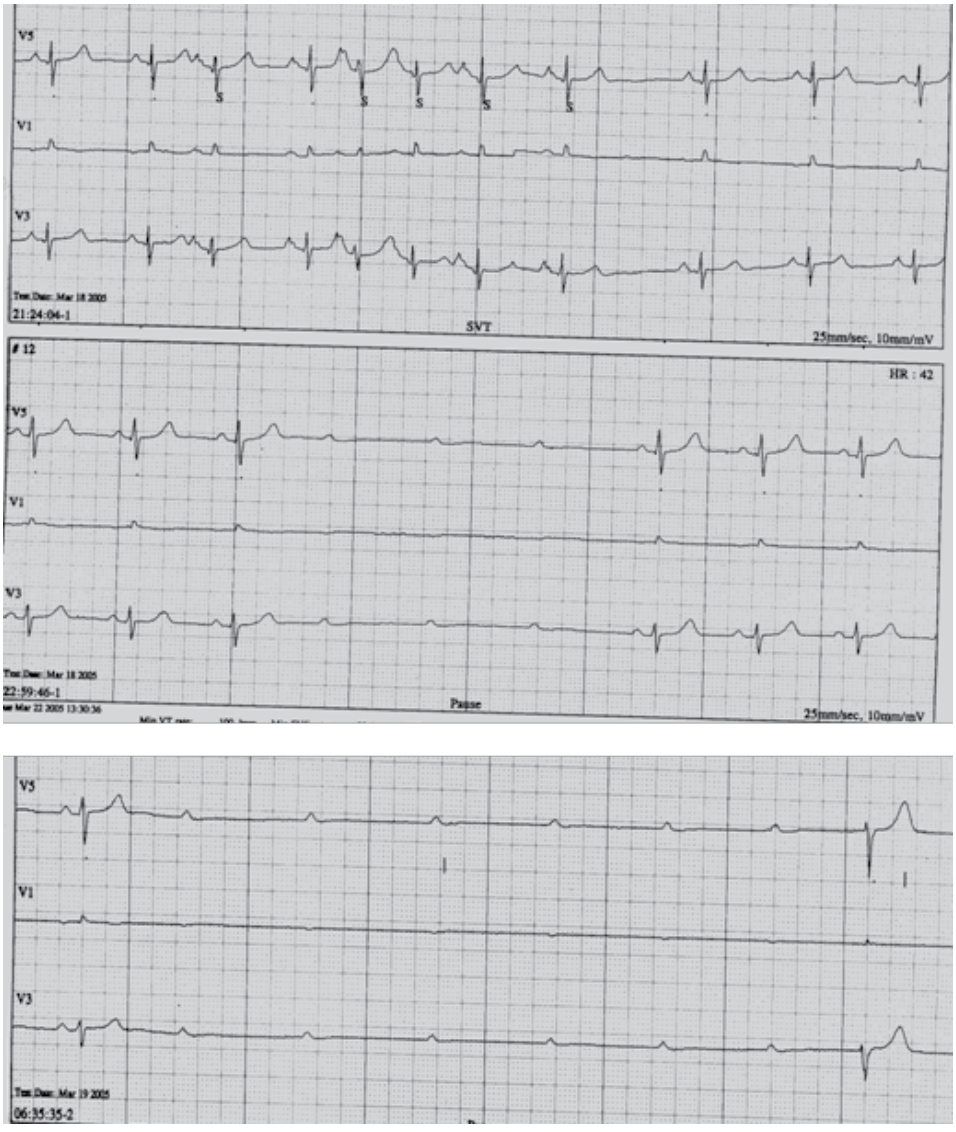
ECG - 11

The correct answer is "d" - Pulmonary embolism

The 12 lead ECG shows sinus tachycardia with a rightward axis. Also, there is a deep S wave in lead I with Q wave and T inversion in lead III. These findings in presence of dull chest pain strongly suggest pulmonary thromboembolism. Fever is a known manifestation in this disorder. Coronary ischemia and myocarditis could have the ST-T changes but axis deviation is unlikely. Pericarditis usually would have ST elevation.

ECG - 12

65 year old lady.
Recent episodes of near-syncope and syncope



12. The AV block is:

a. Paroxysmal
b. Lev / Lenegre's disease
c. Digitalis toxicity
d. Don't know

For correct answer see overleaf

ECG - 12

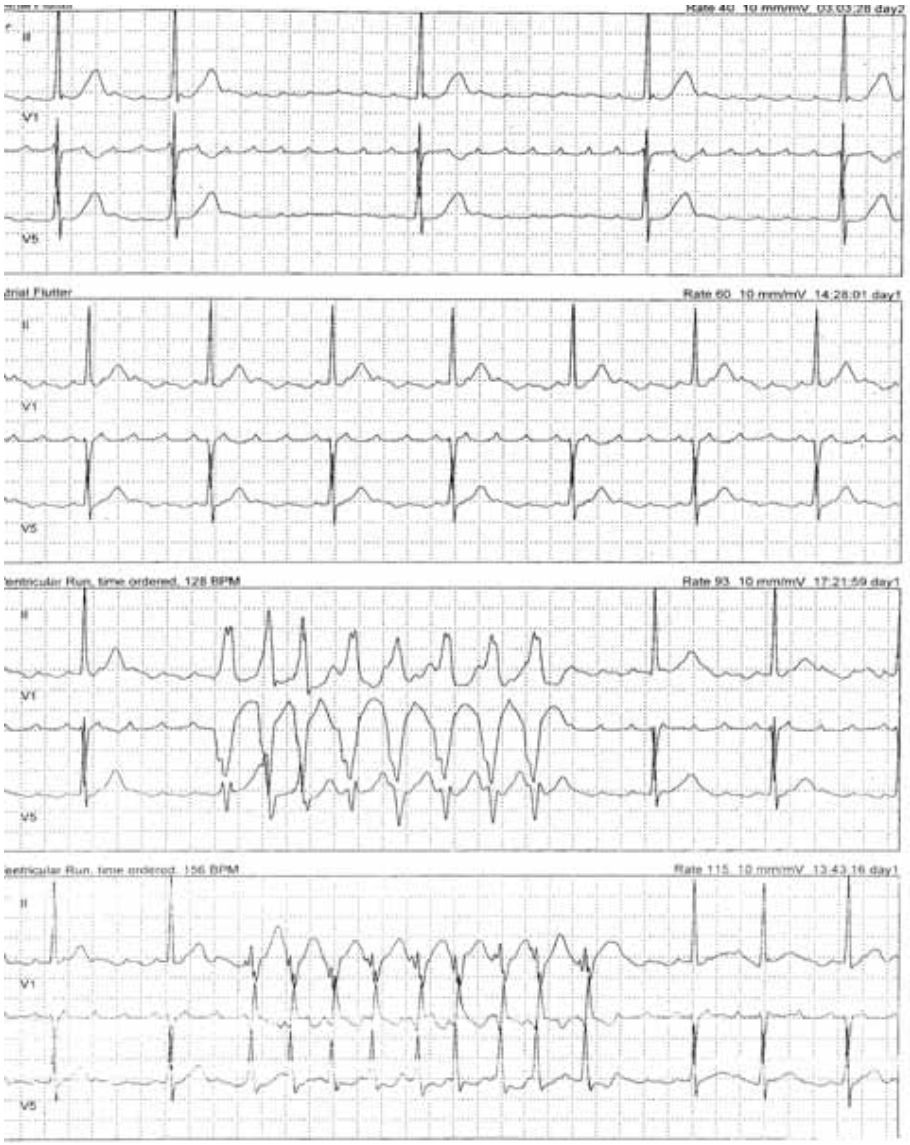
The correct answer is "a" - Paroxysmal AV block

The top Holter ECG strips show a normal sinus rhythm with normal AV conduction with some PACs normally conducted over AV node. In the middle and bottom strip, there is a sudden high grade AV block with long pause and normal P-P interval. This is suggestive of paroxysmal AV block resulting from phase 4 aberrancy in the His bundle fibers and hence narrow QRS. This occurs following lengthening of the cardiac cycle and requires a shift in the transmembrane potential of the His Purkinje fibers to a less negative level and reduced membrane responsiveness, which is often seen in diseased hearts.

Lev- Lenegre's disease produce complete AV block resulting from fibrocalcareous and sclerodegenerative process respectively and is therefore persistent complete AV block. Digitalis toxicity presents with first degree AV block and atrial tachycardia with AV block. The QRS complexes in the Holter strip do not show any evidence of digitalis effect also.

ECG - 13

Asymptomatic 42 year old Sportsman Normal echo



13. The Holter shows:

a. Atrial flutter
b. NSVT
c. A.fl. with BBB
d. Both a + b

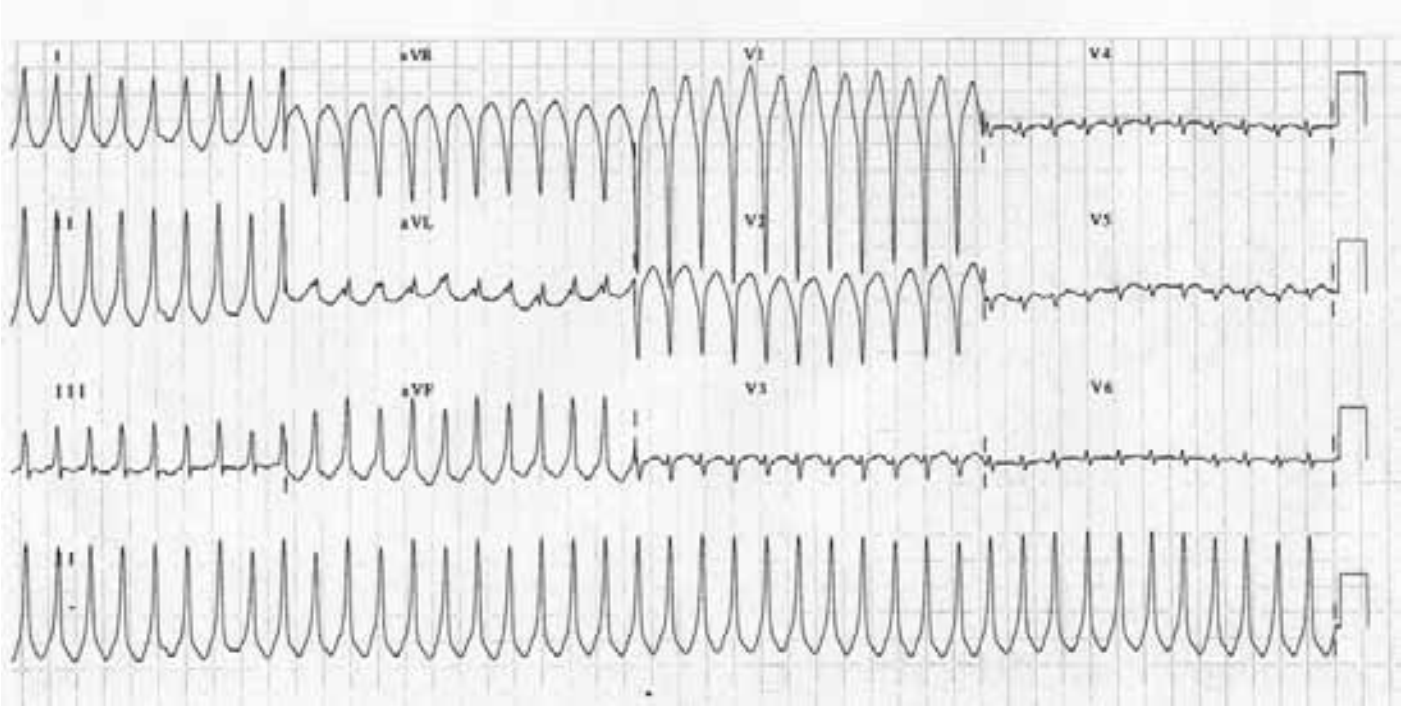
For correct answer see overleaf

ECG - 13

The correct answer is "d" - Both a + b

The four Holter strips show atrial flutter throughout being conducted at variable ventricular rate. The bottom two strips show two episodes of ill-sustained wide QRS tachycardia. The rate of this tachycardia is faster, there is no definite bundle branch block pattern and the tachycardia has two different morphologies; unlikely to be aberrant conduction. Thus, this patient has both atrial flutter and NSVT.

ECG - 14



14. This tachycardia is:

a. SVT with aberrancy
b. VT
c. A.fib with WPW
d. Don't know

For correct answer see overleaf

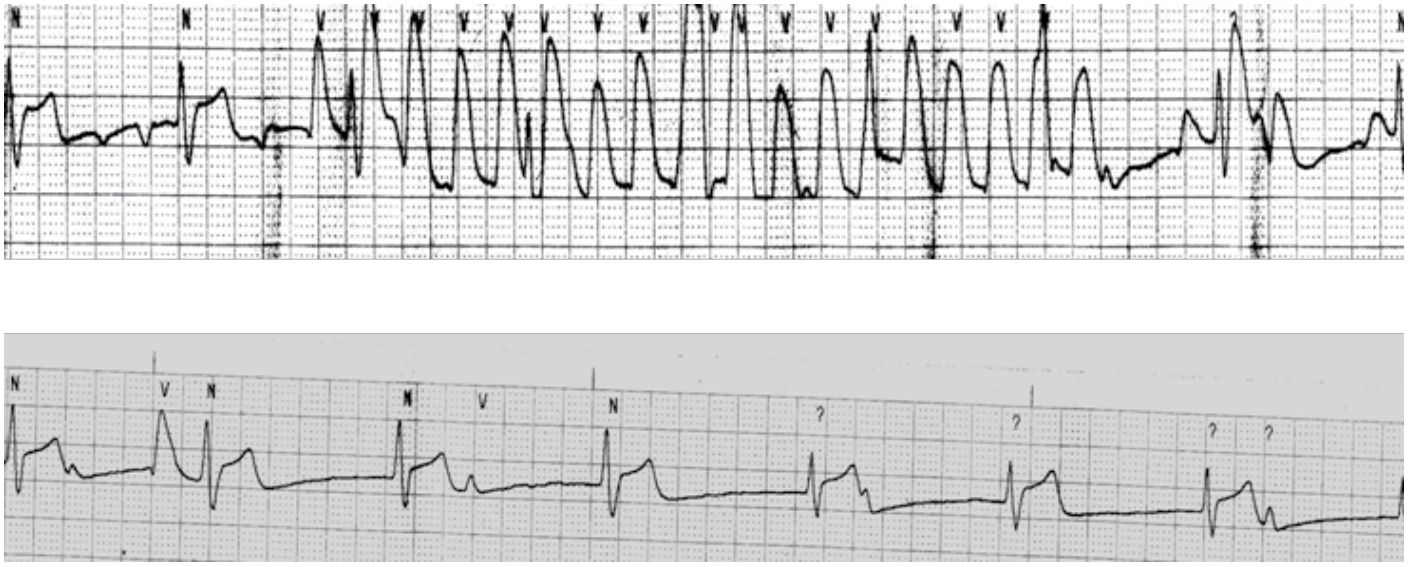
ECG - 14

The correct answer is "b" - VT

The 12 lead ECG shows a wide QRS tachycardia. The tachycardia is regular with no definite bundle branch block pattern. P waves are not well visualized, however if at all there is irregular notching of T waves suggestive of P wave and AV dissociation. This ECG therefore is likely to be VT.

ECG - 15

46 yr old. Old CABG. Admitted for cardiogenic shock.
"Arrest" while on ventilator



15. The first rhythm strip shows:

a. Polymorphic VT
b. Monomorphic VT
c. A.fib with aberrancy
d. None of the above

For correct answer see overleaf

ECG - 15

The correct answer is "d" - None of the above

The top ECG strip shows sudden wide complexes which appear like a ventricular tachycardia. However, on close observation it is seen that intermittently within the wide complexes, there are narrow complexes similar to QRS. Also, these narrow complexes can be timed with the preceding normal QRS complexes, which are regularly present through the bizarre wide complexes. Thus, the wide complexes are artefacts.

Brief Review

Cardiac Arrhythmia and Geomagnetic Activity

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ABSTRACT

Background: The purpose of this paper is a review of a number of studies considering links between life threatening cardiac arrhythmias, sudden cardiac death (SCD) and the level of environmental physical activity factors like geomagnetic activity (GMA) and opposite them cosmic ray and high energy proton flux. This is a part of studies in the field named Clinical Cosmobiology.

Methods: Temporal distribution of cardiac arrhythmias and SCD daily and monthly were compared to the level of GMA, space proton flux, cosmic ray activity according to neutron activity (impulse/min) on the earth's surface. The cosmophysical data was obtained from the cosmic science institutions in the USA, Russia and Finland (cosmic ray data, partially).

Results: As it follows from the results of the quoted studies there is an inverse relationship between the frequency of cardiac arrhythmic events and SCD and the level of daily GMA.

Conclusions: Now studies are in progress considering the role of neutron (cosmic ray) activity in the natural history of the mentioned events. According to the various studies, we can presume that the GMA has some protective effect on cardiac arrhythmias and SCD.

Keywords: sudden cardiac death; cardiac arrhythmia; geomagnetic activity; proton flux; cosmic ray.

Introduction

Geomagnetic activity (GMA) is part of the physical environment surrounding us. In last century many studies were published discussing different links between the level of GMA and human homeostasis. They include studies on cardiac arrhythmia and sudden cardiac death (SCD). The medical community is not equivocal considering those findings. In an editorial comment introducing one recent paper in this field the Editor-in-Chief of "PACE" wrote: "…The question is whether the findings represent a new area for investigation or is a "straw man".[1] In this brief review some data will be presented demonstrating some links between cardiac arrhythmic events and level of GMA.

Geomagnetic activity

The geomagnetic field is a physical phenomenon resulting from different rotation speeds of different layers of our planet . The level of activity of the field can be affected by "geoeffective" parts of magnetic fields coming as a result of solar explosions and accompanied giant magnetic fields. A small portion of them can "disturb" the original geomagnetic field, resulting in active or stormy levels of GMA. The level of GMA is measured in Nanotesla every three hours (8 parameters in 24 hours). The six highest results (in K or integrated A indices of GMA) describe the day as Quiet (I), Unsettled (II), Active (III) or Stormy (IV) day of GMA (**Table 1**). In the last decades many changes in the human hemeostasis related parameters were analyzed in relation

Table 1 : Geomagnetic activity gradation

Category	"A" index range	Typical "K" values	Amplitude (Nanotesla)
1. Quiet (Io)	0< A< 8	Usually No. > 3	0 - 20
2. Unsettled (IIo)	8< A< 16	Usually No. > 3	21-40
3. Active (IIIo)	16< A< 30	Few indices of 4	41 - 70
4. Minor storm (IVo)	30< A< 50	Mostly 4 & 5	71-120
5. Major storm (IVo)	50< A< 100	Some indices 6	121-200

with the level of GMA.[2,3,4] But the level of GMA is not only a factor that can be related with some pathologic effects. The field is also a shield defending our planet from very energetic and potentially harmful space physical activity ingradients, such as cosmic rays and very closely related to them, high energy space proton flux, that can be more active on the surface of our planet when the GMA is extremely low.[5,6,7] For more information special cosmophysical literature must be used, or at least the Glossary of Solar-Terrestrial Terms[8].

Summary of clinical data

Forensic medicine data: Sudden cardiac deaths (n=43) with signs of coronary atherosclerosis, but without acute myocardial infarction occurred more often on days of lowest (Quiet-Io) GMA compared with higher levels of GMA.($p>0.001$).[2]

Holter monitoring data: Hourly atrial premature complex (APC) and ventricular premature complex (VPC) number was more on days of lowest GMA.[9,10]

Sudden deaths (n=480) in three hours after beginning of symptoms (before admission, or at the admission department) were higher on days of lowest GMA ($p<0.01$).[9] Number of sudden deaths dropped on days of severe geomagnetic storm (very high GMA) in July 2000 - the "Bastille day event".[11] Arrival of patients to the emergency department at a tertiary university hospital of patients with atrial fibrillation of new origin (n=653, 1185 days of observation) was inverse correlated with four levels of GMA ($r=-0.97$, $p=0.02$).[12]

Ventricular tachycardia episodes by ambulatory Holter monitoring (n=3019) registered 27% on days of low (Io-IIo) GMA and 17% on days of two higher levels ($chi^2=7.3$, $p=0.006$).[10,13]

In patients admitted for acute myocardial infarction (n=14,529; 8586 men) cardiac arrhythmic events (PAF, AT,VT, VF, n=2024) were relatively higher on days of lowest GMA.[14] SCD that were occurring in one hour after the onset of symptoms (n=261) was more on days of lowest GMA in men < 65 years ($p=0.06$) and women > 65years ($n=0.027$). SCD in woman younger than 65 years was relatively rare.[15]

Discharges (n=402) in 137 days of 25 patients with implanted ICD for ischemic cardiomyopathy, with impaired left ventricular systolic function, for VT , VF were inverse correlated with the level of four daily levels of GMA, ($r=-0.96$-0.97,$p=0.03$-0.04).[16]

The number of SCD (n=516), according to the first aid service data, was significantly correlated with monthly flux of high energy space protons (>90MeV) – a physical parameter closely related to cosmic ray activity (described according to neutron monitoring data on the earth's surface in impulse/min) and inverse related to the levels of solar and GMA.[17]

A new experimental study in pigs has shown that in very low magnetic fields a prolongation of QT interval and changes in P and T waves were registered. Those changes are explained as a result of Calcium ion efflux from the myocytes following channel blocking or inactivation.[18] Also, a possibility was discussed that the electrons that are involved in the electrical activity of the heart, are changing in different levels of magnetic field activity - less active in higher magnetic field.[19]

Now in progress are a number of studies considering the possible role of cosmic ray activity – a factor inverse related to GMA, measured by neutron activity on the earth's surface in the pathogenesis of life threatening cardiac arrhythmias and SCD.[20,21]

The purpose of this short review is to draw attention on possible environmental physical effects that can be an additional pathogenetic factor affecting the time distribution of events related to cardiac arrhythmia and sudden cardiac death.

Conclusion

The presented data, a part of science named Clinical Cosmobiology, has shown that temporal links exist between cardiac arrhythmia, including life threatening events, SCD and level of cosmophysical activity. Atrial fibrillation,ventricular tachycardia/fibrillation and SCD are occurring in inverse relationship to the level of GMA. It is possible that the level of GMA is a factor preventing SCD, especially in patients with damaged heart muscle. The role of factors becoming more active in low GMA, like neutron activity, are an object of further studies.

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Review Article

Rate-Control or Rhythm-Control: Where do we stand?

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ABSTRACT

Atrial fibrillation is the most common sustained rhythm disturbance and its prevalence is increasing worldwide due to the progressive aging of the population. Current guidelines clearly depict the gold standard management of acute symptomatic atrial fibrillation but the best-long term approach for first or recurrent atrial fibrillation is still debated with regard to quality of life, risk of new hospitalizations, and possible disabling complications, such as thromboembolic stroke, major bleeds and death. Some authors propose that regaining sinus rhythm in all cases, thus re-establishing a physiologic cardiac function not requiring a prolonged antithrombotic therapy, avoids the threat of intracranial or extracranial haemorrhages due to Vitamin K antagonists or aspirin. On the contrary, advocates of a rate control approach with an accurate antithrombotic prophylaxis propose that such a strategy may avoid the risk of cardiovascular and non cardiovascular side effects related to antiarrhythmic drugs. This review aims to explore the state of our knowledge in order to summarize evidences and issues that need to be furthermore clarified.

Key words: atrial fibrillation, rate control, rhythm control, secondary prevention

Background

Atrial fibrillation (AF) is the most common serious rhythm disturbance worldwide[1] and its incidence progressively increases with the aging of population by two fold with every decade after 55 year of age[2]. It is associated with increased morbidity and mortality[3], and it is an independent risk factor for stroke: AF increases by 5-fold the overall risk of thromboembolic stroke (TES) and by 12-15% in high risk subsets where further risk factors as hypertension, diabetes mellitus, heart failure, or previous transient ischemic attack, are present[4]. A variety of symptoms such as reduce exercise tolerance, palpitations, dizziness, dyspnoea or other signs of heart failure able to heavily affect the quality of life, may be related to AF[5-8]. Current guidelines clearly state the gold standard approach for symptomatic atrial fibrillation with haemodynamic impairment but, on the contrary, whether rate control approach is preferable to rhythm control strategy is still debated.

An Unresolved Dilemma

Traditionally, a rhythm control approach with electrical or pharmacological cardioversion followed by antiarrhythmic drug prophylaxis was preferred by physician even if strong evidence of its superiority have never been available[9]. Theoretically, restoration and maintenance of sinus rhythm holds some advantages as it re-establishes a physiologic cardiac function, avoids the unfavourable ventricular and atrial remodelling[10,11] due to prolonged tachycardia, and reduce the risk of thromboembolic stroke resuming a normal atrial systole. Current guidelines advocate anticoagulation for 3-4 weeks before and after cardioversion of AF of >48 hours' duration. Alternatively, early cardioversion without anticoagulation may be performed after exclusion of left atrial thrombi by transesophageal echocardiography[1]. Thus, even if evidence of asymptomatic recurrences is growing[12], as of now, prolonged anticoagulation is not yet recommended after restoration of sinus rhythm, due to the known increased risk of haemorrhagic stroke or other major and minor haemorrhages due to Vitamin K antagonists[13,14]. On the other hand, currently available antiarrhythmic drugs have many side effects which in some cases may be life threatening as they are proarrhythmic. Amiodarone is the drugs more frequently used in published trials but also in clinical practice; noteworthy, some reports stated its relation with increased non cardiovascular mortality[15-17]. Advocates of a simple rate control approach associated with an accurate antithrombotic regimen propose that such a strategy would not only avoid the proarrhythmic risk of anti arrhythmic drugs but also ensures a better outcome due to the beneficial effect of warfarin therapy[18]. Rate control approach seems to be equally effective in improving quality of life (QoL) respect to rhythm control approach even if the major predictor of QoL improvement was sinus rhythm[19,20].

Rate Control Strategy

Generally, a rate control approach is considered effective when associated with relief of symptoms and when mean ventricular response ranges between 60 and 80 beats per minute (bpm) at rest and between 90 and 115 bpm during a common moderate exercise test[1,21]. Beta blockers, verapamil, diltiazem, digoxin but also amiodarone are effective in reducing the heart rate: beta blockers are the drugs of choice in patients with coronary artery disease and, in presence of systolic dysfunction, they may be even more valuable[22]. Verapamil is able to rise the serum digoxin levels so the dosage of the latter must be reduced if

administered with verapamil[23]. As sole therapy, digoxin may be suitable for elderly patients[24]. Amiodarone is highly effective in controlling the heart rate but many concerns have been raised about its long-term safety[15-17].

The "ablate and pace" approach consists of the ablation of atrioventricular node followed by implantation of a permanent pacemaker. The ventricular rate is completely controlled by the pace-maker and also a more physiological contraction of the ventricle is restored thus positively affecting the cardiac haemodynamic[25]. As the atria continue to fibrillate, the need of anticoagulant therapy remains unchanged. In patients with heart failure and refractory to other treatment, this approach was found to be associated with better control of palpitations, improvements of dyspnoea and quality of life[26]. No improvement of cardiac performance[26] neither prolonged survival[27] were observe with "ablate and pace" strategy respect to pharmacological therapy. In some cases a modulation instead of ablation of atrioventricular node may be efficacious to improve symptoms[28].

Rhythm Control Strategy

Current guidelines state which antiarrhythmic drugs have proven efficacy in converting atrial fibrillation into sinus rhythm both for atrial fibrillation with less and more than 7 days of duration[1]. Also which drugs are effective in increasing the success rate of electrical cardioversion are described[1]. All of these antiarrhythmic drugs are burdened by the risk of serious cardiovascular and non cardiovascular side effects, including life threatening ventricular arrhythmias (all), lupus like syndrome (procainamide), heart failure (disopyramide, propafenone, flecainide, sotalol), pulmonary toxicity (amiodarone, sotalol). A recent meta analysis supports the efficacy of amiodarone in converting persistent atrial fibrillation[29] but its known thyroid, hepatic, and pulmonary toxicity should make it a second line agent[30].

Electrical cardioversion has a very high initial success rate, but only about 23% of patients at 1 year and 15% at 2 years remain in sinus rhythm[31,32]. Many studies investigated the clinical and instrumental factors able to predict the risk of relapse; the most common accepted are: AF duration more than 1 year, age > 75 year, heart failure and increased atrial dimension[33]. Currently, for patients with unknown atrial fibrillation duration or more than 48 hours, cardioversion is recommended after a 3 weeks period of anticoagulation and it has to be followed by a month of anticoagulation[1]. A transoesophageal echocardiography-guided approach showed a similar risk of stroke compared to conventional management[34] thus suggesting that the risk of stroke is not completely abolished restoring sinus rhythm, even if transoesphageal examination is negative for intra atrial clot.

Recently, newer strategies were developed, including catheter ablation by means of radiofrequency. Ectopic foci, localized within the pulmonary vein, were found in the majority of atrial fibrillation[35] and the ablation of this triggers, despite a little but predictable risk of iatrogenic morbidity[36], seems to be very promising in light of a chance of "real" cure for atrial fibrillation[37]. Device-based therapy[38] but also surgical treatment with the Cox-Maze procedure have been developed in the last years with some promising results[39].

Randomized Trials of Rate vs Rhythm

Several trials addressing this issue were published in the last years[40-45] and 2 other are still ongoing[46,47]. The Pharmacological Intervention in Atrial fibrillation (PIAF)[40] enrolled 252 patients with persistent atrial fibrillation duration up to 360 days. In the first arm the strategy was to control heart rate using diltiazem while, in the second, restoration and maintainance of synus rhythm was obtained using amiodarone or electrical cardioversion as first intervention followed by various antiarrhythmic drugs. Follow up length was 1 year. The primary end point of the study was improvement in symptoms related to atrial fibrillation.

The Strategies of Treatment of Atrial Fibrillation study (STAF)[41] enrolled 200 patients with persistent atrial fibrillation. In the rhythm control arm patients were to be cardioverted by external or internal cardioversion; after restoration of sinus rhythm, prophylaxis was performed with class I antiarrhythmic drugs or sotalol, in the absence of coronary artery disease (CAD) and in the presence of normal left ventricular ejection fraction, while in the presence of impaired ventricular function or CAD, beta-blockers or amiodarone were used. In the rate control arm beta-blockers, digitalis, calcium antagonists, or atrioventricular node ablation/modification with or without pace maker implantation were used. Follow up was of 19.6 + 8.9 months. Primary end point was the combined rate of death, cardiopulmonary resuscitation, cerebrovascular events and systemic embolism. The RACE study[42] enrolled 522 patients with persistent atrial fibrillation after an initial attempt of electrical cardioversion. Rate control was achieved with administration of digitalis, calcium antagonists, and beta-blockers alone or in combination. Rhythm control was obtained with electrical cardioversion without previous treatment with antiarrhythmic drugs. Thereafter, sotalol was used for prophylaxis. At the first recurrence of atrial fibrillation, electrical cardioversion was repeated and sotalol replaced by flecainide. In the presence of a recurrence within 6 months another cardioversion was performed and flecainide replaced by amiodarone. Follow up length was 2.3 + 0.6 years. The primary end point was a composite of cardiovascular death, heart failure, thromboembolic complications, bleeding, implantation of a pace-maker, and severe adverse effects of drugs.

The AFFIRM study[43] enrolled 4060 patients with first or recurrent atrial fibrillation at high risk for stroke in a randomized, multicenter comparison. Risk factors for stroke were considered hypertension, diabetes mellitus, congestive heart failure, prior transient ischemic attack, cerebrovascular accident, systemic embolism history, left atrial size of 50mm or more, LVEF less than 40%. Digitalis, calcium antagonists, and beta-blockers

alone or in combination were the drugs accepted in the rate control arm: the goal was a heart rate not higher than 80 beats per minute at rest and 110 beats per minute during six minute walk test. In the rhythm control arm the antiarrhythmic drug was chosen by the treating physician: attempts to maintain rhythm control could include cardioversion. The following drugs were acceptable: amiodarone, disopyramide, flecainide, moricizine, procainamide, propafenone, quinidine, sotalol, and their combination according to an imposed protocol. Mean follow up length was 3.5 years. The primary end point was overall mortality but several other clinical end-points were reported.

The HOT CAFÉ[44] enrolled 205 patients with a mean time of atrial fibrillation duration of 273 ± 112.4 days. In the rate control arm beta blockers, calcium antagonists, digoxin alone or in combination were the pharmacological treatment. Patients randomised to rhythm control strategy were all treated with electrical cardioversion and subsequent antiarrythmic drugs. Follow up length was 1.7 ± 0.4 years. Primary end point was a composite of death from any cause, thromboembolic complications (especially disabling stroke), and intracranial or other major haemorrhage.

The Control of Rate versus Rhythm in Rheumatic Atrial Fibrillation trial (CRRAFT)[45] trial differs from the others because it enrolled patients with rheumatic heart disease and chronic atrial fibrillation. Forty-eight patients randomized to rate control received 90 mg sustained release diltiazem twice daily to maintain the resting ventricular rate below 90 beats/min, and less than 130 beats/min with activity. No attempt to restore SR with drug therapy or electroversion was made. Those who entered the rhythm control group were further randomized in a double-blind design to receive either amiodarone or placebo and electrical conversion, wherever required. Follow up length was 1 year. Primary end points were: exercise tolerance assessed by Bruce protocol treadmill exercise, NYHA class, QOL score, thromboembolic and bleeding complications, hospitalization rates, and deaths. While the CRRAFT trial suggested that maintenance of sinus rhythm appeared to be superior to ventricular rate control in patients with rheumatic atrial fibrillation in terms of an effect on mortality and morbidity, none of the single published trials on non-rheumatic atrial fibrillation shows statistically significant difference between the two strategies, only suggesting a possible superiority of rate control approach in terms of a trend toward a reduced risk of major adverse cardiovascular events. On the other hand, a recent meta-analysis of our groups[48] stated the superiority of rate control strategy, in patient with non rheumatic atrial fibrillation, in reducing the risk of the combined end point of all cause death and thromboembolic stroke without any increase in the risk of major haemorrhages. This superiority seems even more evident by the low number needed to treat to avoid one combined end point as it results equal to 50. Rate control strategy confirmed its superiority in reducing the combined end point compared to the rhythm control approach even when older patient or longer follow up were considered. Notably, the risk of thromboembolic stroke is strongly reduced in the early period after the beginning of therapy as demonstrated in the studies with mean follow up < 20 months in which we observed a reduction of 82%.

Conclusions

In the evidence-based medicine era the highest level of evidence is that of meta-analytic approach[49] but the results presented above should be cautiously viewed as hypothesis generating not as the definitive answer, for some reasons: 1) the results do not apply to all subsets of patients with atrial fibrillation because patients with Wolff-Parkinson-White syndrome, those who had previously undergone heart surgery, or those with NYHA class IV heart failure were excluded by the trials' designs; 2) all published studies had at least a small percentage of patients crossing over the randomization arms but the statistical analyses were performed by intention to treat thus underestimating this effect, and 3) even if amiodarone was the antiarrhythmic drug more frequently used, other agents have been adopted, anyone with its own risk/benefit profile, thus generating another confounding factor to take into account.

In a post hoc analysis of AFFIRM population the trend toward a lower total mortality in the rate-control versus the rhythm-control group appears entirely explained by non cardiovascular deaths. Independent predictors of non cardiovascular death were rhythm-control strategy, age, male gender, previous smoking history, heart failure, and coronary heart disease[50]. In another analysis, the authors stated that digoxin and antiarrhythmic drugs were directly associated, while the presence of sinus rhythm and warfarin therapy were inversely associated with increased mortality after adjustment for other covariates[51]. The meaning of these separate observations may be that pursuing sinus rhythm may improve prognosis, but this potential advantage has to be weighted against various non cardiovascular adverse effects of the agents used to obtain and maintain it.

As previously stated, current guidelines recommend only a short term anticoagulation therapy after restoration of sinus rhythm while those patients treated with rate control approach continue to take Vitamin K antagonists or aspirin potentially for all the life. This issue rises some concerns: oral anticoagulants, but also aspirin, are associated with at least a two fold risk of major haemorrhages compared to placebo[13], on the other hand, current available antiarrhythmic drug are burdened by a substantial risk of ventricular arrhythmias. A patients treated with rhythm control approach have not an increased risk of haemorragies but an increased risk of sudden death. The contrary may be told for those treated with rate control strategy. In these patients with atrial fibrillation, with so many different clinical features, what is the highest and worst risk? An haemorrhagic stroke or a malignant arrhythmia? We have not enough evidence to answer this pivotal question.

As previously reported[48], there is an early large excess of

thromboembolic stroke in patients randomized to a rhythm-control strategy possibly because the risk of stroke associated with electrical cardioversion is not entirely abolished by short term anticoagulation. Moreover, symptomatic or asymptomatic recurrences are frequent[12] thus increasing the risk of thromboembolic stroke in patients possibly taking neither aspirin nor Vitamin K antagonists.

In the future, new, safer and more effective antiarrhythmic agents, associated with careful and prolonged anticoagulation, will probably make the rhythm-control strategy superior to the rate control one but, according to current evidence, rate-control strategy represents the gold standard strategy, especially for those patients with echocardiographic and clinical features that make unlikely the maintenance of sinus rhythm with or without antiarrhythmic prophylaxis for recurrences.

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Case - 1

Syncope, can you Identify the Mechanism?

R. Pinto, S. Athawale, Amit Vora

A 45-year-old-male, chronic alcoholic was admitted with sepsis and hyponatremia (serum sodium = 110 meq./l). His initial ECG showed Mobitz type I, second degree AV block (figure 1). While he was being treated for sepsis and correction of hyponatremia, patient developed bulbar palsy and the MRI brain scan revealed central pontine myelinosis. Subsequently he developed recurrent episodes of syncope and was shifted to the ICU. What is the likely cause of syncope in this patient?

The ECG showing a Wenckebach phenomenon with a 3:2 AV conduction and the second QRS with a prolonged PR interval showing a right bundle branch block raises the possibility of degeneration in to complete AV block and the likely cause of syncope. The other differential would be any neurologic or metabolic cause in this gentleman, however neither would be transient and spontaneously correct itself. Figure 2a is the ECG recorded as he reached the ICU. It shows complete heart block and markedly prolonged QT interval. The AV block is well identified in long lead II and the prolonged QT interval in precordial leads V2-4. There is also a ventricular bigeminy pattern with the premature complex occurring during the vulnerable phase of prolonged repolarization i.e. descending limb of the T wave. This is a hallmark precursor of polymorphic VT – torsades des pointes. This is what was noted in the ICU – figure 2b. Thus the cause of syncope in this patient was torsades des pointes due to prolonged QT. QT prolongation in this patient is acquired and can be either drug induced or bradycardia (complete AV block) dependant. However, none of the drug treatment received by this patient was known to induce QT prolongation. A permanent pacemaker was implanted. The ventricular rate was kept at 100 bpm to help shorten the QT. At discharge he continued to have a longer QT despite pacing, however the VPCs were completely suppressed and there were no further episodes of polymorphic VT. An extensive search for any drug-metabolic factor responsible for long QT was performed which was apparently normal. It is likely that this gentleman has a form-fruste of long QT, which was precipitated by the complete AV block and his current illness/medications.

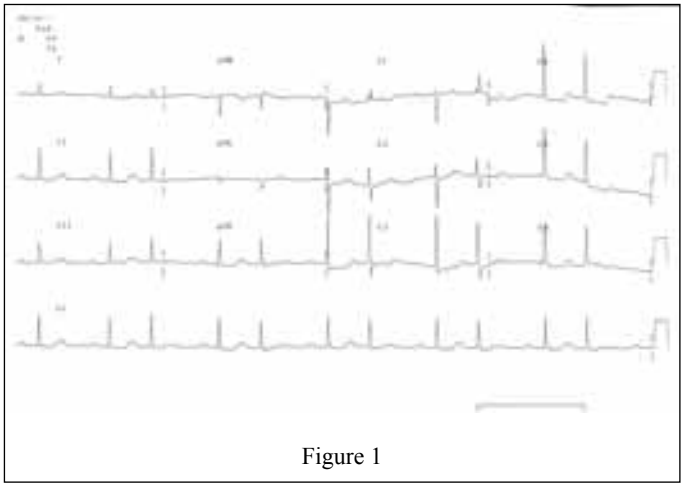

Figure 1

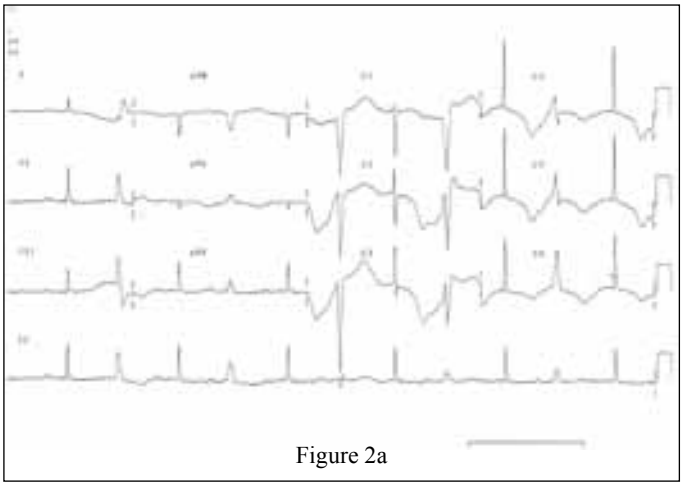

Figure 2a

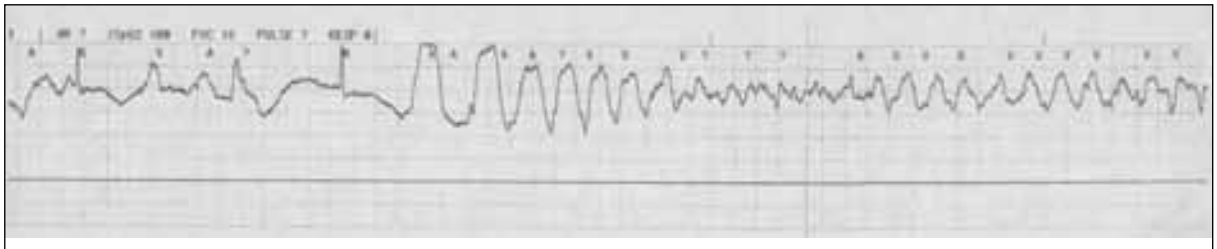

Figure 2b

Case - 2

Can you Identify the Culprit Vessel?

K. Bharti, Amit Vora

A 42-yr-old gentleman, known diabetic, presented with sudden onset chest pain. The ECG (figure 1a) was taken within 2 hours of chest pain. He was given thrombolytic therapy and his subsequent ECG two days later is as shown in figure 1b. Which is the likely coronary artery occlusion responsible for his myocardial infarction? Figure 1a shows ST elevation in leads V_1, V_2 and also suspicious elevation in lead III. There is associated ST depression in leads V_5-V_6, I & avL. Right-sided chest leads V_4R and V_5R also showed ST elevation, suggesting RV infarction. However RV infarction is most often associated with inferior wall MI. The inferior leads do not show significant changes; there is some ST elevation in lead III, a small q in avF and lead II is normal. This suggests not much of inferior myocardium is at jeopardy. How does one explain the antero-lateral wall ST depression? Anterior wall MI due to proximal LAD occlusion can lead to ST depression in antero-lateral leads but in such cases it is a type I LAD falling short of LV apex. In this patient proximal LAD occlusion would not explain the inferior wall ST elevation. ST elevation in inferior leads occurs in anterior wall MI with mid LAD occlusion, where the LAD wraps around the apex (type III LAD), supplying the inferior wall as well. In case of inferior wall MI, ST depression in antero-lateral leads suggest multi-vessel disease. His subsequent ECG (Figure 1b) showed decapitation of R wave in precordial leads V_{1-4}.

The coronary angiogram revealed a 95% proximal RCA occlusion before a large RV branch. The RCA was a non-dominant artery. The RV branch was supplying collaterals to LAD, which was completely occluded in the mid-portion. The LCx was dominant and normal.

Thus in all probability the LAD occlusion was chronic with collateral supply from the RCA. Therefore he remained asymptomatic with no ECG changes of anterior wall MI till the recent admission. The proximal RCA occlusion appears the culprit vessel, which is partly recanalized with thrombolytic therapy. The initial RV infarction & later evolution of anterior wall MI is explained by the proximal RCA occlusion, as the RV branch, which supplies collaterals to the LAD, is occluded. Since the RCA is non-dominant there is not much of inferior wall MI changes noted. Identifying the culprit vessel has its limitations in presence of multiple vessel disease.

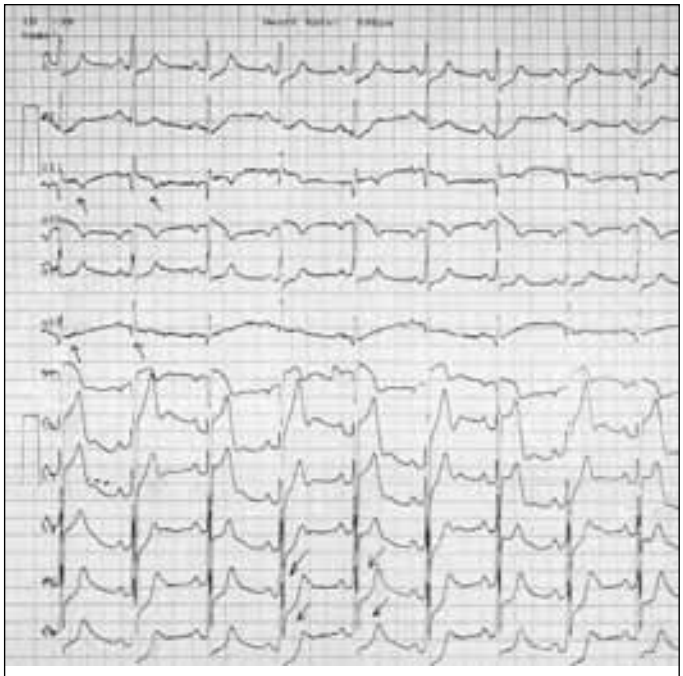

Figure 1a

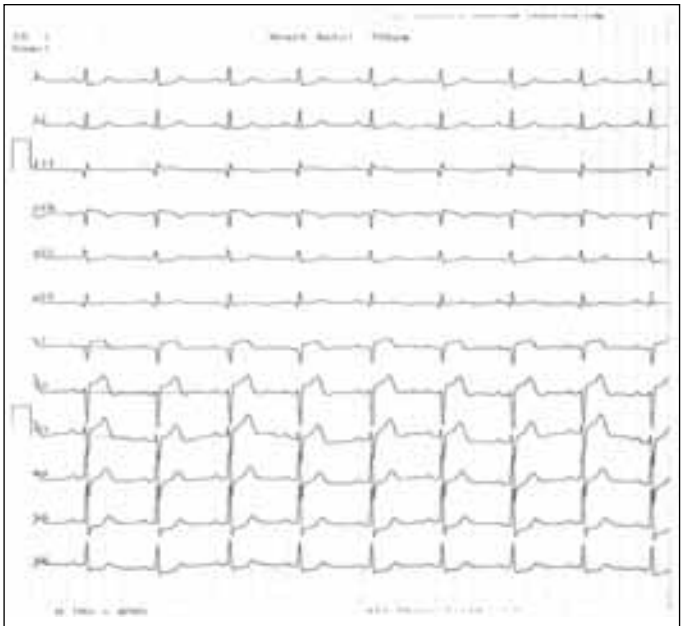

Figure 1b